Encyclopedia of Behavioral Medicine

Marc D. Gellman
Editor

J. Rick Turner
Co-Editor

Encyclopedia of Behavioral Medicine

With 99 Figures and 46 Tables

Springer

Editors
Marc D. Gellman
Behavioral Medicine Research Center
Department of Psychology
University of Miami
Miami, FL, USA

J. Rick Turner
Cardiovascular Safety
Quintiles
Durham, NC, USA

ISBN 978-1-4419-1004-2 ISBN 978-1-4419-1005-9 (eBook)
DOI 10.1007/978-1-4419-1005-9
ISBN 978-1-4419-1380-7 (print and electronic bundle)
Springer New York Heidelberg Dordrecht London

Library of Congress Control Number: 2012944367

Printed on acid-free paper

Springer is part of Springer Science+Business Media (www.springer.com)

Opening Quotations

Some of the unhealthful behaviors that make the greatest contribution to the current burden of disease are cigarette smoking, the abuse of alcohol and drugs, the overeating and underexercise that produce obesity, and Type A behavior. Unfortunately, these behaviors are stubbornly resistant to change and discouragingly subject to relapse. Thus, for behavioral scientists to promise to achieve too much too soon is to court disastrous disillusionment. But any contributions that behavioral scientists can make to reduce any of them will have highly significant implications for health.

Miller, N. E. (1983). Behavioral medicine: Symbiosis between laboratory and clinic. *Annual Reviews of Psychology*, *34*, 1–31.

As behavioral medicine researchers, we must become more directly involved in translating gains in the science of clinical and community (disease) prevention to gains in public policy. We have an unprecedented window of opportunity given the growing recognition at all levels of health care and government that clinical and community interventions that promote and support health behaviors will be essential for success in reducing the nation's most prevalent and costly heath problems and untenable health-care costs and disparities. This is the kind of opportunity that propelled the founders of our field 25 years ago, and we are better prepared than ever in our history to seize it.

Ockene, J. K., & Orleans, C. T. (2010). Behavioral medicine, prevention, and health reform: Linking evidence-based clinical and public health strategies for population health behavior change. In A. Steptoe (Ed.), *Handbook of behavioral medicine: Methods and applications* (pp. 1021–1035). New York: Springer.

The extent to which behavioral medicine can become a successful part of health care delivery systems will in large part depend upon investigators in the field being able to master clinical translational research, moving from efficacy to effectiveness with a high ratio of benefit to cost. . .. Because Behavioral Medicine has been constructed based on the understanding of relationships among behavior, psychosocial processes and sociocultural contexts, the field is well-positioned to take

a leadership role in informing future health care policies. The field of Behavioral Medicine appears to have a bright, important future.

Schneiderman, N. (2012). A personal view of behavioral medicine's future. This volume.

Foreword

Early Developments in the Field of Behavioral Medicine

At the editors' request, this Foreword provides a personal account of the early development of behavioral medicine. With many colleagues, I was fortunate to play a role in bringing together behavioral and biomedical sciences in such a way that the synergism resulting from this interaction resulted in ideas, conceptualizations, models, and ultimately interventions that were truly different from preexisting approaches to health and illness. As noted in the Preface, the contents of this encyclopedia bear witness to the manner in which behavioral medicine has matured during the past 30 years, illustrating current activities in the domains of basic research, clinical investigation and practice, and public health policy.

In 1963, I was a psychology intern in the Department of Medical Psychology at the University of Oregon Medical School (now called the Oregon Health Sciences Center). Under the guidance of Joseph Matarazzo, chair of the department, the relationship between medicine and psychology was undergoing an historic realignment. Joe had a fascinating and exciting perspective on the nature of such relationships and on psychology's potential to make those relationships mutually rewarding for both patients and practitioners. I consider myself fortunate to have been "in the right place at the right time" when a request came from the Division of Cardiothoracic Surgery for psychological and psychiatric consultation on a problem that was mystifying the surgeons.

Under the leadership of Albert Starr, surgeons were performing groundbreaking procedures known as "open heart surgery" on patients who had been incapacitated, typically for many years, by their heart conditions. These surgeries offered them the opportunity to reclaim their earlier lives as active members of society, and, in some cases, to take on roles that were denied to them since childhood. Paradoxically, following surgery, many patients, rather than expressing their gratitude for the opportunity to be "made whole again," become angry, depressed, and suicidal. With colleagues from the departments of psychology and psychiatry, we begin a search for the "underlying mental illness" that must have been uncovered by the stress of the surgery. However, rather than discovering the presence of psychiatric illness, it was found that the *absence of psychological strength* was a key factor associated with the behavioral anomalies. This finding led to the development of a program to psychologically evaluate a candidate's readiness to undergo surgery and to better prepare psychologically vulnerable candidates for the recovery experience.

My dissertation on psychological adjustment following open heart surgery led me to the Division of Psychosomatic Medicine in the Department of Psychiatry at the Johns Hopkins University School of Medicine, and to the application of psychodynamic theory to problems as diverse as diabetes, cardiovascular disease, cancer, and transgender surgery. In 1974, I accepted a position at the National Heart, Lung, and Blood Institute (NHLBI) of the National Institutes of Health (NIH) as chief of a unit that would eventually become the Behavioral Medicine Branch. The first year was *very* difficult since I was essentially the only behavioral scientist at NHLBI, and no one understood exactly what I was supposed to do and why I was there. However, I considered this to be a singular opportunity to bring the behavioral and biological sciences together, if only we could come up with a model, a theoretical framework that made sense to both groups and was scientifically viable.

Good problem-solving strategies break the overall problem down into more manageable pieces. The first was to address the lack of fundable studies in the NHLBI portfolio, which comprised a total of four regular research grants (R01s). The institute director commented to me that behavioral scientists must not be very good scientists as their applications were routinely disapproved or failed to make the funding payline. However, investigation revealed that the 25 "behavioral" applications submitted for the current round were scattered among 14 different study sections. Two issues became evident: (1) many of the behavioral applications were biologically weak and (2) the multidisciplinary expertise necessary to properly review applications that had both behavioral and biological endpoints was missing from the various study sections to which the applications had been assigned.

It became clear that two efforts were needed. First, it was necessary to make both biomedical and behavioral scientists aware of the need for a collaborative "biobehavioral" approach involving top-tier expertise in both areas when submitting grant applications. Second, it was necessary to campaign within the NIH for a study section that could provide relevant peer review for these biobehavioral applications. NIH agreed to convene an "ad hoc" temporary review group (the Behavioral Medicine Study Section) to assess whether there really was a need for such a group. Clearly there was, since 3 years later the study section was formally chartered as a standing study section.

Meanwhile, it became obvious that to develop and sustain meaningful research programs within the NIH would require organized, active, outside constituencies of scientists and clinicians who could provide peer review to all aspects of NIH program development and scientific leadership, i.e., partnerships with academic and professional societies that could provide advice and guidance were needed. With specific regard to biobehavioral research, the need for credible representation led us to Neal Miller, a behavioral scientist who was well known to, and highly respected by, the biomedically oriented Institute staff. Neal had performed landmark studies of learning and biofeedback. He was persuaded to serve as keynote speaker for the 1975 NHLBI Working Conference on Health Behavior. The 3 days of intensive deliberations between senior behavioral and biomedical scientists were

summarized and published as a proceedings to serve as the public blueprint for the Institute's future biobehavioral scientific agenda.

Along with the 1977 Yale Conference on Behavioral Medicine sponsored by NIH, this meeting set the stage for a 1978 organizational meeting hosted by David Hamburg, President of the Institute of Medicine of the National Academy of Sciences. The deliberations of this two-day gathering of highly respected biomedical and behavioral scientists gave birth to two organizations, the Society of Behavioral Medicine (SBM) and the Academy of Behavioral Medicine Research (ABMR). The founding leaderships of these organizations agreed to be complementary rather than competitive in mission and purpose, with SBM serving both scientific and professional interests of all persons interested in the field and ABMR being a small invitation-only group of distinguished senior scientists dedicated to identifying and promoting "gold standard" science in behavioral medicine. SBM created a newsletter that became the high-quality scientific and professional journal *Annals of Behavioral Medicine*, and ABMR published an annual volume, *Perspectives on Behavioral Medicine*, summarizing scientific presentations at their annual retreat meeting.

During this early developmental period, a potentially divisive issue arose among the cadre of behavioral medicine pioneers: Exactly what is meant by the term "behavioral medicine?" Agreement on a common definition of the field was clearly necessary. One contingent defined behavioral medicine primarily as "behavior modification with medical patients," while another contingent took a broader view which included the aforementioned aspect, but challenged both the behavioral and biomedical communities to join forces as "the interdisciplinary field concerned with the development and integration of behavioral and biomedical science knowledge and techniques relevant to the understanding of health and illness, and the application of this knowledge and these techniques to prevention, diagnosis, treatment and rehabilitation." The latter became the agreed-upon definition by both behavioral medicine organizations and survived intact for a decade until the founders of the International Society of Behavioral Medicine proposed in 1990 that "psychosocial" be added to "behavioral and biomedical" to better align the definition with the charters of the emerging European national and regional behavioral medicine organizations.

The underlying concepts of behavioral medicine are perhaps thousands of years old. Prior to the emergence of behavioral medicine in the mid to late 1970s, the most recent effort to capture mind-body interactions can be attributed to those engaged in research and practice of psychosomatic medicine. Primarily psychodynamically oriented psychiatrists, they began to take note of behavioral medicine, initially identifying the fledgling organizations with the first definition mentioned previously (behavior modification with medical patients) whereas their interests were principally focused upon how the principles of psychoanalysis could be applied to the treatment of somatic disorders. However, as the second definition gained traction among the rank and file of the behavioral medicine community, psychosomatic medicine scientists and practitioners were challenged to either resist or join forces with the newcomers. Over the next decade, it became clear that, while

psychoanalytic theory was intellectually provocative, it lacked the tools of modern day science to test its theories, and hence such theorizing remained in the realm of speculation. Behavioral medicine, on the other hand, took full advantage of the new monitoring instrumentation generated in large part by the U.S. space program's need for ambulatory monitoring of physiological processes via telemetry. Such instrumentation facilitated exploration in the laboratory and in real life of how variation in biological processes may be stimulated by behavioral inputs, as well as how biological processes may impact behavior. Over the next 20 years, the membership of the American Psychosomatic Society and the organization's flagship journal, *Psychosomatic Medicine*, shifted their emphasis to one indistinguishable from that of organized behavioral medicine.

During this time, biobehavioral scientific programs were beginning to develop within several institutes at NIH, and funding for biobehavioral research increased exponentially, albeit unevenly. An inter-institute Committee on Health and Behavior was formed, with Matilda White Riley from the National Institute on Aging as its first chair. This committee served in an advisory capacity to the individual institute directors as well as to the NIH director, becoming the precursor for the Office of Behavioral and Social Science Research, Office of the Director, NIH, which is now under the leadership of Robert Kaplan, past president of both SBM and ABMR.

Although research funding was increasing, another challenge became evident: Where were the *training* resources to support new entrants to the field? Typically, research training programs in the biological and biomedical sciences relied on NIH support; it became obvious that such resources needed to be developed to establish a pipeline for "biobehavioral" scientists-in-training to receive both individual and institutional support. Donald Cannon, chief of the training branch at NHLBI, the unit responsible for supporting both types of awards at NHLBI became interested in the issue, and met with senior behavioral medicine researchers who could apply for such awards based on their research programs and the resources of their institutions. Over the next 3 years, 12 institutional awards were made to support cardiovascular behavioral medicine training for both behavioral/social scientists and biomedical/biological scientists, further solidifying the scientific base for the field.

These developments within the United States were mirrored in other parts of the Western world, with emerging organizations in several European countries grappling with the relevance of the behavioral medicine concept to their perspectives on health and illness. In the mid-1980s, discussions at an SBM annual conference with international attendees resulted in an agreement to form an International Society of Behavioral Medicine (ISBM) dedicated to supporting the emergence of new as well as existing national and regional behavioral medicine organizations. Funds to support several planning meetings were provided by the Rockefeller family and the Duke University Behavioral Medicine Research Center, and the first International Congress of Behavioral Medicine took place in 1990 in Uppsala, Sweden. The International Society (members are national or regional societies rather than individuals) represented seven national and regional societies at this first

meeting. *International Journal of Behavioral Medicine* became the scientific outlet for behavioral medicine studies of international relevance. By 2012, 26 (and counting. . .) national/regional societies from every continent formed the membership of ISBM.

Finally, one important element of the behavioral medicine paradigm deserves mention, as it is illustrative of the basic conceptual infrastructure of biomedical and behavioral *integration*. Often, biomedical and behavioral scientists pose the question of treatment efficacy in terms of which is more effective, pharmacologic or behavioral treatments. Rather than "either/or," the behavioral medicine position is to determine how both treatments, perhaps in combination or in sequence, may provide a more effective treatment than either alone. Several examples come to mind, for example, smoking cessation, hypertension treatment, and cardiovascular disease prevention. Using a drug to lower blood pressure or cholesterol can provide a window of opportunity to use non-pharmacologic strategies to maintain lowered blood pressure/cholesterol, thereby reducing/eliminating reliance on the medication. Smoking cessation programs typically are more effective when both behavioral and pharmacologic treatments are combined to sustain cessation. Pharmacologic agents are typically more efficient at creating the desired effect but may have long-term side effects; behavioral treatments may be less efficient at creating change but may be more effective at sustaining conditions that have been achieved pharmacologically. The bottom line is straightforward: Rather than asking which approach is superior, use the strengths of both areas of science creatively to achieve a sustainable treatment effect that minimizes unwanted side effects and could not be attained by using either approach by itself.

In summary, I have tried to provide a few personal insights into the events leading to the formalization of behavioral medicine as a viable, vibrant perspective on the promotion of health and the prevention and treatment of disease and as the multidisciplinary inquiry into the underlying mechanisms involving brain, genes, behavior, and physiology/biology. I hope that this provides a useful historical "snapshot" as you immerse yourself in the impressive array of accomplishments chronicled in this encyclopedia.

The following Foreword by Neil Schneiderman presents a personal view of behavioral medicine's future.

Stephen M. Weiss

Foreword

A Personal View of Behavioral Medicine's Future

The field of behavioral medicine appears to have a bright, important future. That is because contemporary scholarship in behavioral medicine has been constructed upon a solid foundation consisting of basic biological and behavioral science, population-based studies, and randomized clinical trials (RCT). The edifice that is emerging derives its strength and form from its interdisciplinary structure. It derives its reach and potential for future growth from its selection of key building materials and tools including the study of etiology, pathogenesis, diagnosis, treatment, rehabilitation, prevention, health promotion and community health. Because behavioral medicine approaches to prevention, treatment, and health promotion involve important relationships among behavior, psychosocial processes, and the sociocultural context, the roof of this structure will both consist of and benefit from the support of informed patients and populations, thoughtful educated health-care providers and involved communities.

Let us begin with population-based studies. During the second half of the twentieth century, epidemiological studies described important associations between traditional risk factors on the one hand and morbidity and mortality on the other, but elucidated relatively few of the variables mediating these associations. In my own area of cardiovascular disease (CVD) research, considerable attention has now focused upon obesity, inflammation, insulin resistance, oxidative stress, and hemostatic mechanisms as potential mediators. In this respect, traditional large-scale multicenter population-based studies have done a better job of describing the association between traditional risk factors (abnormal lipids, hypertension, smoking, diabetes, age) and CVD and their putative mediators than they have in describing the associations between biobehavioral, psychosocial, and sociocultural risk factors and CVD, and their mediators. However, this is now beginning to be addressed in such National Institute of Health (NIH) multicenter studies as the Hispanic Community Health Study/Study of Latinos (HCHS/SOL), Coronary Risk Development in Young Adults (CARDIA), and Multi-Ethnic Study of Atherosclerosis (MESA). Some of these studies are employing such preclinical measures of disease as carotid intimal-medial wall thickness and plaque by ultrasonography and coronary artery calcium by computed tomography to examine the progression of disease processes relating risk factors and CVD.

The examination of preclinical markers of disease as mediators between biobehavioral, psychosocial, and sociocultural risk factors on the one hand and chronic diseases on the other has been facilitated by the availability of

commercial assays. These assays have permitted the study of biomarkers involved in preclinical disease processes including adhesion molecules, pro-inflammatory cytokines, and oxidative stress in both animal and human studies. We can expect that many further advances will be made in the development of commercially available research methods and that they will increase our understanding of relationships among biobehavioral, psychosocial, and sociocultural risk factors and the pathophysiology of CVD, cancer, and other chronic diseases.

Although a wide range of epidemiological studies have called attention to potentially modifiable risk factors, and most chronic disease risk factors are modifiable (Yusuf et al., 2004), it should be recognized that chronic disease outcomes are the result of the joint effects of risk genes, the environment, and behavior upon these risk factors. One can therefore expect that on the basis of genomic analyses, future studies will begin to identify the extent to which particular individuals are vulnerable to specific risk factors and diseases and may be candidates for targeted behavioral as well as pharmacological interventions. Thus, in the coming era of personalized or tailored medicine, we may expect that behavioral medicine research will play an important role both in understanding the antecedents of disease that interact with genomic predispositions and in selecting appropriate treatment interventions.

The future for behavioral medicine science playing an essential role in population-based observational studies appears to be inevitable. This will occur because both the fields of behavioral medicine and epidemiology have expanded their horizons based upon important scientific findings. Early epidemiological studies focused upon hygiene and infectious diseases. By the middle of the twentieth century, epidemiological studies were examining the prevalence of multiple risk factors (e.g., smoking, dyslipidemia, hypertension) and disease outcomes (e.g., coronary heart disease [CHD], stroke, cancers). However, more recent multicenter observational studies have increasingly identified behavioral, psychosocial, and sociocultural variables as potential risk factors for chronic diseases. Thus, future multicenter observational studies will likely include demographic (e.g., racial/ethnic background, sex, socioeconomic status, neighborhood environments), psychosocial (e.g., temperament and personality, marital and work stressors, social support), lifestyle (e.g., medication adherence, diet, sleep, physical activity, smoking), biomarkers (e.g., immune, inflammatory, hemostatic, imaging), and genomic factors that influence disease outcomes. An important trend that is likely to increase in the future is the development of consortia of population-based studies (e.g., Population of Architecture using Genomics and Epidemiology: PAGE) whose purpose is to investigate mature genetic variants associated with complex diseases in large diverse populations. Such consortium studies are each beginning to include well over 100,000 participants. Perhaps most importantly the constituent studies and the consortia will be able to follow participants over the course of many years, providing important incidence data that will allow us to examine the specific causal variables influencing the course of disease. This represents an important opportunity for behavioral medicine scientists.

Traditional observational studies have often reported findings using odds ratios, which provide estimates (with confidence interval) for the relationship between binary variables. Such studies have also permitted assessment of the effects of other variables on specific relationships using regression analyses. More recently, scientific interest in understanding the role of potential mediators of relationship between risk factors and disease outcomes has increasingly led to the use of analytic techniques such as structural equation modeling including path analysis, which until now have mostly been used in the social sciences. We can expect that a dramatic improvement in our understanding of the mediators between risk factors and disease outcomes will occur in the coming years.

The completion of the Human Genome Project in 2003 led to an increased interest in gene-environment interactions within the behavioral medicine research community. Such interactions occur when genetic factors affect measured phenotypes differentially, for example, when men with the E4 allele of the apolipoprotein E gene (APOE) were shown to have an increased smoking related risk for CHD events (Humphries et al., 2001). Other studies have shown that the interaction of the alpha 2ß-adrenergic receptor polymorphism with job strain is related to elevated blood pressure (Ohlin et al., 2007), and several other studies have related gene polymorphisms with cardiovascular reactivity to mental challenge. Most behavioral studies that have examined gene-environment interactions have been carried out on relatively small samples, but it appears inevitable that a large number of high-quality, well-powered, gene-environment studies of direct relevance to behavioral medicine will be initiated during the next few years.

In addition to the structural genomics exemplified in gene-environment interaction studies, functional genomic studies are also likely to become of increasing interest to behavioral medicine investigators. Briefly, functional genomics focuses on the basics of protein synthesis, which is how genes are "switched on" to provide messenger RNA (mRNA). Francis Crick, who along with James Watson discovered the structure of the DNA molecule, originally thought that each gene, consisting of a particular DNA sequence, codes for one specific mRNA molecule that in turn codes for a specific protein (Crick, 1970). Subsequently, it became evident that after being transcribed, most mRNA molecules undergo an editing process with some segments being spliced out. In this way, a gene can lead to more than one type of mRNA molecule and consequently more than one type of protein. Thus human cells, which each contain about 25,000 genes, are able to synthesize more than 100,000 different proteins.

Epigenetics refers to the altering of gene function without changes in the DNA sequence. This can occur either by methylation of the DNA itself or by remodeling of the chromatin structure in which the DNA is packaged. Because of these processes, in utero exposure to nutrition or social factors can cause permanent modification of gene expression patterns that may lead to increased risk of mental disorders, diabetes, cancer, or cardiovascular diseases (Jirtle & Skinner, 2007). As an example of how social exposure in early life can have long-duration epigenetic and phenotypic influences, Meaney and Szyf (2005) showed that neonatal rodents who received high

levels of postpartum nurturing revealed diminished cortisol responses to stressful experiences when they reached adulthood. Such studies are providing a strong basis for future epigenetic behavioral medicine research.

The important advances made by observational and mechanistic studies relevant to behavioral medicine research are paralleled by a few RCT that have provided evidence that behavioral interventions aimed at modifying lifestyle or psychosocial variables can help prevent morbidity and/or mortality in high-risk populations. Thus, for example, the Diabetes Prevention Program trial (Knowler et al., 2002) in the United States and the Finnish Diabetes Prevention Trial (Tuomilehto et al., 2001) each observed that lifestyle interventions targeting weight loss and an increase in physical activity can reduce the incidence of diabetes in prediabetic patients. Based upon the success of these trials, the NIH has sponsored Look AHEAD (Action for Health in Diabetes), an RCT that is scheduled to last for 11.5 years. This trial is specifically examining whether an intensive lifestyle intervention similar to that used in the Diabetes Prevention Program can prevent major CVD events in obese participants with type 2 diabetes. Whereas the diabetes prevention projects and the Look AHEAD trial are essential for establishing that lifestyle interventions can prevent type 2 diabetes and reduce CVD risk in diabetic patients, subsequent investigation will be needed for us to learn how such interventions can be applied to clinical practice.

Although psychosocial-behavioral RCT conducted upon patients following major adverse coronary events (e.g., myocardial infarction) have yielded both positive and null results, the three major trials that have reported positive results share important similarities that differentiate them from the studies reporting null results (Friedman et al., 1986; Gulliksson et al., 2011; Orth-Gomér et al., 2009). Thus, the participants in the three major RCT reporting positive results all received group-based cognitive behavior therapy that included, in addition to cognitive behavior therapy, relaxation training and attention to lifestyle problems. The interventions all included up to 20 sessions over a year or more and used therapists specifically trained to use behavior change techniques in order to conduct behavioral interventions with cardiac patients. Treatment began at least several months after the CHD event and patients were followed up for an average of 4.5–7.8 years. Although the trials yielding positive results each studied between 237 and 862 participants, the size of each study was insufficient to permit assessment of the efficacy of specific intervention components, the role of potential biological mediators or the applicability of the intervention to populations differing in terms of important demographic characteristics. Thus there is still a need to replicate and amplify the results of the previously successful trials in rigorous, large-scale, multicenter RCT that can identify the demographic, psychosocial, and lifestyle variables that influence specific behavioral and biological determinants of risk.

In the future, evidence-based medicine will play an ever-increasing role in clinical health care. The extent to which behavioral medicine can become a successful part of health-care delivery systems will in large part depend upon investigators in the field being able to master clinical translational

research, moving from efficacy to effectiveness with a high ratio of benefit to cost. Thus, for example, the Diabetes Prevention Program (Knowler et al., 2002) showed that in high-risk patients, a lifestyle intervention reduced the incidence of diabetes significantly better than a pharmacological intervention and that both interventions were superior to a placebo condition. However, the lifestyle intervention was labor intensive and required considerable effort to get participants to maintain improvement. In contrast, maintaining adherence to taking a pill once daily may pose a less daunting task. However, recent advances in web-based intervention research may level the playing field. Thus, automatic e-mail reminders, phone or e-mail based consultations with a health-care professional, interaction with web-based programs, and the instant availability of important specially tailored information on an interactive website can all help patient adherence. To the extent that weight loss programs that involve diet and exercise do more than only decrease the risk of type 2 diabetes but also improve other aspects of CVD risk, such programs are particularly valuable in terms of health promotion.

The RCT that decreased morbidity or mortality rate in CHD patients each required 20 or more group-based sessions (Friedman et al., 1986; Gulliksson et al., 2011; Orth-Gomér et al., 2009). When amortized over the length of the 4.5–7.8 year follow-up period, however, the cost compares favorably with that of most drugs also used in treatment. Participating in 20 or more sessions also poses a personal cost and some hardship for many people. However, the implementation of interactive web-based group sessions using both sound and video could obviate the need for most face-to-face meetings and allow interpersonal interactions to continue over long periods of time. It therefore seems apparent that the rapid advances taking place in science and practice during the internet era will prove helpful in making behavioral medicine an important ingredient of future health-care systems.

Future health-care systems could be strengthened by well-informed patients and by health-care providers who are grounded in behavioral medicine concepts as well as clinical medicine. Attention to the human and health-influencing aspects of neighborhoods (i.e., the built environment) are also important and dependent on informed public policy. Because Behavioral Medicine has been constructed based on the understanding of relationships among behavior, psychosocial processes, and sociocultural contexts, the field is well positioned to take a leadership role in informing future health-care policies. The field of behavioral medicine appears to have a bright, important future.

Neil Schneiderman

References

Crick, F. (1970). Central dogma of molecular biology. *Nature*, *227*, 561–563. PMID: 4913914.

Friedman, M., Thoresen, C. E., Gill, J. J., Ulmer, D., Powell, L. H., & Price, V. A., et al. (1986). Alteration of type A behavior and its effect on cardiac recurrences in post myocardial infarction patients: Summary results of the recurrent coronary prevention project. *American Heart Journal*, *112*, 653–655. PMID: 3766365.

Gulliksson, M., Burell, G., Vessby, B., Lundin, L., Toss, H., & Svärdsudd, K. (2011). Randomized controlled trial of cognitive behavioral therapy vs standard treatment to prevent recurrent cardiovascular events in patients with coronary heart disease: Secondary prevention in Uppsala primary health care project (SUPRIM). *Archives of Internal Medicine*, *171*, 134–140. PMID: 21263103.

Humphries, S. E., Talmud, P. J., Hawe, E., Bolla, M., Day, I. N., Miller, G. J. (2001). Apolipoprotein E4 and coronary heart disease in middle-aged men who smoke: a prospective study. *Lancet*, *385*, 115–119. PMID: 11463413.

Jirtle, R. L., & Skinner, M. K. (2007). Environmental epigenomics and disease susceptibility. *Nature Reviews Genetics*, *8*, 253–262. PMID: 17363974.

Knowler, W. C., Barrett-Connor, E., Fowler, S. E., Hamman, R. F., Lachin, J. M., et al., & Diabetes Prevention Program Research Group. (2002). Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *The New England Journal of Medicine*, *346*, 393–403. PMID: 11832527.

Meaney, M. J., & Szyf, M. (2005). Environmental programming of stress responses through DNA methylation life at the interface between a dynamic environment and a fixed genome. *Dialogues in Clinical Neuroscience*, *7*, 103–123. PMID: 16262207.

Ohlin, S. E., Berglund, G., Nilsson, P., & Melander, O. (2007). Job strain, decision latitude and alpha 2ß-adrenergic receptor polymorphisms significantly interact and associate with high blood pressure in men. *Journal of Hypertension*, *25*, 1613–1619. PMID: 18622236.

Orth-Gomér, K., Schneiderman, N., Wang, H., Walldin, C., Bloom, M., & Jernberg, T. (2009). Stress reduction prolongs life in women with coronary disease: The Stockholm Women's Intervention Trial for Coronary Heart Disease (SWITCHD). *Circulation: Cardiovascular Quality and Outcomes*, *2*, 25–32. PMID: 20031809.

Tuomilehto, J., Lindström, J., Eriksson, J. G., Valle, T. T., Hämäläinen, H., et al., & Finnish Diabetes Prevention Study Group. (2001). Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *The New England Journal of Medicine*, *344*, 1343–1350. PMID: 11333990.

Yusuf, S., Hawken, S., Ounpuu, S., Dans, T., Avezum, A., Lanas, F., & McQueen, M., et al. (2004). Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries: The INTERHEART study: case–control study. *Lancet*, *364*, 937–952. PMID: 15364185.

Preface

The establishment, advancement, and maturation of the field of behavioral medicine bears witness to interest among research scientists, clinicians, and policy makers in psychological, behavioral, and social influences on health and disease from the perspective of both the individual patient and global public health. It has become increasingly clear that such influences may negatively impact health and well-being and, equally importantly, that behavioral interventions may be protective and curative.

Neal Miller (1909–2002), an American psychologist and recipient of the National Medal of Science in 1964, is often credited as being the founder of behavioral medicine. He made significant contributions to our understanding of the relationship between reinforcement mechanisms and the control of autonomic behavior, and in pioneering the field of biofeedback, which is used successfully today to treat a variety of medical conditions. The original definition of behavioral medicine was developed at the Yale Conference on Behavioral Medicine and later published by Gary Schwartz and Stephen Weiss (1977):

> "Behavioral medicine" is the field concerned with the development of behavioral-science knowledge and techniques relevant to the understanding of physical health and illness, and the application of this knowledge and these techniques to diagnosis, prevention, treatment and rehabilitation.

While this definition remains the cornerstone of our interdisciplinary and integrative field, developments in many relevant subfields have advanced at rapid rates, and whole new specialties have arisen. This evolution was well exemplified by the publication in 2010 of the *Handbook of Behavioral Medicine* (Steptoe, 2010). Relevant knowledge and understanding of issues of interest in behavioral medicine is now contributed by the disciplines of and expertise from anthropology, behavioral and molecular genetics, behavioral science, biostatistics, clinical medicine, cultural studies, epidemiology, health economics, general medicine, genomics, psychiatry, psychology, physiology, public health and public health policy, and sociology, to name but a few. It was therefore considered an opportune and appropriate time to create the *Encyclopedia of Behavioral Medicine*, whose publication coincides with the 12th International Congress of Behavioral Medicine, held on August 29th to September 1st, 2012, in Budapest, with attendees representing multiple disciplines and many countries around the globe. The theme of

the meeting is "Behavioral Medicine: From Basic Science to Clinical Investigation and Public Health," the theme around which this *Encyclopedia* has been developed.

Accordingly, the *Encyclopedia* contains entries falling into three categories or domains that represent issues of interest: basic research, clinical investigation and practice, and public health and public health policy. The domain of basic research addresses the key questions of mechanisms of action, both in terms of how behavior can have a deleterious impact on health and how a change in behavior can be beneficial, either preventively or therapeutically. The domain of clinical investigation and practice translates this basic knowledge into clinical interventions on a patient-by-patient basis. Finally, the domain of public health and public health policy takes a broader view of how behavioral medicine research and interventions can impact the health of populations at the community, regional, national, and global levels. This includes addressing the system-wide/public education and advocacy/political activities that are needed to facilitate maximum benefits at the global level.

It can immediately be seen that behavioral medicine is indeed a multidisciplinary and interdisciplinary field. Researching mechanisms of action requires a detailed level of human biology, starting from the molecular genetic level and progressing from cellular to organ to whole-body study. A thorough understanding of environmental interactions with biological functioning is also necessary. The domains of basic research and clinical investigation and practice are linked by the increasingly important concept of translational medicine, that is, how to translate our mechanistic knowledge and understanding into successful clinical interventions most effectively and efficiently. The final challenge, likely the most challenging but ultimately providing the greatest benefit, is to address these interventions at the public health level.

Within these overarching categories, it is possible to group together various entries into categories of interest to individual readers or groups of readers pursuing their own research in cross-cutting areas. One example might be the impact of behavioral medicine research and interventions across the life span, that is, taking a life cycle approach. Entries in the *Encyclopedia* such as Children's Health Study, Elderly, End-of-Life Care, Geriatric Medicine, Life Span, Obesity in Children, and Successful Aging might be instructive in this case.

A second example might be looking at genetic predisposition to the deleterious impact of environmental factors and, equally of interest, to the therapeutic benefit of certain behavioral medicine interventions. Entries of interest here might be Family Studies (Genetics), Gene-Environment Interaction, Gene Expression, Genome-wide Association Study, and Twin Studies. While not always intuitively obvious, one of the most powerful ways to study the effects of environmental (behavioral) factors on a phenotype of interest (e.g., a given disease state or condition of clinical concern) is to study genetic influence on that phenotype (Plomin et al., 1997). Having done so, it is possible to remove from consideration the individual variation attributable to genetic influence and hence to focus on variation attributable to

environmental and gene-environment interaction influences. We are certain that readers will find many such groupings of entries relevant to their own interests and research.

Additional evidence of the growth of the discipline of behavioral medicine is provided by the fact that training in the field can be found in universities around the world, ensuring that the next generation of researchers and practitioners will be trained by current experts. Before going on to specialize in behavioral medicine research or clinical practice, individuals often receive their terminal degrees in disciplines such as medicine, public health, nursing, and psychology. Such diversity is a tremendous strength in this interdisciplinary field.

Like all such printed endeavors, the *Encyclopedia* proves a "snapshot in time" of its subject. Research during the past 30 years has provided the solid foundation from which future advances will be made, and it will be of great interest to all of us in behavioral medicine to follow its further development. We are grateful to Stephen Weiss for providing a Foreword entitled "Early Developments in the Field of Behavioral Medicine," which reviews important events in the discipline's evolution, and to Neil Schneiderman for providing a Foreword entitled "A Personal View of Behavioral Medicine's Future," which provides an insightful view of likely trajectories and benefits of our discipline. We hope that subsequent editions will provide additional snapshots in due course.

Miami, July 2012 Marc D. Gellman and J. Rick Turner

References

Schwartz, G., & Weiss, S. (1977). What is behavioral medicine. *Psychosomatic Medicine*, *39*(6), 377–381.

Steptoe, A. (Ed.) (2010). *Handbook of behavioral medicine: Methods and applications*. New York: Springer.

Plomin, R., DeFries, J. C., McClearn, G. E., & Rutter, M. (1997). *Behavioral genetics* (3rd ed.). New York: WH Freeman & Company.

Acknowledgments

The conceptualization, writing, and publication of the *Encyclopedia of Behavioral Medicine* occurred over a 4-year period. The idea originated with our attentive and thoughtful Senior Editor from Springer, Janice Stern. Without Janice's forward thinking this *Encyclopedia* would not have materialized, and we are deeply grateful for her vision and support throughout its development. She first approached me (MDG) at the International Congress of Behavioral Medicine held in Tokyo, in August 2008. At the time it seemed to be a formidable task, to compile all of the terms that are used in the field of behavioral medicine into an A–Z compendium. My next meeting with Janice occurred in Montreal at a Society of Behavioral Medicine meeting during spring 2009. It was at that time my co-editor (JRT) had agreed to join forces with me in this effort. We then set about creating an advisory board, comprised of leading international experts in the field of behavioral medicine, and selecting our outstanding group of associate editors. They come from various parts of the world and permit this work to be truly an international collaboration. Without their hard work and dedication to this project we would not have the *Encyclopedia of Behavioral Medicine* in the form that you see it. They assisted in the selection of the terms that are included in the *Encyclopedia* and in the selection of the authors who developed each of the entries: our sincere thanks to all of you. We are indebted to the Springer Major Reference Works team including Anil Chandy, Tina Shelton, and Meetu Lall. This is the team that kept us organized, on task, and was instrumental in so many ways in bringing the *Encyclopedia* to fruition.

My journey in the interdisciplinary field of behavioral medicine has allowed me to delve into the fields of epidemiology, medicine, neuroscience, psychology, pharmacology, physiology, and public health. Throughout this entire journey, my mentor and teacher, Neil Schneiderman, has shaped my career. From my time as an undergraduate student, through graduate school, a postdoctoral fellowship, and on to becoming a faculty member, I have been fortunate to be by Neil's side. For this, first and foremost, I would like to acknowledge and dedicate this *Encyclopedia* to him.

To my wife Jill Turner, who has been by my side throughout the development of the *Encyclopedia*, I could not have done this without your patience and support.

MDG

I moved to the United States in 1987 to join Paul Obrist's group at the University of North Carolina at Chapel Hill. I met Neil Schneiderman shortly

thereafter, and he has been a great source of personal and professional support since that time. I am delighted that Marc has dedicated the *Encyclopedia* to him. I would like to acknowledge the scientific training I received at the University of Sheffield and the University of Birmingham. My doctoral work in cardiovascular psychophysiology and cardiovascular behavioral medicine was conducted in Birmingham under the supervision of Doug Carroll, with John Hewitt providing additional guidance in the fields of Statistics and behavioral genetics.

I would also like to thank my wife Karen for her support during the preparation of the *Encyclopedia*.

JRT

About the Editors

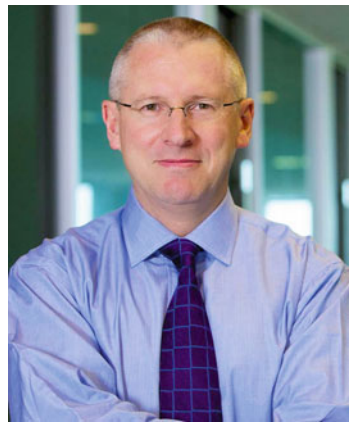
Marc D. Gellman is a Research Associate Professor of Psychology and the Associate Director of the Division of Health Psychology, Department of Psychology, University of Miami, Florida, USA. He is also Associate Director of the Behavioral Medicine Research Center and Associate Director of the Behavioral Medicine Training Program located at the Miller School of Medicine, University of Miami, where he holds a secondary appointment in the Department of Medicine. He has been a member of the faculty of the University of Miami since 1986. Dr. Gellman received all of his formal training (B.S., M.S., and Ph.D. degrees) from the University of Miami.

Since 1986, he has been continuously funded by the National Institutes of Health, primarily in the area of cardiovascular behavioral medicine. Dr. Gellman has published in a variety of journals including: *Psychosomatic Medicine*, *Health Psychology*, *Annals of Behavioral Medicine*, *Psychophysiology*, and others. Dr. Gellman currently serves on the editorial advisory board for the McGraw-Hill *Annual Editions: Drugs, Society, and Behavior*. He previously served on the editorial board of the Sage Publications scientific book series Behavioral Medicine and Health Psychology from 1997 to 2004, edited by J. Rick Turner, his co-editor for this *Encyclopedia*.

Dr. Gellman is a former board member of the International Society of Behavioral Medicine, serving as its secretary 2004–2008 and chair of the communications committee 2000–2004. From 2004 to 2006, he served as program co-chair for the International Congress of Behavioral Medicine. Dr. Gellman is a longtime board member of the Society of Behavioral

Medicine, serving in various capacities from 1996 to 2007. He continues to serve as a member of the Wisdom Council for the Society of Behavioral Medicine. Dr. Gellman is the recipient of the Distinguished Service Award from the Society of Behavioral Medicine and the Outstanding Service Award from the International Society of Behavioral Medicine. Dr. Gellman is a Fellow of the Society of Behavioral Medicine. He is also a member of the American Psychological Association, the American Psychosomatic Society, the Society of Behavioral Medicine, and the International Society of Behavioral Medicine.

In his spare time, he is an avid bicycle rider and enjoys "out of car experiences" with his wife Jill, touring numerous countries on their tandem bicycle. He is a wine aficionado, an enthusiast of rock, jazz, and reggae music, and occasionally lectures on the influence drugs have on culture, being inspired by his attendance at the historic Woodstock Music and Art Festival in 1969.



Rick Turner is Senior Scientific Director, Cardiovascular Safety, at Quintiles, a fully integrated biopharmaceutical services provider. He joined Quintiles after serving as the chairman of the Department of Clinical Research at the Campbell University School of Pharmacy. Prior to that, he was a principal clinical submissions scientist at GlaxoSmithKline, where he received awards for his work on the GlaxoSmithKline Clinical Trial Registry and in product development. He is also Senior Fellow, Center for Medicine in the Public Interest, and President and Chief Scientific Officer, Turner Medical Communications LLC.

Dr. Turner received his Ph.D. in psychophysiology and cardiovascular behavioral medicine from the University of Birmingham, United Kingdom, where he trained under Douglas Carroll. Following a postdoctoral fellowship there, he moved to the University of North Carolina at Chapel Hill to work in the laboratory of the late Paul Obrist and Kathleen Light. His work in the field of cardiovascular behavioral medicine led to 50 peer-reviewed publications and the receipt of two international research awards, the 1998 Distinguished

Scientific Award for an Early Career Contribution to Psychophysiology from the Society for Psychophysiological Research and the 1993 Early Career Award for Contributions to Psychosomatic Medicine from the American Psychosomatic Society. He is the author of the 1994 book *Cardiovascular Reactivity and Stress: Patterns of Physiological Response*, and was a coeditor of the book *Health and Behavior in Childhood and Adolescence*, which received a 2003 *American Journal of Nursing* Book of the Year Award. He became a Fellow of the Society of Behavioral Medicine in 1999.

Since entering the biopharmaceutical industry, Dr. Turner has published extensively in peer-reviewed and professional journals, and authored six books addressing methodological and statistical aspects of randomized, concurrently controlled clinical trials. He was a participant in the 2010 National Heart, Lung, and Blood Institute Clinical Trials Symposium, giving an invited presentation entitled "The Power of the Randomized, Concurrently Controlled Clinical Trial," and he travels extensively to provide clinical trial consulting services to biopharmaceutical companies worldwide and deliver presentations on drug safety topics at international scientific conferences.

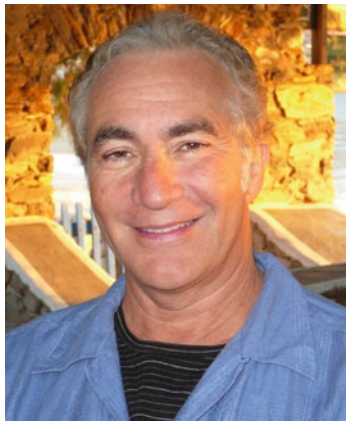
Dr. Turner is particularly interested in the development of drugs for type 2 diabetes mellitus. He has testified before two U.S. Food and Drug Administration committees, the Endocrinologic and Metabolic Drugs Advisory Committee and the Drug Safety and Risk Management Advisory Committee, and is working with both biopharmaceutical companies and regulators to expedite the development of drugs for this disease. He is also an advocate of increasing adherence to drugs for all chronic diseases, including diabetes, by greater use of knowledge and strategies developed in the field of behavioral medicine.

Dr. Turner is on the editorial board of the peer-reviewed *Journal for Clinical Studies* and is editor-in-chief of the peer-reviewed *Drug Information Journal*.

Associate Editors

Mustafa al'Absi
University of Minnesota Medical School
University of Minnesota
235 School of Medicine
Duluth, MN
USA

Alan J. Christensen
Department of Psychology
The University of Iowa Spence Laboratories of Psychology
Iowa City, Iowa
USA

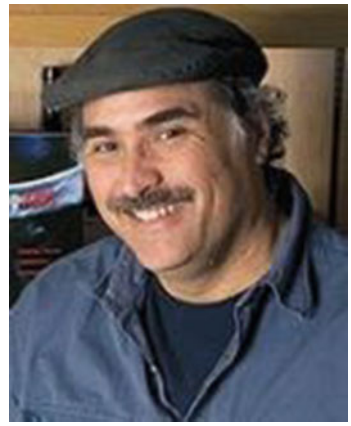


Alan M. Delamater
Department of Pediatrics
University of Miami Miller School of Medicine
Miami, FL
USA

Yori Gidron
Faculty of Medicine & Pharmacy
Free University of Brussels (VUB)
Jette
Belgium

Martica H. Hall
Department of Psychiatry
University of Pittsburgh
Pittsburgh, PA
USA

Peter A. Hall
Faculty of Applied Health Sciences
University of Waterloo
Waterloo, ON
Canada



Seth Kalichman
Department of Psychology
University of Connecticut Center for Health Intervention and Prevention
Storrs, CT
USA

Kevin Masters
Department of Psychology
University of Colorado Denver
Denver, CO
USA

Urs M. Nater
Department of Psychology
University of Marburg
Marburg
Germany

Frank J. Penedo
Feinberg School of Medicine
Northwestern University
Chicago, IL

Anna C. Phillips
Sport & Exercise Sciences
University of Birmingham
Edgbaston, Birmingham
UK

Barbara Resnick
School of Nursing
University of Maryland
Baltimore, MD
USA

Daichi Shimbo
Columbia University
New York, NY
USA

Ingrid Soderback
University Lecturer Emerita
Department of Public Health and
Caring Science
Uppsala University
Uppsala, SE
Sweden

Antti Uutela
Department for Lifestyle and Health
National Institute for Health and Welfare (THL)
Helsinki
Finland

Deborah J. Wiebe
Division of Psychology
Department of Psychiatry
University of Texas Southwestern Medical Center
Dallas, TX
USA

Kazuhiro Yoshiuchi
Department of Stress Sciences & Psychosomatic Medicine
The University of Tokyo
Bunkyo-ku, Tokyo
Japan

Advisory Board

Contributors

David B. Abrams Johns Hopkins Bloomberg School of Public Health, The Schroeder Institute for Tobacco Research and Policy Studies at Legacy, Washington, DC, USA

Howard Aizenstein Geriatric Psychiatry Neuroimaging, Western Psychiatric Institute and Clinic, University of Pittsburgh Medical Center, Pittsburgh, PA, USA

Tatsuo Akechi Department of Psychiatry and Cognitive-Behavioral Medicine, Graduate School of Medical Sciences, Nagoya City University, Nagoya, Aichi, Japan

Mustafa al'Absi University of Minnesota Medical School, University of Minnesota, 235 School of Medicine, Duluth, MN, USA

Melissa A. Alderfer Division of Oncology, The Children's Hospital of Philadelphia, Philadelphia, PA, USA

Sarah Aldred School of Sport and Exercise Sciences, The University of Birmingham, Edgbaston, Birmingham, UK

Julia Allan School of Medicine and Dentistry, University of Aberdeen, Foresterhill, Aberdeen, Scotland, UK

Peter Allebeck Department of Public Health Sciences, Karolinska Institute, Stockholm, Sweden

Josh Allen Care and Compliance Group, Inc. American Assisted Living Nurses Association, Wildomar, CA, USA

Kacie C. Allen Human Nutrition, Foods, and Exercise, Virginia Tech, Roanoke, VA, USA

Leila Anane School of Sport and Exercise Sciences, The University of Birmingham, Birmingham, UK

David E. Anderson Division of Nephrology, Department of Medicine, University of California, San Francisco, CA, USA

Giles M. Anderson School of Psychology, University of Birmingham, Edgbaston, Birmingham, UK

Norman B. Anderson American Psychological Association, Washington, DC, USA

Gerhard Andersson Department of Behavioral Sciences and Learning, Linköping University, Linköping, Sweden

Tetusya Ando Department of Psychosomatic Research, National Institute of Mental Health, National Center of Neurology and Psychiatry, Kodaira-shi, Tokyo, Japan

Mike Antoni Department of Psychology, University of Miami, Sylvester Cancer Center, Miller School of Medicine, Miami, FL, USA

William Arguelles Department of Psychology, University of Miami, Coral Gables, FL, USA

Wiebke Arlt School of Sport & Exercise Sciences, The University of Birmingham, Edgbaston, Birmingham, UK

Arpo Aromaa Health and Functional Capacity, National Institute for Health and Welfare, Helsinki, Finland

Elva Arredondo Division of Health Promotion and Behavioral Sciences, San Diego State University, San Diego, CA, USA

Lisa G. Aspinwall Department of Psychology, The University of Utah, Salt Lake City, UT, USA

Kristin J. August Department of Psychology, Rutgers University, Camden, NJ, USA

Simon Bacon Department of Exercise Science, Concordia University, Montreal Behavioral Medicine Centre, Montreal, QC, Canada

Rachel N. Baek Clinical Psychology, Health Emphasis, Ferkauf Graduate School of Psychology, Yeshiva University, Bronx, NY, USA

Jonathan Z. Bakdash Department of Psychology, University of Utah, Salt Lake City, UT, USA

Center for Human Factors in Patient Safety, VA Salt Lake City Health Care System, Salt Lake City, UT, USA

U.S. Army Research Laboratory, Aberdeen Proving Ground, MD, USA

Elizabeth Baker Department of Psychology, University of Miami, Coral Gables, FL, USA

Austin S. Baldwin Department of Psychology, Southern Methodist University, Dallas, TX, USA

Chad Barrett Department of Psychology, University of Colorado Denver, Denver, CO, USA

Abigail Batchelder Yeshiva University, Bronx, NY, USA

G. David Batty Department of Epidemiology and Public Health, University College London, London, UK

Linda C. Baumann School of Nursing, University of Wisconsin-Madison, Madison, WI, USA

Carolyn Baum School of Medicine in St Louis, Washington University, St. Louis, MO, USA

Elliott A. Beaton Department of Psychiatry and Behavioral Sciences and the M.I.N.D. Institute, University of California-Davis, Sacramento, CA, USA

C. Andres Bedoya Behavioral Medicine Service Department of Psychiatry, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA

Catherine Benedict Department of Psychology, University of Miami, Coral Gables, FL, USA

Ryan M. Beveridge Department of Psychology, University of Delaware, Newark, DE, USA

Stephen Birch Clinical Epidemiology and Biostatistics (CHEPA), McMaster University, Hamilton, ON, Canada

Orit Birnbaum-Weitzman Department of Psychology, University of Miami, Miami, FL, USA

James A. Blumenthal Department of Psychiatry & Behavioral Sciences, Duke University Medical Center, Durham, NC, USA

Guy Bodenmann Department of Psychology, University of Zurich, Binzmuehlestrasse, Zurich, Switzerland

Marie Boltz College of Nursing, New York University, New York, NY, USA

Susan J. Bondy Dalla Lana School of Public Health, University of Toronto, Toronto, ON, Canada

Stephan Bongard Department of Psychology, Goethe-University, Frankfurt am Main, Germany

Brian Borsari Department of Veterans Affairs Medical Center, Mental Health and Behavioral Sciences Service, Providence, RI, USA

Department of Behavioral and Social Sciences, Center for Alcohol and Addiction Studies, Brown University, Providence, RI, USA

Jos A. Bosch Department of Clinical Psychology, Faculty of Social Behavioral Sciences, University of Amsterdam, The Netherlands

Kimberly Bowen Department of Psychology and Health Psychology Program, University of Utah, Salt Lake City, UT, USA

Stephanie Bowlin Department of Psychology, University of Kansas, Lawrence, KS, USA

Nicole Brandt School of Pharmacy, University of Maryland, Baltimore, MD, USA

Dana Brimmer Division of High-Consequence Pathogens and Pathology, Centers for Disease Control and Prevention, McKing Consulting Corporation, Atlanta, GA, USA

Carrie Brintz Department of Psychology, University of Miami, Coral Gables, FL, USA

J. F. Brosschot Clinical, Health and Neuro Psychology, Leiden University, Leiden, Netherlands

Jennifer L. Brown Department of Behavioral Sciences and Health Education, Emory University School of Public Health, Atlanta, GA, USA

Bonnie Bruce Division of Immunology and Rheumatology, Stanford University Department of Medicine, Palo Alto, CA, USA

Vaughn Bryant Behavioral and Social Sciences, Brown University, Providence, RI, USA

Patrícia Cardoso Buchain Occupational Therapist of the Occupational Therapy Service, Institute of Psychiatry, Hospital das Clínicas University of São Paulo Medical School, São Paulo, SP, Brazil

Ross Buck Communication Sciences and Psychology, University of Connecticut, Storrs, CT, USA

Romola S. Bucks School of Psychology, The University of Western Australia (M304), Crawley, WA, Australia

Donna C. Burdzy Department of Psychology, Bowling Green State University, Bowling Green, OH, USA

Rachel J. Burns Department of Psychology, University of Minnesota, Minneapolis, MN, USA

Victoria E. Burns School of Sport and Exercise Sciences, The University of Birmingham, Birmingham, UK

Michelle Nicole Burns Feinberg School of Medicine, Department of Preventive Medicine, Center for Behavioral Intervention Technologies, Northwestern University, Chicago, IL, USA

David Busse Department of Psychology and Social Behaviour, University of California, Irvine, Irvine, CA, USA

Natalie E. Bustillo Department of Psychology, University of Miami, Coral Gables, FL, USA

Colin D. Butler Centre for Epidemiology and Population Health, Australian National University, Canberra, ACT, Australia

Jorie Butler Department of Psychology, University of Utah, Salt Lake City, UT, USA

Melissa M. A. Buttner Department of Psychology, University of Iowa, Iowa City, IA, USA

John T. Cacioppo Department of Psychology, The University of Chicago, Chicago, IL, USA

Demetria Cain Center for Health Intervention and Prevention, University of Connecticut, Storrs, CT, USA

Matthew Calamia Department of Psychology, University of Iowa, Iowa City, IA, USA

David Cameron Department of Psychology, The University of Sheffield, Sheffield, UK

Linda D. Cameron Psychological Sciences, University of California, Merced, Merced, CA, USA

Nerissa Campbell Exercise and Health Psychology Laboratory, The University of Western Ontario, London, ON, Canada

Tavis S. Campbell Department of Psychology, University of Calgary, Calgary, AB, Canada

Rebecca Campo Huntsman Cancer Institute, University of Utah, Salt Lake City, UT, USA

Turhan Canli Department of Psychology, Stony Brook University Psychology B-214, Stony Brook, NY, USA

Elizabeth da Silva Cardoso Department of Educational Foundations & Counseling Programs, The City University of New York-Hunter College, New York, USA

Leeann Carey Melbourne Brain Centre, Heidelberg, VIC, Australia

McKenzie Carlisle Department of Psychology and Health Psychology Program, University of Utah, Salt Lake City, UT, USA

Jordan Carlson Public Health, San Diego State University, University of California San Diego, San Diego, CA, USA

Olveen Carrasquillo Division of General Medicine, Miller School of Medicine, University of Miami, Miami, FL, USA

Adriana Carrillo Department of Pediatrics, Miller School of Medicine, University of Miami, Miami, FL, USA

Linda Carroll Department of Public Health Sciences, University of Alberta, Edmonton, AB, Canada

Douglas Carroll School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Judith Carroll Cousins Center for Psychoneuroimmunology, University of California, Los Angeles, CA, USA

Jennifer Carter The University of Iowa, Iowa City, IA, USA

Charles Carver Department of Psychology, University of Miami, Coral Gables, FL, USA

Pedro C. Castellon Epidemiology and Public Health, Miller School of Medicine, University of Miami, Miami, FL, USA

Sherilynn F. Chan Department of Psychology, University of Miami, Coral Gables, FL, USA

Fong Chan Department of Rehabilitation Psychology and Special Education, University of Wisconsin-Madison, Madison, WI, USA

Matthieu Chansard Department of Psychiatry, The University of Texas Southwestern Medical Center at Dallas Columbia University/New York State Psychiaric Institute, Dallas, TX, USA

Stephenie Chaudoir Department of Psychology, Bradley University, Peoria, IL, USA

Margaret A. Chesney Department of Medicine & Center for Integrative Medicine, University of California, San Francisco, CA, USA

Ornit Chiba-Falek Duke University Medical Center, Durham, NC, USA

Yoichi Chida Research Department of Epidemiology & Public Health, University College London, London, UK

Michael S. Chmielewski Department of Psychology, University of Toronto, Toronto, ON, Canada

Kyung-Eun Choi Kliniken Essen Mitte, Klinik für Naturheilkunde und Integrative Medizin, Universität Duisburg-Essen, Am Deimelsberg 34a, Essen, Germany

Julie Chronister Department of Counseling, San Francisco State University, San Francisco, CA, USA

Cari J. Clark Department of Medicine, University of Minnesota, Minneapolis, MN, USA

Molly S. Clark Department of Family Medicine, University of Mississippi Medical Center, Jackson, MS, USA

Tyler Clark School of Psychology, The University of Sydney, Sydney, NSW, Australia

Benjamin L. Clarke Academic Health Center, School of Medicine-Duluth Campus, University of Minnesota, Duluth, MN, USA

Tainya C. Clarke Department of Epidemiology and Public Health, Miller School of Medicine, University of Miami, Miami, FL, USA

Lindy Clemson Ageing, Work & Health Research Unit, Faculty of Health Sciences, University of Sydney, NSW, Lidcombe, Australia

Lorenzo Cohen Department of General Oncology, Division of Cancer Medicine, The University of Texas MD Anderson Cancer Center, Houston, TX, USA

Susan E. Collins Department of Psychiatry and Behavioral Sciences, University of Washington, Harborview Medical Center, Seattle, WA, USA

Persis Commissariat Clinical Psychology, Health Emphasis, Ferkauf Graduate School of Psychology, Yeshiva University, Bronx, NY, USA

Richard J. Contrada Department of Psychology, Rutgers, The State University of New Jersey, Piscataway, NJ, USA

Michael James Coons Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Susannah D. Copland Obstetrics and Gynecology, Division of Reproductive Endocrinology and Fertility, Duke Fertility Center, Durham, NC, USA

Quirino Cordeiro Department of Psychiatry and Psychological Medicine, Santa Casa Medical School, São Paulo, SP, Brazil

Erin Costanzo Department of Psychiatry, Carbone Cancer Center, University of Wisconsin-Madison, Madison, WI, USA

Jennifer Creek Occupational Therapist, Guisborough, North Yorkshire, UK

Matthew Cribbet Department of Psychology, University of Utah, Salt Lake City, UT, USA

Hugo Critchley Brighton and Sussex Medical School, University of Sussex, Brighton, East Sussex, UK

Crista N. Crittenden Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA

Andrea Croom Department of Psychology, University of Texas Southwestern Medical Center, Dallas, TX, USA

Rick Crosby University of Kentucky, Lexington, KY, USA

Jennifer Cumming School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Cassie Cunningham College of Public Health, University of Iowa, Liberty, IA, USA

Maurizio Cutolo Department of Internal Medicine, Research Laboratories and Academic Unit of Clinical Rheumatology, University of Genova, Genova, Italy

Amber Daigre University of Miami, Miami, FL, USA

Catherine Darker Public Health & Primary Care, Trinity College, The University of Dublin, Dublin, Ireland

Karina Davidson Department of Medicine, Columbia University Medical Center, New York, NY, USA

Gary Davis Medical School Duluth, University of Minnesota, Duluth, MN, USA

Mary C. Davis Department of Psychology, Arizona State University, Tempe, AZ, USA

Karen Dawe School of Social and Community Medicine, University of Bristol, Oakfield House, Oakfield Grove, Bristol, UK

Marijke De Couck Free University of Brussels (VUB), Jette, Belgium

Maartje de Wit Medical Psychology, VU University Medical Center, Amsterdam, North Holland, The Netherlands

Justina Deary Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Scott DeBerard Department of Psychology, Utah State University, Logan, UT, USA

Joost Dekker Department of Psychiatry and Department of Rehabilitation Medicine, VU University Medical Centre, Amsterdam, The Netherlands

Alan M. Delamater Department of Pediatrics, University of Miami Miller School of Medicine, Miami, FL, USA

Kelly S. DeMartini Division of Substance Abuse, School of Medicine, Yale University, New Haven, CT, USA

Andrew DeMott Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Johan Denollet CoRPS – Center of Research on Psychology in Somatic diseases, Tilburg University, Tilburg, The Netherlands

Ellen-ge Denton Department of Medicine Center for Behavioral Cardiovascular Health, Columbia University Medical Center, New York, NY, USA

Stuart Derbyshire School of Psychology, The University of Birmingham, Edgbaston, Birmingham, UK

Martin Deschner Psychiatry, Division of Psychology, The University of Texas Southwestern Medical Center at Dallas, Dallas, TX, USA

Tamer F. Desouky Department of Psychology, The University of Texas at Arlington, Arlington, TX, USA

Mary Amanda Dew School of Medicine and Medical Center, University of Pittsburgh, Pittsburgh, PA, USA

Sally Dickerson Department of Psychology and Social Behavior, University of California, Irvine, Irvine, CA, USA

Andrea F. DiMartini School of Medicine and Medical Center, University of Pittsburgh, Pittsburgh, PA, USA

Joel E. Dimsdale Department of Psychiatry, University of California San Diego, La Jolla, CA, USA

Ding Ding Graduate School of Public Health/Department of Family Preventive Medicine, San Diego State University/University of California San Diego, San Diego, CA, USA

Beate Ditzen Division of Clinical Psychology and Psychotherapy, Department of Psychology, University of Zurich, Binzmuhlestrasse, Zurich, Switzerland

Diane Dixon Department of Psychology, University of Strathclyde, Glasgow, Scotland, UK

Susan Dorsey School of Nursing, University of Maryland, Baltimore, MD, USA

Monica Dowling Miller School of Medicine, University of Miami, Miami, FL, USA

Mark T. Drayson College of Medical and Dental Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Michelle Drerup Clinical Assistant Professor of Medicine, Sleep Disorders Center Neurological Institute, Cleveland Clinic, Cleveland, OH, USA

Frank A. Drews Department of Psychology, University of Utah, Salt Lake City, UT, USA

Center for Human Factors in Patient Safety, VA Salt Lake City Health Care System, Salt Lake City, UT, USA

Suzana Drobnjak Department of Psychology, University of Zurich, Binzmuehlestrasse, Switzerland

Alejandra Duenas School of Management, IESEG, Paris, France

Joan Duer-Hefele Columbia University, New York, NY, USA

Mariam Dum Jackson Memorial Hospital, Miami, FL, USA

Jennifer Duncan Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Valerie Earnshaw Department of Public Health, Yale University, New Haven, CT, USA

Lisa A. Eaton Center for Health, Intervention, and Prevention, University of Connecticut, New Haven, CT, USA

Moritz Thede Eckart General and Biological Psychology, Department of Psychology, University of Marburg, Marburg, Germany

Ulrike Ehlert Department of Psychology, University of Zurich, Binzmuehlestrasse, Zurich, Switzerland

Lorin Elias Department of Psychology, University of Saskatchewan, Saskatoon, SK, Canada

Helio Elkis Department and Institute of Psychiatry, University of São Paulo Medical School, São Paulo, SP, Brazil

Lee Ellington Department of Nursing, College of Nursing, University of Utah, Salt Lake City, UT, USA

Christopher G. Engeland Center for Wound Healing and Tissue Regeneration, University of Illinois, Chicago, IL, USA

Elissa S. Epel University of California, San Francisco, CA, USA

Jennifer Toller Erausquin Chronic Disease and Injury Prevention, NC Division of Public Health, Durham, NC, USA

Alexandra Erdmann Department of Psychiatry, Carbone Cancer Center, University of Wisconsin-Madison, Madison, WI, USA

Sabrina Esbitt Ferkauf Graduate School of Psychology, Yeshiva University, Bronx, NY, USA

Paul A. Estabrooks Translational Obesity Research Program, Virginia Tech Riverside, Roanoke, VA, USA

Susan A. Everson-Rose Department of Medicine, University of Minnesota, Minneapolis, MN, USA

Benjamin I. Felleman Seattle Pacific University, Seattle, Washington, USA

Molly Ferguson Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Cristina A. Fernandez Department of Epidemiology and Public Health, Miller School of Medicine, University of Miami, Miami, FL, USA

Tania C. T. Ferraz Alves Department and Institute of Psychiatry, University of São Paulo Medical School, São Paulo, SP, Brazil

Tiffany Field Touch Research Institute, University of Miami, School of Medicine, Mailman Center for Child Development, Miami, FL, USA

Robyn Fielder Center for Health and Behavior, Syracuse University, Syracuse, NY, USA

David J. Finitsis Department of Psychology, University of Connecticut, Storrs, CT, USA

Simona Fischbacher Klinische Psychologie und Psychotherapie, Universität Zürich, Zürich, Switzerland

Susanne Fischer Department of Psychology, Clinical Biopsychology, Philipps-University of Marburg, Marburg, Germany

Skye Fitzpatrick Department of Psychology, Ryerson University, Toronto, ON, Canada

Kelly Flannery School of Nursing, University of Maryland Baltimore, Baltimore, MD, USA

Magne Arve Flaten Department of Psychology, University of Tromsø, Tromsø, Norway

Serina Floyd Obstetrics and Gynecology, Duke Hospital, Raleigh, NC, USA

Rachel Flurie University of Maryland, Baltimore, MD, USA

Susan Folkman Department of Medicine, School of Medicine, University of California San Francisco, San Mateo, CA, USA

Katherine T. Fortenberry Department of Family and Preventative Medicine, The University of Utah, Salt Lake City, UT, USA

Andrew Fox Recovery and Wellbeing Inpatient Services, Birmingham and Solihull Mental Health NHS Trust, Birmingham, West Midlands, UK

Kristen R. Fox School of Medicine and Medical Center, University of Pittsburgh, Pittsburgh, PA, USA

Christopher France Department of Psychology, Ohio University, Athens, OH, USA

Janis L. France Department of Psychology, Ohio University, Athens, OH, USA

Anne Frankel Robert Stempel College of Public Health and Social Work, Florida International University, Miami, FL, USA

Elizabeth Franzmann Department of Otolaryngology/Division of Head and Neck, Miller School of Medicine, University of Miami, Miami, FL, USA

Fred Friedberg Psychiatry and Behavioral Sciences, Stony Brook University Medical Center, Stony Brook, NY, USA

Georita Marie Frierson Department of Psychology, Southern Methodist University, Dallas, TX, USA

Shin Fukudo Department of Behavioral Medicine, School of Medicine, Tohoku University Graduate, Aoba-ku, Sendai, Japan

Terry Fulmer Bouvé College of Health Sciences, Northeastern University, Boston, MA, USA

Jens Gaab Clinical Psychology and Psychotherapy, Department of Psychology, University of Basel, Basel, Switzerland

Amiram Gafni Department of Clinical Epidemiology and Biostatistics, Centre for Health Economics and Policy Analysis, McMaster University, Hamilton, ON, Canada

Elizabeth Galik School of Nursing, University of Maryland, Baltimore, MD, USA

Stephen Gallagher Department of Psychology, Faculty of Education & Health Sciences, University of Limerick, Castletroy, Limerick, Ireland

Steven Gambert Department of Medicine, School of Medicine, University of Maryland, Baltimore, MD, USA

Luis I. García Center for AIDS Intervention Research, Medical College of Wisconsin, Milwaukee, WI, USA

M. Kay Garcia Department of General Oncology, Division of Cancer Medicine, The University of Texas MD Anderson Cancer Center, Houston, TX, USA

Ryan Garcia University of Texas Southwestern Medical Center at Dallas, Dallas, TX, USA

Luz M. Garcini Ethnic Minority & Multicultural Health SBM SIG Co-Chair, SDSU/UCSD Joint Doctoral Program in Clinical Psychology, San Diego, CA, USA

Stephanie L. Garey Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA

Mariana Garza Department of Psychology, University of North Texas, Denton, TX, USA

Robert J. Gatchel Department of Psychology, College of Science, The University of Texas at Arlington, Arlington, TX, USA

Klaus Gebel School of Education, University of Newcastle, Callaghan, NSW, Australia

City Futures Research Centre, University of New South Wales, Sydney, Australia

Pamela A. Geller Department of Psychology, Drexel University Drexel University College of Medicine, Philadelphia, PA, USA

Marc D. Gellman Behavioral Medicine Research Center, Department of Psychology, University of Miami, Miami, FL, USA

Login S. George Department of Psychology, University of Connecticut, Storrs, CT, USA

William Gerin The College of Health and Human Development, University Park, PA, USA

Denis Gerstorf Institute of Psychology, Humboldt University, Berlin, Germany

Pearl Ghaemmaghami Department of Psychology, University of Zurich, Binzmuehlestrasse, Zurich, Switzerland

Yori Gidron Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Annie T. Ginty School of Sport and Exercise Sciences, The University of Birmingham, Edgbaston, Birmingham, UK

Karen Glanz Schools of Medicine and Nursing, University of Pennsylvania, Philadelphia, PA, USA

Ronald Goldberg Diabetes Research Institute, University of Miami Miller School of Medicine, Miami, FL, USA

Peter M. Gollwitzer Department of Psychology, New York University, New York, NY, USA

Heather Honoré Goltz HSR&D Center of Excellence, Michael E. DeBakey VA Medical Center (MEDVAMC 152), Houston, TX, USA

Department of Social Sciences, University of Houston-Downtown, Houston, TX, USA

Carley Gomez-Meade Department of Pediatrics, Miller School of Medicine, University of Miami, Miami, FL, USA

Jeffrey S. Gonzalez Clinical Psychology, Health Emphasis, Ferkauf Graduate School of Psychology, Yeshiva University, Bronx, NY, USA

Diabetes Research Center, Albert Einstein College of Medicine, Yeshiva University, Bronx, NY, USA

Patricia Gonzalez Institute for Behavioral and Community Health (IBACH), Graduate School of Public Health, San Diego State University, San Diego, CA, USA

Jeffrey Goodie Department of Family Medicine, Uniformed Services University of the Health Sciences, Bethesda, MD, USA

Daniel Gorrin Department of Physical Therapy, University of Delaware, Newark, DE, USA

Elana Graber Department of Psychology, University of Delaware, Newark, DE, USA

John Grabowski Department of Psychiatry, Medical School, University of Minnesota, Minneapolis, MN, USA

Douglas A. Granger Center for Interdisciplinary Salivary Bioscience Research, School of Nursing, Bloomberg School of Public Health, and School of Medicine The Johns Hopkins University, Baltimore, MD, USA

Jessica Haberer Medicine and Center for Global Health, Massachusetts General Hospital, Harvard University, Boston, MA, USA

Tibor Hajos Medical Psychology, VU University Medical Center, Amsterdam, North Holland, The Netherlands

Chanita H. Halbert School of Medicine, University of Pennsylvania, Philadelphia, PA, USA

Martica H. Hall Department of Psychiatry, University of Pittsburgh, Pittsburgh, PA, USA

Judith A. Hall Department of Psychology, Northeastern University, Boston, MA, USA

Katherine S. Hall Durham VA Medical Center Geriatric Research, Education, and Clinical Center, Durham, NC, USA

Peter A. Hall Faculty of Applied Health Sciences, University of Waterloo, Waterloo, ON, Canada

Heidi Hamann Department of Psychiatry, UT Southwestern Medical Center, Dallas, TX, USA

Mark Hamer Epidemiology and Public Health, Division of Population Health, University College London, London, UK

Margaret Hammersla University of Maryland School of Nursing, Baltimore, MD, USA

Reiner Hanewinkel Institute for Therapy and Health Research, Kiel, Germany

Alyssa Haney Department of Psychiatry, School of Medicine, University of Pittsburgh, Pittsburgh, PA, USA

Nelli Hankonen Department of Lifestyle and Participation, National Institute for Health and Welfare University of Helsinki, Helsinki, Finland

Kazuo Hara Department of Metabolic Diseases, Graduate School of Medicine, The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Samantha M. Harden Human Nutrition, Foods, and Exercise, Virginia Tech, Roanoke, VA, USA

Manjunath Harlapur Center of Behavioral Cardiovascular Health, Division of General Medicine, Columbia University, New York, NY, USA

Victoria Harms Department of Psychology, University of Saskatchewan, Saskatoon, SK, Canada

Lisa Harnack Division of Epidemiology and Community Health, School of Public Health, University of Minnesota, Minneapolis, MN, USA

Briain O. Hartaigh School of Sport and Exercise Sciences, The University of Birmingham, Edgbaston, Birmingham, UK

Stacey L. Hart Department of Psychology, Ryerson University, Toronto, ON, Canada

Steven Harulow Royal College of Speech & Language Therapists, London, UK

Toshihide Hashimoto Department of Rehabilitation, Graduate School of Medicine, Gunma University, Maebashi, Gunma, Japan

Masahiro Hashizume Department of Psychosomatic Medicine, Toho University, Ota-ku, Tokyo, Japan

Brant P. Hasler Psychiatry, Western Psychiatric Institute and Clinic University of Pittsburgh School of Medicine, Pittsburgh, PA, USA

Louise C. Hawkley Department of Psychology, The University of Chicago, Chicago, IL, USA

Laura L. Hayman College of Nursing & Health Sciences, University of Massachusetts Boston, Boston, MA, USA

Jennifer Heaney School of Sport and Exercise Sciences, The University of Birmingham, Edgbaston, Birmingham, UK

Christine Heim Institute of Medical Psychology, Charité University Medicine Berlin, Berlin, Germany

Lois Jane Heller Department of Biomedical Sciences, University of Minnesota Medical School – Duluth, Duluth, MN, USA

Miranda Hellman Boston University, Boston, MA, USA

Kimberly M. Henderson Department of Medicine, University of Minnesota, Minneapolis, MN, USA

Whitney M. Herge Department of Psychology, University of Miami, Coral Gables, FL, USA

Patricia Cristine Heyn Department of Physical Medicine and Rehabilitation, University of Colorado Denver Anschutz Medical Campus School of Medicine, Aurora, CO, USA

Emma Hiatt Rehabilitaion Psychology and Special Education, University of Wisconsin-Madison, Madison, WI, USA

Angela M. Hicks Department of Psychology, Westminster College, Salt Lake City, UT, USA

Benjamin Hidalgo Department of Psychiatry, Medical College of Wisconsin, Milwaukee, WI, USA

Catharina Hjortsberg The Swedish Institute for Health Economics, Lund, Sweden

Clare Hocking Faculty of Health and Environmental Sciences, Auckland University of Technology, Auckland, New Zealand

Richard Hoffman Academic Health Center, School of Medicine-Duluth Campus University of Minnesota, Duluth, MN, USA

Maxine Holmqvist Clinical Health Psychology, University of Manitoba, Winnipeg, MB, Canada

Julianne Holt-Lunstad Department of Psychology, Brigham Young University, Provo, UT, USA

Emily D. Hooker Department of Psychology and Social Behavior, University of California, Irvine, Irvine, CA, USA

Stephanie Ann Hooker Department of Psychology, University of Colorado, Denver, CO, USA

Monica Webb Hooper Department of Psychology, University of Miami, Coral Gables, FL, USA

Christiane A. Hoppmann Department of Psychology, University of British Columbia, Vancouver, BC, Canada

M. Bryant Howren Department of Psychology, The University of Iowa & VA Iowa City Healthcare System, Iowa City, IA, USA

Brian M. Hughes School of Psychology, National University of Ireland, Galway, Galway, Ireland

Mann Hyung Hur Public Administration, Chung-Ang University, Seoul, Korea

Seth Hurley Department of Psychology, University of Iowa, Iowa City, IA, USA

Mustafa M. Husain Department of Psychiatry, The University of Texas Southwestern Medical Center at Dallas Columbia University/New York State Psychiaric Institute, Dallas, TX, USA

John Hustad Department of Medicine and Public Health Sciences, Penn State College of Medicine, Hershey, PA, USA

Shannon Idzik University of Maryland School of Nursing and the University of Maryland Medical Center Emergency Department, Baltimore, MD, USA

Shuji Inada Department of Stress Science and Psychosomatic Medicine, Graduate School of Medicine, The University of Tokyo, Tokyo, Japan

Salvatore Insana Western Psychiatric Institute and Clinic, Pittsburgh, PA, USA

Leah Irish Department of Psychiatry, School of Medicine, University of Pittsburgh, Pittsburgh, PA, USA

Makiko Ito Department of Stress Science and Psychosomatic Medicine, Graduate School of Medicine, The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Daisuke Ito Health Service Center, Kanazawa University, Kanazawa, Ishikawa, Japan

Satoru Iwase Department Of Palliative Medicine, The University of Tokyo Hospital, Bunkyo-ku, Tokyo, Japan

Karen Jacobs Occupational Therapy, College of Health and Rehabilitation Science, Sargent College, Boston University, Boston, MA, USA

Farrah Jacquez Department of Psychology, University of Cincinnati, Cincinnati, OH, USA

Lana Jago Department of Psychology, The University of Auckland, Auckland, New Zealand

Denise Janicki-Deverts Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA

Kate L. Jansen Department of Family Medicine, University of Mississippi Medical Center, Jackson, MS, USA

Imke Janssen Department of Preventive Medicine, Rush University Medical Center, Chicago, IL, USA

Elissa Jelalian Department of Psychiatry, Rhode Island Hospital, Brown Medical School, Providence, RI, USA

Chad D. Jensen Department of Psychology, Brigham Young University, Provo, UT, USA

Jason Jent Department of Pediatrics, Mailman Center for Child Development, University of Miami, Miami, FL, USA

Stefanie De Jesus Exercise and Health Psychology Laboratory, The University of Western Ontario, London, ON, Canada

Rong Jiang Department of Psychiatry and Behavioral Sciences, Duke University, Durham, NC, USA

Alvin Jin Department of Psychology, University of South Florida College of Arts & Sciences, Tampa, FL, USA

Jillian A. Johnson Department of Psychology, University of Calgary, Calgary, AB, Canada

Debra Johnson Department of Psychology, University of Iowa, Iowa City, IA, USA

Sara B. Johnson School of Medicine and Bloomberg School of Public Health, Johns Hopkins School of Medicine, Baltimore, MD, USA

Marie Johnston School of Medicine and Dentistry, University of Aberdeen, Aberdeen, Scotland, UK

Derek Johnston School of Psychology, University of Aberdeen, Aberdeen, Scotland, UK

Phil Jones School of Geography, Earth & Environmental Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Randall Steven Jorgensen Department of Psychology, Syracuse University, Syracuse, NY, USA

Vanessa Juth Department of Psychology and Social Behavior, University of California, Irvine, Irvine, CA, USA

Yoshinobu Kanda Division of Hematology, Saitama Medical Center, Jichi Medical University, Omiya-ku, Saitama, Japan

Afton N. Kapuscinski Psychology Department, Syracuse University, Syracuse, NY, USA

Alyssa Karel School of Nursing, University of Wisconsin-Madison, Madison, WI, USA

Mardís Karlsdóttir Department of Psychology, The University of Iceland School of Health Sciences, Reykjavík, Iceland

Yoko Katayori Department of Behavioral Medicine, Graduate School of Medicine, Tohoku University, Aoba-ku, Sendai, Japan

Erin E. Kauffman Department of Psychology, University of North Texas, Denton, TX, USA

Francine Kaufman Medtronic, Northridge, CA, USA

Peter Kaufmann Division of Prevention & Population Sciences, National Heart, Lung, and Blood Institute Clinical Applications and Prevention Branch, Bethesda, MD, USA

Jussi Kauhanen Institute of Public Health and Clinical Nutrition, University of Eastern Finland, Kuopio, Finland

Ulrike Kübler Department of Psychology, University of Zurich, Binzmuehlestrasse, Zurich, Switzerland

Quinn D. Kellerman Department of Psychology, University of Iowa, Iowa City, IA, USA

Riyad Khanfer School of Sport and Exercise Sciences, The University of Birmingham, Edgbaston, Birmingham, UK

Falk Kiefer Department of Addictive Behavior and Addiction Medicine, Central Institute of Mental Health, Mannheim, Baden-Württemberg, Germany

Hiroe Kikuchi Department of Psychosomatic Research, National Institute of Mental Health, National Center of Neurology and Psychiatry, Tokyo, Japan

Kristin Kilbourn Department of Psychology, University of Colorado Denver, Denver, CO, USA

Tereza Killianova Free University of Brussels (VUB), Jette, Belgium

Youngmee Kim Department of Psychology, University of Miami, Coral Gables, Miami, FL, USA

Pamela S. King Pediatric Prevention Research Center, Department of Pediatrics, Wayne State University School of Medicine, Detroit, MI, USA

Megan Kirouac Department of Psychiatry and Behavioral Sciences, University of Washington, Harborview Medical Center, Seattle, WA, USA

Clemens Kirschbaum Chair of Biopsychology, Technische Universität Dresden, Dresden, Saxony, Germany

Mika Kivimaki Epidemiology & Public Health, University College London, London, WC1E 6BT, UK

Maria Kleinstäuber Department of Clinical Psychology and Psychotherapy, Johannes Gutenberg-University of Mainz, Mainz, Germany

Wendy Kliewer Department of Psychology, Virginia Commonwealth University, Richmond, VA, USA

Christopher Kline Department of Psychiatry, School of Medicine, University of Pittsburgh, Pittsburgh, PA, USA

Matthew T. Knauf Department of Psychology, College of Science, The University of Texas at Arlington, Arlington, TX, USA

Carolyn Korbel The Neurobehavioral Clinic and Counseling Center, Lake Forest, CA, USA

Emily Kothe School of Psychology, University of Sydney, Sydney, NSW, Australia

Michael Kotlyar Department of Experimental and Clinical Pharmacology, College of Pharmacy, University of Minnesota, Minneapolis, MN, USA

Marc A. Kowalkouski HSR&D Center of Excellence, Michael E. DeBakey VA Medical Center (MEDVAMC 152), Houston, TX, USA

Tara Kraft Department of Psychology, University of Kansas, Lawrence, KS, USA

Kurt Kroenke Department of Medicine, Indiana University, Regenstrief Institute, VA HSR&D Center for Implementing Evidence-Based Practice, Indianapolis, IN, USA

Stefan Krumm University of Muenster, Muenster, Germany

Laura D. Kubzansky Department of Society, Human Development, and Health, Harvard School of Public Health, Boston, MA, USA

Brigitte M. Kudielka Department of Medical Psychology & Psychological Diagnostics, University of Regensburg, Regensburg, Germany

Masayoshi Kumagai Department of Metabolic Diseases, Graduate School of Medicine The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Keiki Kumano Department of Cell Therapy and Transplantation Medicine, The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Yoshihiko Kunisato Department of Psychiatry and Neurosciences, Hiroshima University, Minami-ku, Hiroshima, Japan

Elyse Kupperman Clinical Psychology, Health Emphasis, Ferkauf Graduate School of Psychology, Yeshiva University, Bronx, NY, USA

Seppo Laaksonen University of Helsinki, Helsinki, Finland

Lara LaCaille Department of Psychology, University of Minnesota Duluth, Duluth, MN, USA

Rick LaCaille Psychology Department, University of Minnesota Duluth, Duluth, MN, USA

Laura H. Lacritz Department of Psychology, The University of Texas Southwestern Medical Center at Dallas, Dallas, TX, USA

Karl-Heinz Ladwig Institut für Epidemiologie, Neuherberg, Germany

Annette M. La Greca Department of Psychology, University of Miami, Coral Gables, FL, USA

Ryan R. Landoll Department of Psychology, University of Miami, Coral Gables, FL, USA

Tanja Lange Department of Neuroendocrinology, University of Luebeck, Lübeck, Germany

Jost Langhorst Kliniken Essen Mitte, Klinik für Naturheilkunde und Integrative Medizin, Universität Duisburg-Essen, Am Deimelsberg 34a, Essen, Germany

David Latini Scott Department of Urology, Baylor College of Medicine, Houston, TX, USA

Kim Lavoie Department of Psychology, University of Québec at Montreal (UQAM); Montreal Behavioural Medicine Centre, Montréal, QC, Canada

Division of Chest Medicine, Hôpital du Sacré-Coeur de Montréal; Research Centre, Montreal Heart Institute, Montréal, QC, Canada

Hannah G. Lawman Department of Psychology, University of South Carolina, Columbia, SC, USA

David J. Lee Department of Epidemiology and Public Health, Miller School of Medicine, University of Miami, Miami, FL, USA

Simon J. Craddock Lee Department of Clinical Sciences, The University of Texas Southwestern Medical Center, Dallas, TX, USA

Carter A. Lennon Department of Psychology, University of Connecticut, Center for Health, Intervention & Prevention, Storrs, CT, USA

Wen B. Leong Diabetes and Endocrinology, University of Birmingham, Heart of England NHS Foundation Trust, Birmingham, West Midlands, UK

Stephen J. Lepore Department of Public Health, Temple University, Philadelphia, PA, USA

Bonnie S. LeRoy Department of Genetics Cell Biology and Development, University of Minnesota, Minneapolis, MN, USA

Yvonne Leung Department of Psychosocial Oncology and Palliative Care, Princess Margaret Hospital, University Health Network/ University of Toronto, Toronto, ON, Canada

Bonnie Levin Department of Neurology, University of Miami Medical Center, Miami, FL, USA

Bingshuo Li University of Minnesota Medical School, University of Minnesota, 235 School of Medicine, Duluth, MN, USA

Roselind Lieb Department of Psychology, Division of Clinical Psychology and Epidemiology, Basel, Switzerland

Julia R. Van Liew Department of Psychology, University of Iowa, Iowa City, IA, USA

Jane Limmer Obstetrics and Gynecology, Duke Hospital, Durham, NC, USA

Bernt Lindahl Occupational and Environmental Medicine, Department of Public Health and Clinical Medicine, Umeå University, Umeå, Sweden

Martin Lindström Department of Clinical Sciences in Malmö, Lund University, Malmö, Sweden

Megan R. Lipe Department of Clinical Health and Psychology, University of Florida, College of Public Health and Health Professions, Gainesville, FL, USA

Steven E. Lipshultz Department of Pediatrics, Epidemiology and Public Health, and Medicine (Oncology), Leonard M. Miller School of Medicine University of Miami Holtz Children's Hospital of the University of Miami-Jackson Memorial Medical Center Batchelor Children's Research Institute Mailman Center for Child Development University of Miami Sylvester Comprehensive Cancer Center, Miami, FL, USA

Cecilia W. P. Li-Tsang Department of Rehabilitation Sciences, The Hong Kong Polytechnic University, Kowloon, Hong Kong, China

Maria Magdalena Llabre Department of Psychology, University of Miami, Coral Gables, FL, USA

Valerie G. Loehr Department of Psychology, Southern Methodist University, Dallas, TX, USA

Joanna Long School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Kristin A. Long Department of Psychology, University of Pittsburgh, Pittsburgh, PA, USA

Sana Loue Department of Epidemiology & Biostatistics, Case Western Reserve University, School of Medicine, Cleveland, OH, USA

William Lovallo Department of Psychiatry and Behavioral Sciences, University of Oklahoma Health Sciences Center Veterans Affairs Medical Center, Oklahoma City, OK, USA

Travis Lovejoy Mental Health & Clinical Neurosciences Division, Portland Veterans Affairs Medical Center, Portland, OR, USA

Tana M. Luger Department of Psychology, University of Iowa, Iowa City, IA, USA

Mark A. Lumley Department of Psychology, Wayne State University, Detroit, MI, USA

M. Kathleen B. Lustyk School of Psychology, Family, and Community, Seattle Pacific University, University of Washington, Seattle, WA, USA

Faith S. Luyster School of Nursing, University of Pittsburgh, Pittsburgh, PA, USA

Kristin L. MacGregor Department of Psychology, Syracuse University, Syracuse, NY, USA

Anna MacKinnon Department of Psychology, McGill University, Montreal, QC, Canada

Shannon Madore Department of Psychology, University of Colorado Denver, Denver, CO, USA

Elizabeth A. Majka Department of Psychology, The University of Chicago, Chicago, IL, USA

Neena Malik Department of Pediatrics, Miller School of Medicine, University of Miami, Miami, FL, USA

Jamil A. Malik National Institute of Psychology, Quaid-i-Azam University/ VU University Amsterdam, Islamabad, Pakistan

Elizabeth M. Maloney Formerly of the Viral and Rickettsial Division, Centers for Disease Control and Prevention, Atlanta, GA, USA

Tsipora Mankovsky Department of Psychology, McGill University, Montreal, QC, Canada

Amy Jo Marcano-Reik Department of Bioethics, Cleveland Clinic, Cleveland, OH, USA

Center for Genetic Research Ethics and Law, Case Western Reserve University, Cleveland, OH, USA

Kristen K. Marciel Department of Psychology, University of Miami, Coral Gables, FL, USA

Judy A. Marciel Perioperative Services, East Tennessee Children's Hospital, Knoxville, TN, USA

Erin N. Marcus Division of General Internal Medicine, University of Miami, Miller School of Medicine, Miami, FL, USA

Seth A. Margolis Clinical Psychology, Health Emphasis, Ferkauf Graduate School of Psychology, Yeshiva University, Bronx, NY, USA

Michela (Micky) Marinelli Department of Cellular and Molecular Pharmacology, Rosalind Franklin University of Medicine and Science, North Chicago, IL, USA

Jacqueline Markowitz Boston University, Boston, MA, USA

G. Alan Marlatt University of Washington, Seattle, Washington, USA

David G. Marrero Diabetes Translational Research Center, Indiana University School of Medicine, Indianapolis, IN, USA

Meghan L. Marsac The Center for Injury Prevention and Research, The Children's Hospital of Philadelphia, Philadelphia, PA, USA

Alexandra Martin Friedrich-Alexander University Erlangen-Nürnberg; University Hospital, Erlangen, Germany

Alexandra Martini de Oliveira Institute of Psychiatry – Hospital das Clínicas University of São Paulo Medical School, São Paulo, SP, Brazil

Kevin S. Masters Department of Psychology, University of Colorado, Denver, CO, USA

Della Matheson Diabetes Research Institute, Miller School of Medicine, University of Miami, Miami, FL, USA

Yoshinobu Matsuda National Hospital Organization, Kinki-Chuo Chest Medical Center, Sakai shi, Osaka, Japan

Hiromichi Matsuoka Department of Psychocomatic Medicine, Kinki University Faculty of Medicine, Osakasayama, Osaka, Japan

Yutaka Matsuyama Department of Biostatistics, School of Public Health, The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Sonia Matwin Department of Psychiatry, Harvard Medical School, Boston, MA, USA

Alfred L. McAlister Behavioral Sciences, University of Texas School of Public Health, Austin, TX, USA

Lisa M. McAndrew Department of Veterans Affairs, NJ Healthcare System, East Orange, NJ, USA

Jeanette McCarthy Community and Family Medicine, Duke University Medical Center, Durham, NC, USA

Shawn McClintock Department of Psychiatry, The University of Texas Southwestern Medical Center at Dallas Columbia University/New York State Psychiaric Institute, Dallas, TX, USA

Lance M. McCracken Psychology Department, Institute of Psychiatry, King's College London, London, UK

James A. McCubbin Department of Psychology, Clemson University, Clemson, SC, USA

Bonnie McGregor Fred Hutchinson Cancer Research Center, Seattle, WA, USA

Brooke McInroy The University of Iowa, Iowa City, IA, USA

David McIntyre School of Sport and Exercise Sciences, The University of Birmingham, Edgbaston, Birmingham, UK

Tara McMullen Doctoral Program in Gerontology, University of Maryland Baltimore and Baltimore County, Baltimore, MD, USA

Marcia D. McNutt Department of Psychology, University of Miami, Coral Gables, FL, USA

Tamar Mendelson Mental Health, Johns Hopkins Bloomberg School of Public Health Johns Hopkins University, Baltimore, MD, USA

Luigi Meneghini Diabetes Research Institute, University of Miami, Miami, FL, USA

Melissa Merrick Division of Violence Prevention, Centers for Disease Control & Prevention, Atlanta, GA, USA

Sarah Messiah Department of Pediatrics, University of Miami, Miami, FL, USA

Miriam A. Mestre Division of Pediatric Clinical Research Department of Pediatrics, Leonard M. Miller School of Medicine University of Miami, Miami, FL, USA

Elizabeth Mezick Department of Psychology, University of Pittsburgh, Pittsburgh, PA, USA

Kathleen Michael School of Nursing, University of Maryland, Baltimore, MD, USA

Susan Michie University College London, London, UK

Eleanor Miles Department of Psychology, The University of Sheffield, Western Bank, Sheffield, UK

Donna Miller Centers for Disease Control and Prevention, National Center for Health Statistics, Hyattsville, MD, USA

Robert Miller Chair of Biopsychology, Technische Universität Dresden, Dresden, Saxony, Germany

Tracie L. Miller Department of Pediatrics and Epidemiology and Public Health Division of Pediatric Clinical Research Department of Pediatrics, Leonard M. Miller School of Medicine University of Miami Holtz Children's Hospital of the University of Miami-Jackson Memorial Medical Center Batchelor Children's Research Institute University of Miami Sylvester Comprehensive Cancer Center, Miami, FL, USA

Rachel Millstein SDSU/UCSD Joint Doctoral Program in Clinical Psychology, University of California, San Diego/San Diego State University, San Diego, CA, USA

Faisal Mir School of Sport & Exercise Sciences, University of Birmingham, Edgbaston, BHAM, UK

Akihisa Mitani Department of Respiratory Medicine, Mitsui Memorial Hospital, Chiyoda-ku, Tokyo, Japan

Laura A. Mitchell Department of Psychology, School of Life Sciences, Glasgow Caledonian University, Glasgow, Scotland, UK

Jason W. Mitchell Center for AIDS Intervention Research, Medical College of Wisconsin, Milwaukee, WI, USA

Koji Miyazaki Department of Hematology, Kitasato University School of Medicine, Sagamihara, Kanagawa, Japan

Marilyn Moffat Department of Physical Therapy, New York University, New York, NY, USA

David C. Mohr Feinberg School of Medicine, Department of Preventive Medicine, Center for Behavioral Intervention Technologies, Northwestern University, Chicago, IL, USA

Kristine M. Molina Department of Psychology, University of Miami, Miami, FL, USA

Arlen C. Moller Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Ivan Molton Department of Rehabilitation Medicine, University of Washington, Seattle, WA, USA

Jane Monaco Department of Biostatistics, The University of North Carolina at Chapel Hill, Chapel Hill, NC, USA

Karla Espinosa de los Monteros Clinical Psychology, SDSU/UCSD Joint Doctoral Program in Clinical Psychology, San Diego, CA, USA

Pablo A. Mora Department of Psychology, The University of Texas at Arlington, Arlington, TX, USA

Theresa A. Morgan Alpert Medical School of Brown University, Department of Psychiatry, Brown University, Providence, RI, USA

Matthis Morgenstern Institute for Therapy and Health Research, Kiel, Germany

Chica Mori Department Of Palliative Medicine, The University of Tokyo Hospital, Bunkyo-ku, Tokyo, Japan

Yoshiya Moriguchi Department of Psychophysiology, National Institute of Mental Health, National Center of Neurology and Psychiatry, Kodaira, Tokyo, Japan

Alexandre Morizio Department of Exercise Science, Concordia University, Montreal Behavioral Medicine Centre, Montreal, QC, Canada

Eleshia J. P. Morrison Department of Psychology, Ethnic Minority & Multicultural Health SBM SIG Chair, The Ohio State University, Columbus, OH, USA

Anett Mueller Department of Psychology, State University of New York at Stony Brook, Stony Brook, NY, USA

Matthew Muldoon Department of Medicine, University of Pittsburgh, Pittsburgh, PA, USA

Barbara Mullan Centre for Medical Psychology & Evidence-based Decision-making, University of Sydney, Sydney, NSW, Australia

Tomohiko Muratsubaki Department of Behavioral Medicine, School of Medicine, Tohoku University Graduate, Aoba-ku, Sendai, Japan

Seema Mutti School of Public Health & Health Systems, University of Waterloo, Waterloo, ON, Canada

Yoko Nagai Brighton and Sussex Medical School, University of Sussex, Brighton, East Sussex, UK

Eun-Shim Nahm School of Nursing, University of Maryland, Baltimore, MD, USA

Motohiro Nakajima University of Minnesota Medical School, University of Minnesota, 235 School of Medicine, Duluth, MN, USA

Misuzu Nakashima Hizen Psychiatric Center, Yoshinogari, Kanzaki, Saga, Japan

Benjamin H. Natelson Department of Pain Medicine & Palliative Care, Beth Israel Medical Center and Albert Einstein College of Medicine, Bronx, NY, USA

Urs M. Nater Department of Psychology, University of Marburg, Marburg, Germany

Astrid Nehlig U666, INSERM, Faculty of Medicine, University of Strasbourg, Strasbourg, France

Alexandra Nelson Drexel University Department of Psychology UNC – Chapel Hill Department of Psychiatry, Chapel Hill, NC, USA

Ashley Nelson Department of Psychiatry, School of Medicine and Public Health, University of Wisconsin-Madison, Madison, WI, USA

Kimberly Nelson Department of Psychology, University of Washington, Seattle, WA, USA

Jonathan Newman Columbia University, New York, NY, USA

Sarah J. Newman Duke University, Durham, NC, USA

Darren Nickel Physical Medicine & Rehabilitation, University of Saskatchewan, Saskatoon, SK, Canada

Nicole Nisly Department of Internal Medicine, University of Iowa, Iowa City, IA, USA

Karen Niven Manchester Business School, The University of Manchester, Manchester, UK

Kyle R. Noll Department of Physical Medicine & Rehabilitation, Baylor College of Medicine, Houston, TX, USA

Wynne E. Norton Department of Health Behavior, School of Public Health, University of Alabama at Birmingham, Birmingham, AL, USA

Kathryn Noth Illinois Institute of Technology, College of Psychology, Chicago, IL, USA

Lindsay Oberleitner Department of Psychology, Wayne State University, Detroit, MI, USA

Eoin O'Brien The Conway Institute, University College Dublin, Belfield, Dublin, Ireland

Julianne O'Daniel Illumina, Inc, San Diego, CA, USA

Gabriele Oettingen Department of Psychology, New York University, New York, NY, USA

Michael O'Hara Department of Psychology, University of Iowa, Iowa City, IA, USA

Ken Ohashi Department of General Internal Medicine, National Cancer Center Hospital, Chuo-ku, Tokyo, Japan

Keisuke Ohta Department of Metabolic Diseases, Graduate School of Medicine The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Michele L. Okun Sleep Medicine Institute and Department of Psychiatry, School of Medicine, University of Pittsburgh, Pittsburgh, PA, USA

Toru Okuyama Division of Psycho-oncology and Palliative Care, Nagoya City University Hospital, Nagoya, Aichi, Japan

Ellinor K. Olander Applied Research Centre in Health and Lifestyle Interventions, Coventry University, Coventry, West Midlands, UK

Brian Oldenburg Department of Epidemiology and Preventive Medicine, Monash University, Melbourne, VIC, Australia

Sheina Orbell Department of Psychology, University of Essex, Colchester, Essex, UK

C. Tracy Orleans Robert Wood Johnson Foundation, Princeton, NJ, USA

Kristina Orth-Gomér Department of Clinical Neuroscience, Karolinska Institute, Stockholm, Sweden

Patricia Osborne Clinical Psychology, Health Emphasis, Ferkauf Graduate School of Psychology, Yeshiva University, Bronx, NY, USA

Kenneth J. Ottenbacher Division of Rehabilitation Sciences, University of Texas Medical Branch, Galveston, TX, USA

Margaret E. Ottenbacher Institute for Translational Sciences, University of Texas Medical Branch, Galveston, TX, USA

Nicole Overstreet Social Psychology, University of Connecticut, Storrs, CT, USA

Jan R. Oyebode School of Psychology, The University of Birmingham, Edgbaston, Birmingham, UK

Gozde Ozakinci Lecturer in Health Psychology, School of Medicine, University of St Andrews, St Andrews, Scotland, UK

Debbie Palmer Department of Psychology, University of Wisconsin-Stevens Point, Stevens Point, WI, USA

Steven C. Palmer Abramson Cancer Center, University of Pennsylvania, Philadelphia, PA, USA

Kenneth Pargament Department of Psychology, Bowling Green State University, Bowling Green, OH, USA

Crystal L. Park Department of Psychology, University of Connecticut, Storrs, CT, USA

Alyssa Parker UTSW Health Systems, South western Medical Center, Dallas, TX, USA

Seema M. Patidar Department of Clinical and Health Psychology, University of Florida, Gainesville, FL, USA

Anna Maria Patino-Fernandez Department of Pediatrics, University of Miami, Miami, FL, USA

David Pearson School of Psychology, University of Aberdeen, Aberdeen, UK

Hollie B. Pellosmaa Department of Psychology , The University of Texas at Arlington, Arlington, TX, USA

Jennifer Pellowski Department of Psychology, University of Connecticut, Storrs, CT, USA

Frank J. Penedo Department of Medical Social Sciences & Lurie Comprehensive Cancer Center, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Watcharaporn Pengchit Faculty of Psychology, Chulalongkorn University, Bangkok, Thailand

Donald Penzien Head Pain Center, University of Mississippi Medical Center, Jackson, MS, USA

Deidre Pereira Department of Clinical and Health Psychology, University of Florida, College of Public Health and Health Professions, Gainesville, FL, USA

Edward L. Perkins Biomedical Sciences, Mercer University School of Medicine, Savannah, GA, USA

Richard Peter Institute of Epidemiology and Medical Biometry, University of Ulm, Ulm, Germany

Anna C. Phillips Sport & Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Alex Pictor Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Bernardine M. Pinto Centers for Behavioral and Preventive Medicine, Brown University, Providence, RI, USA

Sarah Piper Institute of Metabolic Science, Addenbrookes Hospital, Metabolic Research Laboratories, University of Cambridge, Cambridge, UK

Alefiyah Z. Pishori Department of Psychology, University of Connecticut, Storrs, CT, USA

Helene J. Polatajko Department of Occupational Science and Occupational Therapy, Graduate Department of Rehabilitation Science Dalla Lana School of Public Health, University of Toronto, Toronto, ON, Canada

Lynda H. Powell Department of Preventive Medicine, Rush University Medical Center, Chicago, IL, USA

Harry Prapavessis University of Western Ontario, London, ON, Canada

Aric A. Prather Center for Health and Community, University of California, San Francisco, CA, USA

Courtney C. Prather Department of Psychology, University of North Texas, Denton, TX, USA

Sarah D. Pressman Department of Psychology, University of Kansas, Lawrence, KS, USA

James O. Prochaska Clinical and Health Psychology, University of Rhode Island, Kingston, RI, USA

Elizabeth R. Pulgaron Department of Pediatrics, University of Miami, Miami, FL, USA

Naum Purits Stockholm, Sweden

Pekka Puska National Institute for Health and Welfare (THL), Helsinki, Finland

Conny W. E. M. Quaedflieg Faculty of Psychology and Neuroscience, Maastricht University, Maastricht, MD, The Netherlands

Whitney Raglin Department of Psychology, University of Cincinnati, Cincinnati, OH, USA

Jeanetta Rains Center for Sleep Evaluation, Elliot Hospital, Manchester, NH, USA

Amelie Ramirez Department of Epidemiology & Biostatistics, The University of Texas Health Science Center at San Antonio, San Antonio, TX, USA

Ashley K. Randall Family Studies & Human Development, University of Arizona, Tucson, AZ, USA

Sheah Rarback Department of Pediatrics, University of Miami, Miami, FL, USA

Holly Rau Department of Psychology, University of Utah, Salt Lake City, UT, USA

Maija Reblin College of Nursing, University of Utah, Salt Lake City, UT, USA

Gabriela Reed Psychiatry, Children's Medical Center, UT Southwestern Medical Center, Dallas, TX, USA

William Reeves Office of Surveillance, Epidemiology and Laboratory Services Centers for Disease Control and Prevention, Atlanta, GA, USA

Emily W. Reid (Deceased) Department of Psychology, Drexel University, Philadelphia, PA, USA

Ulf-Dietrich Reips Faculty of Engineering; Faculty of Education and Psychology, Universidad de Deusto, Bilbao, Spain

IKERBASQUE, Basque Foundation for Science, Bilbao, Spain

Anthony Remaud Elisabeth Bruyere Research Institute, University of Ottawa, Ottawa, ON, Canada

Laboratory "Motricité, Interactions, Performance", University of Nantes, Nantes, France

Kirsten Rene Department of Psychology, Brandeis University, Waltham, MA, USA

Barbara Resnick School of Nursing, University of Maryland, Baltimore, MD, USA

Spencer M. Richard Department of Psychology, Utah State University, Logan, UT, USA

Michael Richter Department of Psychology, University of Geneva, Geneva, Switzerland

Nina Rieckmann Berlin School of Public Health, Charité Universitätsmedizin, Berlin, Germany

Winfried Rief Department of Clinical Psychology and Psychotherapy, Philipps University of Marburg, Gutenbergstr, Marburg, Germany

Kristen Riley Department of Psychology, University of Connecticut, Storrs, CT, USA

Deborah Rinehart Denver Health and Hospital Authority, Denver, CO, USA

Lynnee Roane School of Nursing, University of Maryland, Baltimore, MD, USA

Denise de Ybarra Rodríguez Department of Psychology, University of Miami, Coral Gables, FL, USA

Laura Rodriguez-Murillo Department of Psychiatry, Columbia University Medical Center, New York, NY, USA

Osvaldo Rodriguez Miami VA Healthcare System, Miami, FL, USA

Kathryn A. Roecklein Department of Psychology, University of Pittsburgh, Pittsburgh, PA, USA

Megan Roehrig Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Nicolas Rohleder Department of Psychology, Brandeis University, Waltham, MA, USA

Karen S. Rook Department of Psychology & Social Behavior, University of California Irvine, Irvine, CA, USA

Jed E. Rose Department of Psychiatry, Duke Center for Nicotine & Smoking Cessation Research, Durham, NC, USA

Leah Rosenberg Department of Medicine, School of Medicine, Duke University, Durham, NC, USA

Debra Roter Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, USA

Alexander J. Rothman Department of Psychology, University of Minnesota, Minneapolis, MN, USA

Eric Roy Department of Kinesiology, University of Waterloo, Waterloo, ON, Canada

Rachel S. Rubinstein Department of Psychology, Rutgers, The State University of New Jersey, Piscataway, NJ, USA

John Ruiz Department of Psychology, University of North Texas, Denton, TX, USA

Stephanie Russell Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

John Ryan Department of Psychiatry, Western Psychiatric Institute and Clinic, University of Pittsburgh, Pittsburgh, PA, USA

Valerie Sabol School of Nursing, Duke University, Durham, NC, USA

Rany M. Salem Broad Institute, Cambridge, MA, USA

Kristen Salomon Department of Psychology, University of South Florida College of Arts & Sciences, Tampa, FL, USA

Janine Sanchez Department of Pediatrics, University of Miami, Miami, FL, USA

Lee Sanders Center for Health Policy and Primary Care Outcomes Research, Stanford University, Stanford, CA, USA

Timothy S. Sannes Department of Clinical and Health Psychology, College of Clinical Health and Health Professions, University of Florida, Gainesville, FL, USA

Amy F. Sato Department of Psychology, Kent State University, Kent, OH, USA

Eve Saucier Department of Psychology, Brandeis University, Waltham, MA, USA

Shekhar Saxena Department of Mental Health and Substance Abuse, World Health Organization, Geneva 27, Switzerland

Chris Dunkel Schetter Department of Psychology, UCLA, Los Angeles, CA, USA

Wolff Schlotz Institute of Experimental Psychology, University of Regensburg, Regensburg, Germany

Havah Schneider Ferkauf Graduate School of Psychology, Yeshiva University, Bronx, NY, USA

Neil Schneiderman Department of Psychology, Behavioral Medicine Research Center, University of Miami, Coral Gables, FL, USA

Beth Schroeder University of Delaware, Newark, DE, USA

James W. Schroeder Genetics and Molecular Biology Program, Emory University, Atlanta, GA, USA

Marie-Louise Schult Karolinska Institute, Department of Clinical Sciences, Department of Neurobiology, Care Sciences and Society, The Rehabilitation Medicine, University Clinic Danderyd Hospital, Stockholm, Sweden

Ingrid Söderback Department of Public Health and Caring Science, Uppsala University, Uppsala, Sweden

M. Di Katie Sebastiano Kinesiology, University of Waterloo, Waterloo, ON, Canada

Sabrina Segal Department of Neurobiology and Behavior, University of California, Irvine, CA, USA

Theresa Senn Center for Health and Behavior, Syracuse University, Syracuse, NY, USA

Jonathan A. Shaffer Department of Medicine/Division of General Medicine, Columbia University Medical Center, New York, NY, USA

Peter A. Shapiro Department of Psychiatry, Columbia University, New York, NY, USA

Leigh A. Sharma Department of Psychology, University of Iowa, Kenosha, WI, USA

Marianne Shaughnessy School of Nursing, University of Maryland, Baltimore, MD, USA

Christopher Shaw Institute of Sport, Exercise and Active Living, Victoria University, Melbourne, Australia

Tamara Goldman Sher The Family Institute at Northwestern University, Evanston, IL, USA

Simon Sherry Department of Psychology, Dalhousie University, Halifax, NS, Canada

Vivek Shetty Oral & Maxillofacial Surgery, University of California, Los Angeles, CA, USA

Akihito Shimazu Department of Mental Health, Graduate School of Medicine, The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Daichi Shimbo Center for Behavioral Cardiovascular Health, Columbia University, New York, NY, USA

Erica Shreck Yeshiva University, Bronx, NY, USA

Koseki Shunsuke Department of School Education, Aichi University of Education, Kariya-shi, Aichi, Japan

Johannes Siegrist Department of Medical Sociology, University of Duesseldorf, Düsseldorf, Germany

Matthew A. Simonson Institute for Behavioural Genetics, Boulder, CO, USA

Kit Sinclair Department of Rehabilitation Sciences, The Hong Kong Polytechnic University, Kowloon, Hong Kong, China

Abanish Singh Duke University Medical Center, Durham, NC, USA

Bengt H. Sjölund University of Southern Denmark, Odense, DK, Denmark

Michelle Skinner Department of Psychology, University of Utah, Salt Lake City, UT, USA

Celette Sugg Skinner Clinical Sciences, The University of Texas Southwestern Medical Center at Dallas Harold C. Simmons Cancer Center, Dallas, TX, USA

Tom Smeets Faculty of Psychology and Neuroscience, Maastricht University, Maastricht, MD, The Netherlands

Alicia K. Smith Psychiatry & Behavioral Sciences, Emory University SOM, Atlanta, GA, USA

Barbara Smith School of Nursing, University of Maryland, Baltimore, MD, USA

Lauren Smith Department of Psychology, University of North Texas, Denton, TX, USA

Timothy W. Smith Department of Psychology, University of Utah, Salt Lake City, UT, USA

Howard Sollins Attorneys at Law, Ober, Kaler, Grimes & Shriver, Baltimore, MD, USA

Colin L. Soskolne Department of Public Health Services, School of Public Health, University of Alberta, Edmonton, AB, Canada

Ana Victoria Soto Medicine – Residency Program, Columbia University Medical Center, New York, NY, USA

Mary Spiers Department of Psychology, Drexel University, Philadelphia, PA, USA

Kevin S. Spink College of Kinesiology, University of Saskatchewan, Saskatoon, SK, Canada

Bonnie Spring Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Sara Mijares St. George Department of Psychology, University of South Carolina, Columbia, SC, USA

Tobias Stalder Chair of Biopsychology, Technische Universität Dresden, Dresden, Saxony, Germany

Annette L. Stanton Department of Psychology, University of California, Los Angeles, CA, USA

Shannon L. Stark Department of Psychology, Arizona State University, Tempe, AZ, USA

Adrienne Stauder Institute of Behavioural Sciences, Semmelweis University Budapest, Budapest, Hungary

Michael E. Stefanek Research and Collaborative Research, Indiana University, Bloomington, IN, USA

Jeremy Steglitz Department of Psychiatry and Behavioral Sciences, Clinical Psychology Division, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Nikola Stenzel Department of Clinical Psychology and Psychotherapy, Philipps University of Marburg, Marburg, Germany

Jana Strahler Clinical Biopsychology, Department of Psychology, University of Marburg, Marburg, Germany

Deborah M. Stringer Department of Psychology, University of Iowa, Iowa City, IA, USA

Victoria Anne Sublette School of Public Health, University of Sydney, Sydney, NSW, Australia

Alyson Sularz Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Michael J. L. Sullivan Department of Psychology, McGill University, Montreal, QC, Canada

Shin-ichi Suzuki Faculty of Human Sciences, Graduate School of Human Sciences, Waseda University, Tokorozawa-shi, Saitama, Japan

Catherine Sykes World Confederation for Physical Therapy, Victoria Charity Centre, London, UK

Sefik Tagay Department of Psychosomatic Medicine and Psychotherapy, University of Duisburg-Essen, Essen, North Rhine-Westphalia, Germany

Shahrad Taheri University of Birmingham, Heart of England NHS Foundation Trust, Birmingham, UK

Misato Takada Department of Health Economics and Epidemiology Research, School of Public Health, The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Yuko Takei Faculty of Medicine, University of Miyazaki Hospital, Miyazaki-shi, Japan

Yoshiyuki Takimoto Department of Stress Science and Psychosomatic Medicine, Graduate School of Medicine, The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Yukari Tanaka Department of Behavioral Medicine, Graduate School of Medicine, Tohoku University, Aoba-ku, Sendai, Japan

Molly L. Tanenbaum Clinical Psychology, Health Emphasis, Ferkauf Graduate School of Psychology, Yeshiva University, Bronx, NY, USA

Asuka Tanoue Advanced Research Center for Human Science, Waseda University, Tokorozawa, Saitama, Japan

Robert N. Taylor Department of Obstetrics and Gynecology, Wake Forest School of Medicine, Winston-Salem, NC, USA

Cortney J. Taylor Department of Psychology, University of Miami, Coral Gables, FL, USA

Marc Taylor Department 163, Behavioral Sciences & Epidemiology, Naval Health Research Center, San Diego, CA, USA

Julian F. Thayer Department of Psychology, The Ohio State University, Columbus, OH, USA

Töres Theorell Stress Research Institute, Stockholm University, Stockholm, Sweden

G. Neil Thomas Department of Public Health, University of Birmingham, Edgbaston, Birmingham, UK

Roland Thomeé Department of Rehabilitation Medicine, Sahlgrenska University Hospital, Öjersjö, Göteborg, Sweden

Rebecca C. Thurston Department of Psychiatry, School of Medicine, University of Pittsburgh, Pittsburgh, PA, USA

Warren Tierney Department of Psychology, Faculty of Education & Health Sciences, University of Limerick, Castletroy, Limerick, Ireland

Jasmin Tiro Department of Clinical Sciences, The University of Texas Southwestern Medical Center, Dallas, TX, USA

Emil C. Toescu Division of Medical Sciences, The University of Birmingham, Edgbaston, Birmingham, UK

Fumiharu Togo Graduate School of Education, The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Akihiro Tokoro Department of Psychosomatic Medicine, National Hospital Organization, Kinki-Chuo Chest Medical Center, Sakai Osaka, Japan

A. Janet Tomiyama Rutgers University, NJ, USA

Hansel Tookes Epidemiology and Public Health, Miller School of Medicine, University of Miami, Miami, FL, USA

George J. Trachte Academic Health Center, School of Medicine-Duluth Campus, University of Minnesota, Duluth, MN, USA

Lara Traeger Behavioral Medicine Service, Massachusetts General Hospital/Harvard Medical School, Boston, MA, USA

Vincent Tran University of Texas, Southwestern Medical Center, Dallas, TX, USA

Wendy Troxel Psychiatry and Psychology, University of Pittsburgh, Pittsburgh, PA, USA

Emiko Tsuchiya Department of Behavioral Medicine, Graduate School of Medicine, Tohoku University, Aoba-ku, Sendai, Japan

Viana Turcios-Cotto Department of Psychology, University of Connecticut, Storrs, CT, USA

Barbara Turner The University of Texas Health Science Center at San Antonio, San Antonio, TX, USA

J. Rick Turner Cardiovascular Safety, Quintiles, Durham, NC, USA

James Turner School of Cancer Sciences, The University of Birmingham, Edgbaston, Birmingham, UK

Bert N. Uchino Department of Psychology and Health Psychology Program, University of Utah, Salt Lake City, UT, USA

C. Renn Upchurch Sweeney VA Salt Lake City Healthcare System, Salt Lake City, UT, USA

Jane Upton School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Antti Uutela Department for Lifestyle and Health, National Institute for Health and Welfare (THL), Helsinki, Finland

Mark Vander Weg Department of Internal Medicine, The University of Iowa and Iowa City VA Health Care System, Iowa City, IA, USA

Kavita Vedhara Institute of Work, Health and Organisations, University of Nottingham, Nottingham, UK

Bart Verkuil Clinical, Health and Neuro Psychology, Leiden University, Leiden, Netherlands

Andrea C. Villanti Johns Hopkins Bloomberg School of Public Health, The Schroeder Institute for Tobacco Research and Policy Studies at Legacy, Washington, DC, USA

Ana Vitlic School of Sport & Exercise Sciences, The University of Birmingham, Edgbaston, Birmingham, UK

Adriana Dias Barbosa Vizzotto Occupational Therapist of the Occupational Therapy Service, Institute of Psychiatry, Hospital das Clínicas University of São Paulo Medical School, São Paulo, SP, Brazil

John P. Vuchetich Department of Psychiatry, University of Minnesota School of Medicine, Minneapolis, MN, USA

Amy Wachholtz Department of Psychiatry, University of Massachusetts Medical School, Worcester, MA, USA

Anton J. M. Wagenmakers School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Andrea Wallace College of Nursing, University of Iowa, Iowa City, IA, USA

Margaret Wallhagen Department of Physiological Nursing, University of California San Francisco School of Nursing, San Francisco, CA, USA

Melissa Walls Biobehavioral Health & Population Sciences, University of Minnesota Medical School – Duluth, Duluth, MN, USA

Kenneth Wallston School of Nursing, Vanderbilt University, Nashville, TN, USA

Jenny T. Wang Department of Medical Psychology, Duke University, Durham, NC, USA

Andrew J. Wawrzyniak School of Nursing & Health Studies, University of Miami, Coral Gables, FL, USA

Thomas Webb Department of Psychology, The University of Sheffield, Sheffield, UK

Stephen M. Weiss Department of Psychiatry and Behavioral Sciences, Miller School of Medicine, University of Miami, Miami, FL, USA

Jennifer Wessel Public Health, School of Medicine, Indiana University, Indianapolis, IN, USA

William Whang Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Anthony J. Wheeler Department of Psychology, Utah State University, Logan, UT, USA

Angela White Department of Psychology, University of Connecticut, Storrs, CT, USA

Timothy Whittaker The International Register of Herbalists & Homeopaths, Cinderford, Glos., UK

Timothy H. Wideman Department of Psychology, McGill University, Montreal, QC, Canada

Deborah J. Wiebe Division of Psychology, Department of Psychiatry, Southwestern Medical Center, University of Texas, Dallas, TX, USA

Friedrich Wieser Department of Gynecology and Obstetrics, Emory University School of Medicine, Atlanta, GA, USA

Diana Wile Department of Pediatrics, University of Miami, Miami, FL, USA

James D. Wilkinson Department of Pediatrics and Epidemiology and Public Health Division of Pediatric Clinical Research Department of Pediatrics, Leonard M. Miller School of Medicine University of Miami Holtz Children's Hospital of the University of Miami-Jackson Memorial Medical Center Batchelor Children's Research Institute University of Miami Sylvester Comprehensive Cancer Center, Miami, FL, USA

Redford B. Williams Department of Psychiatry and Behavioral Sciences, Division of Behavioral Medicine, Duke University, Durham, NC, USA

Virginia P. Williams Williams LifeSkills, Inc., Durham, NC, USA

Paula Williams Department of Psychology, University of Utah, Salt Lake City, UT, USA

Dawn Wilson Department of Psychology, University of South Carolina, Columbia, SC, USA

Oliver J. Wilson School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Kelly Winter Epidemiology, Florida International University, Miami, FL, USA

Katie Witkiewitz University of New Mexico, Albuquerque, New Mexico, USA

Michael Witthöft Psychologisches Institut Abteilung Klinische Psychologie und Psychotherapie, Johannes Gutenberg Universität Mainz, Mainz, Germany

Jutta M. Wolf Department of Psychology, Brandeis University, Waltham, MA, USA

Oliver T. Wolf Department of Cognitive Psychology, Ruhr-Universität Bochum, Bochum, Germany

Timothy Wolf Department of Occupational Therapy and Neurology, Program in Occupational Therapy, St. Louis, MO, USA

Patricia Woltz School of Nursing, University of Maryland, Baltimore, MD, USA

Patricia M. Wong Department of Psychology, University of Pittsburgh, Pittsburgh, PA, USA

Cara Wong School of Psychology, University of Sydney, Sydney, NSW, Australia

Jennifer Wortmann Department of Psychology, University of Connecticut, Storrs, CT, USA

Rex A. Wright College of Arts and Sciences, Department of Psychology, University of North Texas, Denton, TX, USA

Ellen Wuest Boston University, Boston, MA, USA

Naoya Yahagi Department of Metabolic Diseases, Graduate School of Medicine The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Yu Yamada Department of Psychosomatic Medicine, Kyushu University, Fukuoka, Japan

Yoshiharu Yamamoto Educational Physiology Laboratory, Graduate School of Education The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Betina R. Yanez Department of Psychology, University of California, Los Angeles, CA, USA

Samantha Yard Department of Psychology, University of Washington, Seattle, WA, USA

M. Taghi Yasamy Department of Mental Health and Substance Abuse, World Health Organization, Geneva 27, Switzerland

Siqin Ye Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Jason S. Yeh Obstetrics and Gynecology, Division of Reproductive Endocrinology and Fertility, Duke University Medical Center, Durham, NC, USA

Ilona S. Yim Department of Psychology and Social Behaviour, University of California, Irvine, Irvine, CA, USA

Deborah Lee Young-Hyman Department of Pediatrics, Georgia Prevention Institute Georgia Health Sciences Universtiy, Augusta, GA, USA

Lauren Zagorski Department of Psychology, The University of Iowa, Iowa City, IA, USA

Ydwine Zanstra The Amsterdam University College, Amsterdam, The Netherlands

Jet Veldhuijzen van Zanten School of Sport and Exercise Sciences, The University of Birmingham, Edgbaston, Birmingham, UK

Alex Zautra Department of Psychology, Arizona State University, Tempe, AZ, USA

Chris Zehr Department of Health Studies and Gerontology, University of Waterloo, Waterloo, ON, Canada

Kristin A. Zernicke Department of Psychology, University of Calgary, Calgary, AB, Canada

Emily Zielinski-Gutierrez Division of Vector-Borne Diseases, Centers for Disease Control and Prevention, Ft. Collins, CO, USA

Sheryl Zimmerman School of Social Work, The University of North Carolina at Chapel Hill, Chapel Hill, NC, USA

Tanja Zimmermann Department of Clinical Psychology, Psychotherapy and Diagnostics, University of Braunschweig, Braunschweig, Germany

A

A1C

► Glycosylated Hemoglobin
► HbA1c

Abdominal Obesity

► Central Adiposity

Abnormal Psychology

► Psychological Pathology

Abrams, David B. (1951–)

David B. Abrams
Johns Hopkins Bloomberg School of Public Health, The Schroeder Institute for Tobacco Research and Policy Studies at Legacy, Washington, DC, USA

Biographical Information

David Abrams was born in Johannesburg, South Africa, on September 6, 1951. He married Marion Wachtenheim in 1981. He has three children, Tanya, Aaron, and Daniel, who passed away in 2008. He holds a B.Sc. (honors) degree in computer science and psychology from University of Witwatersrand, Johannesburg, South Africa (1974), during which time he studied under Alma Hannon (who also taught Joseph Wolpe, Arnie Lazarus, Terry Wilson, and Ray Rosen). Abrams completed his doctorate in clinical psychology under Terry Wilson at Rutgers University, earning

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

his Ph.D. in 1981, and his internship under David Barlow at Brown University in 1979. Joining the new Division of Behavioral Medicine at Miriam Hospital, founded by Michael Follick, Abrams was the first coordinator of the Behavioral Medicine Risk Factors Clinic.

Abrams is currently Professor, Department of Health, Behavior and Society at The Johns Hopkins Bloomberg School of Public Health and Executive Director of the Schroeder Institute for Tobacco Research and Policy Studies at Legacy®. From 2005 to 2008, he directed the Office of Behavioral and Social Sciences Research (OBSSR) at the National Institutes of Health (NIH). From 1978 to 2004, Abrams rose through the ranks at Alpert Medical School of Brown University, becoming Professor of Psychiatry and Human Behavior, Professor of Community Health, and founding Director of the Transdisciplinary Centers for Behavioral and Preventive Medicine. Abrams is a licensed clinical psychologist specializing in health psychology, tobacco use behavior, addictions, and lifestyle and contextual pathways to population health, conceptualized from a transdisciplinary "systems" framework.

In 1969, Abrams received the Old Parktonian university scholarship from Parktown Boys High School and the IBM undergraduate computer science award in 1973. He has published over 250 scholarly articles. He is the lead author of *The Tobacco Dependence Treatment Handbook: A Guide to Best Practices*, a recipient of a book of the year award. Abrams was a member of the Board of Scientific Advisors of the National Cancer Institute and served on several committees for the Institute of Medicine of the National Academies. He received the Joseph Cullen Memorial Award from the American Society for Preventive Oncology for lifetime contributions to tobacco control. He was President of the Society of Behavioral Medicine in 2003 and a recipient of their Distinguished Scientist, Distinguished Research Mentorship, and Service awards. Abrams is a fellow of the American Psychological Association, The American Academy of Behavioral Medicine Research, The American Academy of Health Behavior, and the Society of Behavioral Medicine. He received the Musiker-Meranda award for contributions to mental health from the Rhode Island Psychological Association and a distinguished alumnus award from Rutgers University.

Major Accomplishments

Abrams is recognized for strategic and scientific contributions to disease prevention, particularly in tobacco control, addictions, and related risk factors. An integrative, "systems thinking" framework permeates his work. His accomplishments fall into two broad dimensions, reflecting the development of strategic research structures, frameworks, and organizations, and his personal research contributions.

He began his focus on strategic research frameworks in basic human physiology and human laboratory studies and in how basic science can inform clinical applications in behavior therapy. He then extended research to dissemination-implementation topics, focusing on worksites, the harnessing new informatics technologies, and the use of policy levers for large-scale impact on population health. John and Sonja McKinlay influenced his public health perspective on frameworks for making a cost-efficient (reach x effectiveness/cost) impact on populations. At Brown University, Abrams envisioned one of the early Transdisciplinary Centers for Behavioral and Preventive Medicine. He advocated for a Center with the organizational structure and function to foster the development of complex systems frameworks and simulation models to improve science-informed policy. He forged partnerships to integrate biopsychosocial and population sciences across disciplines, departments, and institutions.

In 1988, Abrams founded the Centers for Behavioral and Preventive Medicine, bridging basic, clinical, and public health sciences; medical school and campus departments; the Brown-affiliated teaching hospitals; and local institutions (University of Rhode Island; R.I. Dept. of Health). Abrams was instrumental in forging ties to obtain National Institutes of Health (NIH) grants and program project awards, the first of

which was founded in 1989 to establish one of the first National Cancer Institute (NCI) Cancer Prevention Research Units (CPRU). Abrams jointly codirected the CPRU for over a decade with James Prochaska and Wayne Velicer, University of Rhode Island. Regional collaborators were also added from Yale, Brandeis, Tufts, Boston, and Harvard Universities. Abrams was a member of the Robert Wood Johnson's Tobacco Etiology Research Network (TERN), where working with Richard Clayton, Dennis Prager, and the TERN team influenced his own research and vision for transdisciplinary science.

Over the years at Brown University, Abrams nurtured the Centers' faculty and the infrastructure that supported over 30 faculty and many trainees. The Centers evolved in the 2000s to house programs in the leading risk factors and major chronic diseases, including physical activity (Bess Marcus, Director); weight control, obesity, and diabetes (Rena Wing, Director); nicotine dependence and tobacco control (Ray Niaura, Director); and crosscutting programs in cancer, cardiovascular diseases, stress management, underserved populations, comorbidities across psychiatric, alcohol and substance abuse disorders, and HIV-AIDS. Teaching programs ranged from undergraduate and graduate classes to a health psychology internship and postdoctoral and early career fellowships supported by NIH training grant awards. From his arrival at Brown University in 1977 as a psychology intern to his departure in 2004 to become director of OBSSR at NIH, Abrams left a legacy and a culture of support, individual excellence and creativity, and a passion for transdisciplinary team collaboration to address complex problems in health behavior.

In 2005, Abrams was appointed Director of the Office of Behavioral and Social Sciences Research (OBSSR) and Associate Director of the National Institutes of Health (NIH). He was responsible for being the chief spokesperson for the NIH and the nation on matters of behavioral and social sciences and for advising the NIH Director, congress, and other government leaders on matters relating to the role human behavior plays in health and illness. He was responsible for strategic planning of behavioral and social sciences across all 27 of the NIH Institutes and Centers. Abrams spearheaded a new strategic prospectus for OBSSR with Alan Best, John McKinlay, and other consultants. He emphasized that the basic and applied sciences of behavior and behavior change are the bridge between biology and society. The social science scientific disciplines are as much a key to improving population health as are the biomedical and natural science disciplines. Abrams stressed the need for more collaborative interdisciplinary science, integrative thinking, and a robust systems science perspective to address complex pathways to disease prevention and health promotion.

Abrams added a communications office to the mission of OBSSR to showcase the achievements of the behavioral and social sciences across NIH. Returning to his undergraduate roots in computer sciences, he also embraced the use of new informatics, communications, computational, engineering, and mathematical modeling sciences as critical tools for the twenty-first-century transformation of the behavioral social and population sciences. Thus while at OBSSR, Abrams created a transdisciplinary vision for integrating "genomics" and "populomics" via epigenetics (the biological embedding of early experience) over the lifespan and across generations. The OBSSR strategic plan helped make visible and credible the investments in, and rigorous scientific contributions of, the behavioral, social, and population sciences to the NIH mission to improve the nation's health.

At Rutgers from 1974 to 1978, Abrams' early personal line of research interests blossomed under Terry Wilson's able mentorship, focusing on basic science of the cognitive-behavioral and physiological mechanisms in tobacco, alcohol use, and mood states (e.g., the role of stress and expectancy in alcohol and tobacco relapse risk). His masters thesis examined the reactivity of self-monitoring during smoking cessation, and in his doctoral dissertation and related studies, Abrams investigated pharmacological and expectancy effects of alcohol on physiological arousal, stress, and tension reduction theory (under Terry Wilson, Ray Rosen, and Peter Nathan). Moving

to Brown University, Abrams developed ideas (with Ovide Pomerleau) on use of standardized cue exposure and stress reactivity paradigms to elucidate basic mechanisms in nicotine dependence, craving, and relapse and thereby link human laboratory work to clinical treatment. Abrams also collaborated with Peter Monti to develop a parallel line of work in cue reactivity in alcohol use. Human laboratory research on cue reactivity and treatment implications has continued for over a decade with Ray Niaura and others taking a lead role, funded by the National Heart Lung and Blood Institute under Steve Weiss and Sally Shumaker.

Abrams branched out to work on self-help interventions to reach populations on a larger scale (dissemination/implementation and policy research) with grants from the National Cancer Institute under Tom Glynn. He developed and evaluated treatment programs at the worksite in a "systems" conceptual framework (with Lois Biener, Laura Linnan, Mike Follick, and Karen Emmons) to examine multilevel interactions of individual and cluster influences on behavior change in worksites. Abrams researched environmental and policy variables regarding secondhand smoke exposure with Karen Emmons and Bess Marcus. He conducted randomized controlled clinical trials of combined pharmacotherapy and behavior therapy treatment for smoking cessation with Michael Goldstein. In collaboration with Michael Follick, Abrams used randomized trials to evaluate worksite obesity treatments. They developed then evaluated an early form of the concept of harnessing intergroup competition and within-group cooperation to motivate weight loss among teams formed at worksites. Abrams research was consolidated when he became one of the principal investigators in a multicenter cooperative trial of cancer control at the workplace – the Working Well Project, funded by the NCI from 1989 to 1999.

Abrams also continued to collaborate on work in the addictions with interest in the comorbidity of alcohol-tobacco interactions with Damaris Rohsenow and in evaluating the physical activity to prevent weight gain in tobacco cessation treatment for women with Bess Marcus. Abrams helped develop the theme for a National Institutes of Alcoholism and Alcohol Abuse (NIAAA) conference on the need for a strategic research plan to examine alcohol-tobacco interactions from cells to society. He authored several chapters in the 1995 NIAAA conference monograph (No. 30) on Alcohol and Tobacco: from Basic Science to Policy.

In 1999, Abrams became a principal investigator of one of the seven NCI Transdisciplinary Tobacco Use Research Centers (TTURCs). He directed the TTURC with Ray Niaura and Steve Buka until appointed Director of OBSSR at NIH in 2005. This TTURC focused on phenotypes of tobacco use and related comorbidities to inform the intergenerational transmission of nicotine dependence and the tailoring of treatments. The TTURC followed up on the three generations of participants derived from the New England Cohort (originally with Lewis Lipsitt at Brown's psychology department) of the National Collaborative Perinatal Project, begun in 1959. Abrams continues to publish findings from the TTURC project with his colleagues, including a recent 2011 paper with Suzanne Colby on a lifetime measure of tobacco use patterns and trajectories, on generalizing from clinical trials to community samples with Amanda Graham, and on comorbidity of personality and alcohol and substance use disorders with Chris Kahler.

Abrams participated in a Robert Wood Johnson Foundation round table on consumer demand led by Tracy Orleans. He worked with David Levy on a series of computer simulation models to demonstrate the potential impact of putting what is known about evidence-based treatments and policies into widespread practice. These models informed Abrams membership in and contributions to the Institute of Medicine of the National Academies books "Bridging the Evidence Gap in Obesity Prevention (2010) and Ending the Tobacco Problem: A Blueprint for the Nation (2007)."

In 2008, Abrams became the Executive Director of the newly established Steven Schroeder

National Institute for Tobacco Research and Policy Studies at Legacy and Professor at Johns Hopkins Bloomberg School of Public Health. There, he continues to promote the need for efficient delivery of population level interventions and policies, with Donna Vallone, Cheryl Healton, and Legacy colleagues. Abrams continues collaborating with Amanda Graham and Nate Cobb on NCI-funded trials to evaluate Internet smoking cessation treatments and to examine social networks and social media phenomena for making an impact on populations. Together with Ray Niaura, Andrea Villanti, Jennifer Pearson, Mitch Zeller, Tom Kirchner, David Levy, and others, he is also developing and implementing strategic frameworks and studies whereby research can be strategically positioned to inform the Food and Drug Administration's 2009 congressional mandate to regulate tobacco products to reduce their population harms.

Cross-References

- ▶ Addictive Behaviors
- ▶ Diabetes
- ▶ Physical Activity
- ▶ Population Health
- ▶ Tobacco Control

References and Readings

Abrams, D. B. (1986). Roles of psychosocial stress, smoking cues, and coping in smoking-relapse prevention. *Health Psychology, 5*, 91–92.

Abrams, D. B. (1995). Integrating basic, clinical, and public health research for alcohol-tobacco interactions. In J. B. Fertig & J. P. Allen (Eds.), *Alcohol and tobacco: From basic science to policy* (NIAAA alcohol research monograph 30). Bethesda, MD: U.S. Dept. of Health and Human Services, Public Health Service, National Institutes of Health, National Institute on Alcohol Abuse and Alcoholism.

Abrams, D. B. (1999). Nicotine addiction: Paradigms for research in the 21st century. *Nicotine & Tobacco Research, 1*(Suppl. 2), S211–S215. PMID: 11768182.

Abrams, D. B. (2006). Applying transdisciplinary research strategies to understanding and eliminating health disparities. *Health Education & Behavior, 33*(4), 515–531.

Abrams, D. B., & Biener, L. (1992). Motivational characteristics of smokers: A public health challenge. *Preventive Medicine, 21*(6), 679–687. PMID: 1438114.

Abrams, D. B., Boutwell, W. B., Grizzle, J., Heimendinger, J., Sorensen, G., & Varnes, J. (1994). Cancer control at the workplace: The working well trial. *Preventive Medicine, 23*(1), 15–27.

Abrams, D. B., & Follick, M. J. (1983). Behavioral weight-loss intervention at the worksite: Feasibility and maintenance. *Journal of Consulting and Clinical Psychology, 51*(2), 226–233.

Abrams, D. B., Graham, A. L., Levy, D. T., Mabry, P. L., & Orleans, C. T. (2010). Boosting population quits through evidence-based cessation treatment and policy. *American Journal of Preventive Medicine, 38*(3 Suppl), S351–S363. PMID: 20176308.

Abrams, D. B., Leslie, F., Mermelstein, R., Kobus, K., & Clayton, R. R. (2003). Transdisciplinary tobacco use research. *Nicotine & Tobacco Research, 5*(Suppl. 1), S5–S10.

Abrams, D. B., Mills, S., & Bulger, D. (1999). Challenges and future directions for tailored communication research. *Annals of Behavioral Medicine, 21*(4), 299–306. PMID: 10721436.

Abrams, D. B., Monti, P. M., Carey, K. B., Pinto, R. P., & Jacobus, S. I. (1988). Reactivity to smoking cues and relapse: Two studies of discriminant validity. *Behaviour Research and Therapy, 26*(3), 225–233.

Abrams, D. B., Monti, P. M., Pinto, R. P., Elder, J. P., Brown, R. A., & Jacobus, S. I. (1987). Psychosocial stress and coping in smokers who relapse or quit. *Health Psychology, 6*(4), 289–303.

Abrams, D. B., Orleans, C. T., Niaura, R. S., Goldstein, M. G., Prochaska, J. O., & Velicer, W. (1996). Integrating individual and public health perspectives for treatment of tobacco: A combined stepped care and matching model. *Annals of Behavioral Medicine, 18*(4), 290–304.

Abrams, D. B., Rohsenow, D. J., Niaura, R. S., Pedraza, M., Longabaugh, R., Beattie, M. C., et al. (1992). Smoking and treatment outcome for alcoholics: Effects on coping skills, urge to drink, and drinking rates. *Behavior Therapy, 23*(2), 283–297.

Abrams, D. B., & Wilson, G. T. (1979). Effects of alcohol on social anxiety in women: Cognitive versus physiological processes. *Journal of Abnormal Psychology, 88*(2), 161–173.

Cobb, N., & Abrams, D. B. (2011). E-cigarette or drug-delivery device? Regulating novel nicotine products. *New England Journal of Medicine, 365*(3), 193–195.

Mabry, P. L., Olster, D. H., Morgan, G. D., & Abrams, D. B. (2008). Interdisciplinarity and systems science to improve population health: A view from the NIH Office of Behavioral and Social Sciences Research. *American Journal of Preventive Medicine, 35*(2 Suppl), S211–S224.

Niaura, R. S., Rohsenow, D. J., Binkoff, J. A., Monti, P. M., Pedraza, M., & Abrams, D. B. (1988). Relevance of cue reactivity to understanding alcohol and smoking relapse. *Journal of Abnormal Psychology, 97*(2), 133–152.

Villanti, A. C., Vargyas, E. J., Niaura, R. S., Beck, S. E., Pearson, J. L., & Abrams, D. B. (2011). FDA regulation of tobacco: Integrating science, law, policy and advocacy. *American Journal of Public Health, 101*(7), 1160–1162.

Absolute Risk

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

Absolute risk is best defined in conjunction with relative risk. For this example, we can define risk as the likelihood of an adverse consequence in two behavioral medicine interventions, Treatment A and Treatment B. Imagine that the risk is 1 in 10 for Treatment A and 2 in 10 for Treatment B. In this case, a relative risk statement can be made, saying that the probability of the event occurring following Treatment B is twice the probability of the event occurring following Treatment A. However, the same relative risk statement can be made for probabilities of 1 in 1,000,000 and 2 in 1,000,000. However, the absolute risks are vastly different: 1 and 2 in 10; and 1 and 2 in a million.

Description

Imagine that an intervention with beneficial therapeutic properties increased your risk of an adverse consequence (an event) from 1 in 10 to 2 in 10. It is possible that some individuals may consider that the risk is too great, and that they are not prepared to take this risk. Now imagine a different intervention with similarly beneficial properties that increases the risk of an event from 1 in a million to 2 in a million. In contrast to the first scenario, some individuals may feel that, while the relative risk has also doubled, the absolute risk has changed extremely slightly. Therefore, the expression of a risk in different ways, absolute and relative, can influence decisions made upon risk information.

Literature on risk reduction well exemplifies this. Statements of relative risk reduction can look considerably more impressive than statements of absolute risk reduction even though they are based on identical data. Consider that a decrease in risk from 6% to 3% is a 50% relative risk reduction. However, expressed in absolute terms, it is a 3% reduction. The same 50% relative risk reduction would be associated with a decrease in risk from 60% to 30%, but the absolute reduction of 30% would be much more important from a public health perspective. It is therefore very useful to patients and their physicians that risk information be provided in both absolute and relative terms.

Gordon-Lubitz (2003) commented as follows:

> Identical risk information may be presented in different ways, resulting in "framing bias." Perceptions of risk are particularly susceptible to framing effects. For example, patients are much more likely to favor radiation treatment over surgery when radiation is presented as having a 90% survival rate than when it is presented as having a 10% mortality rate. Although both numbers describe identical risks, the latter is perceived as more dangerous. Another common framing effect involves absolute and relative risks. For example, if a medication reduces an adverse outcome from 25% to 20%, then the absolute risk reduction is 5% and the relative risk reduction is 25%. Although the absolute and relative risk estimates are derived from the same data, patients are more strongly persuaded by the larger changes in relative risk.

Cross-References

► Relative Risk

References and Readings

Gordon-Lubitz, R. J. (2003). Risk communication: Problems of presentation and understanding. *Journal of the American Medical Association, 289*, 95.

Abstinence

Linda C. Baumann and Alyssa Karel
School of Nursing, University of Wisconsin-Madison, Madison, WI, USA

Definition

Abstinence is defined as the restraint from indulging in bodily activities that are experienced as giving pleasure. In medical settings this usually refers to drugs, alcohol, and most often sexual activity. Depending on the perspective, abstinence from sex can mean that all sexual activities are avoided, but it can also mean that only sexual intercourse is avoided. Abstinence is also considered a form of contraception; in fact it is the only form of contraception that prevents pregnancy with a 0% failure rate. Abstinence also prevents the spread of sexually transmitted infections.

In recent years the issue between abstinence-only sex education and comprehensive, or abstinence plus sex education has been widely debated. An abstinence-only sex education program focuses on promoting abstinence from sex until marriage due to the possibility of pregnancy and the spread of sexually transmitted infections. Morality is also discussed as a driving factor to remain abstinent. This approach avoids topics such as contraception or condom use and abortion, and rarely acknowledges that many teenagers are sexually active outside of marriage. An abstinence plus sex education program also promotes abstinence, but in addition also provides adolescents with discussion and information about contraception use, abortion, sexually transmitted infections including HIV.

Research on abstinence-only programs shows little evidence that this type of program has much positive impact on teenage behavior and may even put them at risk of being uninformed when it comes to matters of sexual activity. Research on comprehensive sex education programs show that these programs do not increase sexual activity among teens or increase the number of sexual partners, which has been a major concern of advocates of abstinence-only sex education. Research also shows that a comprehensive education program can reduce sexual behaviors that put teens at risk for pregnancy and acquiring sexually transmitted infections and therefore better prepares them to safely deal with the issue of sexual activity and the associated health risks.

As with abstinence and sex education, there is controversy over the effectiveness of many programs that promote abstinence with regards to drugs and alcohol. Drug Abuse Resistance Education (DARE) is one of the most widely implemented programs that teaches abstinence from alcohol, drugs, and violence. DARE reaches kids in over 75% of the United States school districts and is in over 43 countries worldwide. Research evidence, however, raises questions about the effectiveness of the program. In a 2001 report of the US Surgeon General, DARE was categorized as a program that "does not work."

References and Readings

About D.A.R.E. (2010). *D.A.R.E.*. Retrieved December 17, 2010, from http://www.dare.com/home/about_dare.asp

Abstinence. (2010). *Planned parenthood*. Retrieved August 23, 2010, from http://www.plannedparenthood.org/health-topics/birth-control/abstinence-4215.htm

Abstinence and Sex Education. (2010). *Avert*. Retrieved August 25, 2010, from http://www.avert.org/abstinence.htm

Kohler, P. K., Manhart, L. E., & Lafferty, W. E. (2008). Abstinence-only and comprehensive sex education and the initiation of sexual activity and teen pregnancy. *Journal of Adolescent Health, 47*(5), 344–351.

Ott, M. A., & Santelli, J. S. (2007). Abstinence and abstinence-only education. *Current Opinion in Obstetrics and Gynecology, 19*(5), 446–452.

Satcher, D. (2001). Prevention and intervention. *Youth violence, A report of the surgeon general*. Retrieved March 20, from http://www.surgeongeneral.gov/library/youthviolence/chapter5/sec4.html#IneffectivePrimaryPrevention

Abstinence Violation Effect

Susan E. Collins[1] and Katie Witkiewitz[2]
[1]Department of Psychiatry and Behavioral Sciences, University of Washington, Harborview Medical Center, Seattle, WA, USA
[2]University of New Mexico, Albuquerque, New Mexico, USA

Synonyms

AVE

Definition

The abstinence violation effect (AVE) refers to the negative cognitive (i.e., internal, stable, uncontrollable attributions; cognitive dissonance) and affective responses (i.e., guilt, shame) experienced by an individual after a return to substance use following a period of self-imposed abstinence from substances (Curry, Marlatt, & Gordon, 1987).

Description

AVE in the Context of the Relapse Process

The AVE was introduced into the substance abuse literature within the context of the "relapse process" (Marlatt & Gordon, 1985, p. 37). Relapse has been variously defined, depending on theoretical orientation, treatment goals, cultural context, and target substance (Miller, 1996; White, 2007). It is, however, most commonly used to refer to a resumption of substance-use behavior after a period of abstinence from substances (Miller, 1996). The term *relapse* may be used to describe a prolonged return to substance use, whereas *lapse* may be used to describe discrete, circumscribed "slips" during sustained abstinence (Marlatt & Gordon, 1985, p. 32).

As originally described by Marlatt and Gordon (1985), the relapse process typically begins when a person who has achieved abstinence encounters a situation that puts them at high risk for relapse (i.e., a high-risk situation). If the person is able to cope effectively with the high-risk situation, they may experience increased self-efficacy (i.e., confidence to avoid a lapse). If, on the other hand, they are unable to cope with the high-risk situation, they may experience decreased self-efficacy. If this decreased self-efficacy is paired with positive outcome expectancies for substance use, a person may have a heightened risk for a lapse. If a lapse occurs, it may be experienced as a "violation" of self-imposed abstinence, which gave rise to the term, AVE. The AVE may, in turn, precipitate a relapse if the person turns to substances repeatedly to cope with the resulting negative cognitive and affective reactions of the AVE.

AVE: Cognitive and Affective Responses to a Lapse

The AVE is characterized by a lapse paired with a specific constellation of negative cognitive and affective reactions. The role of cognitions stems from attributional theory (Weiner, 1974): a person might attribute their lapse to factors that are internal, global, and uncontrollable. For example, people may believe the lapse occurred due to their own, irreparable character defects or chronic disease determinants. The associated affective component stems from dissonance between the lapse and one's perceived self-image as an abstainer, which together with the attributions, can lead to feelings of guilt, shame, and hopelessness (Marlatt & Gordon, 1985). People who experience the AVE are more likely to progress from a lapse to a relapse (Miller, Westerberg, Harris, & Tonigan, 1996), and several studies have demonstrated the role of the AVE in predicting relapse among drinkers (Collins & Lapp, 1991), smokers (Curry et al., 1987), dieters (Mooney, Burling, Hartman, & Brenner-Liss, 1992), and marijuana users (Stephens, Curtin, Simpson, & Roffman, 1994).

In contrast, if people attribute the lapse to external, unstable (i.e., changeable), and controllable causes, they may not interpret the lapse as a threat to their self-image and may instead view it as a unique occurrence that can be avoided in the future. This attributional style may diffuse the

person's affective response to the lapse and reduces the likelihood of a progression from lapse to relapse (Laws, 1995; Marlatt & Gordon, 1985; Walton, Castro, & Barrington, 1994). Averting the AVE may have lasting effects: as the situation is less affectively charged, the individual might be open to exploring the determinants of the lapse and to experimenting with alternative coping strategies in the future. This may, in turn, lead to increased self-efficacy and more effective coping across various high-risk situations (Marlatt & Gordon, 1985).

Preventing the AVE Response

Clinicians may help clients interrupt the relapse process at various points and ultimately avoid the AVE. First, clinicians can help clients identify and apply effective behavioral and cognitive strategies in high-risk situations to avoid the initial lapse altogether. If a lapse occurs, clinicians should be empathetic and nonjudgmental in their approach (Miller & Rollnick, 2002) and should help clients reframe the lapse as the product of multiple factors (versus only internal factors), as being controllable (versus uncontrollable), and as situation-specific (versus global; Larimer, Palmer, & Marlatt, 1999). A step-by-step exploration may help clients learn how to interrupt the relapse process at various points to avoid future lapses, the AVE and/or relapses (Larimer et al., 1999). Further, the clinician may elicit and positively reinforce clients' existing coping skills to support the clients' self-efficacy and may teach clients additional behavioral and cognitive coping strategies for application in future high-risk situations, as necessary (Witkiewitz & Marlatt, 2007). Finally, clinicians should assess whether clients are coping adequately with the negative affective component of the AVE, which may otherwise precipitate future lapses or relapses.

Cross-References

- ▶ Addictive Behaviors
- ▶ Alcohol Abuse and Dependence
- ▶ Binge Drinking
- ▶ Health Risk (Behavior)
- ▶ National Institute on Alcohol Abuse and Alcoholism
- ▶ Relapse, Relapse Prevention

References and Readings

Collins, R. L., & Lapp, W. M. (1991). Restraint and attributions: Evidence of the abstinence violation effect in alcohol consumption. *Cognitive Therapy and Research, 15*, 69–84.

Curry, S., Marlatt, G. A., & Gordon, J. R. (1987). Abstinence violation effect: Validation of an attributional construct with smoking cessation. *Journal of Consulting and Clinical Psychology, 55*, 145–149.

Larimer, M. E., Palmer, R. S., & Marlatt, G. A. (1999). Relapse prevention: An overview of Marlatt's cognitive-behavioral model. *Alcohol Research & Health, 23*, 151–160.

Laws, D. R. (1995). Central elements in relapse prevention procedures with sex offenders. *Psychology, Crime and Law, 2*, 41–53.

Marlatt, G. A., & Gordon, J. R. (1985). *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors*. New York: The Guilford Press.

Miller, W. R. (1996). What is relapse? Fifty ways to leave the wagon. *Addiction, 91*(Suppl.), S15–S27.

Miller, W. R., & Rollnick, S. (2002). *Motivational interviewing: Preparing people for change* (2nd ed.). New York: US Guilford Press.

Miller, W. R., Westerberg, V. S., Harris, R. J., & Tonigan, J. S. (1996). What predicts relapse? Prospective testing of antecedent models. *Addiction, 91*(Suppl.), 155–171.

Mooney, J. P., Burling, T. A., Hartman, W. M., & Brenner-Liss, D. (1992). The abstinence violation effect and very low calorie diet success. *Addictive Behaviors, 19*, 23–32.

Stephens, R. S., Curtin, L., Simpson, E. E., & Roffman, R. A. (1994). Testing the abstinence violation effect construct with marijuana cessation. *Addictive Behaviors, 19*, 23–32.

Walton, M. A., Castro, F. G., & Barrington, E. H. (1994). The role of attributions in abstinence, lapse and relapse following substance abuse treatment. *Addictive Behaviors, 19*, 319–331.

Weiner, B. (1974). *Achievement motivation and attribution theory*. Morristown, NJ: General Learning Press.

White, W. L. (2007). Addiction recovery: Its definition and conceptual boundaries. *Journal of Substance Abuse Treatment, 33*, 229–241.

Witkiewitz, K., & Marlatt, G. A. (2007). Relapse prevention for alcohol and drug problems. In G. A. Marlatt & D. M. Donovan (Eds.), *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors* (2nd ed.). New York: The Guilford Press.

Abuse, Child

► Child Abuse

Abuse, Elder

Terry Fulmer
Bouvé College of Health Sciences,
Northeastern University,
Boston, MA, USA

Synonyms

Family Violence

Definition

The National Research Council defines elder mistreatment as "intentional actions that cause harm or create a serious risk of harm (whether or not harm is intended) to a vulnerable elder by a caregiver or other person who stands in a trust relationship to the elder or failure by a caregiver to satisfy the elder's basic needs or to protect the elder from harm" (Bonnie & Wallace, 2003, p. 40).

Description

EM may occur in the community setting (domestic EM) or in institutional settings, such as nursing homes and adult family homes. In the United States, data about EM among community-dwelling, vulnerable older adults suggest that victimization will rise from 1.25 million in 2010 to 2.2 million in 2030 based on the aging demographics of America. Further, it is estimated that for every case of EM that is reported, more than five cases go unreported (Tatara, 1997). This means that there will be over 6.6 million unreported cases by 2020 and over 11.7 million unreported cases by 2030, leading to extraordinary suffering and disability among vulnerable older adults.

Elder mistreatment is the outcome of actions which include neglect, physical, sexual, and emotional/psychological abuse; financial and material exploitation; and abandonment. When domestic violence (DV) occurs in situations in which the older adult is vulnerable, domestic violence in later life (DVLL) is a form of elder mistreatment. While self-neglect in community-dwelling older adults, resident-on-resident aggression in long-term care settings and "stranger crimes" (e.g., sweetheart scams; assaults by strangers) are serious issues, they are considered separately from elder mistreatment. Older adults who self-neglect are not necessarily vulnerable adults and there is no caregiving dyad involving a "trusted other" (Bonnie & Wallace, 2003; Dong et al., 2009). Resident-on-resident aggression involves vulnerable adults, but they are not in a caregiving dyad with each other, as they rely on professional staff for care (Lachs, Bachman, Williams, & O'Leary, 2007). Stranger crimes also do not necessarily involve vulnerable older adults or trusted others with a duty of care (Bonnie & Wallace, 2003).

Neglect of community-dwelling vulnerable older adults by trusted others is the most prevalent type of domestic EM, with over 70–80% of cases in that category. Recent estimates of domestic EM place the prevalence since reaching age 60 years and past-year prevalence of psychological mistreatment at 13.5% and 4.6%; physical mistreatment at 1.8% and 1.6%; and sexual mistreatment at 0.3% and 0.6%, respectively. The estimated past-year prevalence of financial mistreatment by family members is 5.2% (Acierno et al., 2010). The prevalence of EM in institutional settings is unknown, but the problem is thought to be widespread and serious (Hawes, 2003). For example, from January 1999 to January 2001, there were 5,283 nursing home citations for almost 9,000 abuse violations (Minority Staff SID, 2001). Regardless of the type(s) of elder mistreatment experienced by vulnerable older adults, EM imposes serious consequences for the health and safety of its victims, including a three-times higher adjusted risk of death for community-dwelling older adults (Lachs, Williams, O'Brien, Pillemer, & Charlson, 1998).

Domestic elder mistreatment is a form of family violence in that the vast majority (90%) of abusers are family members. Adult children are the more prevalent (47%) perpetrators of domestic EM, followed by spouses/partners (19%) and other family members (Tatara, 1997). Perpetrators of institutional EM include staff and family or friends who visit (Hawes, 2003).

EM risk involves characteristics of both the vulnerable older adult and the caregiver. Risk factors in community-dwelling older adults include older age (>70), low social support, and the number of self-care deficits that lead to dependence on others for care (Acierno et al., 2010). Characteristics of adult children who are likely to mistreat their vulnerable parents include dependence of the adult child on the parent for housing and financial support; substance abuse; mental illness; and poor social integration. If the abuser is a spouse or partner, abuse may be the continuation of existing domestic violence into later life; new behavior in the caregiving spouse/partner; or a new relationship in which there is partner violence (Bonnie & Wallace, 2003).

EM risk in institutional settings may be related to characteristics of staff caregivers. For example, psychological abuse of residents was related to higher caregiver work stress (role strain, demand, and work overload) and lower caregiver educational level (Wang, Lin, Tseng, & Chang, 2009). Risk for all mistreatment types is related to the degree to which residents are dependent on staff for care (Cohen, Halevy-Levin, Gagin, Priltuzky, & Friedman, 2010; Post et al., 2010).

Action steps for prevention and early intervention of elder mistreatment include appropriate assessment and screening policies with clear clinical practice protocols for staff to follow; protocols to guide staff in reporting cases to proper authorities; mechanisms for participating in complaint investigations to ensure that accusations of mistreatment are verified and handled appropriately; educational programs, consultation and follow-up protocols for healthcare professionals; and research and public policy actions to address the issue.

Cross-References

▶ Family Violence

References and Readings

Acierno, R., Hernandez, M. A., Amstadter, A. B., Resnick, H. S., Steve, K., Muzzy, W., et al. (2010). Prevalence and correlates of emotional, physical, sexual, and financial abuse and potential neglect in the United States: The National Elder Mistreatment Study. *American Journal of Public Health, 100*(2), 292–297.

Bonnie, R. J., & Wallace, R. (Eds.). (2003). *Elder mistreatment: Abuse, neglect, and exploitation in an aging America*. Washington, DC: The National Academies Press.

Cohen, M., Halevy-Levin, S., Gagin, R., Priltuzky, D., & Friedman, G. (2010). Elder abuse in long-term care residences and the risk indicators. *Ageing & Society, 30*, 1027–1040.

Dong, X., Simon, M., Mendes de Leon, C., Fulmer, T., Beck, T., Hebert, L., et al. (2009). Elder self-neglect and abuse and mortality risk in a community-dwelling population. *Journal of the American Medical Association, 302*(5), 517–526.

Hawes, C. (2003). Elder abuse in residential long-term care settings: What is known and what information is needed? In R. J. Bonnie & R. Wallace (Eds.), *Elder mistreatment: Abuse, neglect, and exploitation in an aging America*. Washington, DC: The National Academies Press.

Lachs, M., Bachman, R., Williams, C. S., & O'Leary, J. R. (2007). Resident-to-resident elder mistreatment and police contact in nursing homes: Findings from a population-based cohort. *Journal of American Geriatrics Society, 55*(6), 840–845.

Lachs, M. S., Williams, C. S., O'Brien, S., Pillemer, K. A., & Charlson, M. E. (1998). The mortality of elder mistreatment. *Journal of the American Medical Association, 280*(5), 428–432.

Minority Staff SID, C. o. G. R., U.S. House of Representatives, (July 30, 2001). *Abuse of residents is a major problem in U.S. nursing homes*. Washington, DC: Author

Post, L., Page, C., Conner, K. O., Prokhorov, A., Fang, Y., & Biroscak, J. (2010). Elder abuse in long-term care: Types, patterns, and risk factors. *Research on Aging, 32*(3), 323–348.

Tatara, T. (1997). *The national elder abuse incidence study: Executive summary*. New York City: Human Services Press.

Wang, J. J., Lin, M. F., Tseng, H. F., & Chang, W. Y. (2009). Caregiver factors contributing to psychological elder abuse behavior in long-term care facilities: a structural equation model approach. *International Psychogeriatrics, 21*(2), 314–320.

Accelerometry

▶ Actigraphy (Wrist, for Measuring Rest/Activity Patterns and Sleep)

Acculturation

Molly L. Tanenbaum[1], Persis Commissariat[1], Elyse Kupperman[1], Rachel N. Baek[1] and Jeffrey S. Gonzalez[1,2]
[1]Clinical Psychology, Health Emphasis, Ferkauf Graduate School of Psychology, Yeshiva University, Bronx, NY, USA
[2]Diabetes Research Center, Albert Einstein College of Medicine, Yeshiva University, Bronx, NY, USA

Definition

Definition and Theoretical Background

Acculturation is the process by which migrants to a new culture develop relationships with the new culture and maintain their original culture (Berry & Sam, 1997). Acculturation has been classically defined as the changes that develop when groups of individuals come into contact with a different culture (Redfield, Linton, & Herskovits, 1936). This process was initially conceptualized as unidimensional, in which retention of the original culture and acquisition of the new host culture were cast at opposing ends of a single continuum (Schwartz, Unger, Zamboanga, & Szapocznik, 2010). According to this unidimensional model, migrants were expected to acquire the values, practices, and beliefs of their new homelands and discard those from their cultural heritage. Acculturation is now more often conceptualized as complex and multidimensional, meaning that both cultures change under the influence of each other and acculturation is influenced by a number of contextual factors (Berry & Sam, 1997; Sam & Berry, 2006).

According to Berry and Sam (1997), acculturation involves four strategies: assimilation, separation, integration, and marginalization. Assimilation means immersion in the new culture and breaking from the original culture; separation refers to the nondominant group distancing themselves from the new, dominant culture and holding onto original cultural practices and beliefs; integration is when individuals maintain both their original cultural identity while taking part in the new culture's practices; marginalization refers to what occurs when individuals leave behind their original cultural identity but do not take part in the new culture.

Some researchers feel that definitions of acculturation need to move beyond behavioral indicators and include other factors such as language use, values, and attitudes (Thomson & Hoffman-Goetz, 2009). Investigators stress the importance of emigration and immigration contexts as modifiers of the acculturation experience (Thomson & Hoffman-Goetz, 2009).

Description

Measuring Acculturation

While no definitive framework for acculturation has been defined, Berry and Sam (1997) suggest a composite framework for measuring acculturation including: societies of origin and settlement, psychological acculturation, and the moderating factors that contribute to and arise from it, and the eventual shift to psychological and sociocultural adaptation. Acculturation can be affected by a number of variables, such as age, gender, race, ethnicity, and socioeconomic status, which in turn affects the behaviors and values of a person (Maxwell, 2006). These variables all play a role in the acculturation process, though no single measurement scale has been able to extensively study all of these factors. The acculturation process is generally accepted to be bidirectional (Sam & Berry, 2006), which assumes that both cultures can change under the influence of each other, but do not necessarily reach a neutral point. Due to the complexity of acculturation, accurate measurement is challenging.

Scales have been developed to measure acculturation for a number of different ethnic groups including Mexican Americans, Chinese Americans, European Americans, and Cuban

Americans. These scales often include items assessing native and host language usage, language usage inside and outside the family, ties to country of origin, cultural familiarity and pride, length of stay in host country, personal values, and interpersonal relations. Some scales also examine concepts of independence, gender, culture, fashion, food, music, and movie preferences (Acculturation Depot, 1998; Maxwell, 2006).

Psychological Acculturation

Graves (1967) makes a distinction between acculturation as a collective or group-level phenomenon, and psychological acculturation, a change in the psychology of the individual. The internal processes of change that immigrants experience when they come into direct contact with members of the host culture constitute psychological acculturation. This construct is conceptualized as a resocialization process involving psychological changes in attitudes, values, and identification; the acquisition of new social skills and norms; changes in reference-group and membership-group affiliations; and adjustment or adaptation to a changed environment (Berry et al., 1992; Sam, 1994). Psychological adaptations to acculturation involve learning a new behavioral repertoire that is appropriate for the new cultural context (Berry & Sam, 1997). If individuals cannot easily change their repertoire, they may experience acculturative stress, the psychological, somatic, and social difficulties that may accompany acculturation processes. This stress may lead to serious psychological disturbances such as clinical depression and anxiety when environmental stressors exceed the individual's capacity to cope.

While the concept of acculturation has become widely used in cross-cultural psychology, it is important to distinguish it from the concepts of assimilation and adaptation. Assimilation is a process of cultural absorption of a minority group into the main cultural body. While acculturation implies a mutual influence in which elements of two cultures merge, assimilation implies a tendency of the ruling cultural group to enforce the adoption of their values rather than the blending of values (Maxwell, 2006). Berry (1984) defined psychological adaptation as the individual behaviors that are linked to acculturation experience, either as "shifts" of the preexisting customs or habits in language, beliefs, attitudes, values, or abilities, or as "acculturative stress" which is generated during acculturation. In the recent literature on psychological adaptation to acculturation, a distinction has been drawn between psychological and sociocultural adaptation. Psychological adaption mostly involves one's psychological well-being and satisfaction in a new cultural context, whereas sociocultural adaptation refers to one's ability to acquire culturally appropriate knowledge and skills and to interact with the new culture and manage daily life (Ward, Bochner, & Furnham, 2001).

Acculturation and Its Effect on Health Behaviors

Acculturation has been linked to changes in health behaviors – including eating, sexual health behaviors, accessing health services, and other behaviors – as well as changes in knowledge and beliefs. Studies have shown that acculturation to the USA may serve as a health risk or as a protective factor depending on the ethnic group, health behavior in question, and other variables such as gender. Acculturation may lead to the adoption of unhealthy behaviors such as smoking and eating a more high-fat diet or may lead to an increase in healthy behaviors such as exercise (Abraído-Lanza, Armbrister, Flórez, & Aguirre, 2006). Recently, Landrine and Klonoff (2004) proposed an operant model of acculturation as a way to guide to health behavior interventions. The operant model of acculturation looks at prevalence of certain health behaviors in an ethnic group's home country and new culture to predict the likelihood of adoption of those health behaviors depending on level of acculturation.

Among Hispanics and Asians in the United States, acculturation has been shown to influence diet, cancer screenings, and smoking, among other health behaviors, often with studies comparing more and less acculturated members of the same ethnic group (Landrine & Klonoff, 2004). The effects of acculturation on health behaviors differ from ethnic group to ethnic group; for example, Japanese-American men who adhere

more to Japanese culture are less likely to develop coronary heart disease (CHD) than more acculturated Japanese-American men, while this change in risk of developing CHD has not been found in Irish-American men. Other factors, such as gender, may play a role in the effect of acculturation on a health behavior. For example, acculturation has been found to increase smoking among Latino women and decrease smoking among Latino men (Perez-Stable et al., 2001). Due to the complex relationship between these variables, acculturation has been shown to have both positive and detrimental effects on an individual's health and well-being, and more research is needed to examine these processes further.

Cross-References

- ▶ Cultural and Ethnic Differences
- ▶ Cultural Competence
- ▶ Cultural Factors
- ▶ Ethnicity
- ▶ Hispanic/Latino Health
- ▶ Latino Health
- ▶ Minority Health
- ▶ Sociocultural Differences

References and Readings

Abraído-Lanza, A. F., Armbrister, A. N., Flórez, K. R., & Aguirre, A. N. (2006). Toward a theory-driven model of acculturation in public health research. *American Journal of Public Health, 96*, 1342–1346.

Acculturation Depot. (1998). University of California, Berkeley. Retrieved February 5, 2011, from http://www.ocf.berkeley.edu/~psych/depot.html#Scales

Berry, J. W. (1984). Multicultural policy in Canada: A social psychological analysis. *Canadian Journal of Behavioral Sciences, 16*, 353–370.

Berry, J. W. (2003). Conceptual approaches to acculturation. In K. M. Chun, P. B. Organista, & G. Marin (Eds.), *Advances in theory, measurement and applied research* (pp. 17–38). Washington, DC: American Psychology Association.

Berry, J. W., Poortinga, Y. H., Segall, M. H., & Dasen, P. R. (1992). *Cross-cultural psychology: Research and applications*. Cambridge: Cambridge University Press.

Berry, J. W., & Sam, D. L. (1997). Acculturation and adaptation. In J. W. Berry, M. H. Segall, & C. Kagitcibasi (Eds.), *Handbook of cross-cultural psychology* (2nd ed., Vol. 3, pp. 291–326). Boston: Allyn and Bacon.

Graves, T. (1967). Psychological acculturation in a tri-ethnic community. *South-Western Journal of Anthropology, 23*, 337–350.

Jasso, G., Massey, D. S., Rosenzweig, M. R., & Smith, J. P. (2004). Immigrant health – selectivity and acculturation. In N. B. Anderson, R. A. Bulatao, & B. Cohen (Eds.), *Critical perspectives on racial and ethnic differences in health in late life* (pp. 227–266). Washington, DC: The National Academies Press.

Landrine, H., & Klonoff, E. A. (2004). Culture change and ethnic-minority health behavior: An operant theory of acculturation. *Journal of Behavioral Medicine, 27*, 527–555.

Maxwell, A. E. (2006). Acculturation. In L. Breslow (Ed.), *Encyclopedia of public health*. Gale Cengage, 2002. Retrieved February 5, 2011, from http://www.enotes.com/public-health-encyclopedia/acculturation

Perez-Stable, E. J., Ramirez, A., Villareal, R., Talavera, G. A., Trapido, E., Suarez, L., et al. (2001). Cigarette smoking behavior among U.S. Latino men and women from different countries of origin. *American Journal of Public Health, 91*(9), 1424–1430.

Redfield, R., Linton, R., & Herskovits, M. (1936). Memorandum on the study of acculturation. *American Anthropologist, 38*, 149–152.

Sam, D. L. (1994). *Acculturation of young immigrants in Norway. A psychological and socio-cultural adaptation*. Bergen: University of Bergen.

Sam, D., & Berry, J. W. (2006). Acculturation: Conceptual background. In D. Sam & J. W. Berry (Eds.), *The Cambridge handbook of acculturation psychology* (pp. 17–18). Cambridge: Cambridge University Press.

Schwartz, S. J., Unger, J. B., Zamboanga, B. L., & Szapocznik, J. (2010). Rethinking the concept of acculturation: Implications for theory and research. *American Psychologist, 65*, 237–251.

Thomson, M. D., & Hoffman-Goetz, L. (2009). Defining and measuring acculturation: A systematic review of public health studies with Hispanic populations in the United States. *Social Science & Medicine, 69*, 983–991.

Ward, C., Bochner, S., & Furnham, A. (2001). *The psychology of culture shock*. London: Routledge.

Acetylcholine

Nicole Brandt[1] and Rachel Flurie[2]
[1]School of Pharmacy, University of Maryland, Baltimore, MD, USA
[2]University of Maryland, Baltimore, MD, USA

Definition

Acetylcholine is a naturally occurring monoamine neurotransmitter found in both the peripheral and

central nervous systems. It is the primary transmitter for the autonomic nervous system and the somatic efferent nerves that innervate skeletal muscle. It was first discovered in 1914 by Sir Henry Dale and colleagues. Acetylcholine is synthesized inside the terminal endings of cholinergic nerve cells where choline is taken up into the nerve terminal and reacts with acetyl coenzyme A via the enzyme choline acetyltransferase.

Description

Once acetylcholine is synthesized, it is stored in vesicles until the nerve is stimulated by calcium entry into the nerve terminal. Stimulation causes the vesicles to release acetylcholine into the synapses between the pre- and postsynaptic nerve fibers. Acetylcholine crosses the synapse and binds to receptors on the postsynaptic cell, exerting its effect. It causes increased permeability of the cell to the cations sodium, potassium, and calcium, resulting in cell depolarization. The two types of receptors on which acetylcholine acts are the nicotinic and muscarinic receptors, so named because they were discovered using the two compounds muscarine and nicotine. The nicotinic receptors are found on all preganglionic autonomic nerve fibers, somatic efferent nerve fibers that connect to skeletal muscle, and the central nervous system. Nicotinic receptors have two subtypes: muscle and neuronal. The muscle subtype is found in skeletal muscle at the neuromuscular junction. The neuronal subtype is found in the peripheral and central nervous systems and in nonneuronal tissues (adrenal medulla). The muscarinic receptors are found on all postganglionic parasympathetic nerve fibers and postganglionic sympathetic nerve fibers that terminate at sweat glands, and in the central nervous system. Muscarinic receptors have five subtypes: M_1, M_2, M_3, M_4, and M_5. The subtypes are distributed throughout different areas of the brain, in autonomic ganglia, and in certain glands (gastric, salivary, and smooth muscle).

The action of acetylcholine is terminated by the enzyme acetylcholinesterase found in the synaptic cleft. The mechanism of action of several drugs, including the drugs used for Alzheimer's disease, relies on the inhibition of this enzyme. Drugs that act on the synthesis, storage, and release process of acetylcholine are not very selective and therefore are not good as systemic therapy.

Acetylcholine affects several organs in the body. The heart has M_2 receptors, and stimulation of those receptors causes vasodilation, decreased heart rate, decreased conduction velocity of the AV node, and decreased force of contraction. Acetylcholine works on M_3 receptors in the lungs to cause bronchoconstriction. It works on M_2 and M_3 receptors in the bladder to cause inhibition of smooth muscle relaxation. In the gastrointestinal tract, acetylcholine works on M_1, M_2, and M_3 receptors to control motility and gastric and salivary gland secretions. Acetylcholine also acts on M_3 receptors in the eye to cause pupillary and ciliary muscle contraction. All five muscarinic receptor subtypes are found in the CNS and cause a variety of actions such as increased cognitive function, increased seizure activity, regulation of dopamine release, neuronal inhibition, analgesia, appetite regulation, and augmentation of drug-seeking behavior and reward.

Drugs that affect the action of acetylcholine are divided according to their physiological site of action. They are muscarinic agonists, muscarinic antagonists, ganglion-stimulating drugs, ganglion-blocking drugs, neuromuscular-blocking drugs, and drugs that enhance cholinergic transmission. Clinically, muscarinic agonists are used locally to treat glaucoma (pilocarpine) by lowering the intraocular pressure and to help with bladder emptying or stimulate gastrointestinal motility (bethanechol). Many more muscarinic antagonists are used clinically. Atropine is used in people with bradycardia and gastrointestinal hypermotility, but it is also used to reduce secretions and inhibit bronchoconstriction in the respiratory tract. Scopolamine is used to treat motion sickness. Ipratropium and tiotropium are used via inhalation in people with asthma and chronic obstructive pulmonary disease to inhibit bronchoconstriction, and ipratropium is additionally used to treat rhinorrhea. Muscarinic antagonists used to reduce frequency of muscle contractions seen in urinary incontinence include oxybutynin, tolterodine, trospium chloride, darifenacin, solifenacin, and

fesoterodine. Pirenzepine inhibits gastric acid secretion and is used to treat peptic ulcers. Drugs that cause pupil dilation and ciliary muscle paralysis are used to treat uveitis and include homatropine hydrobromide, cyclopentolate hydrochloride, and tropicamide. Benztropine mesylate, trihexyphenidyl hydrochloride, and biperiden are used in Parkinson's disease because of their regulation of dopamine. Antipsychotics used for schizophrenia and other neurologic disorders have various degrees of muscarinic antagonism, which can help decrease the extrapyramidal side effects of these drugs but also cause worsening cognition.

Drugs that act at the ganglionic and motor endplate receptors act specifically at nicotinic receptors. The only nicotinic agonists with a therapeutic use are nicotine for smoking cessation and suxamethonium for muscle relaxation, but lobeline, epibatidine, and dimethyphenylpiperazinium are also agonists of the nicotine receptor. Nicotinic antagonists have limited clinical use because nicotinic receptors are found in both divisions of the autonomic nervous system and skeletal muscle; therefore, they cause severe postural and postexercise hypotension. Trimetaphan is used for some types of anesthetic procedures, and pancuronium, atracurium, and vecuronium can be used as muscle relaxants in anesthesia.

Neuromuscular-blocking agents can work either presynaptically or postsynaptically, although all of the drugs used clinically work postsynaptically. They work by either blocking the acetylcholine receptor and ion channels or as agonists at the receptors. They are used mainly in anesthesia to produce muscle relaxation. These drugs are tubocurarine, pancuronium, vecuronium, atracurium, mivacurium, and suxamethonium.

Finally, drugs that enhance cholinergic transmission work either by inhibiting the enzyme acetylcholinesterase or by increasing acetylcholine release from the nerve terminal. These drugs work to increase the effect of acetylcholine in the autonomic nervous system, at the neuromuscular junction, and in the central nervous system. Neostigmine is used after an operation to reverse the anesthesia and for myasthenia gravis. Donepezil, rivastigmine, and galantamine are used to treat Alzheimer's disease dementia.

Acetylcholine is vital to so many systems that understanding the physiology will help to understand the mechanisms of various medications used to address multiple medical conditions.

References and Readings

Brunton, L. L., Chabner, B. A., & Knollmann, B. C. (2010). *Goodman and Gilman's the pharmacological basis of therapeutics* (12th ed.). New York: McGraw-Hill Professional.

Rang, H. P., Dale, M. M., Ritter, J. M., & Flower, R. J. (2007). *Rang and Dale's pharmacology* (6th ed.). Philadelphia: Churchill Livingstone/Elsevier.

Trevor, A. J., Katzung, B. G., & Masters, S. B. (2010). *Pharmacology: Examination and board review* (9th ed.). New York: McGraw-Hill Medical.

ACTH

Jennifer Heaney
School of Sport and Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

Definition

Adrenocorticotrophic hormone (ACTH) is a polypeptide hormone that is synthesized and secreted by the anterior pituitary gland. ACTH forms part the hypothalamic-pituitary-adrenal axis (HPA axis) and its production is stimulated by corticotropin-releasing hormone (CRH) from the hypothalamus. ACTH acts on the adrenal cortex, by increasing the conversion of cholesterol to pregnenolone, to stimulate the release of mineralocorticoids, androgenic steroids, and glucocorticoids, namely, cortisol (Martin, Reichlin, & Brown, 1977).

Along with CRH, ACTH is produced in the response to stress, stimulating an increase in production and secretion of cortisol. The secretion of ACTH is subject to negative feedback, where increased cortisol levels reduce the secretion of ACTH. It also controls its own secretion through short loop feedback via CRH. ACTH is secreted

in a pulsatile manner, which is under neural control rather than a result of stress (Martin, Reichlin, & Brown, 1977). The circadian secretion of ACTH increases prior to awakening and then declines progressively throughout the day.

Overproduction or blunted levels of ACTH can occur as a result of a disease. For example, increased levels of ACTH occur as a result of Addison's disease and Cushing's disease. Alternatively, ACTH levels may be reduced due to adrenal insufficiency or pituitary disease or as a result of a cortisol secreting tumor (Greenspan & Forsham, 1983).

Cross-References

► Hypothalamus

References and Readings

Greenspan, F. S., & Forsham, P. H. (1983). *Basic & clinical endocrinology*. California, CA: Lange Medical Publications.

Martin, J. B., Reichlin, S., & Brown, G. M. (1977). *Clinical neuroendocrinology*. Philadelphia: F.A. Davis Company.

Actigraphy (Wrist, for Measuring Rest/Activity Patterns and Sleep)

Christopher Kline
Department of Psychiatry, School of Medicine, University of Pittsburgh, Pittsburgh, PA, USA

Synonyms

Accelerometry; Actimetry; Activity monitor

Definition

Actigraphy is a method of objective sleep assessment in which sleep/wake status is estimated from bodily movements, typically of the wrist.

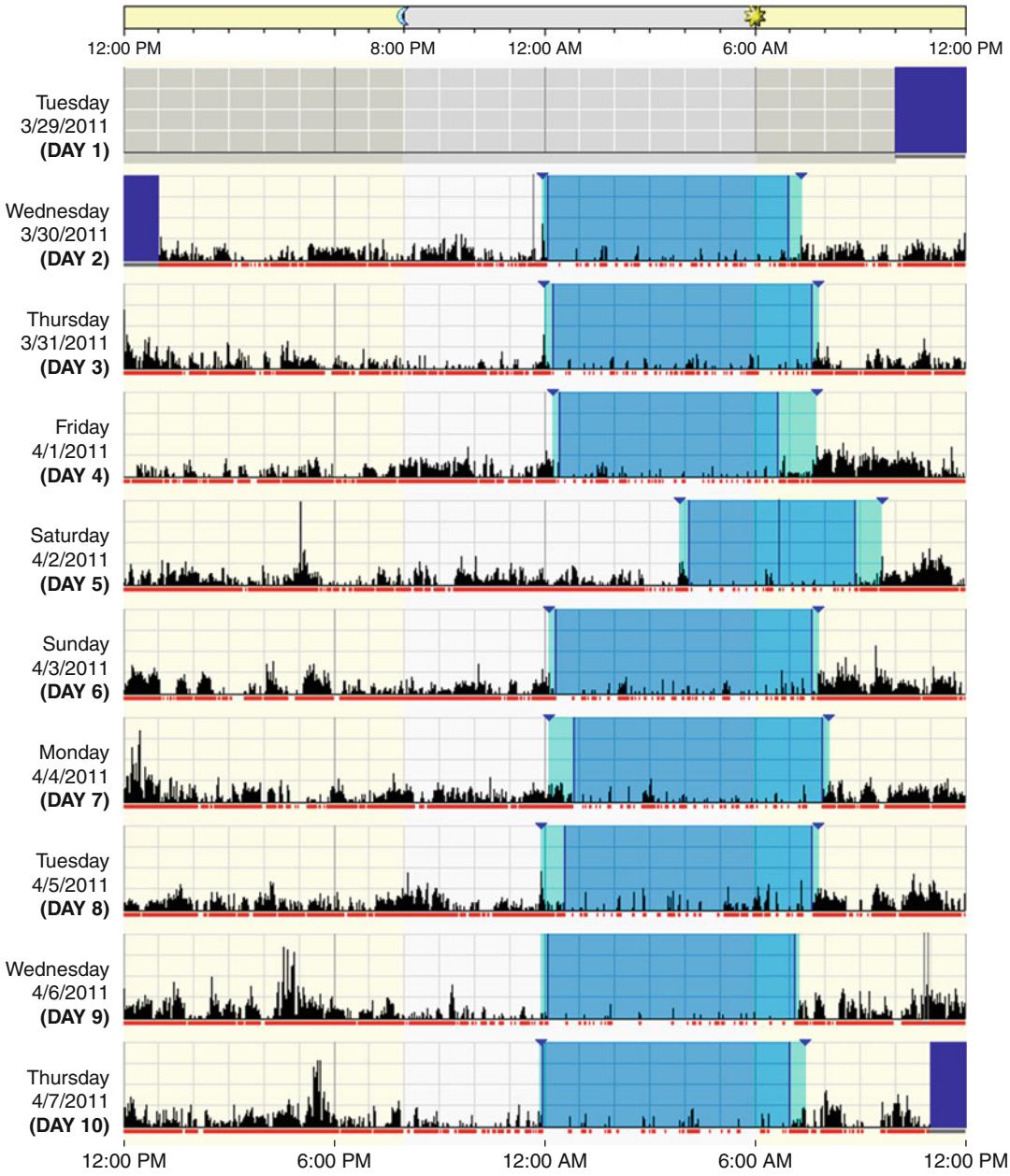
Description

Background and Use

Actigraphy is a common alternative to laboratory polysomnography (PSG) for the objective assessment of sleep/wake patterns, based on the observation that the amount of bodily movement differs between sleep and wakefulness. Although initially developed in the 1970s, there has been an exponential increase in the use and development of actigraphy over the past 15 years. Due to technological developments, actigraphs are now unobtrusive (similar in size and weight to a wrist watch) and inexpensive, capable of collecting data for multiple weeks, and able to provide rapid feedback on sleep patterns due to automated software algorithms. In addition, many actigraphs now record ambient light exposure and have the ability to mark the timing of specific events (e.g., bedtime).

Actigraphs are most commonly used for the evaluation of sleep/wake patterns in the home. Typically worn on the nondominant wrist (or ankle for infants), actigraphs continuously collect information on the frequency and intensity of movement with sensitive internal sensors. Upon download of data, each epoch of activity data is then classified as sleep or wake based upon algorithms that have typically been developed against PSG, the gold standard for objective sleep assessment. The algorithms used to estimate sleep/wake status incorporate movement counts for the epoch in question and the immediate surrounding epochs; the epoch is scored as sleep or wake based on whether the activity counts are below or above a particular threshold, respectively (see Fig. 1 for display).

Common sleep measures obtainable via actigraphy include sleep onset latency (i.e., the length of time it takes for one to fall asleep), wakefulness after sleep onset (i.e., the amount of time spent awake after initially falling asleep), time in bed (i.e., the amount of time during which sleep is attempted), total sleep time (i.e., the total amount of time spent asleep during a specified interval of time), sleep efficiency (i.e., the ratio of time spent asleep to the amount of time



Actigraphy (Wrist, for Measuring Rest/Activity Patterns and Sleep), Fig. 1 Actigraph output, providing a plot of activity counts over multiple days (Actiwatch 2, Philips Respironics Actiware v. 5.59 software, Bend, OR). Each row represents a separate 24-h period (shown here beginning at noon), with activity counts plotted for each minute of data collection. *Triangles* represent times at which the individual pressed an event marker. Light shaded areas indicate times at which the individual was in bed attempting to sleep. Dark shaded areas indicate intervals that were excluded from analysis. Used with permission from Philips Healthcare (Bend, OR)

attempting sleep), the number and duration of nighttime awakenings, and daytime napping duration. However, sleep stage assessment is not possible.

Patients are commonly instructed to wear the actigraph for at least 3 consecutive days, though longer periods of data collection (typically 1–3 weeks) are preferred. Actigraphs should be

continuously worn (i.e., 24 h a day), removing the actigraph only when it will be immersed in water, as most actigraphs are not completely waterproof. Daily sleep logs are often completed concurrent with actigraph wear, providing useful information on, among other things, when the watch was removed, times of daytime napping, the time at which sleep was first attempted (i.e., bedtime) and the time at which one got out of bed for the final time (i.e., rise time).

Although useful for evaluating the sleep/wake patterns of any individual presenting with a sleep complaint, actigraphy is especially useful for characterizing the sleep/wake patterns of insomniacs (Morgenthaler et al., 2007). Self-reported sleep patterns of insomniacs often show poor agreement with objective assessment, with substantial variability from night to night. Laboratory assessment of sleep often leads to reactivity (i.e., the "first night effect," reduced sleep due to the unfamiliar sleep environment), and multiple nights of laboratory sleep assessment would be burdensome and costly. Actigraphy is also valuable for tracking the sleep/wake patterns of individuals with circadian rhythm sleep disorders by documenting the altered timing of sleep/wake patterns.

Finally, actigraphy may serve as a valuable tool when assessing sleep-disordered breathing outside of the laboratory, which is usually performed with limited portable monitoring of respiratory and oximetry signals (Morgenthaler et al., 2007). Actigraphy has been shown to improve the estimate of total sleep time, which is used when calculating the severity of sleep-disordered breathing.

Validity

It is important to note that many actigraph devices are available for use, each utilizing different methods of movement detection and algorithms for estimation of sleep/wake status. Published validation information is not available for all actigraph algorithms, particularly across different populations (e.g., children, older adult insomniacs). Nevertheless, in general, actigraphy has been shown to provide valid estimates of total sleep time, sleep efficiency, and wakefulness after sleep onset in normal adults, with an epoch-by-epoch agreement of >80% between actigraphy and PSG for identification of sleep. Though epoch-by-epoch agreement for correctly identifying wake is low with actigraphy, whole-night averaged correlations are often strong (i.e., >.80) between measures of sleep via actigraphy versus PSG. Algorithm accuracy is reduced for individuals with significantly disturbed sleep (e.g., individuals with frequent awakenings and/or reduced total sleep time) and in populations in which there is substantial activity during sleep (e.g., children, adults with movement disorders) or minimal activity during wakefulness (e.g., insomniacs).

Despite the validity of actigraphy for estimating many common sleep parameters, there is much less validation support for the actigraphic estimation of daytime napping and sleep onset latency, particularly if there is no documentation (via event markers and/or daily log) of the timing of daytime napping or bedtime, respectively. Actigraphy often overestimates sleep during the day, as daytime periods of low waking activity are commonly estimated as sleep via actigraphic algorithms. Likewise, sleep onset latency is often underestimated with actigraphy, particularly if the patient is able to lie awake with minimal movement.

Because actigraphic devices differ in the mechanism by which movement data are collected, algorithms for sleep/wake estimation are device-specific. Furthermore, although the amount of activity during sleep may differ according to developmental stage and/or clinical morbidity (e.g., infants, individuals with sleep apnea), there have been minimal attempts to optimize algorithm accuracy for specific populations of individuals.

Advantages to Other Methods of Sleep Assessment

Laboratory PSG is considered the gold standard for objective sleep assessment. However, for assessment of sleep/wake patterns over multiple days and nights, PSG is often not feasible. In addition, participants often note that sleep is impaired during PSG due to the numerous wires, belts, and devices attached to the head and body during sleep. Therefore, actigraphy

may be a preferred alternative to PSG when large numbers of patients need to be studied, multiple nights of assessment are needed, daytime napping needs to be objectively assessed, and/or when PSG will not be easily tolerated (e.g., in children, insomniacs).

An exclusive reliance on sleep diaries for the evaluation of sleep/wake patterns is often not recommended, because sleep diary reports sometimes show significant divergence from actual sleep/wake patterns. Because actigraphy provides an objective assessment of sleep/wake activity, it is often used as a supplement or alternative to sleep diaries.

Limitations of Actigraphy

There are limits to the amount of information that actigraphy can provide. For instance, whereas PSG is able to provide detailed information on the duration and distribution of specific sleep stages (i.e., rapid eye movement (REM) sleep, stages 1–3 non-REM sleep), sleep stage assessment is not possible with actigraphy. In addition, actigraphy is limited in its ability to successfully identify wakefulness. This could explain the lack of validity for actigraphic assessment of sleep onset latency and pose problems for estimating the sleep of insomniacs and institutionalized adults who remain in bed during the day.

Due to different technologies and algorithms employed by different actigraph manufacturers, the accuracy of sleep/wake estimation can vary considerably across different actigraph models. Many algorithms have minimal to no published validation support. However, when available, it is important to note that actigraphic algorithms are device-, mode-, and (if validated) population-specific. Because there has been little standardization of actigraph models and algorithms across studies, clinicians and researchers should choose actigraphs that provide the desired technological features (e.g., light sensor, event marker) and whose algorithms have been validated in the population of interest.

Currently, there are no established guidelines on the scoring of actigraphy. As a result, considerable variation could occur between technicians and laboratories when evaluating actigraphic data. When scoring actigraphy, standard operating procedures should be developed, specifically addressing how to identify daytime nap periods, when the watch was removed, and the beginning and end of the nocturnal rest period. Use of ambient light data (when available) is helpful in identifying bedtime, since there is often a sharp decrease in light levels at this time. Furthermore, although sleep diaries are typically completed concurrent with actigraphy recording to inform the scorer of sleep/wake patterns, use of event markers should be encouraged to identify intervals of attempted sleep during the day and night. This is especially true if accurate determination of daytime napping is desired, since in some sedentary individuals multiple periods of low daytime activity could be interpreted as napping (Stone & Ancoli-Israel, 2011).

Alternative Uses of Actigraphy

Because actigraphy provides a view of multiple consecutive 24-h periods, the rhythmicity of the sleep/wake pattern can be assessed. Many actigraph software programs now allow for circadian analysis of the rest-activity rhythm. These analyses are particularly helpful in documenting circadian rhythm sleep disorders (e.g., shift work sleep disorder, advanced sleep phase disorder) (Stone & Ancoli-Israel, 2011).

Actigraphy is also able to provide a basic assessment of physical activity. Although not as sensitive as trunk placement for physical activity assessment, wrist actigraphy can estimate overall activity with adequate precision. Activity count per hour of wakefulness is the most commonly used metric, but maximal activity counts, peak activity over a predetermined duration (e.g., 10 min), or amount of time above a threshold of activity (e.g., 1,000 cpm) have been previously utilized.

Cross-References

- ▶ Insomnia
- ▶ Polysomnography
- ▶ Sleep
- ▶ Sleep and Health
- ▶ Sleep Apnea

- ▶ Sleep Continuity
- ▶ Sleep Duration
- ▶ Sleep Fragmentation
- ▶ Sleep Quality

References and Readings

Ancoli-Israel, S., Cole, R., Alessi, C., Chambers, M., Moorcroft, W., & Pollak, C. P. (2003). The role of actigraphy in the study of sleep and circadian rhythms. *Sleep, 26*, 342–392.

Martin, J. S., & Hakim, A. D. (2011). Wrist actigraphy. *Chest, 139*, 1514–1527.

Morgenthaler, T., Alessi, C., Friedman, L., Owens, J., Kapur, V., Boehlecke, B., et al. (2007). Practice parameters for the use of actigraphy in the assessment of sleep and sleep disorders: An update for 2007. *Sleep, 30*, 519–529.

Sadeh, A. (2011). The role and validity of actigraphy in sleep medicine: An update. *Sleep Medicine Reviews, 15*, 259–267.

Stone, K. L., & Ancoli-Israel, S. (2011). Actigraphy. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5th ed., pp. 1668–1675). St. Louis, MO: Elsevier.

Actimetry

▶ Actigraphy (Wrist, for Measuring Rest/Activity Patterns and Sleep)

Activation

▶ Affect Arousal

Active Coping

Linda Carroll
Department of Public Health Sciences, University of Alberta, Edmonton, AB, Canada

Synonyms

Adaptive coping; Constructive coping

Definition

Coping is the set of intentional, goal-directed efforts people engage in to minimize the physical, psychological, or social harm of an event or situation (Lazarus & Folkman, 1984; Lazarus, 1999). There are many different theoretical and empirical frameworks for understanding coping, and many different ways of classifying coping strategies, but one such classification is "active coping." In general, active coping refers to the utilization of those psychological or behavioral coping efforts that are characterized by an attempt to use one's own resources to deal with a problem situation (Zeidner & Endler, 1996). These responses are designed either to change the nature of the stressful situation or event in order to decrease the problematic nature of that situation or event, or to modify how one thinks and feels about it in order to change one's reactions to it. Examples include solving problems, reframing the meaning of the problems, or seeking information. Active coping is thought to be an adaptive way of dealing with stressful events and to be a vital component of resilience in the face of stress, health problems, and other adversity.

Cross-References

- ▶ Coping
- ▶ Problem-Focused Coping

References and Readings

Lazarus, R. S. (1999). *Stress and emotion: A new synthesis*. New York: Springer.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal and coping*. New York: Springer.

Zeidner, M., & Endler, N. S. (1996). *Handbook of coping: Theory, research, applications*. New York: Wiley.

Active Sleep

- ▶ REM Sleep

Active Way of Life

► Lifestyle, Active

Activities of Daily Life Assessment

► Health Assessment Questionnaire

Activities of Daily Living (ADL)

Jane Upton
School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Frailty assessment; Level of occupational performance; Physical ability/disability

Definition

An activities of daily living (ADL) evaluation is an assessment of an individual's physical and sometimes mental skills. In the area of physical or occupational therapy, it reflects how well a disabled patient or someone recovering from disease or accident can function in daily life. It is also used to determine how well patients relate to and participate in their environment (Krapp & Cengage, 2006). Common examples of ADL include personal hygiene and feeding oneself.

Cross-References

► Occupational Therapy

References and Readings

Krapp, K., & Cengage, G. (2006). Activities of daily living evaluation. In *Encyclopedia of nursing & allied health* (2002). Detroit, MI: Gale Group. eNotes.com. Retrieved from http://www.enotes.com/nursing-encyclopedia/activities-daily-living-evaluation

Activity Level

Patrícia Cardoso Buchain[1], Adriana Dias Barbosa Vizzotto[1], Alexandra Martini de Oliveira[2], Tania C. T. Ferraz Alves[3] and Quirino Cordeiro[4]
[1]Occupational Therapist of the Occupational Therapy Service, Institute of Psychiatry, Hospital das Clínicas University of São Paulo Medical School, São Paulo, SP, Brazil
[2]Institute of Psychiatry – Hospital das Clínicas University of São Paulo Medical School, São Paulo, SP, Brazil
[3]Department and Institute of Psychiatry, University of São Paulo Medical School, São Paulo, SP, Brazil
[4]Department of Psychiatry and Psychological Medicine, Santa Casa Medical School, São Paulo, SP, Brazil

Synonyms

Grade of activity

Definition

The level of activity is related to the complexity of the skills that demands and has an impact on an individual's occupational performances. This term is related to several characteristics that might influence the type and amount of effort required from the individual to perform a specific activity, task, or occupation. The term "activity level" is of great importance when designing occupational therapy interventions.

Description

When describing a person's activity level of daily living, it is possible to use the terms occupation and activity synonymously. However, these terms are not fully interchangeable as they describe two different aspects of the same function (Christiansen & Townsend, 2004; Hinojosa & Kramer, 1997). Occupation is the "active process of living: from the beginning to the end of life, . . . occupations are all the active processes of looking after ourselves and others, enjoying life, and being socially and economically productive over the lifespan and in various contexts" (Willard & Spackman's 2008). Activity is a fundamental aspect of human existence, and each activity is usually composed by several tasks to be performed. Different activities might be combined in one routine and contribute to different occupations. Activity synthesis is the integration of some or all of these performance components with an appropriate theory that is consistent with the client's goals and present status (Söderback, 2009).

Factors Influencing Activity Level

Individual's State of Flow

An individual's present activity level is influenced by the interaction between his or her present mental status and the challenge and skill level of an activity, according to Csikszentmihalyi's (1988) proposed theoretical model "Flow: The Psychology of Optimal Experience." There is a complex interaction between the emotional state, the environment, and the ability to perform a specific task. It is suggested that someone might be able to perform a task in one condition and not in another one because of the relation to different aspects, for example, environment, engagement, mood, that is, angst, arousal, flow, worry, control, apathy, boredom, and relaxation. When optimal conditions occur there is a state of flow that might allow people to feel in control of their environment and thus, able to perform a meaningful task. In this context, it is possible to find a person without any disability or mental declining health that due to anxiety or another negative feeling is unable to perform a specific task or fails to complete an assignment. In this case, the person is not per se unable to perform the tasks, but due to his or her own perception it turns into a failure. Here, some factors such as positive thinking, stable mood, and engagement might support a task toward a positive solution, and on the other hand, anxiety, negative thoughts, depression, and cognitive decline might prevent that the task is fulfilled.

Individual's Personal Factors

It is likely that an individual has a great variation in his or her activity level to perform different activities depending on his or her cognitive integrity, familiarity of the present environment, support from other people, and perception of the meaningfulness when an activity is performed (Pool, 2008). For example, in order to keep the person with cognitive impairment engaged in an activity, an enriched environment must be constructed and suitable degree of difficulty chosen to maintain flow and skills; thus, the activity is ended up with an expected product or performance.

Individual's Past Experiences

Level of engagement in an activity is related to the individual's past experiences (Kielhofner et al., 2004, 2008), which can be summarized in positive or negative thoughts and feelings regarding a specific task. An individual with a positive approach interprets difficulty as part of the situation. They will engage in correcting and improving their performances. On the other hand, an individual with a negative approach will experience difficulty as failure and therefore, stop the ongoing activity. The last aspect may be observed among people with depressive or mild cognitive impairment.

Individual's Dysfunctions

Cognitive functions and all mental processes that impair the symbolic operations, perception, and memory, creation of imagery, thinking,

reasoning, and judgment will affect how well activities are realized. Therefore, it is important to identify and assess how these cognitive functions influence the individual's activity level. These assessment's results may be helpful for therapists to guide an individual toward the most appropriate activity level he or she is able to be engaged in and is able to perform as expected (Baum, 1995; Söderback, 1988).

An important context for management of older adults with Alzheimer's disease (AD) activity level is proposed by Csikszentmihalyi (1988). The findings support the importance of keeping the person with AD engaged in occupational pursuits to sustain best functional level, and moderate, appropriate behavior and habits.

Generally, people should keep themselves actively engaged; otherwise disharmony or strain between individual and environment will arise, resulting in negative stress (Baum & Edwards, 1993).

Conclusion

Activity is an essential component of human existence. The activity level is essential to be diagnosed, evaluated, and properly treated among individuals who show a reduced activity level, due to medical diagnosis, present life situation, personality, or inappropriate present environment. In this sense, it is fundamental for therapists and health-care providers to be well prepared and having appropriate knowledge, for example, in medicine, occupational therapy and psychology to meet the challenge of upholding requested activity level.

Cross-References

► Activities of Daily Living (ADL)

References and Readings

Baum, C. M. (1995). The contribution of occupation to function in persons with Alzheimer's disease. *Journal of Occupational Science: Australia, 2*(2), 59–67.

Baum, C. M., & Edwards, D. F. (1993). Cognitive performance in senile dementia of the Alzheimer's type: The kitchen task – American assessment. *The American Journal of Occupational Therapy, 47*(5), 431–436.

Blesedell, C. E., Cohn, E. S., & Boyt S. A. (2008). *Willard and Spackman's Occupational Therapy* (11 ed.). Boston: Lippincott Williams & Wilkins.

Christiansen, C. H., & Townsend, E. A. (Eds.). (2004). *Introduction to occupation: The art and science of living*. Upper Saddle River, NJ: Prentice Hall.

Csikszentmihalyi, M. (1988). *A theoretical model for enjoyment. Beyond boredom and anxiety* (pp. 1–231). San Francisco: Jossey-Bass. 1-?.

Fillenbaum, G. G., Dellinger, D., Maddox, G., & Pfieffer, E. (1978). Assessment of individual functional status in a program evaluation and resource allocation model. In *Multidimensional functional assessment: The OARS methodology* (2nd ed.). Durham, NC: Duke University, Center for the Study of Aging and Human Development.

Hinojosa, J., & Kramer, P. (1997). Fundamental concepts of occupational therapy: Occupation, purposeful activity, and function [Statement]. *The American Journal of Occupational Therapy, 51*(10), 864–866.

Jefferson, A. L., Robert, H. P., Ozonoff, A., & Cohen, R. A. (2006). Evaluating elements of executive functioning as predictors of instrumental activities of daily living (IADLs). *Archives of Clinical Neuropsychology, 21*(2006), 311–332.

Kielhofner, G., Mallinson, T., Crawford, C., Nowak, M., Rigy, M. Henry, A., et al., (2008). Occupational performance history interview II (OPHI-II) Version 2.1. In: Kielhofner, G. *The model of human occupation: Theory and application*. Philadelphia: Lippincot, Williams & Wilkins. Retrieved July 05, 2011, http://www.uic.edu/depts/moho/assess/ophi%202.1.html

Pool, J. (2008). *The pool activity level (PAL) instrument for occupational profiling* (pp. 1–173). Philadelphia: Jessica Kingsley.

Rivlin, A. M., & Wiener, J. M. (1988). *Caring for the disabled elderly: Who will pay?* (pp. 1–318). Washington, D.C: Brookings Institution Press.

Söderback, I., (1988). Intellectual function training and intellectual housework training in patients with acquired brain damage. A study of occupational therapy methods. (Dissertation from Department of Rehabilitation Medicine, Danderyd Hospital; The Department of Social Care and Rehabilitation, Stockholm College of Health and Caring Sciences; Department of Physical Medicine and Rehabilitation, Karolinska Institute: Stockholm 1988). 1–55.

Söderback, I. (Ed.). (2009). *International handbook of occupational therapy interventions* (pp. 1–553). Dordrecht/London: Springer.

Spector, W. D., Katz, S., Murphy, J. B., & Fulton, J. P. (1987). The hierarchical relationship between activities of daily living and instrumental abilities. *Journal of Chronic Diseases, 40*(6), 481–489.

Activity Limitations

▶ Disability

Activity Monitor

▶ Actigraphy (Wrist, for Measuring Rest/Activity Patterns and Sleep)

Acts of Commission

▶ Child Abuse

Acupressure

▶ Acupuncture

Acupuncture

Lorenzo Cohen and M. Kay Garcia
Department of General Oncology, Division of Cancer Medicine, The University of Texas MD Anderson Cancer Center, Houston, TX, USA

Synonyms

Acupressure; Traditional Chinese medicine

Definition

Acupuncture is a traditional therapy used to treat a variety of health-related problems. It involves assessment of the condition and development of a differential diagnosis followed by the insertion and manipulation of thin, solid metal needles at specific locations on the body. Various techniques are used to stimulate the needles and improve the therapeutic effects, including manual manipulation or adding a mild electrical current.

There are many different types of acupuncture. For example, auricular acupuncture involves placement of stainless steel or gold (semipermanent) needles, or "studs" at specific points on the ears. These are often left in place for 3–5 days. Other treatments such as acupressure (applying pressure or massaging acupoints), *gua sha* (scraping), moxibustion, and cupping are also used as adjuncts to acupuncture therapy. Moxibustion is a technique for providing heat to the needles. It involves placement of a special herb onto the needle and igniting it to bring mild warmth to the local area. Cupping involves the creation of a negative vacuum in a jar that is placed onto the skin surface. The negative pressure causes local congestion and vasodilation and may be used to help relieve pain (Deng et al., 1997).

Description

Traditional Chinese medicine (TCM) is a complete system of healthcare delivery used for the prevention and treatment of a wide-range of health-related conditions. The therapeutic approaches of TCM include acupuncture, herbs, food therapy, *tui na* (bodywork), *tai chi* (therapeutic exercise), and *qi gong* (meditative/energy therapy). Acupuncture originated in China over 2,500 years ago and remains one of the most popular therapies within TCM today. According to the World Health Organization, it is used in over 70 countries worldwide (World Health Organization, 2002). There has been documented use of acupuncture in the United States for approximately 200 years, and in 1996, the United States Food and Drug Administration approved acupuncture needles as medical devices (US Food and Drug Administration, Department of Health and Human Services, 1996).

Acupuncture treatments are generally considered safe. Commonly reported risks include fainting, bruising, infection, and mild discomfort. Serious adverse events are rare when treatments are provided by a qualified practitioner. Licensing requirements for acupuncturists vary. Some

countries do not require a license to practice, but in the United States, training programs have a standardized, clinically based curriculum and are formally accredited by the Accreditation Commission for Acupuncture & Oriental Medicine (Accreditation Commission for Acupuncture and Oriental Medicine). The National Certification Commission for Acupuncture and Oriental Medicine (NCCAOM), a nonprofit organization established in 1982, also promotes nationally recognized standards of competence and safety by examining and certifying individuals through national board examinations. Most states require NCCAOM certification in order to obtain a license to practice acupuncture (National Certification Commission for Acupuncture and Oriental Medicine (NCCAOM)).

Acupuncturists use many different models and approaches to understand and apply treatment. These models range from a metaphysical paradigm used by those traditionally trained to a strictly neurophysiologic approach incorporated into pain control regimens. According to ancient theory, acupuncture is based on the belief that energy flows through the body in channels known as meridians. This energy is also referred to as Qi (pronounced chee). A block in the meridians denies the surrounding tissues of Qi and creates an imbalance of health. Qi flow can be restored by inserting needles at specific locations on the body. With restored Qi, imbalances in absorption of nutrients and circulation of blood and fluids to the body's organs can be corrected (Deng et al., 1997).

Although acupuncture is used to treat a wide variety of health problems, human data from rigorous randomized-controlled trials is limited. The most compelling evidence supporting its use is for management of nausea/vomiting and pain. Based on more limited research, there is some evidence suggesting that acupuncture may be useful for stimulating the immune system and for reducing hot flashes, xerostomia (chronic dry mouth), fatigue, mood disorders, sleep disturbances, and peripheral neuropathy (National Institutes of Health (NIH) & National Center for Complementary and Alternative Medicine (NCCAM); National Institutes of Health (NIH) & National Cancer Institute (NCI)).

How acupuncture works is not well understood, but laboratory, animal, and human studies have attempted to differentiate the multiple putative mechanisms involved. In the late 1970s and early 1980s, researchers demonstrated acupuncture analgesia was associated with the stimulation of endogenous opioid peptides and biogenic amines through the central nervous system (Helms, 1997). Although these findings helped give acupuncture scientific credibility, rigorous and systematic research is needed to make clear recommendations as specific mechanisms may be dependent upon the symptom being treated, which point is stimulated, and the type of stimulation used.

Cross-References

- Alternative Medicine
- Complementary and Alternative Medicine
- Integrative Medicine

References and Readings

Accreditation Commission for Acupuncture and Oriental Medicine (ACAOM). Laurel, MD. Accessed September 7, 2011, from http://acaom.org/

Deng, L., Gan, Y., He, S., et al. (1997). Acupuncture techniques. In Y. Cheng (Ed.), *Chinese acupuncture and moxibustion*. Beijing: Foreign Languages Press.

Helms, J. M. (1997). *Acupuncture energetics: A clinical approach for physicians*. Berkeley, CA: Medical Acupuncture.

National Certification Commission for Acupuncture and Oriental Medicine (NCCAOM). Jacksonville, Fl. Accessed September 7, 2011, from http://www.nccaom.org/

National Institutes of Health (NIH) & National Cancer Institute (NCI). Acupuncture PDQ. Bethesda, MD. Accessed September 7, 2011, from http://www.cancer.gov/

National Institutes of Health (NIH) & National Center for Complementary and Alternative Medicine (NCCAM) Bethesda, MD. Accessed September 7, 2011 from http://nccam.nih.gov/

US Food and Drug Administration, Department of Health and Human Services. *Code of federal regulations (21CFR880.5580)*. [61FR 64617, December 6, 1996, revised April 1, 2011].

World Health Organization. (2002). *WHO traditional medicine strategy 2002–2005*. Geneva: World Health Organization.

Acute Care

► Acute Disease

Acute Condition

► Acute Disease

Acute Coronary Syndrome

► Acute Myocardial Infarction

Acute Disease

Amy Jo Marcano-Reik
Department of Bioethics, Cleveland Clinic, Cleveland, OH, USA
Center for Genetic Research Ethics and Law, Case Western Reserve University, Cleveland, OH, USA

Synonyms

Acute care; Acute condition; Acute illness; Acute infection

Definition

Acute diseases tend to have very quick onsets and typically last for only a brief period. By defining a disease as an acute disease, it does not necessarily address the severity of the disease. In fact, it typically only refers to the length of the disease or illness. Acute diseases, as opposed to chronic diseases, include a very rapid onset and/or a short course. Acute diseases can occur throughout all bodily systems. Examples of acute diseases include appendicitis, acute leukemia, and strep throat. Some acute diseases do not require hospitalization or medical treatments, such as influenza, whereas others, such as pneumonia and acute myocardial infarction, may require medical attention and extended treatment.

Cross-References

► Disease Onset

References and Readings

Fisher, S. R., Goodwin, J. S., Protas, E. J., Kuo, Y-F., Graham, J. E., Ottenbacher, K. J., & Ostir, G. V. (2010). ERRATUM. Ambulatory activity of older adults hospitalized with acute medical illness. *Journal of the American Geriatrics Society, 59*(4), 777.

Knaus, W. A., Draper, E. A., Wagner, D. P., & Zimmerman, J. E. (1985). Apache II: A severity of disease classification system. *Critical Care Medicine, 13*(10), 818–829.

Knaus, W. A., Zimmerman, J. E., Wagner, D. P., Draper, E. A., Elizabeth, A., & Lawrence, D. E. (1981). Apache: Acute physiology and chronic health evaluation: A physiologically based classification system. *Critical Care Medicine, 9*(8), 591–597.

Acute Illness

► Acute Disease

Acute Infection

► Acute Disease

Acute Myocardial Infarction

Siqin Ye
Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Synonyms

Acute coronary syndrome; AMI; Heart attack; MI; Non-Q wave myocardial infarction; NSTEMI; Q wave myocardial infarction; STEMI

Definition

Acute myocardial infarction (AMI), also known as heart attack or acute coronary syndrome (ACS), is a clinical condition that occurs when blood flow to regions of the heart is suddenly interrupted, causing myocardial ischemia and eventually cell death. Most commonly, this is caused by coronary atherosclerosis and is initiated by the rupture or erosion of a complex, lipid-laden plaque that then triggers thrombus formation, causing the total or subtotal occlusion of the coronary artery in question. Rarely, non-atherosclerotic processes such as vasospasm, vasculitis, and spontaneous coronary artery dissection may also lead to myocardial infarction (Antman & Braunwald, 2008).

Description

Acute myocardial infarction is further classified into ST elevation myocardial infarction (STEMI) and non-ST elevation myocardial infarction (NSTEMI), depending on the presence of ST segment elevation on the 12-lead electrocardiogram (ECG). ST segment elevation is typically associated with thrombi that are completely occlusive, leading to a large zone of infarction involving the full or nearly full thickness of the affected portion of the ventricle. In the absence of prompt revascularization, most patients will go on to develop pathological Q waves, and the term Q wave myocardial infarction is sometimes used interchangeably with STEMI in the past (Antman & Braunwald, 2008). NSTEMIs, also referred to as non-Q wave myocardial infarction, are thought to be due to thrombi that cause subtotal occlusions severe enough to lead to myocardial necrosis. The term unstable angina (UA) is used to refer to situations in which cell deaths do not occur despite the presence of subtotally occlusive thrombi and clinical symptoms of ischemia such as chest pain (Cannon & Braunwald, 2008). These three entities, STEMI, NSTEMI, and UA, together constitute the spectrum of acute coronary syndrome, a clinically important concept that is the foundation of current diagnostic and management pathways.

Despite recent declines, acute myocardial infarction remains a significant public health burden in the United States, affecting as many as 785, 000 Americans in 2010 (American Heart Association Statistics Committee, 2010). This is in part due to the high burden of risk factors such as hypertension, hyperlipidemia, diabetes mellitus, tobacco smoking, obesity, and physical inactivity in the general population. Patients with acute myocardial infarction typically present with chest pain or pressure radiating to the arm or jaw that is worse with exertion and is associated with shortness of breath, nausea, vomiting, or diaphoresis. Elderly patients and diabetics can often present with atypical symptoms. Upon presentation, timely performance of ECG is essential to determine the presence of ST segment changes, and biomarkers such as cardiac troponins or the muscle–brain (MB) fraction of creatine kinase (CK) are used to confirm myocardial necrosis. Noninvasive imaging modalities such as stress echocardiography or nuclear stress testing can be helpful in equivocal cases (Cannon & Lee, 2008). In order to standardize case definition for both clinical practice and research, current guidelines have recommended that at least two of the following three criteria be met for the diagnosis of acute myocardial infarction: characteristic symptoms, ECG changes, and a typical rise and fall of biomarkers (Thygesen, Alpert, & White, 2007).

For patients with STEMI, the cornerstone of care is timely reperfusion therapy through percutaneous coronary intervention (PCI) or fibrinolysis, which has been shown to improve survival in multiple studies. In the absence of contraindications, it is recommended that patients undergo emergent PCI within 90 min of presentation or receive fibrinolytics within 30 min of arrival in settings where PCI is not available or will be delayed (Antman, 2008). For patients with NSTEMI, an early invasive strategy utilizing PCI is recommended for those with high-risk features such as positive biomarkers or significant ST segment changes (Cannon & Braunwald, 2008). Regardless of the type of myocardial infarction, occasionally, urgent or emergent coronary artery bypass grafting (CABG) may be

required depending on findings on coronary angiography. In terms of pharmacological therapy, both STEMI and NSTEMI patients have been shown to benefit from aspirin, antiplatelet agents such as clopidogrel, beta blockers, statins, ace inhibitors and angiotensin receptor blockers, and anticoagulants such as heparin, low-molecular-weight heparin, and bivalirudin. Nitrates and morphine are often used for symptom relief but have not been shown to improve survival (Anderson et al., 2007; Kushner et al., 2009). Despite these therapeutic advances, myocardial infarction is the cause of death for more than 140,000 Americans annually (American Heart Association Statistics Committee, 2010).

Patients who survive the initial episode of myocardial infarction are also at risk for a number of complications, including heart failure, ventricular free wall rupture or ventricular septal defect, ventricular tachyarrhythmias, left ventricular thrombus, and recurrent myocardial infarction. Careful follow-up and adherence to the prescribed medical regimen is essential for secondary prevention. In addition, lifestyle changes such as smoking cessation, regular exercise, weight loss, and dietary modifications have been shown to be beneficial, but substantial challenges remain in motivating patients to maintain healthy behavioral changes over time. Of note, psychosocial factors such as depression and poor social support have also been shown to be independent risk factors for major adverse cardiovascular events after myocardial infarction, but thus far there is only limited evidence that interventions targeting these can reduce adverse outcomes (Anderson et al., 2007; Kushner et al., 2009).

Cross-References

▶ Coronary Heart Disease

References and Readings

American Heart Association Statistics Committee and Stroke Statistics Subcommittee. (2010). Heart disease and stroke statistics 2010 update: A report from the American heart association. *Circulation, 121*, e46–e215.

Anderson, J. L., Adams, C. D., Antman, E. M., Bridges, C. R., Califf, R. M., Casey, D. E., Jr., et al. (2007). ACC/AHA 2007 guidelines for the management of patients with unstable angina/non-ST-elevation myocardial infarction: A Report of the American College of Cardiology/American Heart Association task force on practice guidelines (writing committee to revise the 2002 guidelines for the management of patients with unstable angina/non-ST-elevation myocardial infarction): Developed in collaboration with the American College of Emergency Physicians, American College of Physicians, Society for Academic Emergency Medicine, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. *Journal of the American College of Cardiology, 50*, e1–e157.

Antman, E. M. (2008). ST-elevation myocardial infarction: Management. In P. Libby, R. O. Bonow, D. L. Mann, D. P. Zipes, & E. Braunwald (Eds.), *Braunwald's heart disease: A textbook of cardiovascular medicine* (pp. 1233–1299). Philadelphia, PA: Saunders Elsevier.

Antman, E. M., & Braunwald, E. (2008). ST-elevation myocardial infarction: Pathology, pathophysiology, and clinical features. In P. Libby, R. O. Bonow, D. L. Mann, D. P. Zipes, & E. Braunwald (Eds.), *Braunwald's heart disease: A textbook of cardiovascular medicine* (pp. 1207–1232). Philadelphia, PA: Saunders Elsevier.

Cannon, C. P., & Braunwald, E. (2008). Unstable angina and non-ST elevation myocardial infarction. In P. Libby, R. O. Bonow, D. L. Mann, D. P. Zipes, & E. Braunwald (Eds.), *Braunwald's heart disease: A textbook of cardiovascular medicine* (pp. 319–1351). Philadelphia, PA: Saunders Elsevier.

Cannon, C. P., & Lee, T. H. (2008). Approach to the patient with chest pain. In P. Libby, R. O. Bonow, D. L. Mann, D. P. Zipes, & E. Braunwald (Eds.), *Braunwald's heart disease: A textbook of cardiovascular medicine* (pp. 1195–1205). Philadelphia, PA: Saunders Elsevier.

Kushner, F. G., Hand, M., Smith, S. C., Jr., King, S. B., 3rd, Anderson, J. L., Antman, E. M., et al. (2009). 2009 focused updates: ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction (updating the 2004 guideline and 2007 focused update) and ACC/AHA/SCAI guidelines on percutaneous coronary intervention (updating the 2005 guideline and 2007 focused update): A report of the American College of Cardiology Foundation/American Heart Association Task Force on practice guidelines. *Circulation, 120*, 2271–2306.

Thygesen, K., Alpert J. S., White H. D., on behalf of the Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction (2007). Universal definition of myocardial infarction. *European Heart Journal*, *28*(20), 2525–2538.

Acute Phase Proteins

▶ C-Reactive Protein (CRP)

Adaptation

▶ Resilience

Adaptive Coping

▶ Active Coping

Addiction

▶ Addictive Behaviors

Addiction Rehabilitation

▶ Substance Abuse: Treatment

Addictive Behaviors

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Synonyms

Addiction

Definition

Addictive behaviors (AB) are a cluster of persistent compulsive behaviors which people feel an uncontrolled urge to perform, without which they fear experiencing loss of control or a very negative outcome. The American Psychiatric Association defines substance dependence as: "When an individual persists in use of alcohol or other drugs despite problems related to use of the substance, substance dependence may be diagnosed. Compulsive and repetitive use may result in tolerance to the effect of the drug and withdrawal symptoms when use is reduced or stopped. This, along with Substance Abuse are considered Substance Use Disorders. . .." (DSM-IV & DSM-IV-TR: Substance Dependence).

Description

AB include smoking, ingesting street and medical drugs, alcohol consumption, or even other normally healthy activities such as sports, among other behaviors. There is often comorbidity between AB and mental health problems. In the AB, there is a repetitive form of behavior lasting various periods of time from perhaps months to years, and an eventual dependence on the sought material or activity. The prevalence of AB varies according to the type of AB, gender, age group, and geographic region. For example, a national survey conducted in the USA in over 43,000 people found a lifetime prevalence of alcohol dependence of 12.5% (Hasin, Stinson, Ogburn, & Grant, 2007). Among 12th grade American students, 6.1% take marijuana daily (National Institute of Drug Abuse). When researching risk factors of AB, one needs to distinguish between abuse of certain agents (e.g., drug abuse) and dependence on such agents, resulting in AB. Risk factors of AB vary between ages and genders. In youth, these can include peer pressure and parental behavior (in favor and against). In adults, unmarried people, men, and low income are among the socioeconomic correlates of alcohol dependence (Hasin et al., 2007). A review of studies on life events and alcohol found that the type of event mattered – while health and financial problems predicted reduced alcohol intake, life events related to the spouse, friends, or retiring predicted increases in alcohol consumption in longitudinal studies

(Veenstra et al., 2006). Overattention to environmental cues associated with alcohol in alcoholics, as measured by the emotional stroop test, was found in another study to be associated with alcoholism (Lusher, Chandler, & Ball, 2004). Among the different mechanisms proposed to account for AB are operant conditioning – the consumed material produces a feeling of reward, pleasure, or reduction in distress, all which serve as reinforcements and eventually form the basis for the dependence on the consumed material and the AB. Neural mechanisms underlying AB include activation of dopamine-rich regions in the brain. One study found that the mesencephalon, a dopamine-rich region, showed higher activity specifically in response to drug-related words in cocaine users than in healthy controls (Goldstein et al., 2009). Thus, AB is a severe public health problem worldwide, with multiple risk factors spanning from socioeconomic, perceptual, environmental, and biological, hence requiring a multilevel type of intervention.

Cross-References

► Alcohol Abuse and Dependence

References and Readings

Goldstein, R. Z., Tomasi, D., Alia-Klein, N., Honorio Carrillo, J., Maloney, T., Woicik, P. A., Wang, R., Telang, F., & Volkow, N. D. (2009). Dopaminergic response to drug words in cocaine addiction. *Journal of Neuroscience, 29*, 6001–6006.

Hasin, D. S., Stinson, F. S., Ogburn, E., & Grant, B. F. (2007). Prevalence, correlates, disability, and comorbidity of DSM-IV alcohol abuse and dependence in the United States: Results from the national epidemiologic survey on alcohol and related conditions. *Archives of General Psychiatry, 64*, 830–842.

Lusher, J., Chandler, C., & Ball, D. (2004). Alcohol dependence and the alcohol Stroop paradigm: Evidence and issues. *Drug and Alcohol Dependence, 75*, 225–231.

Veenstra, M. Y., Lemmens, P. H., Friesema, I. H., Garretsen, H. F., Knottnerus, J. A., & Zwietering, P. J. (2006). A literature overview of the relationship between life-events and alcohol use in the general population. *Alcohol and Alcoholism, 41*, 455–463.

Additional or Assisting Medication

► Adjuvant Chemotherapy

Ader, Robert

Marc D. Gellman
Behavioral Medicine Research Center, Department of Psychology, University of Miami, Miami, FL, USA

Biographical Information

Robert Ader was born in 1932. He was a native of the Bronx, New York, and a graduate of Tulane University. He received his Ph.D. at Cornell University. He then joined the faculty at the University of Rochester Medical Center and quickly rose through the ranks, becoming a professor of psychiatry and psychology in 1968. He held numerous positions and titles during his tenure at the University of Rochester, including the George Engel Professor of Psychosocial Medicine and Distinguished University Professor. He retired in July 2011 as professor emeritus. He had received an honorary doctor of science degree from Tulane and an honorary medical degree from Trondheim University in Norway.

Major Accomplishments

Ader coined the word psychoneuroimmunology to describe the field of study he helped create.

He was editor and later co-editor of the book *Psychoneuroimmunology*, first published in 1981, that details the research supporting the proposition that the brain and immune system are an integrated system. He was the founder and past president of the Psychoneuroimmunology Research Society, and also past president of the Academy of Behavioral Medicine Research and the American Psychosomatic Society. He launched the journal *Brain, Behavior and Immunity*, and in 2002, the Psychoneuroimmunology Research Society created an award, the Robert Ader New Investigator Award, to be given to promising young scientists.

His theories that the human mind could significantly affect the ability of the immune system to fight disease were initially greeted with heated skepticism and sometimes scorn when he proposed them more than 30 years ago. However, they are now applied and studied in many medical specialties, as well as by researchers around the world.

In the early 1970s, in what would become one of his most distinctive experiments, Ader was studying taste aversion conditioning in rats. In the experiment, rats drank different volumes of a saccharin solution and also were injected with a dose of Cytoxan, an immunosuppressive drug that induces gastrointestinal upset. The rats "learned," or were conditioned, to avoid consuming the solution. When he stopped giving the rats the drug but continued to give them the saccharin solution, not only did the rats avoid drinking the solution but also some of the animals died. The magnitude of the avoidance response of the rats was directly related to the volume of solution consumed. Additionally, the mortality rate varied with the amount of solution consumed. Ader believed that this orderly relationship could not be due to chance. It was through these experiments that he discovered that the rat immune system can be conditioned to respond to external stimuli. This was one of the first scientific experiments that demonstrated that the nervous system can affect the immune system.

In an interview conducted in 2010 that appeared in the newsletter of the American Institute of Stress, Ader commented as follows: "As a psychologist, I was unaware that there were no connections between the brain and the immune system so I was free to consider any possibility that might explain this orderly relationship between the magnitude of the conditioned response and the rate of mortality. A hypothesis that seemed reasonable to me was that, in addition to conditioning the avoidance response, we were conditioning the immunosuppressive effects (of Cytoxan). It seems to me that basic research on the interactions among behavior, neuroendocrine and immune processes has a bright future that promises new developments in our understanding of adaptive processes with profound consequences for the maintenance of health and for the treatment of disease."

This hypothesis was tested and confirmed in a classic study employing deliberately immunized animals, the results of which were published in 1975 in the journal *Psychosomatic Medicine*. Conditioning is one form of learning, and, as such, involves the higher centers of the brain. Ader's study, clearly demonstrating that immune responses could be modified by classical conditioning, meant there were connections between the brain and the immune system and that the mind could have profound effects on the body's functions that were thought to be independent.

In his paper published in 2010 in the journal *Psychosomatic Medicine*, Ader and his fellow Medical Center researchers described the use of a placebo effect to successfully treat psoriasis patients with a quarter to a half of the usual dose of a widely used steroid medication. Early results in human patients suggest that this new technique could improve treatment for several chronic diseases that involve mental state or the immune system. Following publication of this paper, Ader observed that "Our study provides evidence that the placebo effect can make possible the treatment of psoriasis with an amount of drug that should be too small to work. . ..While these results are preliminary, we believe the medical establishment needs to recognize the mind's reaction to medication as a powerful part of many drug effects, and start taking advantage of it."

Editors' Note: Dr. Ader passed away in 2011.

Cross-References

- ▶ Behavioral Immunology
- ▶ Immune function
- ▶ Immune Responses to Stress
- ▶ Neuroimmunology
- ▶ Neuroimmunomodulation
- ▶ Psychoneuroimmunology

References and Readings

Ader, R. (2003). Conditioned immunomodulation: Research needs and directions. *Brain, Behavior, and Immunity, 17*(Suppl. 1), 51–57.

Ader, R., & Cohen, N. (1982). Behaviorally conditioned immunosuppression and murine systemic lupus erythematosus. *Science, 215*, 1534–1536.

Ader, R., Felten, D. L., & Cohen, N. (2006). *Psychoneuroimmunology, Vol. 1–2* (4th ed.). Burlington, MA: Academic Press. ISBN 0-12-088576-X.

Ader, R., Mercurio, M. G., Walton, J., James, D., Davis, M., Ojha, V., et al. (2010). Conditioned pharmacotherapeutic effects: A preliminary study. *Psychosomatic Medicine, 72*, 192–197.

Adherence

M. Bryant Howren
Department of Psychology, The University of Iowa & VA Iowa City Healthcare System, Iowa City, IA, USA

Synonyms

Patient compliance

Definition

Adherence is a term used to describe the extent to which an individual's behavior coincides with health-related instructions or recommendations given by a health care provider in the context of a specific disease or disorder. The term has been used extensively in psychology and medicine in reference to acute, chronic, and preventive treatment regimens (e.g., a course of prescribed medication, wound self-care), preventive health screenings, dietary restriction, exercise recommendations, smoking cessation, and other health behaviors. Although *adherence* is synonymous with *compliance* in many contexts, the former is often preferred by behavioral scientists and allied health professionals given its emphasis of patient-provider collaboration as opposed to a more authoritarian, provider-centered exchange.

Description

Extent and Implications of Nonadherence

Despite significant advances in biomedical science related to the treatment of disease, the problem of nonadherence remains pervasive. Although estimates of nonadherence vary considerably as a function of (a) the length and complexity of the treatment regimen, (b) setting and population, and (c) assessment method, data suggest that rates of nonadherence range from 20% to 80%. From an economic standpoint, medication nonadherence has been estimated to cost upwards of $300 billion annually in avoidable medical spending in the USA alone, a figure that does not include the cost implications of nonadherence to all health care regimens. In addition to the enormous monetary cost, nonadherence also has implications for the efficacy and effectiveness of medical treatment regimens. Specifically, not only has patient nonadherence been linked to treatment failures but it also may directly undermine future treatment efficacy due to its contribution to the development of drug-resistant disease strains as well as complications regarding the establishment of empirically based guidelines for medication usage.

Assessing Adherence

Numerous strategies for assessing adherence behavior have been employed by researchers and clinicians alike. These include patient self-report, clinician judgment, clinical/health outcomes, medication measurement, electronic medication monitors, computerized pharmacy

records, biological indicators, and directly observed therapy. Each strategy is reviewed here in turn.

Patient Self-Report

Subjective patient reports remain the most widely used assessment method largely because they are quick, inexpensive, and can be administered by persons with little or no technical expertise. Furthermore, self-report may be the only method of ascertaining patient attitudes and experiences related to a specific medication regimen. For example, a patient choosing to split dosages in order to share medication with a significant other will not necessarily be identified as nonadherent through objective assessment methods (reviewed below). Despite these benefits, however, evidence indicates that patients significantly *underestimate* rates of nonadherence. In addition to memory biases inherent in retrospective recall of behavioral events, patients may be hesitant to report nonadherent behavior to health care providers or behavioral scientists for fear of rebuke.

Recognizing the limitations associated with patient self-report – particularly when requested over an extended period, such as days or weeks – many have advocated the use of *ecological momentary assessment* (EMA), a method in which patients are asked to report about their behavior over several, discrete points in time. Because EMA captures events in "real time," it minimizes recall biases; however, it is important to note that EMA does require considerably greater patient burden and still may be subject to certain methodological issues, such as poor adherence to the self-assessment protocol itself.

Clinician Judgment

Similar to evidence regarding patient self-report, health care providers also significantly underestimate rates of nonadherence in their patients. Evidence suggests that less experienced providers are the least likely to recognize nonadherence, but identification of such patients is difficult for providers at all levels and specialties. This may be due, in part, to stereotypes about what constitutes an adherent patient including gender and race, attributes which are not consistently related to adherence behavior. Collectively, this overall lack of provider awareness of patient nonadherence has clinical implications regarding adjustments to patient treatment regimens as well as the frequency and aggressiveness of patient follow-up over time.

Clinical Outcomes

Adherence researchers have also utilized clinical outcomes – such as infection resolution or changes in blood pressure – as proxies of treatment adherence. These measurements must be interpreted with caution, however. Evidence indicates that poor physical health is significantly predictive of treatment nonadherence, clouding any interpretation of an association between clinical outcomes and adherence behavior. In addition, the extent to which many clinical and health outcomes are related to adherence behavior either varies considerably or is largely unknown, further complicating the use of such measures as indicators of adherence.

Medication Measurement

Medication measurement methods, such as pill counts or the weighing of liquid medication, represent a straightforward, objective way of estimating adherence. Much like self-report methods, however, medication measurement is subject to several limitations. In particular, pill counts have been repeatedly shown to be an insensitive index of nonadherence. One possible explanation is the phenomenon of medication "dumping" in which patients choose to discard unused medication for fear of appearing nonadherent. Patients may also share medications with family members on the same (or similar) treatment regimen. Even if patients are not actively engaging in deception or sharing, medication measurement methods are further limited because they provide no indication of regimen fidelity (i.e., whether the medication, if ingested, was taken as directed).

Electronic Medication Monitors

A more advanced method of tracking medication usage is through the use of electronic medication monitors. In contrast to more simplistic

medication measurement – such as pill counts – electronic monitoring allows a clinician or researcher to capture some information regarding regimen fidelity. For example, one of the most widely used systems is the Medication Event Monitoring System (MEMS), which may be used with any regimen consisting of pills or capsules. The MEMS "Track Cap" records the date and time of each cap removal, which is stored in a small chip affixed to the bottle and may later be downloaded to a computer for analysis. Evidence indicates that systems like MEMS are a more sensitive measure of adherence when compared to other measurement methods including clinician judgment, patient self-report, and pill counts. Although electronic monitoring methods may alleviate some concerns associated with other assessment strategies, they are not without limitations. Much like pill counts, electronic methods also are tied to the assumption that medication removal may be equated with medication ingestion.

Computerized Pharmacy Records

Behavioral scientists have developed methods that utilize prescription refill records from computerized pharmacy databases to obtain estimates of adherence. These methods, often referred to as *refill compliance* (RC) measures, ascertain the timing of medication refills and calculate the percentage of time patients have a necessary supply of medication(s) during a specific time frame. RC estimates utilize a considerable amount of information including the drug name, dosage, quantity dispensed, and date of refill; thus, RC estimates may be computed for individual medications, specific classes of medications, and/or across all prescription agents taken by a particular patient.

Much like the methods already reviewed, RC estimates, too, are limited. For example, while a patient may obtain prescription refills on a regular basis, RC measures do not provide information as to whether a patient takes the drug(s) as directed. Furthermore, abrupt, provider-directed changes to a patient's regimen may not be accurately captured. Lastly, RC methods are not useful for estimating adherence to short-term or discretionary treatments, such as a brief course of antibiotics or prescription analgesics used "as needed." On balance, however, increasing evidence supports the validity of RC methods with strong associations reported between pharmacy records and other measures of adherence including medication measurement, biochemical assays, and other clinical outcomes.

Biological Indicators

Clinical analyses, such as biochemical assays and other laboratory tests, may be used to estimate adherence through measurement of medication, metabolites, or drug tracers in serum or urine. Such methods are free of subjective biases, but may be limited in several other ways. Biochemical assays are, at present, only available for a limited number of patient drugs and are influenced by individual differences in drug metabolism. Moreover, these methods are often quite costly, precluding their use in routine clinical care. Lastly, even biological indicators may be compromised if a patient alters adherence behavior close to the time of analysis.

One example of an oft-used, widely available laboratory test is the hemoglobin A1C assay (a.k.a., glycosylated hemoglobin), a reliable and valid clinical indicator of glycemic control in diabetic patients. Because diabetic patients must adhere to a complex self-care regimen in order to maintain blood glucose control (e.g., insulin injections, restricted diet, exercise, frequent blood glucose testing), self-reported adherence may be especially biased and/or difficult to capture given the array of relevant behaviors to be measured. The hemoglobin A1C assay provides a more stable – though imperfect – proxy of adherence (i.e., glycemic control) over the previous 2–3 months. Hemoglobin A1C levels are now routinely used in both clinical care and research and have become the gold standard with respect to diabetes diagnosis and care.

Directly Observed Therapy (DOT)

Finally, DOT – as indicated by its name – requires the direct observation of patients as they complete each treatment or dose to confirm adherence. DOT was developed in the context of

tuberculosis (cf. Bayer & Wilkinson, 1995), an infectious disease requiring complex, months-long treatment and, consequently, is fraught with challenges to patient adherence. In particular, those most affected by tuberculosis (e.g., IV drug users, the homeless) were also those least likely to adhere to treatment. Besides tuberculosis treatment, DOT has proven a successful adherence strategy in studies of patients with HIV, pertussis, and hepatitis C. Overall, DOT may be most useful in the context of those illnesses that mutate quickly, are highly contagious, or where patient adherence is the primary barrier to treatment effectiveness.

Determinants of Adherence

Over the past 50 years, much research has worked to identify determinants of patient adherence. Although health care providers typically attribute nonadherent behavior to patient characteristics, the determinants of nonadherence are multifaceted and quite complex. Reviewed below are several characteristics known to be associated with patient adherence.

Characteristics of the Treatment Regimen

Relative to the other general categories of adherence determinants, characteristics of the treatment regimen have been less studied. Despite the paucity of data in this context, however, research consistently indicates that the complexity of the specific treatment regimen appears to substantially influence adherence behavior. For example, much evidence demonstrates that patients have more trouble adhering to prescribed treatments when multiple (vs single) doses are required throughout the day or are attached to certain caveats (e.g., "take with food"). Moreover, multifaceted regimens have been shown to yield poor adherence behavior as well. For example, diabetic patients (i.e., those required to meet multiple, complex self-care responsibilities) often have the highest levels of nonadherence compared to other patient populations. Of note, the correlations among various facets of complex treatment regimens are known to be quite low, suggesting that otherwise adherent patients may have trouble navigating multiple, complex treatment demands.

Patient Characteristics

Research regarding patient characteristics has typically focused on either (a) sociodemographic or (b) psychological correlates of adherence behavior. With respect to the former, few consistent patterns have emerged, perhaps with the exceptions of patient age and socioeconomic status (SES). Across numerous treatment settings and patient populations, younger individuals tend to exhibit poorer adherence behaviors as compared to older adults, although not uniformly. Patients of lower SES also tend to have increased rates of nonadherence irrespective of the treatment setting.

Patient psychological characteristics, such as personality traits and individual differences related to patient beliefs and expectancies, have been extensively studied in the context of treatment adherence. For example, the Five Factor Model personality trait of conscientiousness – reflecting self-control, dependability, deliberation, and the will to achieve – has been related to adherence in some (e.g., dietary adherence in end-stage renal disease patients), but not all, contexts. In addition, *health locus of control* (HLC), or the extent to which one believes that good health is a product of one's own behaviors as opposed to external or chance factors, has been shown to be associated with better adherence in several studies. In some instances, patients believing that health outcomes are due largely to their own behaviors (i.e., *internal* health locus of control; IHLC) exhibit more favorable adherence; however, other research has failed to demonstrate any association between IHLC and adherence while still others have found it to be associated with *worse* adherence. At best, the relationship between HLC and adherence is unclear. Some have speculated that patients with an IHLC orientation may demonstrate poorer adherence in contexts where self-care demands are minimal and patient control over health outcomes is limited.

Adherence researchers have also shown a decided interest in patient self-efficacy or the extent to which an individual believes he/she is capable of performing the behavior(s) needed to bring about a certain outcome. Much evidence

has consistently demonstrated the importance of patient self-efficacy in multiple treatment contexts, including diabetes, chronic kidney disease, HIV, transplant recipients, and post-MI recovery. Notably, some evidence suggests that locus of control and self-efficacy – distinct, yet complementary constructs – best predict adherence when considered in tandem, suggesting avenues for future research.

Patient Depression

Patient experience of psychological distress, particularly depression, has been investigated extensively in the context of patient adherence. Hallmarks of clinical depression include decreased motivation, psychomotor retardation, and cognitive deficits, all of which seemingly may impact adherence intentions and subsequent behaviors. Similar to other patient characteristics reviewed above, however, evidence of any relationship between depression and patient adherence is inconsistent.

Such findings may be explained, to some extent, by the way in which depression is captured (i.e., via self-report or diagnostic interview) and the context in which adherence is measured. Any relationship between depression and nonadherence is likely to be a function of the neurovegetative symptoms of depression noted above, each of which may be less likely in patients with subclinical depression. Consistent with this line of thought, research using self-report measures to capture depressive affect (vs structured clinical interview) tends to show weaker associations with adherence outcomes. Furthermore, some evidence demonstrates that as self-care demands increase, so do associations between depression and nonadherence, making the specific disease context in which adherence is measured extremely important.

Provider Characteristics

Health care provider (and practice style) characteristics have not been extensively considered in the context of adherence, nor have many significant associations materialized. While provider age, gender, and race/ethnicity seem to have little to do with the level of patient adherence, limited evidence suggests that the degree of information provision afforded patients is related to adherence; however, this variable seems to be more strongly associated with patient satisfaction than patient adherence.

Patient-Provider Interaction

Perhaps more important is the *interaction* between patient and provider. Health care delivery is decidedly interpersonal; thus, it seems remiss not to consider the patient-provider dyad in the context of adherence. Indeed, a growing body of evidence suggests that the symmetry, or match, between a patient and his/her provider on health-related attitudes toward illness and the health care context itself may be important for patient adherence. Christensen and his colleagues have done considerable work in this area, finding that patients and providers with similar attitudes regarding control over one's health (i.e., IHLC; see above) as well as preference for self-management and shared decision-making regarding treatment are predictive of adherence. Future research regarding patient-provider interaction in the context of treatment adherence may shed additional light on some of the issues reviewed heretofore.

Socio-Environmental Characteristics

Although considerable research underscores the importance of perceived quality and availability of social support in the context of adherence, the totality of evidence is, at best, mixed. Several studies in various disease contexts have found social support to be either unrelated or inconsistently related to adherence; still, others have reported social support to be related to poorer treatment adherence within certain contexts. In such instances, researchers have speculated that social support may confer a barrier to adherence – such as increased social obligations or stigma – particularly when a regimen requires changes in dietary behavior, which is known to be heavily influenced by social factors.

Facilitating Adherence

Strategies aimed at facilitating, or increasing, patient adherence may be classified in one of

three general categories: behavioral, psychoeducational, and socio-environmental. Behavioral strategies – such as patient self-monitoring, contingency contracting, stimulus control, and behavioral cues/reminders – have been implemented widely in the context of chronic treatment regimens. Collectively, such strategies have yielded modest success in facilitating patient adherence. For example, behavioral techniques have been commonly used to help improve adherence among diabetic patients; in particular, self-monitoring strategies have resulted in improved adherence to dietary guidelines and, as such, have been touted as a crucial component of modern diabetic therapy.

Psychoeducational strategies, including the provision of written or computer-based education, appear to be most effective when patients are given explicit, tailored recommendations relative to their disease and treatment versus interventions that impart more general health-related information. Although briefer psychoeducational interventions have demonstrated reasonable success, clinicians and researchers should be aware that multifaceted regimens (e.g., those associated with diabetes, transplant recovery) likely require considerably more instruction to confer a significant benefit.

Finally, several socio-environmental strategies have also been used to facilitate adherence. The most common strategy increases contact between the patient and health care provider, such as the treating physician or study nurse, with mixed success. Similarly, patient support groups and enhancement of the patient's family support structure through counseling have been attempted with some success. Much of this work, however, has been plagued by inattention to theory and poor methodology, highlighting the need for further investigation.

Conclusions

Patient adherence is as fundamental a component of effective health care as the treatment regimen itself. However, despite extensive study over five decades, nonadherence remains a significant problem. In both research and clinical care settings, measurement of patient adherence behavior may take many forms, all with considerable strengths and weaknesses. Many determinants of adherence remain unknown, underscoring not only the remarkable complexity of patient adherence but also the difficulty in reliably predicting behaviors often associated with enormous health-related consequences and, ultimately, the need for further investigation of this dynamic phenomenon.

Cross-References

- ▶ Health Promotion
- ▶ Medical Utilization

References and Readings

Christensen, A. J. (2004). *Patient adherence to medical treatment regimens: Bridging the gap between behavioral science and biomedicine*. New Haven, CT: Yale University Press.

Christensen, A. J., Howren, M. B., Hillis, S. L., Kaboli, P., Carter, B. L., et al. (2010). Patient and physician beliefs about control over health: Association of symmetrical beliefs with medication regimen adherence. *Journal of General Internal Medicine, 25*, 397–402.

DiMatteo, M. R. (2004). Variations in patients' adherence to medical recommendations: A quantitative review of 50 years of research. *Medical Care, 42*, 200–209.

DiMatteo, M. R., Giordani, P. J., Lepper, H. S., & Croghan, T. W. (2002). Patient adherence and medical treatment outcomes: A meta-analysis. *Medical Care, 40*, 794–811.

Dunbar-Jacob, J., & Schlenk, E. (2001). Patient adherence to treatment regimen. In A. Baum, T. A. Revenson, & J. E. Singer (Eds.), *Handbook of health psychology* (pp. 571–580). Mahwah, NJ: Lawrence Erlbaum.

Eisenthal, S., Emery, R., Lazare, A., & Udin, H. (1979). "Adherence" and the negotiated approach to patienthood. *Archives of General Psychiatry, 36*, 393–398.

Haynes, R. B., Ackloo, E., Sahota, N., McDonald, H. P., & Yao, X. (2008). Interventions for enhancing medication adherence. *Cochrane Database of Systematic Reviews*, (2), Article CD000011. doi:10.1002/14651858.CD000011.pub3.

Steiner, J. F., & Prochazka, A. V. (1997). The assessment of refill compliance using pharmacy records: Methods, validity, and applications. *Journal of Clinical Epidemiology, 50*, 105–116.

Stone, A. A., Turkkan, J., Jobe, J., Kurtzman, H., & Cain, V. (2000). *The science of self-report*. Mahwah, NJ: Lawrence Erlbaum.

Van Dulmen, S., Sluijs, E., Van Dijk, L., De Ridder, D., Heerdink, R., & Bensing, J. (2007). Patient adherence to treatment: A review of reviews. *BMC Health Services Research, 7*, 55.

Adhesion Molecules

Manjunath Harlapur[1] and Daichi Shimbo[2]
[1]Center of Behavioral Cardiovascular Health, Division of General Medicine, Columbia University, New York, NY, USA
[2]Center for Behavioral Cardiovascular Health, Columbia University, New York, NY, USA

Synonyms

Cell adhesion molecule

Definition

Adhesion molecules are the protein molecules that are situated on the surface of the cells or the intracellular organelle.

Description

The adhesion molecules attach to adjacent cells and help with binding with these cells or extracellular structures. There are several functions described like transmitting the information (cellular signaling pathways) to communicate between cells and within the cells. The adhesion molecules usually share a common basic structure that they have extracellular and intracellular domains that are connected to transcellular domain. This facilitates their function to act as messenger across the cellular membrane on either side.

Adhesion molecules are classified based on the function, structure, and location. Cadherins, integrins, selectins, and immunoglobulin (Ig)-related cell adhesion molecules (CAMS) are four major types of adhesion molecules. Brief summary of these molecules are described below.

The cadherins are calcium-dependent adhesion molecules, and they are activated when the extracellular domain is attached to calcium. The intracellular domain connects to various intracellular proteins to perform several functions. The details about epithelial (E)-cadherin, neural (N)-cadherin, and placental (P)-cadherin have been described in relation to several tissue function and also embryogenesis.

The integrins are transmembrane proteins that are composed of different subunits (e.g., alpha and beta subunits). These have been described in the signal transfer from within cell to extracellular matrix and also from extracellular to intracellular structures.

The selectins are the type of adhesion molecules, and they are glycoproteins in their composition. When the specific carbohydrate related to cellular signaling pathway attaches to the extracellular domain, these selectins transmit the signal across the cellular membrane. Platelet-selectins, leukocyte- selectins, and endothelial-selectins are subtypes of selectins, and they are involved with various functions related to platelet, leukocyte, and platelet activity at the cellular level.

The immunoglobulin (Ig)-related cellular adhesion molecules (CAM) are independent of calcium for their activity. Vascular-cell adhesion molecules (VCAM-1), neural (N)-cell adhesion molecule (NCAM), intercellular adhesion molecule (ICAM), and platelet-endothelial cell adhesion molecule (PECAM) have been related to dysregulations of the vascular system, nervous system, and platelet-thrombosis.

These adhesion molecules may be involved with each other in the process of various functions of the cellular signaling pathway. For example, during the inflammatory process, white blood cells (leukocytes) are transferred across the endothelial lining to the subendothelial layer of the vasculature which is a multistep process and an important part of the early pathogenesis of atherosclerosis. Initially, leukocytes come in close proximity to endothelial cells which are selectin mediated. The integrins activate the surface adhesion molecules in the presence of

several pro-inflammatory factors including various extracellular proteins and cytokines. The integrins also help with the attachment of the leukocytes to the endothelium. With the help of PECAM, the leukocytes migrate across the endothelium (diapedesis) to the subendothelial space. Subsequent complex changes occur, leading to the development and progression of atherosclerosis. Various adhesion molecules have been described in the context of cancer metastasis, growth of tumor cells, and embryogenesis.

Cross-References

- ▶ Atherosclerosis
- ▶ Heart Disease
- ▶ Inflammation

References and Readings

Fuster, V., Lois, F., & Franco, M. (2010). Early identification of atherosclerotic disease by noninvasive imaging. *Nature Reviews Cardiology, 7*(6), 327–333.

Gahmberg, C. G., Valmu, L., Kotovuori, A., Kotovuori, P., Hilden, T. J., Fagerholm, S., & Tian, L. (1999). Leukocyte adhesion–an integrated molecular process at the leukocyte plasma membrane. *Bioscience Reports, 19*(4), 273–281.

Gonzalez-Amaro, R., & Sanchez-Madrid, F. (1999). Cell adhesion molecules: Selectins and integrins. *Critical Reviews in Immunology, 19*(5–6), 389–429.

Worthylake, R. A., & Burridge, K. (2001). Leukocyte transendothelial migration: Orchestrating the underlying molecular machinery. *Current Opinion in Cell Biology, 13*(5), 569–577.

Adipose Tissue

Keisuke Ohta and Naoya Yahagi
Department of Metabolic Diseases, Graduate School of Medicine The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Synonyms

Body fat

Definition

Adipose tissue is a loose connective tissue composed of adipocytes. It is composed of roughly only 80% fat. Adipocytes are the cells specialized in storing energy as fat. Adipose tissue also serves as an important endocrine organ by producing hormone such as leptin (Kershaw & Flier, 2004).

There are two types of adipose tissue, white adipose tissue (WAT) and brown adipose tissue (BAT). White adipose tissue is involved in the storage of energy, whereas brown adipose tissue serves as a thermogenic organ.

Cross-References

- ▶ Leptin

References and Readings

Kershaw, E. E., & Flier, J. S. (2004). Adipose tissue as an endocrine organ. *Journal of Clinical Endocrinology and Metabolism, 89*(6), 2548–2556.

Adjuvant Chemotherapy

Elizabeth Franzmann
Department of Otolaryngology/Division of Head and Neck, Miller School of Medicine, University of Miami, Miami, FL, USA

Synonyms

Additional or assisting medication

Definition

Antineoplastic medication given following the primary cancer treatment, usually surgery or radiation, with the goal to improve relapse-free survival.

Description

Paul Ehrlich, a famous German chemist, was the first to coin the term "chemotherapy" or the use of chemicals to treat disease in the early 1900s (DeVita & Chu, 2008). While known for his work on drugs to treat infectious disease, he also worked with anticancer agents. However, his work and the work of others that followed him, usually with single agents, were fraught with challenges (DeVita & Chu). The idea that cancers could be cured with chemotherapy first became widely accepted around 1970 with successes in both childhood leukemia and Hodgkin's lymphoma using multi-agent regimens (DeVita & Chu; Frei, 1985). Prior to this time, surgery and radiation were the mainstay for solid tumor treatment. However, even for the most aggressive surgical or radiotherapy regimens, cure rates did not exceed 33% (DeVita & Chu, 2008).

Interest in adjuvant chemotherapy arose from experiments in animal models showing that chemotherapy, though minimally effective against large tumors, may be curative against microscopic disease (DeVita & Chu, 2008; Frei, 1985). Work by Howard Skipper showed that a given dose of chemotherapy killed a constant fraction of tumor cells rather than a constant number (DeVita & Chu, 2008; Skipper, 1978). This inverse relationship between tumor cell number and curability suggested that drugs used against advanced disease might work better after the tumor was eradicated with primary treatment such as surgery (DeVita & Chu, 2008).

Even after optimal local control is achieved with surgery and/or radiotherapy, advanced stage tumors and aggressive pathology findings are often associated with a high likelihood of disseminated micrometastases or microscopic residual disease (Skipper, 1978). In such cases, administration of additional or adjuvant treatment may eradicate microscopic disease and decrease chances of relapse. These concepts were first tested and reported for breast cancer in the mid-1970s with successful results (DeVita & Chu, 2008; Frei, 1985). This success triggered a plethora of adjuvant studies in breast and other tumors such as colorectal cancer (DeVita & Chu, 2008). The resulting therapies have contributed significantly to the national decline in breast and colorectal cancer mortality that has been witnessed in recent years (DeVita & Chu, 2008). Other solid tumors have also shown benefit with adjuvant chemotherapy (DeVita & Chu). These include cervical cancer, gastric cancer, head and neck cancers, pancreas cancer, melanoma, non-small cell lung cancer, osteogenic sarcoma, and ovarian carcinoma (DeVita & Chu).

Chemotherapy, radiation therapy, immunotherapy, hormonal therapy, and targeted therapy have all been used as adjuvant treatments. Each of these is associated with specific side effects that can be severe and must be weighed against the potential benefits. Side effects of chemotherapy, for example, can include nausea, vomiting, hair loss, and drops in blood cell counts. Patients who experience treatment-related adverse effects are more likely to discontinue adjuvant therapy (Burstein et al. 2010). Thus the decision to proceed with adjuvant treatment can be complicated, especially when the disease appears to be eradicated and may indeed never return, even without further treatment (Burstein et al.). This is even a more difficult decision if the primary treatment was physically and emotionally draining for the patients, such as a disfiguring and debilitating surgery (Burstein et al.). The decision to proceed with adjuvant therapy requires a thorough discussion between the patient and their surgical, medical, and radiation oncologists with thorough explanation of the potential risks and benefits.

References and Readings

Burstein, H. J., Prestrud, A. A., Seidenfeld, J., Anderson, H., Buchholz, T. A., Davidson, N. E., et al. (2010). American Society of Clinical Oncology clinical practice guideline: Update on adjuvant endocrine therapy for women with hormone receptor-positive breast cancer. *Journal of Clinical Oncology, 28*, 3784–3796.

DeVita, V. T., & Chu, E. (2008). A history of cancer chemotherapy. *Cancer Research, 68*, 8643–8653.

Frei, E., III. (1985). Curative cancer chemotherapy. *Cancer Research, 45*, 6523–6537.

Skipper, H. E. (1978). Adjuvant chemotherapy. *Cancer, 41*, 936–940.

Admixture

Abanish Singh
Duke University Medical Center,
Durham, NC, USA

Definition

The use of racial classification in medicine and biomedical research has become a popular tool and is very helpful in understanding racial and ethnic differences in the causes, expression, and prevalence of disease traits. Usually, human society is classified based on color and linguistic and cultural differences. However, these classifications often ignore the availability of interbreeding. Breeding between members of different classes can result in the exchange of genetic information which can further affect genetic disease profiles.

The phenomenon of interbreeding between the members of two or more different population groups is known as genetic admixture. It results in continued, long-term exchange of genes among the various human society classifications. The admixture process creates linkage disequilibrium between loci in a hybrid population and its magnitude is guided by several factors such as time duration, dynamics, recombination rate, and allele frequency differential in parental populations. Admixture can be estimated reliably from the genetic similarities if the accurate identities of parental populations in the hybrid population are available.

Cross-References

- ▶ Allele
- ▶ Gene
- ▶ Genome-Wide Association Study (GWAS)
- ▶ Locus (Genetics)

References and Readings

Burchard, E. G., Ziv, E., Coyle, N., Gomez, S. L., Tang, H., Karter, A. J., et al. (2003). The Importance of race and ethnic background in biomedical research and clinical practice. *The New England Journal of Medicine, 348*(12), 1170–1175.

Chakraborty, R., & Weiss, K. M. (1988). Admixture as a tool for finding linked genes and detecting that difference from allelic association between loci. *Genetics, 85*, 9119–9123.

Indrani, Halder, & Shriver, M. D. (2003). Measuring and using admixture to study the genetics of complex diseases. *Human Genomics, 1*(1), 52–62.

McKeigue, P. M., Carpenter, J., Parra, E. J., & Shriver, M. D. (2000). Estimation of admixture and detection of linkage in admixed populations by a Bayesian approach: Application to African-American populations'. *Annals of Human Genetics, 64*, 171–186.

Patterson, N., et al. (2004). Methods for high-density admixture mapping of disease genes. *American Journal of Human Genetics, 74*, 979–1000.

Patterson, N., et al. (2010). Genetic structure of a unique admixed population: Implications for medical research. *Human Molecular Genetics, 19*(3), 411–419. doi:10.1093/hmg/ddp505.

Pfaff, C. L., Parra, E. J., Bonilla, C., et al. (2001). Population structure in admixed populations: Effects of admixture dynamics on the pattern of linkage disequilibrium. *American Journal of Human Genetics, 68*, 198–207.

Stephens, J. C., Briscoe, D., & O'Brien, S. J. (1994). Mapping by admixture linkage disequilibrium in human populations: Limits and guidelines. *American Journal of Human Genetics, 55*, 809–824.

Adolescent Psychology

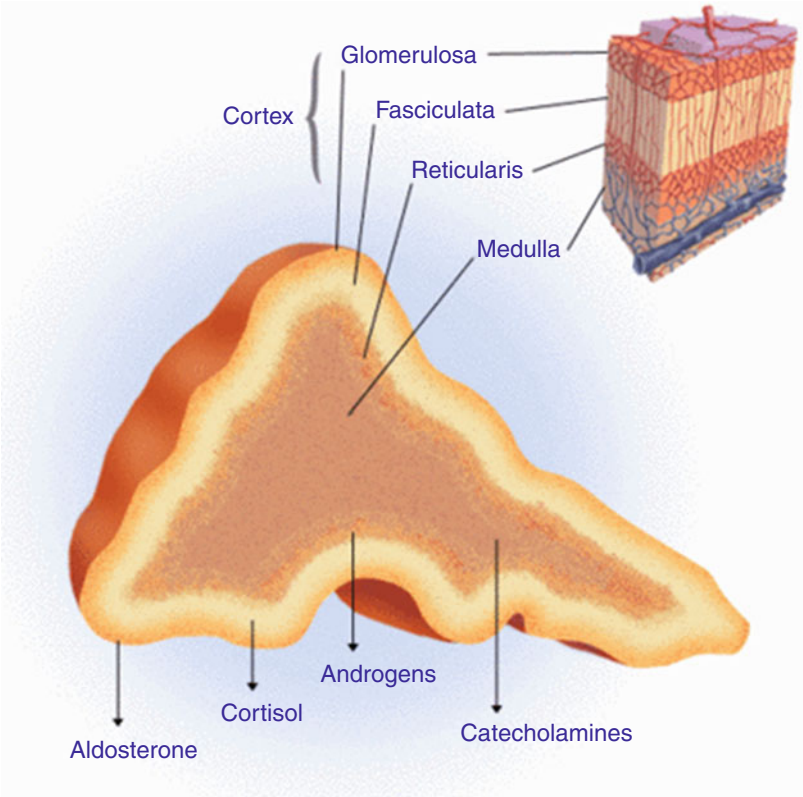
- ▶ Child Development

Adrenal Glands

Linda C. Baumann and Alyssa Karel
School of Nursing, University of Wisconsin-Madison, Madison, WI, USA

Definition

The adrenal glands are part of the endocrine system which is comprised of various glands



Adrenal Glands, Fig. 1 The zona glomerulosa, the zona fasciculata, and the zona reticularis

that are located throughout the body. These glands are responsible for synthesizing and producing hormones which communicate regulatory information to cells and organs. The adrenal glands are primarily responsible for the stress response in the human body secondary to physical or emotional stimuli. The two adrenal glands are pyramid-shaped organs located directly anterior to the kidneys and behind the peritoneum. Each adrenal gland consists of two distinct portions: an inner medulla and an outer cortex. Each portion has differing structures and hormonal functions, but are interrelated.

Description

The adrenal medulla synthesizes and secretes the catecholamines epinephrine and norepinephrine. Major effects of catecholemines are increased rate and force of contraction of the heart muscle, constriction of blood vessels, dilation of bronchioles, stimulation of lipolysis in fat cells, increased metabolic rate, dilation of the pupils, and inhibition of nonessential processes.

The adrenal cortex is subdivided into three layers which are primarily responsible for producing corticosteroids (see Fig. 1). The outer layer, the zona glomerulosa, is responsible for secreting the mineralocorticoid aldosterone. The middle layer, the zona fasciculata, and the inner layer, the zona reticularis, produce adrenal androgens and estrogens, and glucocorticoids such as cortisol. Aldosterone, the major mineralocorticoid, is necessary for survival and is responsible for increases in sodium reabsorption from the renal tubule, saliva, and gastric juice which results in increased reabsorption of water. Secondary actions of aldosterone include maintenance of blood pressure and potassium regulation. Cortisol, the most potent of the

glucocorticoids, is responsible for stimulation of gluconeogenesis, mobilization of amino acids from extrahepatic tissues, inhibition of glucose uptake in muscle and adipose tissue, and stimulation of fat breakdown. Cortisol also has potent anti-inflammatory and immunosuppressive properties. In the absence of corticosteroids, the stress response would induce hypotension, shock, and death.

In general, an individual will experience a stress response when a stimulus exceeds their coping abilities which can result in disturbances of cognition, emotion, and behavior. The stress response starts in the central nervous system and endocrine system. It is cyclical in nature and will continue as long as the stimulus is present. Stress responses can be either acute or chronic in nature. Acute stress responses are a result of an immediate threat; subconscious, false, or perceived. The process will elicit a reaction most commonly known as the fight or flight response. In this circumstance, individuals may exhibit behaviors secondary to physiologic changes including anxiety, rapid speech, restlessness, facial tics, teeth grinding, and nail biting to name a few. Once an acute stressor is eliminated or overcome, the body shuts down the process through a negative feedback system and hormone levels eventually return to normal. Not all stressful situations are detrimental and at times may be desirable. It can prompt individuals to work toward worthwhile goals, relieve monotony, and can play a part in pleasurable activities.

In a chronic stress response, the stress cycle is continually activated leading to elevated hormone levels. Chronic stress can be related to weight gain and obesity. Individuals crave salt, fat, and sugar in an attempt to counteract tension secondary to the sustained release of cortisol into the blood stream. Sustained levels of stress hormones have also been detected in individuals with eating disorders such as anorexia nervosa and bulimia. Chronic stress can lead to insomnia and impaired memory and concentration. It can also contribute to major anxiety, depression, and suicidal ideations, as well as behaviors such as alcoholism and drug abuse.

Personality traits, such as Type A personality, can contribute to a maladaptive stress response. Type A personality is characterized by hostility, impatience, and competitiveness. These traits can lead to an increased risk of hypertension, heart disease, job stress, alcoholism, and social alienation.

Individual reactions to stress depend on factors such as knowledge about the stress response, learned behaviors, personality type, and attitudes about controlling, altering, and adapting to stressful situations. The importance of healthy stress management can reduce some of the maladaptive behaviors associated with the adrenal glands and the stress response.

Cross-References

▶ Stress Reactivity

References and Readings

Ebstrup, J., Eplov, L., Pisinger, C., & Jorgensen, T. (2011). Association between the five factor personality traits and perceived stress: Is the effect medicated by general self-efficacy? *Anxiety Stress Coping, 6*, 1–13.

Howard, J. (1990). Type A behavior, personality, and sympathetic response. *Behavioral Medicine, 16*(4), 149–160.

McCance, K., & Huether, S. (2006). *Pathophysiology: The biologic basis for disease in adults and children* (5th ed.). St. Louis: Mosby.

McPhee, S., & Papadakis, M. (2010). *Current medical diagnosis and treatment*. New York: McGraw Hill.

Pathology Outlines (2010). *Adrenal gland and paraganglia*. Retrieved January 20, 2010, from www.pathologyoutlines.com/adrenal.html#top

Adrenaline

▶ Catecholamines
▶ Epinephrine

Adrenergic Activation

Debra Johnson
Department of Psychology, University of Iowa, Iowa City, IA, USA

Definition

Adrenalin/epinephrine is a neurotransmitter produced by and released from the adrenal glands. Release of this chemical activates the sympathetic nervous system through the alpha-adrenergic and beta-adrenergic receptor families and produces the classic "fight-or-flight response" including increased blood pressure, heart rate, and respiratory rate. While this system is adaptive in the context of acutely stressful events, prolonged stress can produce a chronic overactivation of the adrenergic system. This overactivation is implicated in the development and progression of chronic health problems including hypertension and coronary artery disease.

Cross-References

► Sympatho-Adrenergic Stimulation

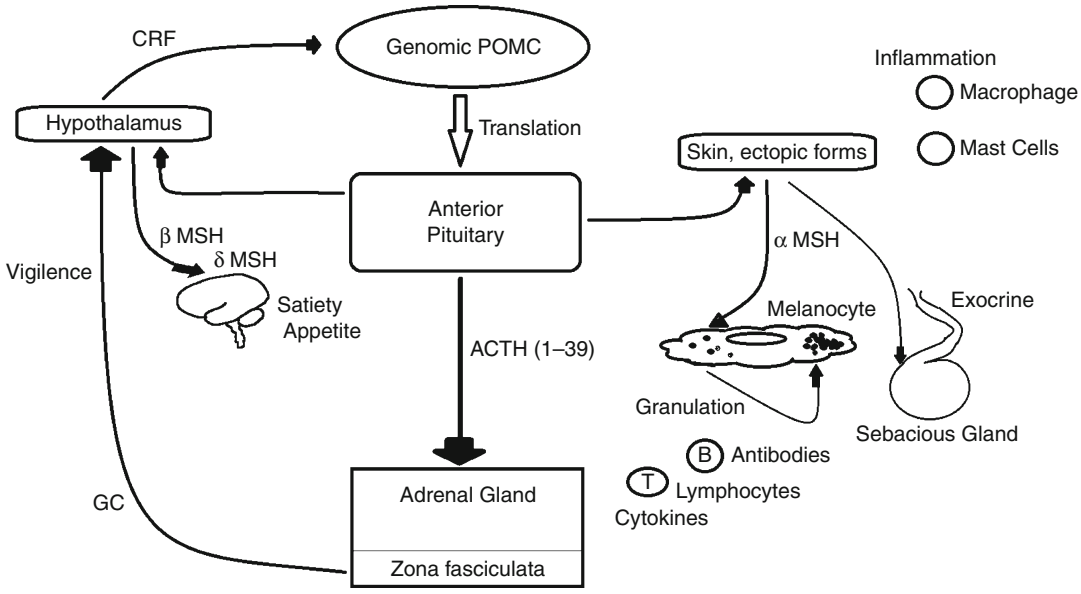
Adrenocorticotropin

Benjamin L. Clarke
Academic Health Center, School of Medicine-Duluth Campus, University of Minnesota, Duluth, MN, USA

Definition

Adrenocorticotropic hormone (ACTH) is a peptide hormone produced by corticotropin cells of the anterior pituitary. Discovery of ACTH and subsequent characterization focused on a pituitary substance known to stimulate glucocorticoid synthesis from the adrenal cortex; hence, the name reflects this tropic hormone function. Pituitary production of ACTH is stimulated by corticotropin-releasing hormone (CRH), a peptide hormone emanating from the "nuclei" neurons found in the hypothalamus (see Fig. 1). Released CRH then passes through the hypophyseal portal vessels into the anterior pituitary to target corticotropin cells. The hypothalamus provides a cognitive nexus for translating central nervous system (CNS) activity into an endocrine signaling pathway responsible for stimulating the adrenal cortex. This triad of hypothalamus-pituitary-adrenals activity is often referred to as the HPA axis. A large number of behavioral and physiological responses are controlled by the HPA in order to sustain physiological homeostasis. The canonical HPA activity is CNS control of glucocorticoid release following physical or emotional stress to promote physical responses. The "fight or flight" response is a common behavioral pattern regulated by the HPA. Heightened HPA activity increases cognitive vigilance, plasma glucose, and metabolic activity while suppressing tissue repair and anabolic processes. Excessive HPA activity is often associated with behavior conditions like anxiety and depression, and medical conditions such as fluid retention and obesity.

ACTH is coded by the proopiomelanocortin (POMC) gene located on Chromosome 2 in humans. The initial gene product is a pro-form protein that is differentially processed into active peptide hormones within corticotropin cells of the anterior pituitary. In the precursor form, POMC is a polypeptide consisting of 241 amino acids. Embedded in the polypeptide POMC are several different peptide hormones. Due to "tissue-specific processing" of POMC, a large number of peptide hormones are produced at the site of action. Specific POMC-derived hormones are revealed by the action of pro-hormone convertases (PC). This differential processing is controlled by the expression of two different PCs, (PC1 and PC2) that are localized to different tissues. The majority of POMC peptide is produced in the anterior pituitary and the proteolytic fragments are redistributed to distal sites for additional processing. Corticotropin cells in the anterior pituitary express PC1 which



Adrenocorticotropin, Fig. 1 Generation of ACTH

hydrolyzes POMC to first form pro-ACTH and beta lipotropin hormone (β-LPH). A second round of hydrolysis by PC1 on pro-ACTH produces ACTH and N-POMC. A third round of hydrolysis by PC1 on N-POMC produces pro-γ-MSH and a joining peptide (JP) fragment. The most abundant form of ACTH has 39 amino acids, ACTH (1–39); however, minor amounts of several smaller sized ACTH are also produced. Several other peptide hormones are also derived from POMC using a second convertase expressed in the hypothalamus and skin. Products from differential processing by PC1 in the pituitary are distributed to distal tissues to be further processed by PC2. This process of disseminating ACTH peptides to other sites provides specificity for biological activity. The pro-γ-MSH is converted to γ-MSH by PC2. ACTH (1–39) is converted by PC2 to form ACTH (1–17) and corticotropin-like intermediate peptide (CLIP). ACTH (1–17) is then converted into α-MSH by the sequential action of carboxypeptidase E, peptidyl-amidating monooxygenase, and N-acetyl transferase. Beta-lipotropin hormone is converted into γ-LPH and beta-endorphin by PC2, and γ-LPH is converted by PC2 into beta-melanocyte-stimulating hormone (β-MSH). The two major corticotropins, ACTH and α-MSH, share the first 13 amino acids. However, α-MSH has two important chemical changes that alter solubility and transport properties; the amino terminal serine is acylated and the carboxyl terminal valine is amidated. Common to all corticotropins is a tetra-amino acid sequence of histidine-phenylalanine-arginine-tryptophan (H-F-R-W). Point substitutions within the HFRW sequence completely oblates the activity of ACTH, α-MSH, β-MSH, and γ-MSH.

Cellular recognition of ACTH and MSH molecules has been attributed to melanocortin receptors (MCRs) (see Table 1). Five different G-protein-coupled receptors have been cloned and characterized for biological activity. Specific binding of POMC-derived peptides are dependent on the presence of the HFRW sequence embedded within the hormone sequence (see Table 2). Selectivity between potential hormone ligands is the HFRW sequence, peptide length, and chemical modification of the peptide termini. The steroidogenic receptor for ACTH is MC2R has the highest stringency requiring both the HFRW sequence plus a highly anionic tetra peptide sequence of lysine-lysine-arginine-arginine (KKRR) at positions 15–18. The MSH peptides do not possess the KKRR sequence and are not

Adrenocorticotropin, Table 1 Melanocortin receptors

	MC1R	MC2R	MC3R	MC4R	MC5R
Predominate agonist	α/β-MSH	ACTH (1–39)	γ-MSH	α-MSH	α-MSH
Site of expression	Skin	Adrenal glomerulosa, adrenal fasiculata, adipose	Hypothalamus, gut, heart, kidney, and placenta	Brain and spinal cord	Widely expressed at low density
Physiological activity	Pigmentation, anti-inflammation, anti-pyretic	Steroidogensis	Satiety, cardiovascular, energy homeostasis	Appetite, energy homeostasis, anti-pyretic, pain, penile erections	Exocrine, immunoregulatory
Behavioral activity	Coat/skin color	Hypervigilance	Feeding	Feeding	Male dominance
Native antagonist	Agouti protein		Agouti protein	Agouti protein	

Adrenocorticotropin, Table 2 Melanocortin peptides

MC(1–5)R MC2R only
ACTH (1–39) NH_2-SYSME*HFRW*GKPVG*KKRR*PVKV PNGAEDESAEAFPLEF-OH
α-MSH Ac-SYSME*HFRW*GKPV-NH_2
β-MSH NH2-AEJJKEGPYRME*HFRW*GSPPKD-OH
γ-MSH NH_2-YVMG*HFRW*DRF-OH

ligands for MC2R. The other four receptors (MC1, MC3, MC4, and MC5) have comparably binding affinities for all ACTH and MSH peptides. Each MC receptor is localized to different tissues. The MC1R is abundant in melanotropic cells found in the skin and regulates skin and coat pigmentation. Adrenal glands express the MC2R high expression levels in the fasiculata zone (glucocorticoids) and the glomerulosa zone (mineralocorticoids), plus lower expression of MC5R. The brain expresses the MCRs is several regions.

Adversarial Growth

► Benefit Finding
► Posttraumatic Growth

Adverse Drug Events

► Drug, Adverse Effects/Complications

Adverse Drug Reaction

Debra Johnson
Department of Psychology, University of Iowa, Iowa City, IA, USA

Definition

An adverse event is a negative change in health observed in individuals participating in clinical drug trials or trials of medical devices. These events may occur during the trial or within a short time after the trial ends. They may or may not be due to the drug or device and can be categorized as minor (i.e., hypotension) or serious (i.e., life-threatening complications or even death). Minor adverse events are reported to the facility's institutional review board and to the sponsor of the trial. Serious events must additionally be reported to the regulatory agencies (i.e., FDA). In recent years, several large clinical trials have been halted because of serious adverse events observed in the patient groups.

Adverse Drug Reactions

► Drug, Adverse Effects/Complications

Adversity, Early Life

► Stress, Early Life

Aerobic Exercise

Rachel Millstein
SDSU/UCSD Joint Doctoral Program in Clinical Psychology, University of California, San Diego/San Diego State University, San Diego, CA, USA

Synonyms

Exercise; Moderate-vigorous physical activity

Definition

Aerobic exercise refers to the type of repetitive, structured physical activity that requires the body's metabolic system to use oxygen to produce energy. Aerobic exercise improves the capacity of the cardiovascular system to uptake and transport oxygen. Aerobic activity can be undertaken in many different forms, with the common feature that it is achieved at a heart rate of 70–80% of a person's age-appropriate maximum. Aerobic exercise is considered the cornerstone of endurance training, characterized by moderate energy expenditure over a prolonged period of time. Aerobic power or endurance is measured by VO_2 max, a person's maximal oxygen uptake.

Description

Aerobic exercise is different from, though related to, physical activity and exercise in general. Physical activity is a broad category that refers to all bodily movements that require skeletal muscle contraction and energy expenditure. Exercise is a subset of physical activity, requiring specifically planned, structured, and repetitive movements with a goal of improving performance or fitness (Caspersen, Powell, & Christensen, 1985). Aerobic exercise can be undertaken in many different forms, such as brisk walking, running, cycling, swimming, cross-country skiing, and dancing. In contrast, anaerobic exercise involves high-intensity work done for shorter periods of time. The anaerobic metabolic system does not require oxygen to be used for energy.

Aerobic exercise was popularized by Dr. Kenneth Cooper, following the publication of his 1968 book, *Aerobics*. Since that time, the research and knowledge of the health benefits of aerobic exercise have grown and are widely known. The many health benefits of regular aerobic exercise include improved cardiorespiratory endurance, improved muscular endurance and strength, improved body composition, reduced risk of type 2 diabetes, reduced risk of osteoporosis and several cancers, and improved mental health (anxiety and depression symptom reduction).

Several authoritative groups have set forth recommended aerobic physical activity guidelines for health benefits. The United States' Centers for Disease Control and Prevention (2008) recommend that adults achieve at least 150 min of moderate-intensity or 75 min of vigorous-intensity aerobic physical activity each week. Youth are recommended to accumulate 60–90 min or more, or physical activity a day. Moderate-intensity physical activity requires about 3–6 times as much energy as resting (i.e., brisk walking), and vigorous-intensity physical activity requires about 7 times as much energy as resting (i.e., jogging).

Cross-References

► Exercise
► Physical Activity and Health
► Physical Fitness

References and Readings

Bouchard, C., Shepard, R. J., & Stephens, T. (Eds.). (1994). *Physical activity, fitness, and health: International proceedings and consensus statement*. Champaign, IL: Human Kinetics Books.

Bouchard, C., Shepard, R. J., Stephens, T., & McPherson, B. (Eds.). (1990). *Exercise, fitness, and health: A consensus of current knowledge*. Champaign, IL: Human Kinetics Books.
Caspersen, C. J., Powell, K. E., & Christensen, G. M. (1985). Physical activity, exercise, and physical fitness: Definitions and distinctions for health-related research. *Public Health Reports, 100*, 126–131.
Cooper, K. H. (1968). *Aerobics*. New York: Bantam Publishing.
Physical Activity Guidelines for Americans. (2008). Retrieved on October 20, 2010 from www.health.gov/paguidelines/default.aspx
Sallis, J. F., & Owen, N. (1999). *Physical activity & behavioral medicine*. Thousand Oaks, CA: Sage.
U.S. Department of Health and Human Services. (1996). *Physical Activity and Health: A Report of the Surgeon General*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion
Wilmore, J. H., & Costill, D. L. (1994). *Physiology of sport and exercise*. Champaign, IL: Human Kinetics.

Affect

Karen Niven
Manchester Business School, The University of Manchester, Manchester, UK

Synonyms

Affective state; Emotion; Feeling; Feeling state; Mood

Definition

Affect is the collective term for describing *feeling states* like emotions and moods. Affective states may vary in several ways, including their duration, intensity, specificity, pleasantness, and level of arousal, and they have an important role to play in regulating cognition, behavior, and social interactions.

Description

Affect is the experiential state of feeling. In everyday language, terms like affect, emotion, and mood are often used interchangeably. Affect is the superordinate category; emotions and moods are states belonging to this category. Emotions and moods are mainly distinguished by their duration, and by whether they are directed at a specific cause. Emotions are fairly fleeting and intense experiences that are elicited in response to specific external stimuli (i.e., objects or events), and may arise relatively automatically, or following a cognitive appraisal of a stimulus (e.g., How does the stimulus relate to my goals? How personally relevant is this stimulus?). Moods last somewhat longer than emotions, and are more diffuse in nature. For instance, a generalized feeling of sadness with no specific cause would be considered a mood state. People's experiences of affect over a long period of time can be summarized to represent their subjective well-being, i.e., their global assessment of happiness and satisfaction with life.

Describing and Distinguishing Affective States

Affective states are typically distinguished along the dual dimensions of pleasure and arousal. Pleasure concerns the hedonic properties of the state, ranging from unpleasant to pleasant, and arousal concerns the level of engagement or alertness of the state, ranging from activated to deactivated. So, for example, "anxiety" is a low pleasure high arousal state. Pleasure is usually measured by self-report (i.e., asking people how pleasant they feel), while arousal can be measured by self-report or by using physiological data (e.g., heart rate, epinephrine).

There are at least five "basic" emotions – anger, disgust, fear, happiness, and sadness – that are thought to be highly idiosyncratic and distinguishable according to a particular pattern of cognitions, physiology, facial expressions, and "action tendencies" that predispose a person to act in a certain way. For instance, the emotion of fear is distinguished by the cognitive appraisal of danger, physiological arousal in terms of higher heart rate, blood pressure, and perspiration, a facial expression involving widened eyes, dilated pupils and drawn brows, and the action tendency of fleeing a situation. More complex emotions like jealousy, shame, and pride may have distinguishable

cognitive and expressive elements, but may not be reliably associated with specific patterns of physiological arousal or action tendencies.

Functions of Affect

Affect has important cognitive functions, being used as a source of information when judging the value or valance of objects or people, priming congruent memories, and influencing decision-making and information processing and possibly decisions. Positive affect appears to be linked to broadening of attention, with benefits for creativity and problem solving, while negative affect is associated with narrowing of attention, with benefits for more focused tasks. Affect also serves a function in regulating behavior. Mood states give rise to broad tendencies toward approach or avoidance behaviors (pleasant moods are associated with a drive to approach a stimulus, whereas unpleasant moods are associated with avoidance), while emotions are associated with specific action tendencies (e.g., anger is associated with the tendency to "fight"). Similarly, anticipated affect drives our behavior, such that we pursue those behaviors we deem likely to result in desirable affective outcomes, and avoid those likely to result in undesirable affect. Affect may also function to regulate social behavior, by communicating information to others about how we would like them to engage with us (e.g., guilt signals a desire to be forgiven).

Individual Differences in Affect

Some people are more prone than others to experiencing particular affective states. Those with high negative affectivity tend to experience unpleasant affect much of the time, while those with high positive affectivity tend to experience pleasant affect. People may also be more or less reactive to affective stimuli. Those who are high in trait neuroticism are thought to have a lower threshold for reacting to stimuli, and also appear to react more intensely. Individual differences in the extent to which people outwardly communicate their affect (expressivity), and the extent to which people are able to deliberately control their experienced and expressed affect (affect regulation), have also been observed.

Cross-References

- ▶ Affect Arousal
- ▶ Emotional Responses
- ▶ Emotions: Positive and Negative
- ▶ Mood

References and Readings

Batson, C. D., Shaw, L. L., & Oleson, K. C. (1992). Differentiating affect, mood, and emotion. In M. S. Clarke (Ed.), *Emotion* (pp. 294–326). Newbury Park, CA: Sage.

Lewis, M., Haviland-Jones, J. M., & Feldman Barrett, L. (2008). *Handbook of emotions* (3rd ed.). New York: Guilford Press.

Mauss, I. B., & Robinson, M. D. (2009). Measures of emotion: A review. *Cognition and Emotion, 23*, 209–237.

Russell, J. A. (1980). A circumplex model of affect. *Journal of Personality and Social Psychology, 39*, 1161–1178.

Affect Arousal

Karen Niven[1] and Eleanor Miles[2]

[1]Manchester Business School, The University of Manchester, Manchester, UK

[2]Department of Psychology, The University of Sheffield, Western Bank, Sheffield, UK

Synonyms

Activation; Arousal; Energy; Tension

Definition

Affect arousal is the state of being activated, either physiologically or psychologically, and is one dimension of our affective response to emotional stimuli. Psychological characteristics of arousal include feelings of vigor, energy, and tension. Physiological symptoms of arousal include increased heart rate and blood pressure, among other changes.

Description

Affect arousal describes the state of feeling awake, activated, and highly reactive to stimuli. There are both psychological and physiological components to the state of arousal. Psychologically, the state of arousal is associated with the subjective experience of feelings including high energy and tension. Physiologically, the body is in a state of relative heightened responsiveness, and is prepared for action through the activation of various neural (limbic) and bodily systems (e.g., the sympathetic nervous system). The state of arousal is usually prompted by external, typically highly emotive, stimuli (such as being in a dangerous situation, or watching a scary movie). Some argue that arousal occurs immediately after exposure to an emotive stimulus; others suggest that exposure to a high arousal stimulus first prompts the person to appraise the personal meaning of the stimulus (Is it congruent or incongruent to my goals? Is it important to my survival?), with arousal experienced as a result of the appraisal.

Psychological and Physiological Components of Arousal

Affective states are typically described in terms of their valence (pleasure-displeasure) and their arousal (high activation-low activation). Thus, psychological arousal is one dimension of our affective response to external stimuli. High activation or arousal is characterized by feelings of energy for pleasurable states (e.g., excitement), or tension for unpleasant states (e.g., fear). These can be distinguished from low arousal states such as calmness and depression. Arousal can also be distinguished from intensity, which is a separate dimension; both high and low arousal states may be experienced more or less intensely. It has been argued that there is a second arousal dimension of sleepiness energy that follows circadian rhythms.

Several systems underlie physiological arousal, including the autonomic nervous system, the reticular activating system, and also the endocrine system, which releases hormones including adrenalin and noradrenalin into the bloodstream. The physiological symptoms of arousal are diverse, and include increases in heart rate, blood pressure, perspiration, respiration rate, muscle tension and metabolic rate, and changes in the electrical activity of the brain – in its regions, hemispheres, and in the connectivity between regions.

Functions of Arousal

High affective arousal prepares the body to respond to stimuli, priming us for "fight or flight." Arousal also influences cognitive processing in ways that may be adaptive for survival. Arousal appears to influence attention, with high arousal stimuli capturing attention more efficiently, thus recruiting more processing and coping resources; this may be why high arousal stimuli are also evaluated and responded to more quickly than low arousal stimuli. Arousal also has implications for memory. Studies have observed enhanced memory for arousing events, although high states of arousal can also cause short-term impairments in retrieval of memories. In addition, retrieval of memories is facilitated when experiencing a similar level of arousal to that during encoding. Arousal may also function to regulate task performance. Most agree that the relationship between arousal and performance takes the form of an inverted U-shaped curve, such that states of either extremely low or extremely high arousal are detrimental to performance.

Individual Differences in Arousal

Individual differences in arousal may underlie core personality traits. According to a theory originally proposed by Eysenck, differences in baseline physiological levels of arousal (i.e., autonomic nervous system and reticular activating systems) cause people to be more or less extraverted. For example, low baseline arousal is associated with high extraversion, because people seek the stimulation they lack from their external environment. Likewise, the trait of neuroticism is thought to reflect a person's threshold for activation in the autonomic nervous system. People with higher levels of neuroticism have a lower threshold, and are therefore less able to inhibit their emotional reactions.

Cross-References

- ▶ Affect
- ▶ Energy
- ▶ Physiological Reactivity

References and Readings

Cannon, W. B. (1932). *The wisdom of the body*. New York: WW Norton.
Russell, J. A. (1980). A circumplex model of affect. *Journal of Personality and Social Psychology, 39*, 1161–1178.
Storbeck, J., & Clore, G. L. (2008). Affective arousal as information: How affective arousal influences judgments, learning, and memory. *Social and Personality Psychology Compass, 2*, 1824–1843.
Strelau, J., & Eysenck, H. J. (1987). *Personality dimensions and arousal*. New York: Plenum Press.
Thayer, R. E. (1978). Toward a psychological theory of multidimensional activation (arousal). *Motivation and Emotion, 2*, 1–34.

Affective Hostility

- ▶ Hostility

Affective Responses

- ▶ Emotional Responses

Affective State

- ▶ Affect
- ▶ Mood

Affiliation

- ▶ Interpersonal Circumplex

Aged

- ▶ Elderly

Age-Related Cognitive Decline

- ▶ Coffee Drinking, Effects of Caffeine

Aggregate Data

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Aggregate measures; Descriptive data; Summary data

Definition

Aggregate measures, or data, are summaries of observations, or measurements, derived from individuals in the group or groups of interest.

This is a wide-ranging term used in many circumstances. The key concept is that data used are not individual measures, but a summary statistic reached by aggregating large amounts of data from individual subjects, geographic regions, socioeconomic classes, etc.

Measures of central tendency are useful aggregate data. Commonly used measures include the arithmetic mean (usually called simply the mean), median, and mode. For example, median family income in a given region, state, or country would be aggregate data. Proportions are also useful, e.g., the proportion of a given identified population that smokes cigarettes.

Cross-References

- ▶ Data
- ▶ Median
- ▶ Mode

Aggregate Measures

▶ Aggregate Data

Aggression

▶ Hostility
▶ Hostility, Psychophysiological Responses

Aging

Barbara Resnick
School of Nursing, University of Maryland, Baltimore, MD, USA

Synonyms

Alter; Changing; Grown; Progress

Definition

Aging relates to the developmental process of growth and senescence over time. *Age-related* refers to how age is taken into account in health and social systems.

Description

Many of the changes associated with aging result from gradual loss. These losses may often begin in early adulthood, but individuals are usually not affected by changes until the loss is fairly extensive. Most organ systems seem to lose function at about 1% a year beginning around age 30 years. The loss of function in an organ, for example, does not become significant until it crosses a given level. Thus the functional performance of an organ in an older person depends on two principal factors: (1) the rate of deterioration and (2) the level of performance needed. It is not surprising then to learn that most older persons will have normal laboratory values, normal heart rate and blood pressure, and normal vision and hearing. It is possible, however, when stressed such as by doing exercise that the heart then does not respond appropriately or when it is dark the eyes do not adjust in a timely fashion. The health care provider must appreciate and utilize the notion of variability in aging to help individuals make lifestyle and treatment choices to optimize their own aging. As noted, changes occur over time but there is no known way in which to predict the rate of decline in any individual. There is, however, much evidence to support the benefit of lifestyle interventions, specifically diet and physical activity that will help to overcome some of the physical changes that can occur with age, and may improve overall health and quality of life. With regard to diet, repeatedly it has been noted that there are protective effects to diets low in saturated fats and high in fruits and vegetables. Likewise, engaging in regular physical activity, at least 30 min daily, has been noted to have not only physical but mental health benefits. Behavior change interventions are critical to facilitate adherence to these behaviors at any point in the lifespan.

It is impossible to address aging without considering the psychosocial aspects that occur in addition to the more visible biological and physical changes. Transitions associated with aging are commonly noted around retirement, loss of a spouse or significant other, pet, home, car, and ability to drive, as well as the loss of sensory function (hearing and vision) or ambulatory ability or capacity. Many fear the loss of independence with age, cognitive decline and worry about having an acute catastrophic problem such as a hip fracture or stroke. Conversely, many older adults are quite resilient in the face of these losses and have much to teach the younger generation on how to respond to loss, optimize remaining function and ability, and adjust.

Recognizing the consequences and anticipated changes that will occur with age are important to help facilitate the process and optimize outcomes in adults as they progress through the lifespan. Critical to the process is adherence to healthy lifestyle behaviors as well as a willingness to

adjust and adapt to the changes that are occurring. In so doing adults can age successfully, regardless of underlying disease or disability.

At this point in time there is still relatively little known about the aging process and how to separate aging and age-related changes from disease. Behavioral medicine can help manage both sources of change, although understanding and knowing the difference is critical so as to optimize outcomes. For example, it is possible that cognitive changes are occurring because of an elevated blood sugar in the individual or a low sodium. Treating this with cognitive interventions may help but will not optimize outcomes as much as combining this treatment with medical management. Conversely, it is critical to avoid treating changes medically when behavioral interventions would result in better and safer outcomes.

Many of the changes associated with aging result from gradual loss. These losses generally begin in young to middle adulthood. Fortunately, however, the changes are not noted until there is a critical mass of cell death or functional change that alters the underlying system. The changes in organ function depend on two principal factors: (1) the rate of deterioration and (2) the level of performance needed. Thus under normal circumstances the older adult may function within normal. However, when he or she undergoes some type of stress the body is not able to compensate and changes are noted in major organs such as the brain, kidney, heart, lungs, or liver. Behavioral interventions can help to optimize response in times of stress by preparing individuals for such these experiences. An older adult who exercises regularly prior to breaking a hip or undergoing a joint replacement will recover quicker than an individual who has not been regularly exercising.

Aging is a complex and multifactorial process that combines life experiences and behaviors and genetics particularly. It is critical to remember, however, that the genetic impact of life span accounts for 35% of its variance, whereas environmental and behavioral factors account for >65% of the variance.

Changes associated with aging are believed to be a combination of normal change over time and disease. Often individuals do not notice aging until they hit a certain threshold of loss or change. For example, the loss of function does not become significant until it crosses a given level. Likewise, the loss of function in an organ such as the liver is not noticed until there is sufficient amount of cell death that functional change occurs. Thus aging is noted based on two principal factors: (1) the rate of deterioration and (2) the level of performance needed. A good example of the impact of aging on the system occurs with regard to cardiovascular function and health. An older individual may have a normal resting pulse and normal cardiac output when engaging in routine daily activities. When he or she tries to exercise, however, the heart rate and cardiac output do not respond in the way that would be anticipated in a younger individual (i.e., neither the pulse or the output increase sufficiently to withstand the activity).

Unfortunately, much of what we know about aging is based on studies doing using cross-sectional samples of individuals of different ages that are compared in terms of group averages. Such an approach generally reveals a gradual decline in organ function with age, beginning in early middle life. A few studies have followed cohorts of people longitudinally as they age. Their conclusions are quite different. When evaluated over time some characteristics and aspects of aging may actually improve rather than decline. Individual variation may be particularly important to aging. Individuals who have been physically active throughout their lifespan will be more likely to recover optimally following a hip fracture, for example, than those who have been sedentary.

Aging is not simply a series of biological changes. It is also an accumulation of life experiences and accrued knowledge. It may also be associated with multiple losses such as the loss of social roles such as work, motherhood, loss of income, loss of friends and relatives. These losses can result in fear of loneliness, financial insecurity, fear of dependency, and fear of one's own

death. Despite these fears and challenges most older adults cope with multiple losses and limitations and enjoy this time in life.

Increasingly it is recognized that aging occurs differently depending on the person. The changing composition of today's older adults compared with that of a generation ago may actually reflect a bimodal shift wherein there are both more disabled people and more healthy older people. We continue to learn more and more about healthy or successful aging through hearing the stories of the growing number of centenarians. Generally the consensus is that moderation in all areas (e.g., food intake, alcohol intake), regular physical activity, and an engaging social life are critical to successful aging.

Cross-References

▶ Older Adult
▶ Successful Aging

References and Readings

American Geriatrics Society. *Geriatrics review syllabus*. Retrieved September, 2011, from https://fulfillment.frycomm.com/ags/grs7_order_form.asp.
Goldsmith, T. C. (2006). *The evolution of aging* (2nd ed.). Annapolis, MD: Azinet Press.

Agonist

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

An agonist is any molecule which binds to a receptor on a cell, which then can potentially lead to subsequent changes in the cell's functions. While agonists activate a process that follows their binding to a receptor, an antagonist inhibits these effects, and an inverse agonist results in opposite effects to those of the agonist. In pharmacology, this issue is pivotal, as certain medications can act as agonists of receptors, where they mimic the effects of a natural compound or ligand that normally binds to the same receptor. However, the synthetic compound can possibly lead to similar cellular changes without unwanted side effects. Receptors can be activated by endogenous agonists – a natural compound which binds to a receptor. In contrast, receptors can also be activated by exogenous agonists – synthetic medications or compounds which activate a receptor. An example of an endogenous agonist is acetylcholine which activates the acetylcholine receptor. One important measure concerning agonists is their affinity to a receptor – the degree to which they structurally and functionally fit a receptor. Consequently, there are full and partial agonists. These influence the subsequent effects of an agonist on a cell's function. Another measure of the efficacy of an agonist is its EC_{50} – the concentration of agonist needed to elicit half the biological response to that agonist. The potency of an agonist is inversely related to the EC_{50} value, as a more potent agonist requires lower concentrations to yield a certain response than a weaker agonist. Multiple agonists are used in research and clinical applications, of relevance to behavioral medicine as well. For example, isoproterenol is a drug that stimulates the sympathetic response since it is an agonist of beta-adrenoreceptors, mimicking the effects of epinephrine (Goodman, Gilman, & Brunton, 2008). Some compounds can have both agonist and antagonist characteristics. An intriguing example is tamoxifen, which is an antagonist of estrogen used in cancer treatment, yet it is also an agonist of breast cancer cells, which induces cell-cycle-related gene activity (Hodges et al., 2003). Thus, agonists reflect a basic biochemical process at cellular levels and play numerous roles in health and disease.

Cross-References

▶ Neurotransmitter

References and Readings

Goodman, L. S., Gilman, A., & Brunton, L. L. (2008). *Goodman and Gilman's manual of pharmacology and therapeutics* (11th ed., p. 14). New York: McGraw-Hill Medical.

Hodges, L. C., Cook, J. D., Lobenhofer, E. K., Li, L., Bennett, L., Bushel, P. R., et al. (2003). Tamoxifen functions as a molecular agonist inducing cell cycle-associated genes in breast cancer cells. *Molecular Cancer Research, 1*, 300–311.

AIDS Prevention

► HIV Prevention

AIDS Wasting

► Cachexia (Wasting Syndrome)

AIDS: Acquired Immunodeficiency Syndrome

Carter A. Lennon
Department of Psychology, University of Connecticut, Center for Health, Intervention & Prevention, Storrs, CT, USA

Synonyms

Human immunodeficiency virus (HIV); Opportunistic infections; Sexually transmitted disease/infection (STD/STI)

Definition

Acquired immunodeficiency syndrome (AIDS) is the final stage of the human immunodeficiency virus (HIV) defined by a marked decline in an individual's immune system. Specifically, when an individual's CD4 lymphocyte/helper T cell count falls under 200 per microliter (μL) of blood, the individual is diagnosed with AIDS (Crooks & Baur, 2005; Shibley Hyde & Delamater, 2008). AIDS is defined as a syndrome because its presence is often accompanied by a grouping of illnesses that signal the progression of HIV to AIDS (Kelly, 2008). These AIDS-defining illnesses, termed opportunistic infections, include, but are not limited to, *Pneumocystis carinii*, Kaposi's sarcoma, cervical cancer, and *Mycobacterium tuberculosis* (Crooks & Baur, 2005; Jekel, Katz, & Elmore, 2001; Shibley Hyde & Delamater, 2008; World Health Organization, 2010b).

Description

History of AIDS

AIDS was first recognized as a disease in 1981 by the Centers for Disease Control and Prevention. It was not until 1984 when Luc Montagnier's team at the Pasteur Institute in France and Robert Gallo's team at the National Institute of Health in the United States discovered that HIV was the cause of AIDS (Shibley Hyde & Delamater, 2008). In the United States, the epidemic was first extensively identified in men who have sex with men (MSM) and was therefore called "gay-related immune deficiency" (GRID; Shilts, 1987). As outbreaks of HIV/AIDS were also seen among injection drug users (IDUs), Haitian immigrants, and hemophiliacs, the disease was renamed AIDS (Shilts, 1987). Around the world, AIDS is mainly a disease affecting heterosexual individuals; in the United States, MSM still comprise the majority of HIV/AIDS cases (Crooks & Baur, 2005).

Epidemiology

HIV/AIDS is largely considered a global pandemic (Crooks & Baur, 2005). It is now the leading cause of death worldwide in women between the ages of 15 and 49 (UNAIDS, 2010a). According to the 2010 UNAIDS Global Report (2010b), there were 1.8 million AIDS-related deaths worldwide in 2009. Sub-Saharan

Africa accounts for the overwhelming majority of these deaths. By region, AIDS-related deaths are as follows (UNAIDS, 2010b):

- Sub-Saharan Africa: 1.3 million (72.2%)
- South and Southeast Asia: 260,000 (14.4%)
- Eastern Europe and Central Asia: 76,000 (4.2%)
- Central and South America: 58,000 (3.2%)
- East Asia: 36,000 (2.0%)
- North America: 26,000 (1.4%)
- Middle East and North Africa: 24,000 (1.3%)
- Caribbean: 12,000 (0.67%)
- West and Central Europe: 8,500 (0.47%)
- Oceania: 1,400 (0.07%)

On the whole, AIDS-related deaths worldwide have stabilized, mainly due to the advent of highly active antiretroviral therapy (HAART) in 1996 (UNAIDS, 2010b). North America and Central and Western Europe have seen a decline in AIDS-related deaths since 1996, while deaths in Sub-Saharan Africa and the Caribbean have been decreasing since 2005 (UNAIDS, 2010b). AIDS-related deaths in Central and South America and parts of Asia have remained constant; however, deaths in Eastern Europe and Central Asia are still increasing (UNAIDS, 2010b). Globally, 70% of infections are transmitted through heterosexual sex, especially in Africa and Asia (Crooks & Baur, 2005). In the United States, the epidemic is still predominantly in MSM, but rates are rising in heterosexuals, especially ethnic minority women (Crooks & Baur, 2005). In Russia, and other parts of Eastern Europe, the epidemic is due mainly to injection drug use.

Diagnosis

AIDS is the final stage of HIV and is diagnosed when the individual's immune system becomes severely compromised. Specifically, a diagnosis of AIDS is given when CD4 levels fall below 200 per microliter (μL) of blood (normal levels are between 600 and 1,200; Crooks & Baur, 2005; Shibley Hyde & Delamater, 2008). On average, HIV progresses to AIDS 8–11 years after contracting HIV (Crooks & Baur, 2005). An AIDS diagnosis is often accompanied by a number of opportunistic infections and other AIDS-related illness. Opportunistic infections are illnesses that are usually not present in humans and signal a severely weakened immune system (Shibley Hyde & Delamater, 2008); these include *Pneumocystis carinii*, Kaposi's sarcoma, toxoplasmosis, advanced cervical cancer, meningitis, encephalitis, and *Mycobacterium* tuberculosis (Crooks & Baur, 2005, Jekel et al., 2001; Maartens, 2008; Shibley Hyde & Delamater, 2008). Other AIDS-related symptoms include severe weight loss ("wasting syndrome"), diarrhea, neurological decline, and infection of most organs (NIAID, 2009b). AIDS is the end stage of HIV and is eventually fatal. However, life can be prolonged with antiretrovirals, which suppress the virus, usually resulting in an increase in CD4 cell count.

Treatment

There is no known cure for HIV/AIDS. In 1996, highly active antiretroviral therapy (HAART) was introduced (Wood, 2008), which combines multiple types of antiretrovirals to suppress HIV viral load in order to stop the progression of AIDS. There are five classes of antiretrovirals (NIAID, 2009a):

1. Reverse transcriptase inhibitors: prevent HIV from replicating in healthy cells
2. Protease inhibitors: block protease, which is used to replicate HIV
3. Fusion/entry inhibitors: block HIV from binding to healthy cells
4. Integrase inhibitors: block integrase, which helps HIV combine its RNA with the healthy cell's DNA
5. Multidrug combination products (HAART): a combination of the above classes of drugs; the World Health Organization currently recommends combining at least three classes of drugs (2010a)

Adherence to these medications is vitally important. Nonadherence can result in resistance to antiretrovirals and a resurgence of the virus in the individual (NIAID, 2010). Adherence is difficult, due to the many side effects that accompany these drugs (Shibley Hyde & Delamater, 2008) and myths that circulate about the dangers of antiretroviral use (Kalichman et al., 2009). It is also important to keep in mind the treatment

of psychological side effects of HIV/AIDS, including depression (Shibley Hyde & Delamater, 2008).

Prevention

There are two types of prevention relevant to HIV/AIDS interventions. Primary prevention is concerned with preventing an HIV− individual from contracting the disease; secondary prevention is concerned with preventing someone who is HIV+ from transmitting the virus to someone who is HIV− (Jekel et al., 2001). Behavioral HIV/AIDS prevention interventions typically include three components: information, motivation, and behavioral skills training (Fisher & Fisher, 2000). Interventions typically address how HIV/AIDS is transmitted and how to prevent it. Known behaviors that can decrease the likelihood of HIV/AIDS transmission include consistent condom use, using clean needles to inject drugs, abstinence, decreasing number of sexual partners, and remaining monogamous (NIAID, 2009c). Additionally, interventions are conducted at multiple levels (individual, dyadic, small group, community, mass media, structural).

The Centers for Disease Control and Prevention (CDC, 2009b) have put forth criteria to define what constitutes an effective behavioral HIV/AIDS prevention intervention. These criteria set guidelines for quality of design, implementation, and data analysis, as well as what constitutes support for intervention effectiveness. In addition to targeting high-risk behaviors (usually sex- or drug-related), the results of these interventions must show (a) a marked decrease in risk behaviors (sex- or drug-related), (b) a decrease in the rate of new infections, and/or (c) an increase in protective behavior (e.g., condom use). To date, there are over 40 best evidence prevention interventions that meet these criteria and can be found on two websites (CDC, 2009a; DEBI, 2010). Known as DEBIs (Diffusion of Effective Behavioral Interventions), these HIV/AIDS prevention interventions are conducted at the individual, group, and/or community level and are targeted toward specific groups (HIV + individuals, HIV − individuals, heterosexuals, MSM, IDUs, males, females, racial minorities, transgender individuals, couples, etc.).

Biologically, using antiretroviral therapy can prevent transmission of HIV/AIDS from mother to child (Shibley Hyde & Delamater, 2008). A vaccine to cure AIDS is not yet available, though research continues. Recently, preexposure prophylaxis (PrEP; CDC, 2010), microbicides (Kelly, 2008; Shibley Hyde & Delamater, 2008), and circumcision (CDC, 2008) have all offered promising results in the prevention of HIV/AIDS. PrEP is a chemoprophylaxis; it is thought that by having antiretrovirals present in the body's system before HIV enters the body, infection can be prevented (CDC, 2010). Microbicides are gels or other substances that can be inserted into the vagina or rectum with the potential to kill HIV before it can enter the individual's body (Shibley Hyde & Delamater, 2008). Finally, circumcision has been repeatedly shown in interventions to decrease the rate of HIV transmission because circumcision decreases the amount of Langerhans cells and skin tears present on the male penis, where HIV can enter the body (CDC, 2008). Research is continuing to make great strides in the fight to prevent and treat HIV/AIDS.

Cross-References

- ▶ Cachexia (Wasting Syndrome)
- ▶ Cancer, Cervical
- ▶ Condom Use
- ▶ HIV Infection
- ▶ HIV Prevention
- ▶ Human Immunodeficiency Virus (HIV)
- ▶ Immune Function
- ▶ Kaposi's Sarcoma
- ▶ Prevention: Primary, Secondary, Tertiary
- ▶ Sexual Risk Behavior

References and Readings

CDC. (2008). *HIV/AIDS fact sheets: Male circumcision and risk for HIV transmission and other health conditions: Implications for the United States*. Retrieved December 2010, from http://www.cdc.gov/hiv/resources/factsheets/circumcision.htm

CDC. (2009a). *Best-evidence interventions*. Retrieved December 2010, from http://www.cdc.gov/hiv/topics/research/prs/best-evidence-intervention.htm#completelist

CDC. (2009b). *PRS efficacy criteria for best-evidence (tier 1) individual-level and group-level interventions (ILIs/GLIs)*. Retrieved December 2010, from http://www.cdc.gov/hiv/topics/research/prs/efficacy_best-evidence_ILIs-GLIs.htm

CDC. (2010). *HIV/AIDS: Pre-exposure prophylaxis (PrEP)*. Retrieved December 2010, from http://www.cdc.gov/hiv/prep/

Crooks, R., & Baur, K. (2005). *Our sexuality* (9th ed.). Belmont, CA: Thomson Wadsworth.

DEBI. (2010). *Diffusion of effective behavioral interventions*. Retrieved December 2010, from: http://effectiveinterventions.org

Fisher, J. D., & Fisher, W. A. (2000). Theoretical approaches to individual-level change in HIV risk behavior. In R. J. DiClemente (Ed.), *Handbook of HIV prevention* (pp. 3–55). Dordrecht: Kluwer.

Jekel, J. F., Katz, D. L., & Elmore, J. G. (2001). *Epidemiology, biostatistics, and preventive medicine* (2nd ed.). Philadelphia: Saunders.

Kalichman, S. C., Amaral, C. M., White, D., Swetsze, C., Pope, H., Kalichman, M. O., et al. (2009). Prevalence and clinical implications of interactive toxicity beliefs regarding mixing alcohol and antiretroviral therapies among people living with HIV/AIDS. *AIDS Patient Care and STDs, 23*, 449–454. doi:10.1089/apc.2008.0184.

Kelly, G. F. (2008). *Sexuality today* (9th ed.). New York: McGraw-Hill.

Maartens, G. (2008). Prevention of opportunistic infections in adults. In S. S. Abdool Karim & Q. Abdool Karim (Eds.), *HIV/AIDS in South Africa* (pp. 454–462). New York: Cambridge University Press.

NIAID. (2009a). *HIV/AIDS: Classes of HIV/AIDS antiretroviral drugs*. Retrieved December 2010, from http://www.niaid.nih.gov/topics/HIVAIDS/Understanding/Treatment/Pages/arvDrugClasses.aspx

NIAID. (2009b). *HIV/AIDS: More on how HIV causes AIDS*. Retrieved December 2010, from http://www.niaid.nih.gov/topics/HIVAIDS/Understanding/howHIVCausesAIDS/Pages/howhiv.aspx

NIAID. (2009c). *HIV/AIDS: Prevention*. Retrieved December 2010, from http://www.niaid.nih.gov/topics/HIVAIDS/Understanding/Prevention/Pages/prevention.aspx

NIAID. (2010). *HIV/AIDS: Adherence and drug resistance*. Retrieved December 2010, from http://www.niaid.nih.gov/topics/HIVAIDS/Understanding/Treatment/Pages/adherence.aspx

Shibley Hyde, J., & Delamater, J. D. (2008). *Understanding human sexuality* (10th ed.). New York: McGraw-Hill.

Shilts, R. (1987). *And the band played on: Politics, people and the AIDS epidemic*. New York: St. Martins Press.

UNAIDS. (2010a). *Fact sheet: Women, girls and HIV*. Retrieved December 2010, from http://data.unaids.org/pub/FactSheet/2010/20100302_fs_womenhiv_en.pdf

UNAIDS. (2010b). *Global report: UNAIDS report on the global AIDS epidemic 2010*. Retrieved December 2010, from http://www.unaids.org/globalreport/Global_report.htm

Wood, R. (2008). Antiretroviral therapy. In S. S. Abdool Karim & Q. Abdool Karim (Eds.), *HIV/AIDS in South Africa* (pp. 504–523). New York: Cambridge University Press.

World Health Organization. (2010a). *HIV/AIDS: Antiretroviral therapy*. Retrieved December 2010, from: http://www.who.int/hiv/topics/treatment/en/index.html

World Health Organization. (2010b). *HIV/AIDS: Online Q&A*. Retrieved December 2010, from http://www.who.int/features/qa/71/en/index.html

All

▶ Angiotensin

Alcohol

▶ Binge Drinking

Alcohol Abuse

▶ Lifestyle Changes

Alcohol Abuse and Dependence

Kelly S. DeMartini[1] and Kristin L. MacGregor[2]
[1]Division of Substance Abuse, School of Medicine, Yale University, New Haven, CT, USA
[2]Department of Psychology, Syracuse University, Syracuse, NY, USA

Synonyms

Substance abuse; Substance dependence

Definition

Alcohol abuse (AA), as defined by the DSM-IV, is characterized by a maladaptive pattern of alcohol use that leads to clinically significant impairment or distress, manifesting in at least one of the following recurrent symptoms: failure to fulfill major role obligations at work, school, and/or home; use in situations in which it would be physically hazardous (e.g., while driving); alcohol-related legal problems; and/or interpersonal problems associated with alcohol use (American Psychiatric Association [APA], 2000). The most commonly endorsed symptom of alcohol abuse is use in physically hazardous situations, and the most common situation is driving while under the influence (Harford, Grant, Yi, & Chen, 2005; Hasin & Paykin, 1999). A diagnosis of AA, therefore, is characterized not by obtaining a certain level of alcohol consumption but by the experience of alcohol-related problems. It is a widespread problem in the United States that has estimated 12-month and lifetime prevalence rates of 4.7% and 17.8%, and prevalence rates have increased steadily over the past two decades (Grant et al., 2004; Hasin, Stinson, Ogburn, & Grant, 2007). It is most prevalent in younger, white, unmarried men, yet there is evidence to suggest that this gender difference is decreasing in younger age cohorts. AA often co-occurs with other mental health disorders, including other substance abuse, nicotine dependence, panic disorder, depression, and bipolar disorder (Swendsen et al., 2010; Zvolensky, Bernstein, Marshall, & Feldner, 2006). The existence of preexisting mental health disorders predicts the transition from regular alcohol use to AA, and the odds of developing AA greatly increase when three or more mental health disorders are present (Swendsen et al., 2010).

Alcohol dependence (AD), as defined by the DSM-IV, is characterized by a maladaptive pattern of alcohol use leading to clinically significant impairment or distress. The pattern is manifested by the existence of three or more of the following symptoms: tolerance; withdrawal; using alcohol in larger amounts or over a longer period of time than intended; persistent desire or unsuccessful efforts to cut down; spending a great deal of time to obtain, use, or recover from alcohol; giving up social, occupational, or recreational activities because of alcohol use; continuing alcohol use despite knowledge of a persistent or recurrent physical or psychological problem that is likely to have been caused by alcohol. AD can occur with or without physiological dependence. Once a diagnosis of AD exists, a person cannot again meet criteria for AA (APA, 2000). Epidemiological estimates from 2001 to 2002 indicate that the 12-month prevalence rate of AD is 3.81%, which is a decrease from the 1991 to 1992 12-month prevalence estimate of 4.38% (Hasin et al., 2007). Rates of AD are higher in males (5.42%) than females (2.32%). Whites, Native Americans, and Hispanics have higher rates of AD than Asians. Lifetime prevalence of AD is estimated to be 12.5% (Hasin et al., 2007). The clinical course of AD includes fluctuations in the intensity of difficulties with alcohol and a high likelihood of recurrent cycles of abstinence that last several months or more followed by recurrent use (see Schuckit, Tipp, Smith, & Bucholz, 1997). Broadly, interpersonal and occupational problems, as well as tolerance, escalate by the mid- to late 20s, and more serious medical problems and alcohol-related abstinence syndromes are experienced by the mid-30s to early 40s (Schuckit, Daeppen, Tipp, Hesselbrock, & Bucholz, 1998). Those with physiological dependence experience more binge drinking, alcohol-related problems, physiological complications, and more alcohol-related psychiatric problems (Schuckit et al., 1998).

References and Readings

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.

Cassidy, F., Ahearn, E. P., & Carroll, B. J. (2001). Substance abuse in bipolar disorder. *Bipolar Disorder, 3*(4), 181–188.

Cosci, F., Schruers, K. R. J., Abrams, K., & Griez, E. J. L. (2007). Alcohol use disorders and panic disorder: A review of the evidence of a direct relationship. *Journal of Clinical Psychiatry, 68*, 874–880.

Grant, B. F., Dawson, D. A., Stinson, F. S., Chou, S. P., Dufour, M. C., & Pickering, R. P. (2004). The 12-month prevalence and trends in DSM-IV alcohol abuse and dependence: United States, 1991–1992 and 2001–2002. *Drug and Alcohol Dependence, 74*, 223–234.

Harford, T. C., Grant, B. F., Yi, H. Y., & Chen, C. M. (2005). Patterns of DSM-IV alcohol abuse and dependence criteria among adolescents and adults: Results from the 2001 National Household Survey on drug abuse. *Alcoholism: Clinical and Experimental Research, 29*, 810–828.

Hasin, D., & Paykin, A. (1999). Alcohol dependence and abuse diagnoses: Concurrent validity in a nationally representative sample. *Alcoholism: Clinical and Experimental Research, 23*, 144–150.

Hasin, D. S., Stinson, F. S., Ogburn, E., & Grant, B. F. (2007). Prevalence, correlates, disability, and comorbidity of DSM-IV alcohol abuse and dependence in the United States: Results from the National Epidemiologic Survey on alcohol and related conditions. *Archives of General Psychiatry, 64*(7), 830–842.

Kessler, R. C., Berglund, A., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of *DSM-IV* disorders in the national comorbidity survey replication. *Archives of General Psychiatry, 62*, 593–602.

Keyes, K. M., Grant, B. F., & Hasin, D. S. (2008). Evidence for a closing gender gap in alcohol use, abuse, and dependence in the United States population. *Drug and Alcohol Dependence, 93*, 21–29.

Schuckit, M. A., Daeppen, J. B., Tipp, J. E., Hesselbrock, M., & Bucholz, K. K. (1998). The clinical course of alcohol-related problems in alcohol dependent and nonalcohol dependent drinking women and men. *Journal of Studies on Alcohol, 59*, 581–590.

Schuckit, M. A., & Smith, T. L. (2001). The clinical course of alcohol dependence associated with a low level of response to alcohol. *Addiction, 96*, 903–910.

Schuckit, M. A., Smith, T. L., Daeppen, J., Eng, M., Hesselbrock, V. M., Nurnberger, J. I., et al. (1998). Clinical relevance of the distinction between alcohol dependence with and without a physiological component. *American Journal of Psychiatry, 155*, 733–740.

Schuckit, M. A., Tipp, J. E., Smith, T. L., & Bucholz, K. K. (1997). Periods of abstinence following the onset of alcohol dependence in 1,853 men and women. *Journal of Studies on Alcohol, 58*, 581–589.

Swendsen, J., Conway, K. P., Degenhardt, L., Glantz, M., Jin, R., Merikangas, K. R., et al. (2010). Mental disorders as risk factors for substance use, abuse and dependence: Results from the 10-year follow-up of the National Comorbidity Survey. *Addiction, 105*, 1117–1128.

Zvolensky, M. J., Bernstein, A., Marshall, E. C., & Feldner, M. T. (2006). Panic attacks, panic disorder, and agoraphobia: Associations with substance use, abuse, and dependence. *Current Psychiatry Report, 8*, 279–285.

Alcohol Consumption

Susan E. Collins and Megan Kirouac
Department of Psychiatry and Behavioral Sciences, University of Washington, Harborview Medical Center, Seattle, WA, USA

Synonyms

Alcohol use; Drinking

Definition

Alcohol consumption, as the term is used in clinical and research applications, refers to the act of ingesting – typically orally – a beverage containing ethanol. Ethyl alcohol or ethanol (CH_3CH_2OH) is the only type of alcohol that is safe for human consumption. Other types of alcohol, such as isopropyl and methyl alcohol, are toxic and potentially lethal. Alcoholic beverages that are typically consumed may include beer, wine, distilled spirits, and beverages that contain combinations of these or other additives, including malt liquor, fortified wine, liqueur, and cordials.

Description

Relevance to Behavioral Medicine

Alcohol consumption is an important construct in behavioral medicine because alcohol is a psychoactive substance that affects the body in various ways. In addition to its acute effects, it can have longer-term medical, psychiatric/psychological, social, economic, and occupational effects on individuals, families, communities, and society at large.

Cultural and Historical Factors Influencing Alcohol Consumption

Alcoholic beverages have been crafted and consumed for millennia. Alcohol consumption patterns have, however, varied widely depending on

broader macrofactors, such as historical time, cultural context, and geopolitical forces, as well as microfactors such as individuals' gender, family background, local environment, religion, and socioeconomic position (Edwards, 2000; Gately, 2008). Alcohol consumption is often regulated by law and may be shaped by cultural norms, values, and local knowledge. Some early cultures and developing communities have relied on alcoholic beverages to quench thirst when clean water was unavailable, to supplement their diet, and to stave off hunger when food was scarce (Blocker, 2006; Denning, 2000; Martin, 2006; Molamu & Macdonald, 1996). Consequently, alcohol has been provided in rations as a form of payment to workers throughout history, from ancient Egyptian builders to feudal European serfs to US troops during the Vietnam War (Gately, 2008). Alcohol consumption has also played key roles in religious and spiritual ceremonies, social celebrations, and medicine (Gately, 2008). Recent statistics from the World Health Organization (World Health Organization [WHO], 2004) suggest that per capita alcohol consumption is currently highest in certain areas in Europe (see Fig. 1 for per capital alcohol consumption in various areas of the world).

Neurobiological Effects of Alcohol Consumption

When alcohol is consumed, it is absorbed into the bloodstream via the stomach lining and small intestines, and then crosses the blood–brain barrier to affect the central nervous system. Its effects are dose dependent and include changes in memory, cognition, perception, coordination, and emotion (Dodgen & Shea, 2000). Table 1 shows possible effects of alcohol at different blood alcohol levels. These changes in the brain likely result from ethanol's effect on dopamine, acetylcholine, serotonin, NMDA, and GABA receptors (Shuckit, 2000). Studies have suggested that the activation of $GABA_A$, a specific GABA subtype, and decreased NMDA glutaminergic neurotransmission both lead to the increased sedation and decreased anxiety that are hallmarks of alcohol intoxication (Nestler & Self, 2010).

Alcohol Metabolism

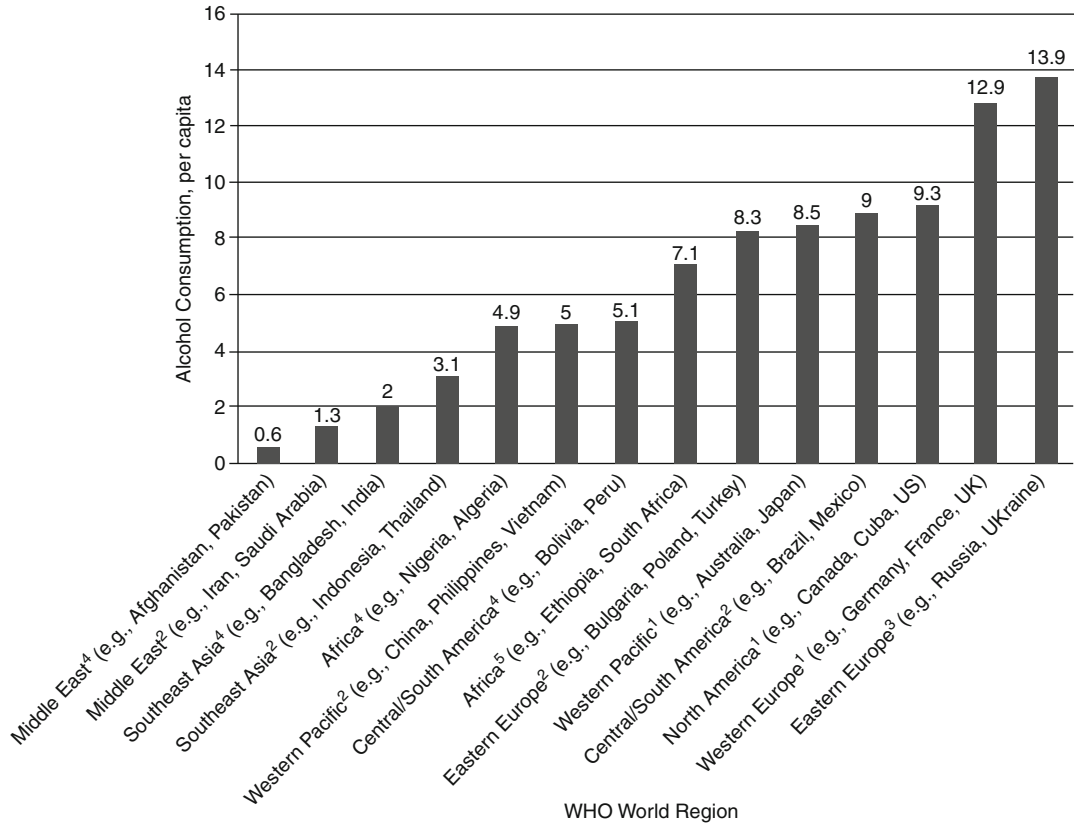
Alcohol is metabolized in the liver, where an enzyme called alcohol dehydrogenase (ADH) transforms it into acetaldehyde (CH_3CHO). Acetaldehyde is a toxic compound that is, in turn, quickly metabolized by another enzyme, aldehyde dehydrogenase (ALDH), into a less toxic compound called acetate. Acetate is finally broken down into water and carbon dioxide by various other tissues and eliminated from the body. The toxic compound, acetaldehyde, is believed to play a causal role in some alcohol-related morbidity, such as liver cirrhosis and cancer (Zakhari, 2006).

Subjective Effects of Alcohol Consumption

Although pharmacological research has indicated that alcohol is a depressant, alcohol consumption has been associated with subjectively perceived biphasic effects (Winger, Woods, & Hofmann, 2004). Thus, drinkers may report experiencing feelings of stimulation and euphoria during the ascending curve of their blood alcohol level. At higher levels of alcohol consumption (blood alcohol levels $>.08$) and/or during the descent of the blood alcohol curve, drinkers may report experiencing more depressant effects of alcohol, including sedation and/or dysphoria. Drinkers with lower sensitivity to these depressant effects of alcohol may be at greater risk for alcohol-use disorders (Schuckit & Smith, 2000). Although recent research has indicated some support for these biphasic effects, it has also shown that subjective experiences of alcohol effects vary widely and warrant further study (Morean & Corbin, 2009).

Consequences Associated with Alcohol Consumption

According to the WHO (2004), alcohol consumption is responsible for 3.2% of deaths recorded worldwide and a global loss of 58.3 million disability-adjusted life years. The alcohol-related disease burden is precipitated in part by acute intoxication, which is known to decrease reaction time, perceptual/motor skills, and inhibitions and is thereby associated with increased risk for traffic accidents, self-inflicted injury/suicide, falls,



Alcohol Consumption, Fig. 1 Numbers are based on WHO (2004) data. Alcohol consumption data are represented in liters of pure alcohol consumed per capita (inhabitants ages 15 and older) based on both recorded and unrecorded estimates. Statistics are grouped by approximate geographical regions and adult and child mortality rates

drownings, alcohol poisoning, and interpersonal violence. Longer-term effects of alcohol consumption also contribute to the disease burden by way of various medical conditions (e.g., cancer, cardiovascular disease, and liver cirrhosis) and psychiatric disorders (e.g., depression; alcohol dependence and abuse).

While the global alcohol-related disease burden is considerable, low-to-moderate alcohol consumption has been shown to have protective effects against cardiovascular heart disease, ischemic stroke, diabetes, and gallstones. "Moderate" alcohol consumption has been variously defined across cultures. According to a report on national health agency guidelines in over 30 countries, moderate alcohol consumption guidelines range from 14 g (one standard drink) to 70 g (approximately five standard drinks) per day (International Center for Alcohol Policies, 2003). According to US measurement standards, a "standard drink" refers to a 12 oz of beer, 5 oz of wine, or 1.5 oz of distilled spirits (National Institutes on Alcoholism and Alcohol Abuse [NIAAA], 2005). US guidelines distinguish between (a) "moderate drinking," which is defined as daily alcohol consumption $\leq$1 standard drink for women and $\leq$2 for men (US Department of Agriculture & US Department of Health and Human Services, 2010); and (b) "low-risk drinking," which is defined as consuming $\leq$3 standard drinks a day and $\leq$7 per week for women or $\leq$4 per day and $\leq$14 per week for men (National Institutes on Alcoholism and Alcohol Abuse [NIAAA], 2005).

Alcohol Consumption, Table 1 Possible and/or common effects of alcohol consumption at various blood alcohol levels

Blood alcohol level	Possible and/or common effects
.02	Subtle effects that may be detected with special tests
.04	Effects of intoxication, especially among people with lower alcohol tolerance
.08	Relaxation, concentration difficulties and impaired judgment about one's own capabilities
.10	Nausea, slurred speech and decreased reasoning and depth perception
.20	Impaired balance and movement; increased risk for memory loss (blackouts) and accidental injury
.30	Extreme physical and cognitive impairment. Memory loss (blackouts) and alcohol poisoning are common among young adults
.40	Loss of consciousness; increased risk for alcohol poisoning and alcohol-induced coma
.45	Median lethal dose (LD_{50} = .45)

Assessment of Alcohol Consumption

Measuring alcohol consumption and its potential effects on an individual is an important and challenging task for health-care professionals that often requires triangulation among self-report measures, diagnostic interviews, behavioral observation, psychological testing, examination of archival data, collection of collateral data, and biological measurement. Measures used should evince adequate reliability and validity, and if applicable, sensitivity and specificity. Screening measures such as the 4-item CAGE (Mayfield, McLeod, & Hall, 1974), 25-item Michigan Alcoholism Screening Test (MAST; Selzer, 1971), and Alcohol Use Disorder Identification Test (AUDIT; Babor, Higgins-Biddle, Saunders, & Monteiro, 1991) are often used to indicate the need for further questioning regarding alcohol consumption. Health-care providers should then inquire about the quantity and frequency of alcohol consumption during typical and peak drinking occasions within a clinically relevant time period to document current use. Quantity and frequency questions also assess for potential heavy-drinking episodes, which put a people at increased risk for alcohol-related negative consequences (e.g., four or more for women and five or more for men on a single drinking occasion; Wechsler et al., 2002). Retrospective (e.g., Timeline Followback; Sobell & Sobell, 1992) or prospective (drinking diary) measures may be used to document daily drinking over time and thereby identify individuals' longitudinal drinking patterns. Assessment of drinking patterns is important because research has indicated that alcohol consumption – even among heavier drinkers – may be best conceptualized as fluid and dynamic (Klingemann, Sobell, & Sobell, 2010; Pandina & Johnson, 2005).

The potential negative psychological effects of alcohol consumption may be assessed using structured diagnostic interviews, such as the Structured Clinical Interview for the DSM-IV-TR (SCID; First, Spitzer, Gibbon, & Williams, 2002) or the WHO's Composite International Diagnostic Interview (CIDI; World Health Organization [WHO], 1990), which systematically assess the lifetime and current presence of alcohol-use disorders (e.g., hazardous drinking, alcohol dependence, abuse, alcohol withdrawal, and/or intoxication) according to either DSM-IV-TR or ICD-10 criteria. Finally, negative physiological effects of alcohol consumption may be assessed using blood tests to detect elevated liver enzymes (GGT, ALT, AST), increased red blood cell size (MCV), and/or carbohydrate-deficit transferrin (CDT), which, taken together, may indicate damage due to heavy alcohol use (Warner & Sharma, 2009).

Cross-References

- ▶ Addictive Behaviors
- ▶ Alcohol Abuse and Dependence
- ▶ Binge Drinking
- ▶ Health Risk (Behavior)
- ▶ National Institute on Alcohol Abuse and Alcoholism

References and Readings

Babor, T. F., Higgins-Biddle, J. C., Saunders, J. B., & Monteiro, M. G. (1991). *The alcohol use disorders identification test: Guidelines for use in primary care* (2nd ed.). Geneva, Switzerland: World Health Organization.

Blocker, J. S. (2006). Kaleidoscope in motion: Drinking in the United States, 1400–2000. In M. P. Holt (Ed.), *Alcohol: A social and cultural history* (pp. 225–240). Oxford, UK: Berg.

Denning, P. (2000). *Practicing harm reduction psychotherapy: An alternative approach to addictions*. New York: Guilford Press.

Dodgen, C. E., & Shea, W. M. (2000). Clinical pharmacology and clinical epidemiology of psychoactive substances. In *Substance use disorders: Assessment and treatment* (pp. 1–28). San Diego, CA: Academic Press.

Edwards, G. (2000). *Alcohol: The world's favorite drug*. London: Penguin.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (2002). *Structured clinical interview for DSM-IV-TR Axis I disorders, research version, patient edition (SCID-I/P)*. New York: Biometrics Research, New York State Psychiatric Institute.

Gately, I. (2008). *Drink: A cultural history of alcohol*. New York: Gotham Books.

International Center for Alcohol Policies. (2003). *ICAP reports 14: International drinking guidelines*. Washington, DC: Author.

Klingemann, H. K.-H., Sobell, M. B., & Sobell, L. C. (2010). Continuities and changes in self-change research. *Addiction, 105*, 1510–1518. doi:10.1111/j.1360-0443.2009.02770.x.

Martin, A. L. (2006). Drinking and alehouses in the diary of an English mercer's apprentice, 1663–1674. In M. P. Holt (Ed.), *Alcohol: A social and cultural history* (pp. 93–106). Oxford, UK: Berg.

Mayfield, D., McLeod, G., & Hall, P. (1974). CAGE questionnaire: Validation of a new alcoholism screening instrument. *American Journal of Psychiatry, 131*, 1121–1123.

Molamu, L., & Macdonald, D. (1996). Alcohol abuse among the Basarwa of the Kgalagadi and Ghanzi districts of Botswana. *Drugs: Education, Prevention, and Policy, 3*, 145–152.

Morean, M. E., & Corbin, W. R. (2009). Subjective response to alcohol: A critical review of the literature. *Alcoholism: Clinical and Experimental Research, 34*, 385–395. doi:10.1111/j.1530-0277.2009.01103.x.

National Institutes on Alcoholism and Alcohol Abuse. (2005). *Helping patients who drink too much: A clinician's guide updated* (2005th ed.). Bethesda, MD: Author.

Nestler, E. J., & Self, D. W. (2010). Neuropsychiatric aspects of ethanol and other chemical dependencies. In S. C. Yudofsky & R. E. Hales (Eds.), *Essentials of neuropsychiatry and behavioral neurosciences* (2nd ed.). Arlington, VA: American Psychiatric Publishing.

Pandina, R. J., & Johnson, V. L. (2005). Lifespan development and drugs. In R. H. Coombs (Ed.), *Addiction counseling review: Preparing for comprehensive, certification and licensing* (pp. 105–128). Mahwah, NJ: Lawrence Erlbaum Associates.

Schuckit, M. A., & Smith, T. L. (2000). The relationships of a family history of alcohol dependence, a low level of response to alcohol and six domains of life functioning to the development of alcohol use disorders. *Journal of Studies on Alcohol, 61*, 827–835.

Selzer, M. L. (1971). The Michigan Alcoholism Screening Test (MAST): The quest for a new diagnostic instrument. *American Journal of Psychiatry, 127*, 1653–1658.

Shuckit, M. A. (2000). *Drug and alcohol abuse: A clinical guide to diagnosis and treatment* (5th ed.). New York: Kluwer Academic/Plenum.

Sobell, L. C., & Sobell, M. B. (1992). Timeline followback: A technique for assessing self-reported ethanol consumption. In J. Allen & R. Z. Litten (Eds.), *Measuring alcohol consumption: Psychosocial and biological methods* (pp. 41–72). Totowa, NJ: Humana Press.

US Department of Agriculture, US Department of Health and Human Services. (2010). *Dietary Guidelines for Americans, 2010* (7th ed.). Washington, DC: US Government Printing Office.

Warner, E. A., & Sharma, N. (2009). Laboratory diagnosis. In R. K. Ries, S. C. Miller, D. A. Fiellin, & R. Saitz (Eds.), *Principles of addiction medicine*. Philadelphia: Lippincott Williams & Wilkins.

Wechsler, H., Lee, J. E., Kuo, M., Seibring, M., Nelson, T. F., & Lee, H. (2002). Trends in college binge drinking during a period of increased prevention efforts. *Journal of American College Health, 50*, 203–217.

WHO. (1990). *Composite International Diagnostic Interview (CIDI)*. Geneva, Switzerland: Author.

WHO. (2004). *Global status report on alcohol 2004*. Geneva, Switzerland: Author. Retrieved from http://www.who.int/substance_abuse/publications/global_status_report_2004_overview.pdf

Winger, G., Woods, J. H., & Hofmann, F. G. (2004). Depressants of the central nervous system: Alcohol, barbiturates, and benzodiazepines. In G. Winger, J. H. Woods, & F. G. Hofmann (Eds.), *A handbook on drug and alcohol abuse: The biomedical aspects* (4th ed., pp. 55–80). Oxford, UK: Oxford University Press.

Zakhari, S. (2006). Overview: How is alcohol metabolized by the body? *Alcohol Research & Health, 29*, 245–254.

Alcohol Use

► Alcohol Consumption

Alertness

▶ Coffee Drinking, Effects of Caffeine

Allele

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

Alleles are alternate forms of a gene, with any given gene having one or more alleles. While humans, for example, share the same genes, each human does not have an identical pattern of alleles. If the two alleles at a particular locus are the same, that locus is homozygous; if they are different, it is heterozygous. Alleles influence phenotypes and contribute to the differences between individuals. Alleles for genes occur in pairs.

Imagine that a specific gene has two alleles, represented by the upper case letter G and the lower case letter g. At a certain address on each of two homologous chromosomes, there will be one of these alleles. There are four possible combinations:
G and G
G and g
g and G
g and g

The combination of the two alleles determines what phenotype results from a particular pairing of alleles. If the allele G is dominant (and the allele g is therefore recessive), the phenotype encoded by the G allele will occur for each of the first three combinations. Only in the fourth combination, where the recessive allele is present on both chromosomes, will the phenotype encoded by the g allele occur. The terms autosomal dominant inheritance and autosomal recessive inheritance are used in this context.

Cross-References

▶ Chromosomes
▶ Dominant Inheritance
▶ Gene
▶ Recessive Inheritance

References and Readings

Britannica. (2009) *The Britannica guide to genetics* (Introduction by Steve Jones). Philadelphia: Running Press.
Edelson, E. (1999). *Gregor Mendel and the roots of genetics*. New York: Oxford University Press.

Allele Heterogeneity

Abanish Singh
Duke University Medical Center, Durham, NC, USA

Definition

The complement of an individual's genes is known as the genotype, and observable traits such as physical characteristics (height, weight, skin color and eye color, etc.) and disease status such as diabetes and heart disease are known as phenotypes. A process known as natural selection provides a mechanism of evolution along with mutation, migration, and genetic drift in DNA. Mutations cause changes in genetic code and create diversity or allelic variation in genotypes. The alteration in DNA sequence at a single nucleotide is known as single nucleotide polymorphism (SNP). Though often it was believed that a phenotype is caused by a single SNP or a cluster of SNPs in linkage disequilibrium, it remains no more a rule. There have been several lines of evidence that different SNPs within a gene can cause the same phenotype. This phenomenon of different mutations at a single locus causing the same phenotype is known as allelic heterogeneity.

There are several examples where allelic heterogeneity has been observed, such as β-thalassemias, in which several mutations in β-globin locus cause the disease phenotype. Different mutations in dystrophin locus causes Duchenne dystrophy, and multiple mutations in CFTR cause cystic fibrosis.

Cross-References

- ▶ Genotype
- ▶ Phenotype
- ▶ Single Nucleotide Polymorphism (SNP)

References and Readings

1. Cystic Fibrosis (CF). (2010). In: *Online mendelian inheritance in man, OMIM*. Baltimore: Johns Hopkins University Press. Accessed June 9, 2010, from http://www.ncbi.nlm.nih.gov/omim/219700. Updated April 29, 2010.
2. Faucz, F. R., Souza, D. A. S., Olandoski, M., & Raskin, S. (2010). CFTR allelic heterogeneity in Brazil: Historical and geographical perspectives and implications for screening and counseling for cystic fibrosis in this country. *Journal of Human Genetics, 55*, 71–76. doi:10.1038/jhg.2009.123.

Allergy: Behavioral Treatment, Risk Factors, Psychosocial Aspects

Yori Gidron
Faculty of Medicine and Pharmacy,
Free University of Brussels (VUB), Jette,
Belgium

Definition

Allergies are a group of immune-mediated diseases characterized by excessive inflammatory responses to otherwise innocuous environmental compounds. Allergies include excessive white blood cell recruitment, due to elicitation of immunoglobulin E, upon exposure to such compounds. Allergies include allergic rhinitis, asthma, hay fever, eczema, food sensitivities, and hypersensitivities to insect bites (Kay, 2000). Allergic disorders are highly prevalent, and some estimate that 25% of children are affected by them (Torres-Borrego, Molina-Terán, & Montes-Mendoza, 2008). Symptoms of allergic rhinitis include excessive sneezing, tearing, runny nose, and itching nose, throat, eyes, and ears. Symptoms of asthma include wheezing, coughing, shortness of breath, and chest pain and tightness. Symptoms of food allergies are very heterogeneous and include swelling or tingling in the mouth and lips, swelling in other body parts, wheezing and nasal congestion, breathing problems, abdominal pain, nausea, vomiting or diarrhea, dizziness, and in rare occasions shock or fainting.

No clear consensus exists for their risk factors, but these may include genetic predisposition and exposure to pollution (e.g., diesel fuel). Several studies have proposed that psychosocial factors increase the risk of various types of allergies including asthma. Yet, many studies were cross-sectional, making the inferential validity questionable. One important research has also shown that family (parental) stress synergistically interacts with exposure to traffic pollution in prospectively predicting new onset of asthma: the effects of pollution on asthma onset were stronger and significant only in children with parental stress (Shankardass et al., 2009). Furthermore, allergies have profound effects on patients' physical, psychological, and social dimensions of quality of life.

A few meta-analyses were conducted testing the overall effects of psychological interventions on asthma. While some promising effects emerged on medication needs, other effects were weaker, and study quality and heterogeneity did not enable to draw firm conclusions (Yorke, Fleming, & Shuldham, 2007). Clearly, the role of psychosocial factors in allergies is an important domain for further investigation, given the high prevalence and impact of such health problems.

Cross-References

- ▶ Asthma and Stress
- ▶ Asthma: Behavioral Treatment

References and Readings

Kay, A. B. (2000). Overview of 'allergy and allergic diseases: with a view to the future'. *British Medical Bulletin, 56*, 843–864.

Shankardass, K., McConnell, R., Jerrett, M., Milam, J., Richardson, J., & Berhane, K. (2009). Parental stress increases the effect of traffic-related air pollution on childhood asthma incidence. *Proceedings of the National Academy of Sciences of the United States of America, 106*, 12406–12411.

Torres-Borrego, J., Molina-Terán, A. B., & Montes-Mendoza, C. (2008). Prevalence and associated factors of allergic rhinitis and atopic dermatitis in children. *Allergologia et Immunopathologia, 36*, 90–100.

Yorke, J., Fleming, S. L., & Shuldham, C. (2007). Psychological interventions for adults with asthma: A systematic review. *Respiratory Medicine, 101*, 1–14.

Allostasis, Allostatic Load

Yoshiharu Yamamoto
Educational Physiology Laboratory, Graduate School of Education The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Definition

Allostasis: Achieving stability through change; the ability to adapt successfully to the challenges of daily life by feedforward mechanisms to maintain viability, emphasizing the biological imperative that "an organism must vary all the parameters of its internal milieu and match them appropriately to environmental demands" (Sterling & Eyer, 1988). This is an extension of homeostasis, i.e., stability through constancy, maintaining constancy of a vital variable by sensing its deviation from a set point and providing feedback to correct the error. Allostasis describes mechanisms that change the variable by predicting what level will be needed and then overriding local feedback to meet anticipated demand (Sterling, 2004). As such mechanisms require higher brain functions, in most cases, the allostasis deals with cephalic involvement in systemic physiological regulation, including behavioral and/or psychosocial impact. Feedforward mechanisms associated with fear, anxiety, addiction, etc., are typical examples. The neuroendocrine system, autonomic nervous system, and immune system are the primary mediators. When they are in a state of heightened activity, this is referred to as an allostatic "state."

Allostatic Load: A measure of the cumulative burden that reflects the continued operation of the allostatic state or over-activation of allostatic responses. When the adaptive responses to challenge lie chronically outside normal operating ranges, wear and tear on regulatory systems occurs, and the allostatic load accumulates as a "cost" of adaptation (McEwen, 1998). Best known and studied is the effect of the primary hormonal mediators of the stress response, glucocorticoids and catecholamines, where in the short term, they are essential for adaptation, maintenance of homeostasis, and survival, but over longer periods of time, they exact a cost that can accelerate disease processes (McEwen, 2000). The resultant secondary outcomes associated with increased risk of diseases include neuronal atrophy or hippocampal loss, atherosclerotic plaques, abdominal fat deposition, left ventricular hypertrophy, glycosylated hemoglobin, high cholesterol, low high-density lipoprotein, and chronic pain and fatigue associated with imbalance of immune mediators (McEwen, 2004). This diversity of the secondary outcomes – which demarcates the allostatic load from Selye's general adaptation syndrome – sharing the primary mediators is considered to explain the presence of a variety of comorbidity patterns of chronic illnesses (e.g., depression and diabetes, colon cancer and coronary heart disease, depression and cardiovascular disease), especially in the elderly. Thus, the allostatic load, if successfully measured, is expected to be an early warning system of biomarkers that can signal early signs of dysregulation across multiple biological systems (Singer, Ryff, & Seeman, 2004). Various attempts have been made to quantify the cumulative burden by accounting for abnormalities in the primary mediators (e.g., urinary cortisol and catecholamines) and the secondary outcomes (e.g., blood pressure and glycosylated hemoglobin), but there is as yet no established measure for the allostatic load. This is partially because

the primary mediators also vary dramatically as normal physiological responses in the allostatic state, and profiling the secondary outcomes tends to focus on specific, not diverse, pathophysiology. The characterization of the allostatic load (and state) to overcome these limitations is the future challenge.

Cross-References

- ▶ Homeostasis
- ▶ Stress Response

References and Readings

McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine, 338*, 171–179.

McEwen, B. S. (2000). Allostasis and allostatic load: Implications for neuropsychopharmacology. *Neuropsychopharmacology, 22*, 108–124.

McEwen, B. S. (2004). Protective and damaging effects of the mediators of stress and adaptation: Allostasis and allostatic load. In J. Schulkin (Ed.), *Allostasis, homeostasis, and the cost of physiological adaptation*. New York: Cambridge University Press.

Singer, B., Ryff, C. D., & Seeman, T. (2004). Operationalizing allostatic load. In J. Schulkin (Ed.), *Allostasis, homeostasis, and the cost of physiological adaptation*. New York: Cambridge University Press.

Sterling, P. (2004). Principles of allostasis: Optimal design, predictive regulation, pathophysiology, and rational therapeutics. In J. Schulkin (Ed.), *Allostasis, homeostasis, and the cost of physiological adaptation*. New York: Cambridge University Press.

Sterling, P., & Eyer, J. (1988). Allostasis: A new paradigm to explain arousal pathology. In S. Fisher & J. Reason (Eds.), *Handbook of life stress, cognition, and health*. New York: Wiley.

Alpha-Amylase

Urs M. Nater
Department of Psychology
University of Marburg, Marburg, Germany

Description

Salivary measures have become increasingly important in behavioral medicine. Substances such as the hormone cortisol or the immune parameter salivary IgA can be measured in saliva as meaningful markers for various normal and pathological processes in the body. The salivary enzyme alpha-amylase (sAA) has been suggested to reflect stress-related changes in the body. Its secretion is known to be elicited by activation of the autonomic nervous system which controls the salivary glands.

Salivary alpha-amylase (a-1,4-a-D-glucan 4-glucanohydrolase; EC 3.2.1.1) is one of the most important enzymes in saliva. It accounts for 40–50% of the total salivary gland-produced protein, most of the enzyme being synthesized in the parotid glands (80% of the total). It is a calcium-containing metalloenzyme that hydrolyzes the a-1,4 linkages of starch into glucose and maltose. However, sAA has also been shown to have an important bacterial interactive function.

The components of saliva are primarily produced by acinar cells. Acinar cells are innervated by both the sympathetic and the parasympathetic branches of the autonomic nervous system (ANS). The afferent pathways for the transmission of taste are via the facial and glossopharyngeal nerves to a solitary nucleus in the medulla. Salivary glands receive their input from higher centers in response to smell, sight, etc. As parasympathetic efferences, the facial nerve innervates the sublingual and submandibular glands via the submandibular ganglion, while the glossopharyngeal nerve innervates the parotid glands via the otic ganglion. The sympathetic postganglionic pathways are from the cervical ganglion of the sympathetic chain. Norepinephrine released from sympathetic neurons binds to both alpha- and beta-adrenergic receptors on the acinar cell leading to an increase of the second messenger cyclic adenosine monophosphate (cAMP) and thus increasing saliva secretion.

In the release of sAA, beta-adrenergic mechanisms are the main contributing factors, as numerous animal and human studies have demonstrated. For example, studies have shown a sympathetic response by cold water immersion or by administering propranolol (a beta-adrenergic blocker). Exposure to cold water raises sAA activity in the parotid gland, whereas

propranolol leads to a reduction of sAA concentrations. It has also been shown that yohimbine hydrochloride, an alpha-2-adrenergic receptor antagonist, resulted in significant increases in sAA concentration relative to a placebo condition. These findings suggest that changes in sAA might be regarded as an indirect indicator of changes in autonomic activation.

Since the release of sAA is governed by activation of the ANS, an increase in sAA may be expected during psychological stress, i.e., when autonomic activation is high. In a seminal paper, Chatterton, Vogelsong, Lu, Ellman, and Hudgens (1996) published their findings of increases in sAA during a written examination. sAA increases have also been reported in response to other psychologically stressful conditions, such as experience of medical procedures, adverse musical stimuli, mothers watching their children being exposed to a stressful task, the cold pressor test, achievement and interpersonal stress, a driving simulation, use of noise exposure, a mental arithmetic task, oral academic examination, affective picture viewing, etc. Almost uniformly, these tests have resulted in sAA increases. This suggests that sAA is a highly sensitive parameter reflecting changes caused by *acute* psychological stressors. In contrast, only a handful of studies have reported associations between *chronic* stress and sAA. According to this small number of studies, there is a broad potential of measuring sAA as an index of chronic stress in selected high-risk populations.

Applications of Salivary Alpha-Amylase Measurement

Based on the knowledge that has been accumulated about the role of stress and its underlying physiological mechanisms in the secretion of sAA, it seems reasonable to conclude that sAA activity can serve as an index for pathological dysregulation of the ANS in specific clinical and subclinical conditions. As an example, anxiety-related conditions are accompanied by autonomic changes. Consequently, the measurement of sAA might provide insight into alterations in the autonomic nervous system in anxiety patients.

In addition, sAA might also be measured in the context of somatic disorders, in which autonomic dysregulation has been observed. Exaggerated autonomic responses to different stimuli or basal autonomic dysregulation can be observed in hypertensive patients, or patients with HIV or with atopic diseases. Also, a number of functional somatic syndromes, such as chronic fatigue syndrome or irritable bowel syndrome, have been shown to be associated with a dysregulation of the ANS. It will be particularly interesting to see whether these alterations are reflected in altered sAA levels. Given findings of the role of stress in clinical populations, the use of sAA to measure the effects of stress management training is expected to be a promising approach for studies of treatment effects. In general, measurement of sAA in clinical populations seems very useful in ambulatory settings where saliva collection might present an easy, noninvasive, and efficient sampling method.

Summary

sAA activity is a potential indicator for autonomic activation. Numerous studies have shown that changes in sAA can be elicited by stressful stimuli, whether they are physiological or psychological in nature. The biological meaning of this phenomenon remains to be clarified, though. Physiological stress reactions comprise orchestrated actions throughout the body, putting the organism in a state of overall preparedness to engage in fight or flight. Thus, increases in amylase activity may be one of many actions involved in activating the body's resources to cope with stressful events or threats to homeostasis. However, this explanation only applies to responses to short-term, acute stressors. Further studies are needed to examine long-term changes in sAA activity which are of particular interest in disorders associated with autonomic dysfunction.

Further studies are needed to elucidate the mechanisms underlying elevations in sAA in response to stress. Although a variety of studies have examined the physiological mechanisms of sAA production and secretion in animals, studies

in humans are scarce. Use of pharmacological agents that inhibit or activate the ANS may prove particularly useful here, providing more detailed insights into the branches of the ANS responsible for increases in sAA. Also, electrical stimulation techniques in awake or anesthetized humans, e.g., in the clinical context of a hospital, may be useful. Measurement of direct sympathetic nerve activity via microneurography is considered to be the most accurate technique for assessing sympathetic activation. Beyond peripheral measurements, the relationship between central parameters and changes in sAA might prove very interesting.

Future studies will show to what extend sAA will play a role within the research of pathophysiological mechanisms and treatment of stress-related disorders.

References and Readings

Chatterton, R. T., Jr., Vogelsong, K. M., Lu, Y. C., Ellman, A. B., & Hudgens, G. A. (1996). Salivary alpha-amylase as a measure of endogenous adrenergic activity. *Clinical Physiology, 16*(4), 433–448.

Ehlert, U., Erni, K., Hebisch, G., & Nater, U. (2006). Salivary alpha-amylase levels following yohimbine challenge in healthy men. *The Journal of Clinical Endocrinology and Metabolism, 91*(12), 5130–5133.

Granger, D. A., Kivlighan, K. T., el-Sheikh, M., Gordis, E. B., & Stroud, L. R. (2007). Salivary alpha-amylase in biobehavioral research: Recent developments and applications. *Annals of the New York Academy of Sciences, 1098*, 122–144.

Limm, H., Gundel, H., Heinmuller, M., Marten-Mittag, B., Nater, U. M., Siegrist, J., et al. (2010). Stress management interventions in the workplace improve stress reactivity: a randomised controlled trial. *Occupational and Environmental Medicine, 62*(8), 126–133.

Nater, U. M., Bohus, M., Abbruzzese, E., Ditzen, B., Gaab, J., Kleindienst, N., et al. (2010). Increased psychological and attenuated cortisol and alpha-amylase responses to acute psychosocial stress in female patients with borderline personality disorder. *Psychoneuroendocrinology, 35*(10), 1565–1572.

Nater, U. M., La Marca, R., Florin, L., Moses, A., Langhans, W., Koller, M. M., et al. (2006). Stress-induced changes in human salivary alpha-amylase activity: Associations with adrenergic activity. *Psychoneuroendocrinology, 31*(1), 49–58.

Nater, U. M., & Rohleder, N. (2009). Salivary alpha-amylase as a non-invasive biomarker for the sympathetic nervous system: Current state of research. *Psychoneuroendocrinology, 34*(4), 486–496.

Nater, U. M., Rohleder, N., Gaab, J., Berger, S., Jud, A., Kirschbaum, C., et al. (2005). Human salivary alpha-amylase reactivity in a psychosocial stress paradigm. *International Journal of Psychophysiology, 55*(3), 333–342.

Rohleder, N., & Nater, U. M. (2009). Determinants of salivary alpha-amylase in humans and methodological considerations. *Psychoneuroendocrinology, 34*(4), 469–485.

van Stegeren, A., Rohleder, N., Everaerd, W., & Wolf, O. T. (2006). Salivary alpha amylase as marker for adrenergic activity during stress: Effect of betablockade. *Psychoneuroendocrinology, 31*(1), 137–141.

Alpha-linolenic Acid

▶ Omega-3 Fatty Acids

Alter

▶ Aging

Alternative Medicine

Nicole Nisly
Department of Internal Medicine, University of Iowa, Iowa City, IA, USA

Synonyms

Complimentary and alternative medicine; Integrative medicine

Definition

Complementary and alternative medicine (CAM) encompasses a broad and diverse range of therapies, practices, and products which are not considered a part of conventional scientific medicine. It may be utilized in conjunction with

(complementary) or in place of (alternative) conventional health care. They are widely utilized by patients to improve health and well-being; however, they have not yet had adequate research, are not commonly taught in the health-care professional curriculum, and are not incorporated in health-care systems, although this has been rapidly changing over the last two decades, with rising patient interest, federally funded research, and education initiatives. Patient utilization is significant, with approximately 38% of US adults aged 18 years and over and approximately 12% of children using some form of CAM (Barnes, Powell-Griner, McFann, & Nahin, 2004; Eisenberg 2005).

What Are the Types of Modalities Encompassed in the CAM Definition?

The NCCAM classifies CAM modalities into four broad groups. Some modalities may belong to more than one group (http://nccam.nih.gov/health/whatiscam/#types).

1. Natural products: This include herbs, vitamins, minerals, fatty acids, and digestive enzymes, to name a few. Many of these products are regulated as dietary supplements (see below).
2. Mind-body medicine: These practices bring together the mind, body, and brain to improve health. Some of the modalities include meditation, Yoga, Tai Chi, acupuncture, and guided imagery. Some of these modalities are thousands of years old.
3. Manipulative or body-based therapies such as chiropractic care and massage therapy.
4. Other forms of healing include energy medicine such as healing touch and Reiki, movement therapies such as Alexander technique and Feldenkrais, and whole systems of healing such as TCM, Ayurvedic medicine, and naturopathy.

What Is a Dietary Supplement, and How Are These Regulated by FDA ?

The 1994 Dietary Supplement and Health Education Act (DSHEA) defines dietary supplements as products intended to supplement diet; they may include a vitamin or mineral, a herb or botanical, enzymes, amino acid, or other extracts. They are regulated under the general umbrella of food and not drugs. Manufacturers are responsible for appropriate product labeling and following good manufacturing practices. The FDA must prove that a product is unsafe before it can be removed from the market. Patient's use of dietary supplement is substantial. In the US adult population aged 20 and over, the percentage of people who used at least one dietary supplement increased from 42% in 1988–1994 to 53% in 2003–2006 (www.fda/gov Gahche et al., 2011).

Description

As health-care complexity, knowledge basis, and technology grow with resulting improved life expectancy and quality of life, patients are growing more interested in a health care perceived as more holistic and congruent with their values and philosophical beliefs (Eisenberg 2005).

Unfortunately, this growth in patient engagement and health-care knowledge is not being matched at the same speed by health-care training in a more participatory and collaborative health care. While medicine is moving toward a patient-centered care, this is too slow of a movement in a field where health-care students and practitioners still have very limited curriculum preparing them to incorporate patients' beliefs, values, and cultural background in the health-care plan. Many providers have very limited knowledge and skills in addressing the use of complementary and alternative care by patients, including use of dietary supplements and nutrition and collaboration with holistic medicine providers, while their patients' knowledge and CAM use are growing rapidly (Kronenberg 2008; Wallace 2007).

In an age of spiraling health-care costs and national debt, health-care reform will be inevitable. This is a time where patient engagement, empowerment, and knowledge provide a significant opportunity to explore a health-care system geared toward wellness and prevention and to pursue healthier living in body and mind, where patients and health-care providers can work collaboratively toward a more sustainable and

holistic health care. Patient and consumer interest and engagement are reflected on the fact that out-of-pocket expenditure for CAM exceeds that for traditional health-care expenditure (Davis, West, Weeks, & Sirovich, 2011; Eisenberg 2007; Nahim 2007).

A new discipline called integrative medicine has emerged over the last two decades where health-care providers bring together scientific evidence-based medicine with complementary and alternative therapies to provide patients a more holistic and values-centered care. A growing number of academic centers now offer integrative medicine programs, and medical schools' curriculum and residencies are offering more training programs on how to integrate the evidence-based medical knowledge with CAM. The academic organization "Consortium of Academic Health Centers for Integrative Medicine" has brought together academic centers dedicated to conducting research, education, and providing patient care in this exciting new health-care field, which brings together many conventional health-care practitioners such as physicians, behavioral health specialists, nutritionists, and CAM providers such as acupuncturists, chiropractors, and massage therapists.

While some of the modalities utilized in CAM and integrative medicine are ancient, Yoga and Tai Chi, for example, have been practiced for thousands of years. Others have emerged in the last decade or so. What unifies this vast and diverse field of treatments and techniques is the lack of adequate research on efficacy, safety, and the understanding of their mechanisms of action. Since 1998, however, the National Institutes of Health through the National Center for Complementary and Alternative Medicine (NCCAM) has been funding and conducting extensive research on this broad field and better understanding on how these therapies work is growing (http://nccam.nih.gov/).

Cross-References

- ▶ Acupuncture
- ▶ Dean Ornish
- ▶ Integrative Medicine
- ▶ Spirituality and Health
- ▶ Yoga

References and Readings

Bardia, A., Nisly, N. L., Zimmerman, M. B., Gryzlak, B. M., & Wallace, R. B. (2007). Use of herbs among adults based on evidence-based indications: Findings from the National Health Interview Survey. *Mayo Clinic Proceedings, 82*(5), 561–566.

Barnes, P. M., Powell-Griner, E., McFann, K., & Nahin, R. L. (2004). Complementary and alternative medicine use among adults: United States, 2002. *Advance Data, 343*, 1–19.

Chao, M. T., Wade, C., & Kronenberg, F. (2008). Disclosure of complementary and alternative medicine to conventional medical providers: Variation by race/ethnicity and type of CAM. *Journal of the National Medical Association, 100*(11), 1341–1349.

Clinical practice guidelines on complementary and alternative medicine. Retrieved from http://nccam.nih.gov/health/providers/clinicalpractice.htm

Complementary and alternative medicine for HealthCare providers reliable information. Retrieved from http://nccam.nih.gov/health/providers/

Consortium of Academic Health Centers for Integrative Medicine, with member5ship list. Retrieved from http://www.imconsortium.org/ and http://www.imconsortium.org/members/home.html

Davis, M. A., West, A. N., Weeks, W. B., & Sirovich, B. E. (2011). Health behaviors and utilization among users of complementary and alternative medicine for treatment versus health promotion. *Health Services Research, 46*(5), 1402–1416.

Eisenberg, D. M. (2005). The Institute of Medicine report on complementary and alternative medicine in the United States–personal reflections on its content and implications. *Alternative Therapies in Health and Medicine. 11*(3), 10–15.

Federal drug administration definition of dietary supplements. Retrieved from http://www.fda.gov/Food/DietarySupplements/ConsumerInformation/ucm110417.htm

Gahche, J., Bailey, R., Burt, V., Hughes, J., Yetley, E., Dwyer, J., et al. (2011). *Dietary supplement use among U.S. adults has increased since NHANES III (1988–1994)* (NCHS Data Brief No. 61). Hyattsville, MD: National Center for Health Statistics.

Nahin, R. L., Barnes, P. M., Stussman, B. J., & Bloom, B. (2009). Costs of complementary and alternative medicine (CAM) and frequency of visits to CAM practitioners: United States, 2007. *National Health Statistics Reports, 18*, 1–14.

National Center for Complementary and Alternative Medicine (NCCAM) at National Institutes of Health.

Retrieved from http://nccam.nih.gov/about/ataglance/index.htm
National Center for Complementary and Alternative Medicine (NCCAM) at National Institutes of Health. Retrieved from http://nccam.nih.gov/health/whatiscam/#types
Tindle, H. A., Davis, R. B., Phillips, R. S., & Eisenberg, D. M. (2005). Trends in use of complementary and alternative medicine by US adults: 1997–2002. *Alternative Therapies in Health and Medicine, 11*(1), 42–49.

Alzheimer's Caregivers

▶ Williams LifeSkills Program

Alzheimer's Disease

Debra Johnson
Department of Psychology, University of Iowa, Iowa City, IA, USA

Synonyms

Cortical dementia; Dementia

Definition

Alzheimer's disease, the most common form of cortical dementia, was first described by Alois Alzheimer in 1906. It is a progressive dementia characterized by a downward decline in cognitive functioning, typically ending in death within 15 years.

Description

While memory loss is a common symptom of Alzheimer's disease, memory loss by itself is not pathognomic. Patients experience significant impairments in intellectual functioning that interfere with normal activities and relationships. They lose functionality in many realms of cognitive functioning – including the ability to use language, to think abstractly, to solve problems, and to maintain emotional control. Additionally, they may experience personality changes and behavioral problems, such as agitation, delusions, and hallucinations.

Early in the disease, word-finding difficulties are common. Memory impairments impact short-term memories, while long-term memories tend to remain intact until much later in the progression of the disease. Eventually, long-term memory and knowledge bases are compromised. Communication becomes more and more difficult, and the person loses the ability to perform activities of daily living (dressing, preparing meals, personal hygiene). In the very advanced stages of the disease, individuals with the disease may become incommunicative and require significant care taking.

Alzheimer's disease is characterized by profound changes in the brain. At a gross level, the brain shows significant volume reduction. The loss of tissue results in widening of the sulci and gyri and smoothing of the brain surface. Microscopic inspection of brain tissue reveals loss of neurons throughout the brain as well as the presence of large numbers of pathological changes. Neurofibrillary tangles (aberrant strands of intracellular tau protein) and senile plaques (aggregates of extracellular amyloid protein) are evident throughout the brain. The development of these pathologies is thought to be mediated by abnormal proteins (tau and beta-amyloid) (Tiraboschi, Hansen, & Thal, 2004). Many neurotransmitter systems including acetylcholine, glutamate, and norepinephrine have been implicated in the development and progression the disease.

There are two widely recognized forms of the disease – a sporadic/age-related form and a genetic/familial form. In the vast majority of cases, the slow steady buildup of risk factors over a lifetime are thought to produce pathological brain changes which in turn produce changes in cognitive functioning. Not surprisingly, prevalence of the disease increases dramatically with advancing age. Overall prevalence in the USA is 1.6% in the 65–74 age group but in it rises to 19% in people aged 75–84 and 42% in the 84+ age group (Liddell, Williams, Bayer, Kaiser, & Owen, 1994).

The genetic form (also called "early-onset AD") is atypical – accounting for less than 5% of all cases – and is associated with onset of symptoms before the age of 65. This form of the disease is particularly aggressive – with progression to death happening more rapidly than in the age-related form. Autosomal dominance patterns involve three gene families – presenilin 1, presenilin 2, and amyloid precursor protein (APP). All of these gene families code for the production of brain proteins thought to produce the cellular level changes associated with the disease.

Although sporadic cases lack autosomal dominant genetic determination, genetic predispositions have been identified. For example, individuals with the E4 allele of the apoprotein E (APOE) gene are at an increased risk of developing AD (Hebert, Scherr, Bienias, Bennett, & Evans, 2003). People with one copy of the E4 allele have a three times greater risk than those without the E4 allele, while people with two copies of the E4 allele have a 15 times greater risk of developing the disease. It is important to note that the E4 allele is one of many risk factors that have been identified. Other risk factors include increased age, poverty, and history of head injury. It is most likely that environmental and lifestyle factors interact with genetic risk factors to produce the disease.

Studies find that people who lead interesting, active lives (intellectually stimulating, socially involved, physically active) have a lower risk of developing the disease.

Several pharmaceutical agents are available to palliate the cognitive decline associated with disease, but no cure exists. Most of these drugs are cholinergic agonists, although one is an NMDA receptor antagonist.

Affecting more than 26 million people worldwide, Alzheimer's disease places enormous strains on families and social institutions. Governmental agencies in many countries struggle to meet the needs of the increasing numbers of patients as our populations continue to age (Brookmeyer, Johnson, Ziegler-Graham, & Arrighi, 2007).

Cross-References

▶ Coffee Drinking, Effects of Caffeine
▶ Dementia

References and Readings

Brookmeyer, R., Johnson, E., Ziegler-Graham, K., & Arrighi, H. M. (2007). Forecasting the global burden of Alzheimer's disease. *Alzheimer's and Dementia, 3*(3), 186–191.

Hebert, L. E., Scherr, P. A., Bienias, J. L., Bennett, D. A., & Evans, D. A. (2003). Alzheimer disease in the US population: Prevalence estimates using the 2000 census. *Archives of Neurology, 60*(8), 1119–1122.

Liddell, M., Williams, J., Bayer, A., Kaiser, F., & Owen, M. (1994). Confirmation of association between the e4 allele of apolipoprotein E and Alzheimer's disease. *Journal of Medical Genetics, 31*(3), 197–200.

Tiraboschi, P., Hansen, L. A., & Thal, L. J. (2004). Corey-Bloom J "The importance of neuritic plaques and tangles to the development and evolution of AD". *Neurology, 62*(11), 1984–1989.

Ambulatory Blood Pressure

Derek Johnston[1] and Ydwine Zanstra[2]
[1]School of Psychology, University of Aberdeen, Aberdeen, Scotland, UK
[2]The Amsterdam University College, Amsterdam, The Netherlands

Synonyms

Real-life blood pressure monitoring

Definition

Ambulatory blood pressure is arterial blood pressure measured in real-life settings by an automatic device.

Description

Blood pressure (BP) was first measured in the eighteenth century by Halles following Harvey's

work on the circulation of blood. It has been measured in the clinic and operating theater since the early part of the twentieth century and its utility as a predictor of cardiovascular disease established in the second half of the century. The ambulatory measurement of BP (ABPM) outside the clinic or laboratory is a development of the later part of the century. Despite its comparatively recent origin, it is now regarded as the measure of choice clinically since it provides a more reliable and valid measure of an individual's BP and is a better predictor of later disease, perhaps because it reduces "white coat" hypertension, the elevation of BP produced in some individuals when BP is measured in a medical setting. The majority of ambulatory BP monitors are automatic miniaturized versions of the standard sphygmomanometer and can measure systolic and diastolic BP at predetermined times, often every 20 or 30 min during the day, less frequently when the participant is asleep. There are also devices that measure BP continuously, the most successful of which use the vascular unloading technique first described by Penaz (1973). Continuous measurement is not widely used but has considerable advantages for psychophysiological studies since it greatly improves the power of studies to detect the relationship between BP and environmental events or psychological phenomena that might be transitory, as well as providing repeated measurement during more enduring events. The Penaz-derived devices also enable one to determine the mechanisms, vascular or cardiac, that underlie the elevations in blood pressure. Alternatively, the underlying mechanisms can be determined by combining intermittent ABPM with cardiac output measured by ambulatory measures of cardiac impedance. Whether infrequent or continuous ambulatory blood pressure is affected by many factors that are often noise with respect to the question under study. The most important of these are movement and posture, and they are usually controlled either through questionnaires completed at the time of measurement or, more satisfactorily, through direct measurement and recording with accelerometer-based devices.

While ABPM was developed to deal with clinical issues, it has great relevance for behavioral medicine. Most psychophysiological studies of the cardiovascular system are conducted in the laboratory for reasons of convenience, control, and the accuracy of measurement. ABPM allows the study of the psychological processes that in part determine BP to be extended to real life with obvious benefits in ecological validity (although at the cost of loss of control) and a potential increase in understanding of the role of stress in hypertension and cardiovascular disease. ABPM can also provide insight into psychological process that have effects on the cardiovascular (CV) system by providing sophisticated measures of autonomic arousal that illuminate or index processes that cannot be studied by observation or self-report.

BP is elevated in many situations that are conventionally seen as stressful, such as public speaking or examination. This was originally shown in laboratory studies but has been confirmed in field studies using ABPM when it is often found that the responses are considerably larger than in the laboratory simulations of these situations. Interpersonal conflict, often difficult to study meaningfully in the laboratory, is also associated with elevated BP in field settings. Perhaps unsurprisingly, it has also been shown that heightened subjective feelings of anxiety or arousal are associated with increased BP, as are variations in the demand that people feel they are under or their perception of the control that they feel they have over the situation since high demand and low control has been widely shown to be associated with increased strain. Such effects are moderated by personality with, for example, some studies showing that highly hostile people had high BP whatever their mood, while the less hostile had high BP only when in a negative mood. The highly hostile are also less likely to show a reduction in stress-related BP with social support. There is in additional evidence of gender moderating the effects of stress on BP with women benefiting more than men from social support during stressful situations. The effects of social interaction are subtle with interactions with a person with whom one has an ambivalent relationship leading, in one study, to greater BP elevations than interactions with

people that were more clearly either liked or disliked. Such studies have also shown that it can be the nature of the relationship rather than the nature of a specific interaction that relates to BP. This information on complex and subtle social situations could only be obtained in real life using ABPM.

BP is determined by the interplay of cardiac output and vascular resistance. Laboratory studies suggest that objectively different situations and subjectively different appraisal of these situations can affect the determinants of BP. Tasks that involve active coping or the related appraisal of situations as challenging are associated with cardiac effects, while tasks involving passive coping or the appraisal of a situation as threatening, leading to a vascular response. While this has not been studied extensively in real life, in one study, challenge appraisals were related to cardiac effects in people making an academic presentation and threat with a more vascular response. More vascular responses have been reported in lonely people who are hypothesized to adopt passive coping strategies.

An enduring issue in laboratory and ambulatory studies of the effects of stress has been the extent to which responses seen in the laboratory generalize to real life. The response to laboratory stressors is a poor candidate as an index of the risk of disease if it relates only weakly, or not at all, to the response in real life. The issue is controversial, but the most recent studies using the best available measurement and analytic techniques suggested that reliable CV (heart rate and BP) responses to laboratory stressors, obtained by combining the responses to several stressors, do relate to the CV response to objective stressful environments, negative emotions and perceptions of the situation as stressful.

The reactivity hypothesis has been the dominant theory in cardiovascular behavior medicine since 1980. In its simplest form, this theory states that individuals who show an excessive CV response to stress, the hyperreactive, are at increased risk of CV disease. Prospective studies using the CV response to laboratory stressors to predict cardiovascular disease endpoint have had mixed findings at best. However, hyperreactivity is not enough since a hyperreactive person has to be reacting to something. A vulnerability factor, like hyperreactivity, needs an appropriate environment to actually become a risk factor. Laboratory studies only establish the vulnerability. Ambulatory studies can go some way to establishing if the appropriate environment also exists. Unfortunately, while ambulatory BP is a better predictor of CV disease than clinic BP, there do not appear to be studies seeking to link BP elevations to stress in real life and subsequent disease.

Cross-References

▶ Ambulatory Monitoring
▶ Cardiovascular Disease

References and Readings

Penaz, J. (1973). Photoelectric measurement of blood pressure volume and flow in the finger. In *Digest of the International Conference on Medicine and Biological Engineering*, Dresden, pp 104–104.

Steptoe, A. (2001). Ambulatory monitoring of blood pressure in daily life: A tool for investigating psychosocial processes. In J. Fahrenberg & M. Myrtek (Eds.), *Progress in ambulatory monitoring* (pp. 257–269). Seattle: Hogrefe & Huber.

White, W. B. (2007). *Blood pressure monitoring in cardiovascular medicine and therapeutics*. Totowa, NJ: Humana Press.

Zanstra, Y. J., & Johnston, D. W. (2011). Cardiovascular reactivity in real life settings: Measurement, mechanism and meaning. *Biological Psychology, 86*, 98–105.

Ambulatory Monitoring

Derek Johnston
School of Psychology, University of Aberdeen, Aberdeen, Scotland, UK

Definition

In behavioral medicine, ambulatory monitoring has two components: the measurement in real life

of some physiological parameter and the real-time measurement of behavior, emotion, or psychological process. Studies can, but do not always, involve both types of measurement

Description

The initial drivers for ambulatory physiological recorders were largely clinical. The ambulatory recording of physiological signals started with Holter's experimental development, shortly after the Second World War, of a very bulky physiological ambulatory monitor which later became a useful device to measure and record the electrocardiogram (ECG, Holter, 1961). Such devices, often still called Holter monitors, were developed primarily for diagnostic purposes and are routinely used in the diagnosis of coronary heart disease. Much later, this was followed by ambulatory blood pressure monitors (ABPM) which are now the method of choice in the diagnosis and management of hypertension. Advances in the miniaturization of electronic devices and developments in solid-state memory (and to a lesser extent telemetry) have lead to the development of a variety of multipurpose ambulatory recorders that can record and store virtually all the systems that were once measured solely in the laboratory. This includes ECG, electroencephalogram (EEG), electromyogram (EMG), blood pressure (BP) measured both discontinuously and continuously, the impedance cardiogram, measures of local blood flow with photoelectric devices, skin temperature, and skin conductance. These measures are derived from conventional electrophysiological techniques, impedance technology, BP from sophisticated fast-acting pumps on finger cuffs, and many functions from devices placed in purposely built vests or shirts worn by participants (sometimes called smart clothes). From these devices, one can measure a wide range of cardiovascular and respiratory parameters including heart rate and heart rate variability, blood pressure, cardiac output and vascular resistance, and features of cardiac activity like pre-ejection time, as well neural signals. Many of these measures reflect the functioning of the autonomic nervous system. The advances in electronic technology has been matched by developments in software, and most systems are sold with sophisticated software packages to handle signal detection, data reduction, and other aspects of data processing. There are also standalone software packages that work with a variety of ambulatory recording systems.

As well as measures of physiological systems, there are devices based on accelerometers that can measure activity, posture, and details of limb movement and gait. The simplest of these are single axial accelerometers mounted in some convenient place, such as the waist, to measure activity through to complex systems of multiple accelerometers attached to different parts of the body that are claimed to be able to measure different categories of movement such as speed of walking or running, cycling, standing, and rate of change of such activities. One device uses the combination of heart rate from the ECG and activity from a chest-mounted accelerometer to derive well-validated measures of energy expenditure. Such activity measures are also important in interpreting the autonomic measures since heart rate and blood pressure are profoundly influenced by metabolic demand, and it is very helpful to account for this when interpreting changes in cardiac activity.

The measurement of some aspects of behavior can be achieved by direct objective measurement of limb movement and, in much prescribed circumstances, by body-mounted cameras or by direct observation. However, most behavioral measurement in real life is through self-report; participants' record in real time what they are doing, thinking, and feeling. Such methods, often called ecological momentary assessment or experience sampling, are most often achieved by used electronic devices on which the participants complete questionnaires about their current behavior. Such devices, which are readily programmed using specialist software or purchased from specialist companies, are generally acceptable and provide high-quality time-stamped information. Traditional paper and pencil diaries can also be used and have the obvious advantage of cheapness but lack time stamping, and hence, the investigator cannot know then the

diary entry was actually made. However, with well-motivated participants and when information is gathered quite infrequently, perhaps weekly, then they may well be the most cost-effective method. The electronic measurement is usually on PDAs (personal data assistant), smart phones, or, if recording is infrequent and of a summary nature, on home personal computers using the internet. Responses can be self-ratings, text, or brief audio recordings. The smart phone and internet realizations of EMA allow the possibility of interaction with the recording system so that it can respond to missing data or particular responses or to deliver interventions, perhaps contingent on behavior. Global positioning systems are also being used in conjunction with EMA devices to gather information on a participant's behavior and to target appropriate interventions.

An interesting variant of EMA is the Day Reconstruction Method (DRM) developed by Kahneman, Krueger, Schkade, Schwartz, and Stone (2004) and colleagues for use in large surveys where EMA is impractical. In DRM, participants retrospectively structure their day into meaningful units then recall and rate their behavior or mood during these units. This method has been implemented face to face, singly, or in groups and can be used online. It produces systematic data that relates sensibly to known diurnal variations in mood and arousal, heart rate measured throughout the day, and mood assessed in real time using EMA. The method has been influential in attempts to estimate national well-being by economists.

The rapid developments in ambulatory physiological and behavioral measurement technology have been matched by the increasing sophistication of the statistical tools used to analyze such repeated, heavily autocorrelated, and multilevel data. Multilevel random effects modeling of ambulatory data is now almost universal, and most widely used statistical packages allow some form of multilevel modeling, and the specialist programs have become much more user friendly.

The methods of ambulatory physiological and activity recording and EMA have been successfully applied to a wide range of practical and theoretical problems in many areas of behavioral medicine and related fields such as clinical, health, and occupational psychology. Among the issues illuminated by such methods are cardiovascular reactivity, hypertension, addiction, disability, pain, adherence to treatment regimes, sleep, occupational and other kinds of stress, the effects of surgery, and patient and staff safety in medical settings.

Cross-References

- ▶ Ambulatory Blood Pressure
- ▶ Blood Pressure, Measurement of

References and Readings

Bolger, N., Davis, A., & Rafaeli, E. (2003). Diary methods: Capturing life as it is lived. *Annual Review of Psychology, 54*, 579–616.

Holter, N. J. (1961). A new method for recording in heart studies: Continuous electrocardiography of active subjects. *Science, 134*, 1214–1220.

Kahneman, D., Krueger, A. B., Schkade, D. A., Schwartz, N., & Stone, A. A. (2004). A survey method for characterizing daily life experience: The day reconstruction method. *Science, 306*, 1776–1780.

Wilhelm, F. H., & Grossman, P. (2010). Emotions beyond the laboratory: Theoretical fundamentals, study design, and analytic strategies for advanced ambulatory assessment. *Biological Psychology, 84*, 552–569.

American Cancer Society

Youngmee Kim
Department of Psychology, University of Miami, Coral Gables, Miami, FL, USA

Basic Information

The American Cancer Society (ACS) is the nationwide, community-based, voluntary health organization dedicated to eliminating cancer as a major health problem by preventing cancer, saving lives, and diminishing suffering from cancer through research, education, advocacy, and service. Headquartered in Atlanta, Georgia, the ACS has 12 chartered divisions, more than

900 local offices nationwide, and a presence in more than 5,100 communities. The Society's Research Program is composed of two main divisions: extramural and intramural research. The Extramural Grant Department reviews and administers both Research Grants and Health Professional Training Grants. The ACS focuses its extramural funding on investigator-initiated, peer-reviewed proposals. Intramural Research is composed of six programs: Epidemiology, Surveillance Research, Health Services Research, International Tobacco Control Research, the Behavioral Research Center, and the Statistics and Evaluation Center. All intramural department staff conduct applied cancer research in-house.

Major Impact on the Field

The Extramural Grant Department has supported groundbreaking studies. Forty-four investigators who were supported by ACS went on to win the Nobel Prize. The Society's Intramural Research Program has provided population-based surveillance systems and other national and international databases to evaluate trends and population variability in cancer incidence and mortality, behavioral risk factors, early detection and treatment patterns, and economic factors in tobacco control. This research has helped monitor progress in cancer control and provide evidence for policy and advocacy initiatives. In addition, a series of Cancer Prevention Studies (CPS-I, CPS-II, CPS-II Nutrition Survey, and CPS-3) have been landmarks in the field, providing mortality trends and identifying dietary factors related to cancer incidence and mortality. Overall, findings from the CPS-I and CPS-II have made major contributions to the field by (a) advancing our knowledge of the environmental, behavioral, and biological causes of cancer and cancer survivorship and (b) informing the development of cancer prevention and survivorship strategies, policies, and practice. The ACS is one of the first organizations to recognize the importance of psychosocial and behavioral factors in cancer prevention and control.

The ACS has also implemented numerous programs in communities, guided by its mission statement. For example, to help people stay healthy, ACS distributes the Healthy Living Newsletter, which provides educational materials about healthy lifestyle habits that have been found to reduce the risk of cancer and other major diseases. Other programs for this effort are for employees (the Active For Life program) and school-age children (the School Health program) to become more active and to improve healthy diet. The Society's website, cancer.org, provides the most up-to-date information about cancer information and support available at the local and the national level for patients, survivors, and caregivers. Some example programs include the Cancer Survivors Network, the online community by and for people with cancer and their family; the Road to Recovery, which provides transportation service for cancer patients who need rides to treatment; the Hope Lodge, which is free lodging available for patients and their families; the I Can Cope, which provides educational classes about cancer and treatment, and which is available in person or on line; and the Patient Navigator Program, a personalized cancer guide.

Several events organized by the ACS, such as Making Strides Against Breast Cancer and Relay For Life, raise funds to support the community and research effort in making a difference in the fight against cancer.

Cross-References

- ▶ Cancer Prevention
- ▶ Cancer Survivorship
- ▶ Epidemiology
- ▶ Quality of Life

References and Readings

Blanchard, C. M., Courneya, K. S., & Stein, K. D. (2008). Cancer survivors' adherence to lifestyle behavior recommendations and associations with health-related quality of life: Results from the ACS SCS-II. *Journal of Clinical Oncology, 26*, 2198–2204.

Calle, E. E., Feigelson, H. S., Hildebrand, J. S., Teras, L. R., Thun, M. J., & Rodriguez, C. (2009). Postmenopausal hormone use and breast cancer associations differ by hormone regimen and histologic subtype. *Cancer, 115*, 936–945.

Fedewa, S. A., Ward, E. M., & Edge, S. B. (2010). Delays in adjuvant chemotherapy treatment among black cancer patients are more likely in black and Hispanic populations: A National Cohort Study 2004–2006. *Journal of Clinical Oncology, 28*, 4135–4141.

http://www.cancer.org. Accessed Mar 18, 2012.

Kim, Y., & Given, B. A. (2008). Quality of life of family caregivers of cancer survivors across the trajectory of the illness. *Cancer, 112*(Suppl. 11), 2556–2568.

Rodriguez, C., Jacobs, E. J., Deka, A., et al. (2009). Use of blood-pressure-lowering medication and risk of prostate cancer in the Cancer Prevention Study II Nutrition Cohort. *Cancer Causes & Control, 20*, 671–679.

Smith, R. A., Cokkinides, V., Brooks, D., Saslow, D., & Brawley, O. W. (2010). Cancer screening in the United States, 2010: A review of current American Cancer Society guidelines and issues in cancer screening. *CA: A Cancer Journal for Clinicians, 60*, 99–119.

Thun, M. J., Hannan, L. M., & DeLancey, J. O. (2009). Alcohol consumption not associated with lung cancer mortality in lifelong nonsmokers. *Cancer Epidemiology, Biomarkers & Prevention, 18*, 2269–2272.

Zhang, X., Albanes, D., Beeson, W. L., et al. (2010). Risk of colon cancer and coffee, tea, and sugar-sweetened soft drink intake: Pooled analysis of prospective cohort studies. *Journal of the National Cancer Institute, 102*, 771–783.

American Diabetes Association

Della Matheson
Diabetes Research Institute, Miller School of Medicine, University of Miami, Miami, FL, USA

Basic Information

The American Diabetes Association (ADA) is a leading United States-based nonprofit organization providing funding for diabetes research, professional and lay education, and advocacy for people with diabetes. Their mission as stated is: "to prevent and cure diabetes and to improve the lives of all people affected by diabetes."

The organization was founded in 1940 by a group of physicians and scientists with the goal of providing education and support to physicians and health-care professionals. In the 1960s, the organization expanded its membership to include general members and heightened its services to provide education and support to the community at large. There are currently 97 local affiliate offices distributed throughout 47 states.

Major Impact on the Field

ADA programs and activities include:

Publications – a large library of informational books, magazines, and journals for both medical professionals and consumers are available.

Professional meetings – serve to educate and stimulate collaborative efforts in the delivery of health care to people with diabetes and to enhance research efforts of scientists involved in diabetes research. The two largest meetings that occur annually are the ADA Postgraduate Course held in winter of each year and the ADA Scientific Sessions held in the summer each year.

Public meetings – in addition to multiple local chapter offerings, the Diabetes Expo provides a 1-day public program in major markets throughout the USA that includes lectures, large vendor display area, and informational services.

Funding of research – supports basic and clinical diabetes research aimed at prevention, better treatment, and a cure. The research funding program is designed to complement government-funded research through the National Institutes of Health which serves to amplify the effectiveness of the millions of dollars provided by ADA ($42.5 million in 2008).

Family link – a program that provides information to families about living with diabetes and provides information and tool kits for families of children newly diagnosed with diabetes, parent to parent mentoring programs, and school initiatives aimed at enhancing safety for children with diabetes while in school.

Diabetes camps – there are over 60 camps for children between the ages of 4 and 17 years supported by the ADA. Camps consist of residential week-long programs, day camps, family camps, and teen adventure camps. The camping experience offers children with diabetes the opportunity to interact with other children with diabetes, increase their knowledge of diabetes self-management, and enhance independence in a safe environment, while also having fun!

Fund-raising events – Walk to Fight Diabetes, Tour de Cure, School Walk for Diabetes, and Bikers Against Diabetes. The ADA website also includes links for personal gifts as well. ADA currently receives over $250 million dollars through their fund-raising efforts annually.

Advocacy – this mission involves a goal of improving access to health care for people with diabetes and to eliminate discrimination against people at school, in the workplace, or elsewhere in their lives. On a biennial bases, the ADA has organized an event, the Association's Call to Congress, that facilitates the meeting of advocates and members of the ADA to meet with their US Representatives and Senators to discuss how diabetes affects their lives and how health-care legislation can be directed to improve living with diabetes.

How to contact ADA:

National Call Center (1-800-DIABETES or 1-800-342-2383)

Website: http://www.diabetes.org

Cross-References

► Diabetes

References and Readings

American Diabetes Association 2008–2011 Strategic Plan

American Diabetes Association Website. Retrieved from http://www.diabetes.org

Pickup, J., & Williams G. (1991). *Textbook of diabetes* (Vol. 2, Chap. 102, pp. 965–968) Malden, MA: Blackwell Scientific Publications.

American Heart Association

Brooke McInroy
The University of Iowa, Iowa City, IA, USA

Basic Information

The American Heart Association (*AHA*) is a nonprofit health organization in the United States. Its headquarters are in Dallas, Texas, and it maintains offices in 48 states and Puerto Rico. Founded in 1924 by a group of cardiologists, the current mission of the organization is to "build healthier lives, free of cardiovascular diseases and stroke." Its main website is www.heart.org.

The main expenditures of the AHA are on research and educational programs, including an emphasis on cardiopulmonary resuscitation (CPR) and first aid training. The AHA publishes scientific journals and offers a membership program for science and health-care professionals as well as research grants and fellowships. The AHA is affiliated with the American Stroke Association (ASA), which was founded in 1997 with a focus on "prevention, diagnosis, and treatment to save lives from stroke."

Cross-References

► Cardiology
► Cardiovascular Disease

American Psychological Association Division 38 (Health Psychology)

Christopher France
Department of Psychology, Ohio University, Athens, OH, USA

Basic Information

With more than 154,000 members as of 2011, the American Psychological Association (APA) represents the largest scientific and professional organization of psychologists in the world. The mission of APA is to advance the creation, communication, and application of psychological knowledge to benefit society and improve people's lives. In addition to this primary association, there are currently 54 divisions of APA which represent specific interest groups and maintain their own memberships, eligibility criteria, and officers.

APA Division 38 (Health Psychology) was established in 1978 and currently has approximately 3,000 members. From the beginning, APA Division 38 has been broadly focused on issues related to both the science and practice of health psychology, and this broad mission has included the following three components: (1) advancing psychology's contributions to the understanding of health and illness through basic and clinical research and through the integration of biomedical information about health and illness with current psychological knowledge, (2) promoting professional education and services related to health and illness, and (3) ensuring that the psychological, biomedical, and lay public communities are aware of the results of current research and service activities in this area.

The importance of health psychology as a discipline is best illustrated by the fact that behavioral factors predispose, precipitate, and perpetuate many of the leading causes of illness and death in the USA and around the world. And, perhaps more importantly, behavioral and psychological interventions have been shown to encourage disease prevention, enhance coping with acute and chronic illness, and improve health outcomes when delivered in isolation and in conjunction with existing medical procedures. To promote further progress in each of these areas, APA Division 38 supports the educational, scientific, and professional efforts within psychology to understand the etiology, promotion, and maintenance of health in the prevention, diagnosis, treatment, and rehabilitation of physical illness; conduct research related to the psychological, social, emotional, and behavioral factors that contribute to physical illness; make active contributions to improving the health care system; and assist in the formulation of health policy.

Consistent with its goal of promoting the science and practice of health psychology, APA Division 38 maintains an active website (www.health-psych.org); publishes the leading scientific journal in the field, *Health Psychology* (www.apa.org/pubs/journals/hea/index.aspx), as well as a Division newsletter, *The Health Psychologist* (http://www.health-psych.org/ResourcesNewsletters.cfm); and maintains a range of educational and training resources for those interested in the profession. Participation and affiliation with APA Division 38 is encouraged through a variety of mechanisms, including professional membership (open to existing APA members), professional affiliates (including psychologists, physicians, and other health professionals who are not APA members), international affiliates (including health psychologists living and working outside of the United States or Canada), and student affiliates (including those enrolled in accredited programs of psychology, medicine, and related fields).

As models of health care evolve in the United States and around the world, APA Division 38 is working to establish liaisons between researchers, clinicians, and policymakers to encourage the use of psychological science in the promotion of health and prevention of illness.

American Psychosomatic Society

Shin Fukudo, Emiko Tsuchiya and Yoko Katayori
Department of Behavioral Medicine, School of Medicine, Tohoku University Graduate, Aoba-ku, Sendai, Japan

Basic Information

History

There has been a perception of mind–body interaction in many fields of study in recent centuries (Levenson, 1994, p. 1). The American Psychosomatic Society (APS) was founded in response to the desire for cross-discipline study of the people of psychiatry, internal medicine, physiology, and other fields.

With philanthropic support, *Psychosomatic Medicine* was published in 1939 and the journal's board voted to establish the "American Society for Research in Psychosomatic Problems" in December 1942. These founders included Drs. George Daniels, George Draper, and Helen Dunbar.

The name was changed to "The American Psychosomatic Society" in 1948 (American Psychosomatic Society, 2010a).

Today, APS has become an international society. It offers a website, journal, and annual meeting. Researchers and clinicians use various approaches to investigate the links among mind, brain, body, and social issues for curing disease and promoting health (American Psychosomatic Society, 2011a). The Council members have expanded pediatrics, neuroanatomy, physiological sciences, neurophysiology, psychophysiology, clinical psychology, sociology, anthropology, and public health. (American Psychosomatic Society, 2010a).

Major Impact on the Field

Mission

The mission of the APS is "to promote and advance the scientific understanding and multidisciplinary integration of biological, psychological, behavioral and social factors in human health and disease, and to foster the dissemination and application of this understanding in education and health care (American Psychosomatic Society, 2011b)."

Awards and Scholarships

Awards include Alvin P. Shapiro Award, American Psychosomatic Society Scholar Awards, Cousins Center Global Outreach Awards, Donald Oken Fellowship, Herbert Weiner Early Career Award, Medical Student/Resident/Fellow Travel Scholarships, Minority Initiative Awards, Patricia R. Barchas Award in Sociophysiology, Paul D. MacLean Award, President's Award, and Travel Awards for MacLean Scholars (American Psychosomatic Society, 2011b).

Annual Meeting

The APA holds an annual 3-day open meeting in March. In this scientific and clinical assembly, investigators communicate, consider problems of conceptual relationships, and develop ideas that will stimulate further research (American Psychosomatic Society, 2011b). During the meeting, the APS members present scientific papers, participate in symposia, workshops, poster sessions, and invited lectures and addresses.

Journal

Psychosomatic Medicine: *Journal of Biobehavioral Medicine* founded in 1939 by the editor Dr. Dunbar, is the official and international peer-reviewed journal of APS. It is devoted to experimental and clinical research of interdisciplinary fields: behavioral biology, psychiatry, psychology, physiology, anthropology, and clinical medicine. It includes experimental and clinical studies on various perspectives and effects of the relationships among social, psychological, and behavioral factors, and physical processes in humans and animals. It publishes in print nine times a year, and most articles are online ahead of print (American Psychosomatic Society, 2010b).

Committees and Memberships

There are 10 committees such as Ad-Hoc Journal, Ad-Hoc Website, Awards, Fundraising, Liaison, Membership, Nominating, Past Leaders, Professional Education, and Program Committees (American Psychosomatic Society, 2010a).

There are four categories of memberships – Regular, Emeritus, Corresponding, and Associate (for students and trainees). Committee members are professionals and specialists from medical and health-related fields in behavioral and social sciences. A short membership is available as well (American Psychosomatic Society, 2011b).

Membership benefits (American Psychosomatic Society, 2011b) include:

- Subscription to printed and online *Psychosomatic Medicine*.
- Discount registration fees for the APS annual meeting.
- Networking and professional development in the society.
- Newsletter and e-newsletter (both three times a year).
- Opportunity of becoming a member of committees.
- Awards for professionals, students and trainees.
- Access to the APS international online membership directory.

References and Readings

American Psychosomatic Society (APS). (2010a). *About APS*. Retrieved March 20, 2011, from http://www.psychosomatic.org/about/index.cfm

American Psychosomatic Society (APS). (2010b). *About psychosomatic medicine: Journal of biobehavioral medicine*. Retrieved March 20, 2011, from http://www.psychosomaticmedicine.org/site/misc/about.xhtml

American Psychosomatic Society (APS). (2011a). New Editor-in-Chief for *Psychosomatic Medicine*. Retrieved April 3, 2011, from http://www.psychosomaticmedicine.org/site/misc/kopeditor.xhtml

American Psychosomatic Society (APS). (2011b). *69th annual scientific meeting, March 9–12, 2011, biobehavioral processes and health: Understanding mechanisms, implementing interventions [Brochure]*. McLean, VA: American Psychosomatic Society.

Levenson, D. (1994). *Mind, body, and medicine: A history of the American Psychosomatic Society*. McLean, VA: American Psychosomatic Society.

AMI

► Acute Myocardial Infarction

Anabolic Resistance

► Sarcopenia

Analgesia

Martin Deschner
Psychiatry, Division of Psychology,
The University of Texas Southwestern Medical Center at Dallas, Dallas, TX, USA

Definition

The term "analgesia" derives from the Greek words *an* (without) and *algesis* (pain) for "without pain" (Webster's Ninth New Collegiate Dictionary, 1988). It refers to relief from the sensation of pain or the loss of ability to feel pain while still remaining conscious. This term is to be distinguished from the broader term "anesthesia," which refers to a loss of sensation of all types, including pain, with or without loss of consciousness.

Analgesia can refer to partial or total relief from pain. When pain is reduced, some sensation persists but often without it being experienced as painful. Analgesia is often discussed in terms of medications or medical procedures. For example, opiate medications such as morphine, oxycodone, or hydrocodone are frequently used for their analgesic effects as are steroidal and nonsteroidal anti-inflammatory medications. Devices such as spinal cord stimulators, TENS units, and intrathecal pain medication pumps, as well as injections such as selective nerve root blocks, facet injections, or epidural steroid injections are also used for their analgesic effects, with varying degrees of success.

Behavioral medicine addresses analgesia from a biopsychosocial perspective. Psychologists and other behavioral health specialists frequently work in conjunction with medical and other professionals such as physical therapists, to help patients reduce and learn to cope with their acute or chronic pain. By improving the patient's understanding of his or her pain and emotional coping behaviors while also improving his or her capacity to reduce somatic tension and arousal, perceived pain is often reduced. Capacity to cope with remaining pain is generally increased through greater understanding of issues related to pain and mastery of pain management techniques.

Behavioral medicine approaches frequently involve education to improve understanding of the pain-causing condition. Education tends to reduce the patient's fear, feelings of powerlessness, and tendency to distort or catastrophically appraise their painful condition based on faulty information or assumptions.

Behavioral medicine uses cognitive behavioral therapy (CBT) approaches to help the patient to examine and change his or her irrational beliefs generally, and dysfunctional pain-specific beliefs in particular. CBT focuses on improving emotional coping skills, using cognitive behavioral training techniques such as reframing, correcting negative thinking patterns such as catastrophizing

or overgeneralizing, and improving communication and assertiveness skills. Correcting negative thinking patterns improves the patient's sense of mastery and provides him or her with effective tools to cope rationally with pain (Okifuji & Ackerlind, 2007).

Stress management training reduces the patient's somatic reactivity to pain by improving the patient's capacity to activate their parasympathetic response to pain. Patients are often taught such skills as diaphragmatic breathing, progressive muscle relaxation, meditation, and imagery techniques to help them break the vicious cycle of stress intensifying pain. Biofeedback training is often used to help the patient graphically see his or her somatic reactivity and also provides the patient with objective evidence as to the efficacy of the stress management techniques they are learning.

Cross-References

- ▶ Behavioral Medicine
- ▶ Cognitive Behavioral Therapy (CBT)
- ▶ Stress Management

References and Readings

Gatchel, R. J. (Ed.). (2004). *Clinical essentials of pain management*. Washington, DC: APA Books.

Okifuji, A., & Ackerlind, S. (2007). Behavioral medicine approaches to pain. *Anesthesiology Clinics, 25*, 709–719.

Turk, D. C., & Gatchel, R. J. (Eds.). (2002). *Psychological approaches to pain management: A practitioner's handbook* (2nd ed.). New York: Guilford Press.

Webster's ninth new collegiate dictionary. (1988). Springfield, MA: Merriam-Webster.

Weiner, R. S. (Ed.). (2002). *Pain management: A practical guide for clinicians* (6th ed.). New York: CRC Press.

Analytes

- ▶ Salivary Biomarkers

Anderson, Norman B. (1955–)

Norman B. Anderson
American Psychological Association, Washington, DC, USA

Biographical Information

A graduate of the North Carolina Central University in Durham, NC, Norman B. Anderson earned masters and doctoral degrees in clinical psychology from the University of North Carolina at Greensboro. He received additional clinical and research training at the Schools of Medicine at Brown and Duke Universities, including postdoctoral fellowships in psychophysiology and aging at Duke.

Anderson is the Chief Executive Officer and Executive Vice President of the American Psychological Association (APA). With 137,000 members and affiliates, APA is the largest and oldest of the world's psychological societies. Headquartered on Capitol Hill in Washington, DC, APA has a staff of over 550 and an annual budget of over $112 million. As the Chief Executive Officer, he is responsible for overseeing the overall management of the Association and works closely with the APA Board of Directors.

Major Accomplishments

Among his numerous accomplishments at APA, Anderson recently led a successful effort to create the first strategic plan in the 120-year history of the Association and oversaw APA's efforts to foster the inclusion of integrated care, health promotion and disease prevention, and mental health care in the new health care reform legislation.

Anderson was the founding Associate Director of the National Institutes of Health (NIH) in charge of behavioral and social science and was the first Director of the NIH Office of Behavioral and Social Sciences Research (OBSSR). At NIH, he was charged with facilitating behavioral and social sciences research across all of the [then] 24 Institutes and Centers of the National Institutes of Health. Under his purview was behavioral and social research in such areas as cancer, heart disease, child health, mental health, diabetes, aging, oral health and others. His special interest at NIH was advancing an integrated, transdisciplinary approach to health science, prevention, and health care.

Appointed to NIH in 1995, Anderson worked closely with the scientific community nationally to quickly establish the Office's long-term goals and to develop strategies for achieving them, resulting in the first OBSSR strategic plan. Under his leadership, the Office organized trans-institute funding initiatives totaling over $90 million in 5 years. The success of the Office prompted Congress to triple its budget, enabling it to have greater latitude in developing NIH-wide collaborative funding activities.

Anderson has held faculty appointments at both Duke University School of Medicine and the Harvard School of Public Health. He is well-known for his research and writings on health disparities and health behavior. He has received several awards for his research, including the 1986 New Investigator Award from the Society of Behavioral Medicine, the 1991 Award for Outstanding Contributions to Health Psychology from the American Psychological Association, and at least ten other significant awards from scientific societies and universities He has been awarded three honorary doctorate degrees.

Anderson is a Fellow of the American Association for the Advancement of Science, American Psychological Association, Association for Psychological Science, the Society of Behavioral Medicine, and the Academy of Behavioral Medicine Research. He is a Past President of the Society of Behavioral Medicine, served as President of the Board of Directors for the Starbright Foundation of Los Angeles (now the Starlight Children's Foundation), and is currently a Trustee of the Starlight Children's Foundation. He currently serves on the National Advisory Council for the National Institute on Aging at NIH the National Academic Affiliations Council in the Department of Affairs, the Board of Directors for the American Psychological Foundation, and the Board of Directors for the Excellence Foundation of the University of North Carolina at Greensboro. He also chaired the National Academy of Sciences Panel on Understanding Racial and Ethnic Health Disparities in Late Life.

He is the author and editor of several books, including serving as Editor-in-Chief of the two-volume *Encyclopedia of Health and Behavior*. He is also Editor-in-Chief of the flagship journal of APA, the *American Psychologist*.

Androgen

Chad Barrett
Department of Psychology, University of Colorado Denver, Denver, CO, USA

Synonyms

Androgenic hormone; Testoid

Definition

Androgens are a group of hormones that are present in males and females (though at lower levels)

and which primarily promote the development, and maintenance, of masculine traits. Androgens are produced in the testes, ovaries, placenta, and adrenal cortex. The most prominent androgen is testosterone. Other androgens include androstenedione, androstenediol, androsterone, dehydroepiandrosterone (DHEA) and dehydroepiandrosterone sulfate (DHEA sulfate), and dihydrotestosterone (DHT). Androgens are necessary for differentiation of male reproductive organs during fetal development, sexual maturation, spermatogenesis, genital function, and the development of male secondary sexual characteristics (Chang, 2002). Increases in androgens are associated with the maturational changes that occur in puberty. In addition, they play a role in the growth and functioning of muscles, bones, kidneys, liver, and the regeneration of red blood cells (Bagatell & Bremer, 2003). Androgens also influence aggression (Giammanco, Tabacchi, Giammanco, Di Majo, & La Guardia, 2005) and sexual desire, performance, and satisfaction (Hutchinson, 1995).

Androgens can reduce the risk of cardiovascular diseases, obesity, diabetes, bone loss, and Alzheimer's (Clarke & Khosla, 2009; Manolakou, Angelopoulou, Bakoyiannis, & Bastounis, 2009). In men with prostate cancer, hormone therapies may be used that reduce androgen levels in order to slow the growth of cancer cells. Low levels of androgen can increase the risk of cardiovascular diseases, obesity, diabetes, bone loss, Alzheimer's, and high cholesterol (Jordan & DonCarlos, 2008). Low levels of androgens are also related to reduced libido, erectile dysfunction, anemia, loss of muscle mass, weight gain, fatigue, and depression. Androgen supplements can often protect against these risks.

In women, high levels of androgen are associated with hirsutism, balding, acne, menstrual disorders, ▶ insulin resistance, ▶ diabetes, high ▶ cholesterol, high ▶ blood pressure, and cardiovascular diseases. Low levels are associated with low libido, ▶ fatigue, depression, and increased susceptibility to bone disease (Cheung, 1999).

Cross-References

▶ Sex Hormones

References and Readings

Bagatell, C. J., & Bremer, W. J. (Eds.). (2003). *Androgens in health and disease*. Totowa, NJ: Humana.

Chang, C. (2002). *Androgens and androgen receptor: Mechanisms, functions, and clinical applications*. Norwell, MA: Kluwer Academic.

Cheung, T. (1999). *Androgen disorders in women: The most neglected hormone problem*. Alameda, CA: Hunter House.

Clarke, B. L., & Khosla, S. (2009). Androgens and bone. *Steroids, 74*, 296–305.

Giammanco, M., Tabacchi, G., Giammanco, S., Di Majo, D., & La Guardia, M. (2005). Testosterone and aggressiveness. *Medical Science Monitor, 11*, 136–145.

Hutchinson, K. A. (1995). Androgens and sexuality. *The American Journal of Medicine, 98*(Suppl. 1), 111–115.

Jordan, C. L., & Don Carlos, L. (2008). Androgens in health and disease: An overview. *Hormones and Behavior, 53*, 589–595.

Manolakou, P., Angelopoulou, R., Bakayiannis, C., & Bastounis, E. (2009). The effects of endogenous and exogenous androgens on cardiovascular disease risk factors and progression. *Reproductive Biology and Endocrinology, 7*, 44. Available from www.rbej.com/content/7/1/44.

Androgenic Hormone

▶ Androgen

Ang II

▶ Angiotensin

Anger

▶ Anger Management
▶ Hostility
▶ Hostility, Psychophysiological Responses
▶ Negative Thoughts

Anger Assessment

► Anger, Measurement

Anger Expression

► Anger, Measurement

Anger Management

Mark A. Lumley and Lindsay Oberleitner
Department of Psychology, Wayne State University, Detroit, MI, USA

Synonyms

Anger; Emotion; Emotion regulation; Stress management

Definition

Anger management is a broad term referring to various techniques designed to help individuals manage or reduce their experience and expression of anger so that they will have better psychological, physical, and social health.

Description

Anger is a normal human emotion that is adaptive when elicited by appropriate social circumstances, specifically threatened or actual violation of something that one values. The experience and expression of anger in such circumstances can be healthy, but anger that is experienced or expressed too intensely or frequently and in inappropriate circumstances can contribute to many problems, including mood and anxiety disorders, cardiovascular disease, persistent pain, digestive problems, substance abuse, relational difficulties, and social disorder (Miller, Smith, Turner, Guijarro, & Hallet, 1996). Anger management, therefore, is targeted for patients whose anger is viewed by themselves or others as excessive, out of control, or having negative effects. People sometimes recognize their excessive anger and seek to manage it better, but more often, anger management is recommended or required by others who recognize that a person is too angry or expressing it too often or intensely (Saini, 2009).

Anger management typically refers to a program of specific intervention techniques that target different processes in the sequence of anger elicitation, experience, and expression. Anger management strategies can be placed in two general categories – strategies that directly avoid or reduce the experience and expression of anger, and strategies that facilitate identifying, experiencing, and adaptively expressing anger. The first category is more popular and is how anger management is traditionally defined (Glancy & Saini, 2005). This approach stems from research and practice on people who have excessive anger, such as cardiac patients with the Type A behavior pattern or people referred for treatment of aggressive behavior. The second category represents a newer, alternative view of the value of anger and other emotions (Gross, 2002). These two categories are discussed next.

Anger Avoidance and Reduction Strategies

A basic strategy to decrease anger is to identify and avoid specific anger triggers. For some individuals, anger is reliably elicited by certain people, situations, and activities, such as interacting with difficult coworkers or participating in competitive sports. An initial strategy, especially if other approaches are less successful, is to simply avoid those triggers.

Yet, behavioral avoidance often is not feasible or desirable and typically is considered a rather unsophisticated approach. Other anger management strategies can be used when encounters with anger triggers are inevitable. One such strategy is to reduce physiological activation when angered.

This typically is done by applying relaxation techniques (e.g., deep breathing, imagery, and progressive muscle relaxation) or distraction by engaging in pleasurable activities (e.g., exercise, playing a game, and reading). Such downregulation techniques can lower cardiovascular activity, increase calmness, and reduce tension until thoughts and feelings of anger have subsided. These techniques are particularly appropriate when situations are not controllable or when anger is only experienced occasionally ("state anger") (Del Vecchio & O'Leary, 2004).

Cognitive reappraisal techniques are used to identify, question, and modify the thoughts or interpretations that underlie anger. Reappraisal often involves evaluating and changing patterns of faulty thinking that are especially potent in anger, such as overgeneralization and exaggeration. For example, beliefs like "the world is always against me" can be examined and challenged with contradictory information. A specific cognitive reappraisal program has been developed by Williams and Williams (1993) that teaches people to question whether an anger-inducing situation is important, whether anger is appropriate and will change the outcome, and whether the expected outcome is worthwhile. Such questioning requires a pause in the anger experience, and allows the person to examine the situation and his or her reactions more logically. If any of the key questions are answered in the negative, then the anger experience can be aborted, or some alternative anger management strategy can be used, such as downregulation. Affirmative answers to these questions can lead to a decision to express anger in an adaptive manner.

There are several higher order, more sophisticated cognitive strategies to help manage anger. Perspective-taking or empathy-building refers to the ability to "walk in someone else's shoes," to feel what another person is feeling, and to see a situation from another's point of view. This approach may be helpful when the behavior of the anger-inducing person is not changeable, or when anger stems from victimization or abuse during childhood or when committed by strangers (e.g., being mugged or assaulted). The angry person is challenged to understand the circumstances that may have led to the offender's actions. This technique may help to attenuate anger, especially when added to the next technique.

An approach that stems from perspective-taking is to engage in forgiveness or "letting go." Anger typically follows victimization, and although anger may be justified, chronic anger eventually harms the person experiencing it. Forgiveness exercises help offended people free themselves from ongoing resentment, first by perspective-taking, and then by volitionally deciding to reduce the blame of the other person and to forgive them or let go of the resentment, whether or not the other person has apologized or made amends. Forgiveness or letting go returns control to victims, allowing them to view themselves as having the power and ability to heal. Although not necessary for forgiveness, many people incorporate this technique as part of their religious or spiritual practices (Lin, Mack, Enright, Krahn, & Baskin, 2004).

Anger Awareness, Experience, and Expression Strategies

It is important to recognize that anger is a vital, evolutionarily based, adaptive emotion when experienced and expressed appropriately. The experience of anger informs the person about actual or potential victimization or unjust experiences and motivates action to protect oneself or loved ones. Anger energizes and directs needed defense, protection, and the righting of social wrongs. Thus, it can be maladaptive to deny, disavow, or suppress anger, or transform it into sadness, guilt, or shame. Although in many social situations it is wise to suppress the expression of anger, a lack of anger awareness and chronic anger suppression can be detrimental (Iver, Korin, Higginbotham, & Davidson, 2010). Research suggests that the suppression of anger can increase pain, disrupt cognition, trigger depression, and impair intimacy (Burns, Quartana, & Bruehl, 2008). Furthermore, chronic anger sometimes stems from the failure to express one's needs, opinions, or dissatisfactions directly and effectively toward the appropriate person or target. Therefore, alternative approaches to managing anger

involve strategies or techniques that facilitate the awareness and adaptive expression of anger. Anger awareness and expression strategies can be divided into two types – assertiveness training and experiential exercises.

Assertiveness training is a popular approach found in many anger management programs, but it is fundamentally different from the anger reduction strategies described above. Training in assertion helps a person directly and honestly express thoughts and feelings to another person, while remaining mindful of the desired outcome and respecting the other person's experience. Assertive communication requires identifying one's own thoughts or desires and then directly yet skillfully expressing them verbally and nonverbally, without excessive apology, blame, or threat. Such direct assertion adaptively expresses anger, decreases feelings of victimization and helplessness that trigger mood and health problems, and helps prevent the inappropriate transfer or generalization of anger to innocent targets (Rakos, 1991).

Experiential techniques are relatively new approaches to dealing with anger. Training in mindfulness or meditation appears to help people recognize and experience their anger and other emotions in a nonjudgmental manner, and to distinguish awareness from action. Written emotional disclosure, or expressive writing, is a technique that helps people voice suppressed thoughts and feelings and narrate them into a story, thereby facilitating extinction of anger and/or the making of meaning and changes in understanding (Graham, Lobel, Glass, & Lokshina, 2008). Finally, experiential psychotherapy has developed several techniques that help people to experience and process unexpressed anger, including empty chair work and two-chair dialogues. All of these techniques can help people identify, clarify, and voice emotions, including anger, thereby helping them to develop insight, resolve conflicts, and make needed behavioral and interpersonal changes.

It is likely that the optimal approach to dealing with anger depends on individual differences among people in their usual anger regulation style. People who experience excessive anger or express it too readily likely need some of the traditional anger reduction techniques such as trigger avoidance, arousal downregulation, distraction, cognitive reappraisal, perspective-taking, and forgiveness. In contrast, people who are prone to excessive anger inhibition or suppression are more likely to benefit from techniques that help them experience and adaptively express anger, including assertiveness training and experiential exercises. This proposal, however, awaits empirical study.

Cross-References

► Anger, Measurement

References and Readings

Burns, J. W., Quartana, P. J., & Bruehl, S. (2008). Anger inhibition and pain: Conceptualizations, evidence, and new directions. *Journal of Behavioral Medicine, 31*, 259–279.

Del Vecchio, T., & O'Leary, K. D. (2004). The effectiveness of anger treatments for specific anger problems: A meta-analytic review. *Clinical Psychology Review, 24*, 15–34.

Glancy, G., & Saini, M. A. (2005). An evidenced-based review of psychological treatments of anger and aggression. *Brief Treatment and Crisis Intervention, 5*, 229.

Graham, J., Lobel, M., Glass, P., & Lokshina, I. (2008). Effects of written anger expression in chronic pain patients: Making meaning from pain. *Journal of Behavioral Medicine, 31*, 201–212.

Gross, J. J. (2002). Emotion regulation: Affective, cognitive, and social consequences. *Psychophysiology, 39*, 281–291.

Iyer, P., Korin, M. R., Higginbotham, L., & Davidson, K. W. (2010). Anger, anger expression, and health. In J. M. Suls, K. W. Davidson, & R. M. Kaplan (Eds.), *Handbook of health psychology and behavioral medicine* (pp. 120–132). New York: Guilford Press.

Lin, W.-F., Mack, D., Enright, R. D., Krahn, D., & Baskin, T. W. (2004). Effects of forgiveness therapy on anger, mood, and vulnerability to substance use among inpatient substance-dependent clients. *Journal of Consulting and Clinical Psychology, 72*, 1114–1121.

Miller, T., Smith, T., Turner, C., Guijarro, M., & Hallet, A. (1996). A meta-analytic review of research on hostility and physical health. *Psychological Bulletin, 119*, 322–348.

Rakos, R. F. (1991). *Assertive behavior: Theory, research, and training*. New York: Routledge.
Saini, M. (2009). A meta-analysis of the psychological treatment of anger: Developing guidelines for evidence-based practice. *The Journal of the American Academy of Psychiatry and the Law, 37*, 473–488.
Williams, R., & Williams, V. (1993). *Anger kills: Seventeen strategies for controlling the hostility that can harm your health*. New York: HarperCollins.

Anger, Measurement

Stephan Bongard
Department of Psychology, Goethe-University, Frankfurt am Main, Germany

Synonyms

Anger assessment; Operationalization of anger

Definition

Assessment of states of anger or of the personality disposition for anger responses.

Description

Anger is usually considered as a basic emotion that emerged during evolution because it served the function of preparing the organism for fight against enemies and to overcome obstacles (Plutchik, 1994). Therefore, it is associated with a specific response pattern that communicates to the environment preparedness for fight and that supplies the body with metabolic demands for fight or flight. The response pattern is thought to be independent of cultural influences while so-called display rules regulate under which conditions the expression of anger is appropriate or not. Another component characteristic of the state of anger is specific cognitions (appraisals). Measurements of anger aim at assessing responses of one or more of these components. Consequently, instruments for collecting self-reports, behavioral observations, and physiological measures for the assessment of anger have been developed. Measures of anger should be able to discriminate anger from other emotional states, provide some quantification of anger, and deliver results that are free from deliberate manipulations by respondents.

Self-reports

The most common way to assess anger is the use of psychometric self-reports in which individuals respond to statements describing their cognitions, feelings, attitudes, and behavior. Anger can be measured as a state, that is, an acute condition of feelings ranging in intensity from mild irritation or annoyance to intense fury and rage, or anger can be measured as a trait, that is, an enduring behavior disposition for anger states. Individuals high in trait anger are assumed to experience state anger more often, more intensely and longer than individuals low in state anger (Spielberger et al., 1985). Table 1 lists some often-used instruments for the assessment of anger.

Self-reports are usually specific to anger and provide an easy means of quantification (e.g., counting the "Yes" answers) but they can also easily be manipulated by respondents.

Behavioral Observations

The experience of anger often goes along with a characteristic facial expression but it does not have to. Depending on situational conditions, social norms, and individual differences, the expression of anger is more or less appropriate. However, if anger is expressed in the face, the inner eyebrows are lowered and brought closer together. Often, this is accompanied by glaring eyes, widened nostrils, lips that are tightly pressed together, or flashing of the teeth. This facial behavior pattern can be coded using the Facial Action Coding System (FACS; Ekman & Friesen, 1978). The central and most important movement for anger is the constriction of the eyebrows. The facial expression of basic emotions such as anger is assumed to be hereditary. It can already be observed in young children. Figure 1 shows the facial expression of a 5-year-old boy after he was asked to display anger.

Anger, Measurement, Table 1 Examples of self-report instruments for the assessment of anger (in chronically order)

Scale	Author/s
Picture-Frustration Test (PFT)	Rosenzweig (1945)
Cook-Medley Hostility Scale (Ho-Scale)	Cook and Medley (1954)
Buss-Durkee Hostility Scale (BDHS)	Buss and Durkee (1957)
Harburg-Items	Harburg et al. (1973)
Novaco Anger Inventory	Novaco (1975)
Framingham Anger Items	Haynes et al. (1978)
Subjective Anger Scale (SAS)	Knight et al. (1985)
Multidimensional Anger Inventory (MAI)	Siegel (1986)
State Trait Anger Expression Inventory (STAXI)	Spielberger et al. (1985)
State Trait Anger Expression Inventory 2 (STAXI-2)	Spielberger (1999)

Anger, Measurement, Fig. 1 Facial expression of a 5-year-old boy after he was asked to display anger

While facial expression can validly be assessed by trained raters, this measure provides only a moderate quantification of anger. Usually, one can discriminate between weak and strong anger. Though humans can control facial expressions, they are difficult to manipulate convincingly to an experienced rater.

Physiological Measures

The state of anger is associated with feelings of hyperactivation, restlessness, tension, and power. These feelings are caused by an activation of the sympathetic branch of the autonomic nerve system combined with vagal withdrawal. This activation can be measured using, for example, blood pressure and heart rate readings or by registration for electrodermal activity. Physiological response patterns of emotional activation are usually unspecific and it is not possible to draw conclusions from the observed pattern of activation to the underlying emotional quality. However, when people report being angry it is usually accompanied by increases in systolic and particularly diastolic blood pressure and total peripheral resistance. Also, increased heart rates, number of skin conductance responses, and muscle activity are often reported (Stemmler, 2010).

Studies using brain-imaging methods (fMRI, PET) report activation of the orbitofrontal cortex during episodes of anger (Murphy, Nimmo-Smith, & Lawrence, 2003). Further, anger seems to be associated with an asymmetric activation of the frontal cortex. Measuring spontaneous EEG activity, Harmon-Jones (2003) reported greater activation of the left frontal brain relative to the right frontal brain.

Physiological measures provide very exact, but unspecific quantifications. Many physiological responses can be observed during other emotional states as well. So far, no response pattern has been defined that is unique to the state of anger. Though biofeedback studies show that physiological states, too, can be manipulated after extensive training, there is almost no way to do this spontaneously.

Conclusion

Anger manifests itself on the level of subjective experiences, observable behavior, and physiological activation. For each of these qualities, there are standardized measures for the assessment of anger: self-reports, facial expression coding, and physiological registration. However, since each of these measures has specific strengths and weaknesses, anger should be assessed on at least two different levels.

Cross-References

- ▶ Affect
- ▶ Affect Arousal
- ▶ Emotional Control
- ▶ Emotional Responses

References and Readings

al'absi, M., & Bongard, S. (2006). Neuroendocrine and behavioral mechanisms mediating the relationship between anger expression and cardiovascular risk: Assessment consideration and improvements. *Journal of Behavioral Medicine, 29*, 573–591.

Buss, A., & Durkee, A. (1957). An inventory for assessing different kinds of hostility. *Journal of Consulting Psychology*, *21*, 343–349.

Cook, W.W., & Medley, D.M. (1954). Proposed hostility and pharisaic-virtue scales for the MMPI. *The Journal of Applied Psychology*, *38*, 414–418.

Ekman, P., & Friesen, W. V. (1978). *Facial action coding system*. Palo Alto, CA: Consulting Psychologists Press.

Harburg, E., Erfurt, J.C., Hauenstein, L.S., Chape, C., Schull, W.J., & Schork, M.A. (1973). Socio-ecological stress, suppressed hostility, skin color, and black-white male blood pressure: Detroit. *Psychosomatic Medicine*, *35*, 276–296.

Harmon-Jones, E. (2003). Anger and the behavioural approach system. *Personality and Individual Differences, 35*, 995–1005.

Haynes, S., Levine, S., Scotch, N., Feinleib, M., & Kannel, W.B. (1978). The relationship of psy- chosocial factors to coronary heart disease in the Framingham Study. I. Methods and risk factors. *American Journal of Epidemiology, 107*, 362–383.

Knight, R. G., Ross, R.A., Collins, J. I., & Parmenter, S. A. (1985). Some norms, reliability and preliminary validity data for an S-R inventory of anger: The Subjective Anger Scale (SAS). *Personality and Individual Differences*, *6*, 331–339.

Murphy, F. C., Nimmo-Smith, I., & Lawrence, A. D. (2003). Functional neuroanatomy of emotions: A meta-analysis. *Cognitive, Affective, & Behavioral Neuroscience, 3*, 207–233.

Novaco, R.W. (1975). Anger Control: The development and evaluation of an experimental treatment. Lexington, Mass.: Lexington Books.

Plutchik, R. (1994). *The psychology and biology of emotion*. New York: Harper.

Potegal, M., Stemmler, G., & Spielberger, C. D. (Eds.). (2010). *International handbook of anger*. New York: Springer.

Rosenzweig, S. (1945). The Picture-Association Method and its application in a study of reactions to frustration. *Journal of Personality*, *14*, 3–23.

Siegel, J.M. (1986). The multidimensional anger inventory. *Journal of Personality and Social Psychology*, *51*, 191–200.

Spielberger, C. D. (1999). Professional manual for the State-Trait Anger Expression Inventory-2 (STAXI-2). Odessa, FL: *Psychological Assessment Resources*.

Spielberger, C. D., Johnson, E. H., Russell, S. F., Crane, R. J., Jacobs, G. A., & Worden, T. J. (1985). The experience and expression of anger: Construction and validation of an anger expression scale. In M. A. Chesney & R. H. Roseman (Eds.), *Anger and hostility in cardiovascular and behavioral disorders* (pp. 5–30). New York: Hemisphere/McGraw-Hill.

Stemmler, G. (2010). Somatovisceral activation during anger. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *International handbook of anger* (pp. 103–121). New York: Springer.

Anger-In

- ▶ Anger, Measurement

Anger-Out

- ▶ Anger, Measurement

Angina Pectoris

Siqin Ye
Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Synonyms

Chest pain

Definition

Angina pectoris is the classic manifestation of cardiac ischemia, which occurs when there is a mismatch between myocardial oxygen supply and demand. The sensation is typically described as substernal chest pressure or tightness, often accompanied by shortness of breath, and often radiates to the jaw, neck, and left arm or shoulder.

It can also be associated with nausea, vomiting, and diaphoresis. The onset typically occurs over several minutes and is frequently triggered by physical activity and emotional stress (Panju, Hemmelgarn, Guyatt, & Simel, 1998).

The most common cause of angina pectoris is coronary atherosclerosis, either via stable but flow-limiting stenotic lesions or via acute rupture of a vulnerable plaque that causes total or subtotal occlusion of a coronary artery. Other conditions that limit myocardial oxygen supply and thereby cause angina include coronary vasospasm, vasculitides of the coronary vasculature, myocardial bridge, dissection of the coronary arteries, or other congenital anomalies of the coronary arteries that can cause impairment of blood flow. Conditions that can increase myocardial oxygen demand include aortic stenosis or hypertrophic cardiomyopathy or general conditions such as tachycardia, anemia, sepsis, or thyrotoxicosis that often exacerbate cardiac ischemia in patients with underlying coronary atherosclerotic disease (Cannon & Lee, 2008). The appropriate management of angina pectoris depends on the underlying cause.

Cross-References

► Chest Pain

References and Readings

Cannon, C. P., & Lee, T. H. (2008). Approach to the patient with chest pain. In P. Libby, R. O. Bonow, D. L. Mann, D. P. Zipes, & E. Braunwald (Eds.), *Braunwald's heart disease: A textbook of cardiovascular medicine* (pp. 1195–1205). Philadelphia: Saunders Elsevier.

Panju, A. A., Hemmelgarn, B. R., Guyatt, G. H., & Simel, D. L. (1998). Is this patient having a myocardial infarction? *Journal of American Medical Association, 280*(14), 1250–1263.

Angiogram

► Angiography/Angioplasty

Angiography/Angioplasty

Amy Jo Marcano-Reik
Department of Bioethics, Cleveland Clinic, Cleveland, OH, USA
Center for Genetic Research Ethics and Law, Case Western Reserve University, Cleveland, OH, USA

Synonyms

Angiogram; Angioplasty; Arteriography

Definition

Angioplasty is a medical technique that allows for visualization of the inside of blood vessels and various organs of the body. This is a moderately invasive procedure, which involves a widening of narrowed or obstructed blood vessels to increase blood flow (http://www.nhlbi.nih.gov/health/health-topics/topics/angioplasty/). This procedure may involve the insertion of a mesh stent or balloon to open the blocked arteries (Fischman et al., 1994, New England Journal of Medicine). Angioplasty is a common procedure for atherosclerosis, which is the buildup of a fatty substance, called plaque, in the arteries. The angiogram is the physical record produced from the procedure.

Cross-References

► Atherosclerosis
► Cardiac Surgery
► Cardiovascular Disease
► Coronary Artery Disease

References and Readings

Cleland, J. G., Calvert, M., Freemantle, N., Arrow, Y., Ball, S. G., Bonser, R. S., et al. (2011). The Heart Failure Revascularization Trial (HEART). *European Journal of Heart Failure, 13*(2), 227–233.

Fischman, D. L., Leon, M. B., Baim, D. S., Schatz, R. A., Savage, M. P., Penn, I., et al. (1994). A randomized comparison of coronary-stent placement and balloon angioplasty in the treatment of coronary artery disease. Stent restenosis study investigators. *New England Journal of Medicine, 331*(8), 496–501.

Retrieved from http://www.nhlbi.nih.gov/health/health-topics/topics/angioplasty/

Angioplasty

► Angiography/Angioplasty

Angiotensin

Seth Hurley
Department of Psychology, University of Iowa, Iowa City, IA, USA

Synonyms

AII; Ang II

Definition

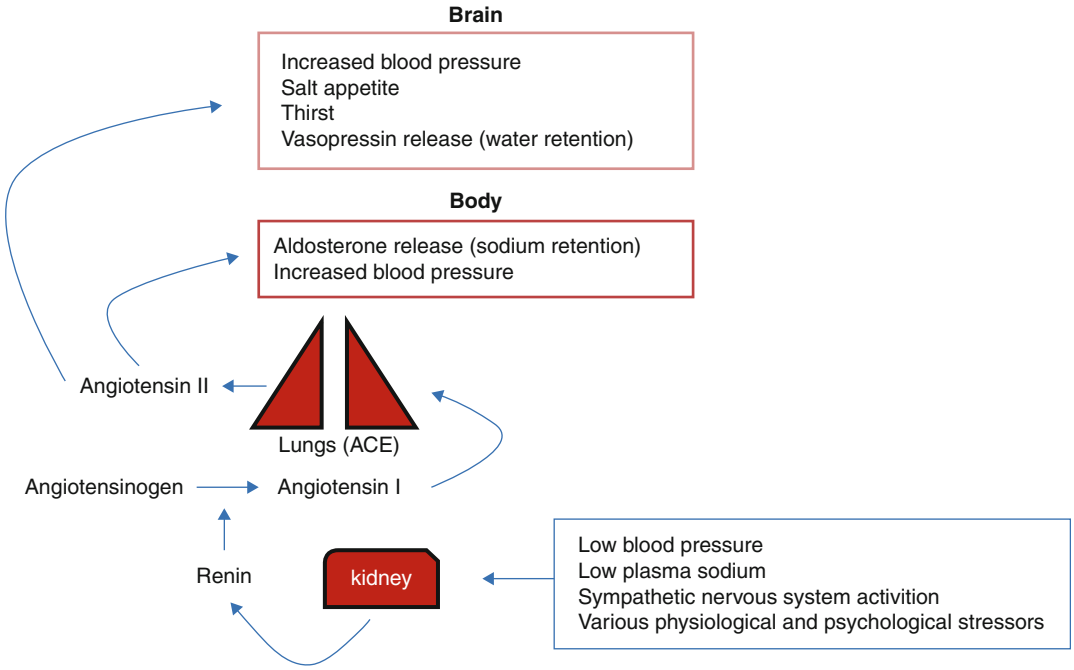
Angiotensin II is a polyfunctional octapeptide generated in response to stress and challenges to body fluid homeostasis. This peptide primarily acts on metabotropic angiotensin II type 1 (AT_1) receptors to accomplish its behavioral and physiological effects. Angiotensin II acts as both a hormone and, in the brain, as a neuromodulator.

Biosynthesis – Angiotensin II is the product of a multienzyme, multisubstrate biosynthesis pathway known as the renin-angiotensin system (RAS). In the classic and best studied RAS, renin is released from the granular cells of the juxtaglomerular apparatus cells of the kidney by various stimuli, including activation of the sympathetic nervous system. Renin acts on constitutively present angiotensinogen in the plasma to catalyze the conversion of angiotensinogen to angiotensin I, an inactive precursor to angiotensin II. Circulating angiotensin I is converted by angiotensin-converting enzyme, located primarily in the lungs, into the bioactive peptide angiotensin II. Researchers are now aware that there are many RASs. In contrast to angiotensin II's system-wide generation, many cells including cardiac myocytes and brain neurons have localized intracellular components of the RAS.

Stimuli for release – Common stimuli for activation of the RAS are perturbations of body fluid homeostasis. For example, loss of extracellular fluid and low plasma sodium activate the RAS. Environmental stressors also stimulate renin release from the kidneys and downstream angiotensin II synthesis via activation of the renal sympathetic nerve. Thus, angiotensin II is a stress hormone. Furthermore, pathologies such as hypertension and heart failure are associated with elevated activity of the systemic or cellular RASs.

Physiological effects – Initially, circulating angiotensin II was studied for its pressor effects in the periphery. In addition to a direct effect on cardiovascular smooth muscle, angiotensin II enhances the release of norepinephrine from sympathetic neurons and potentiates the effects of norepinephrine on vasoconstriction. Importantly, through AT_1 receptors in the brain, angiotensin II acts to promote water- and salt-seeking behaviors, release vasopressin, and initiate a centrally mediated pressor response. Vasopressin acts as a hormone at the kidney to decrease diuresis and thus conserve water. In addition, angiotensin II stimulates the adrenal gland to release aldosterone. Aldosterone is a hormone that signals the kidney to retain sodium and the brain to promote salt-seeking behaviors (Jackson, 2010).

Psychological effects – Since the discovery that angiotensin II acts as a neuromodulator in the brain, researchers have been interested in psychological effects of angiotensin II, for example, recent evidence has supported a role for angiotensin II in the stress response. Stressors cause increased angiotensin II synthesis and release. Administration of candesartan, an AT_1 receptor antagonist, in doses that access the brain attenuates the hormonal response to stressors in rats, including decreases in corticosterone, adrenocorticotropic hormone, epinephrine,



Angiotensin, Fig. 1 Synthesis and effector pathway for hormonal angiotensin II. Various stimuli cause renin release from the kidneys which acts on constitutively present angiotensinogen in blood plasma to generate angiotensin I. Angiotensin I is converted by angiotensin converting enzyme into bioactive angiotensin II. Angiotensin II acts in the brain and in the body to protect body fluid homeostasis

norepinephrine, and aldosterone. Evidence from preclinical models also indicates that angiotensin II produces anxiety-like behavior, and blocking angiotensin II ameliorates these effects. Furthermore, it is likely that angiotensin II has a role in depression. Preclinical models of depression show that angiotensin II antagonists reduce depressive-like behavior and antidepressants antagonize the thirst and smooth muscle contraction effects of angiotensin II (Gard, 2002; Saavedra et al., 2005). Drugs that inhibit the synthesis of angiotensin II are also reported to be mood enhancing. Finally, in nonhuman animals learning and memory are enhanced through central AT_4 receptor activation (Wright & Harding, 2004).

Given the various psychological effects of angiotensin II, it is likely that individuals with cardiovascular disorders associated with increased circulating angiotensin II, such as hypertension and chronic heart failure, are more likely to suffer psychological disorders. Numerous studies have found an association between heart failure and depression, and preclinical models show that heart failure can cause anhedonia, a symptom of major depressive disorder (Johnson & Grippo, 2006). Furthermore, evidence indicates an association between hypertension and depression (Lobo-Escolar et al., 2008). Whether blocking angiotensin II ameliorates psychological pathologies in patients with cardiovascular disorders remains to be validated (Fig. 1).

The classic renin-angiotensin system. Stimuli such as low blood pressure, plasma sodium, or environmental stressors cause the kidney to release renin into blood plasma. Renin is an enzyme that catalyzes the conversion of plasma angiotensinogen into angiotensin I. Circulating angiotensin I is converted by angiotensin-converting enzyme, located primarily in the lungs, into angiotensin II. Angiotensin II acts through the periphery and central nervous system to expand body fluids.

Cross-References

- ▶ Heart Disease and Stress
- ▶ Heart Failure
- ▶ Hypertension
- ▶ Stress

References and Readings

Gard, P. (2002). The role of angiotensin II in cognition and behaviour. *European Journal of Pharmacology, 438* (1–2), 1–14.

Jackson, E. (2010). Renin and Angiotensin. In J. G. Hardman & L. E. Limbird (Eds.), *Goodman and Gilman's the pharmacological basis of therapeutics* (Chap. 30, 11th ed.). New York: McGraw-Hill. Retrieved from http://www.accessmedicine.com/content.aspx?aID=944099

Johnson, A. K., & Grippo, A. J. (2006). Sadness and broken hearts: Neurohumoral mechanisms and co-morbidity of ischemic heart disease and psychological depression. *Journal of Physiology and Pharmacology, 57*(Suppl. 11), 5–29.

Lobo-Escolar, A., Roy, J., Saz, P., De-la-Cámara, C., Marcos, G., & Lobo, A. (2008). Association of hypertension with depression in community-dwelling elderly persons: Results from the ZARADEMP project. *Psychotherapy and Psychosomatics, 77*(5), 323–325.

Saavedra, J., Ando, H., Armando, I., Baiardi, G., Bregonzio, C., Juorio, A., et al. (2005). Anti-stress and anti-anxiety effects of centrally acting angiotensin II AT1 receptor antagonists. *Regulatory Peptides, 128*(3), 227–238.

Wright, J. W., & Harding, J. W. (2004). The brain angiotensin system and extracellular matrix molecules in neural plasticity, learning, and memory. *Progress in Neurobiology, 72*(4), 263–293.

Angiotensin-Converting Enzyme Inhibitors (ACE Inhibitors)

Seth Hurley
Department of Psychology, University of Iowa, Iowa City, IA, USA

Definition

Angiotensin-converting enzyme (ACE) inhibitors are a class of drugs that inhibit ACE activity. ACE is a key enzyme of the renin-angiotensin system (RAS), the synthesis pathway of angiotensin II. Antagonizing ACE activity prevents the conversion of biologically inactive angiotensin I into bioactive angiotensin II (for details of the RAS and the effects of angiotensin II, see ▶ Angiotensin, this volume).

Therapeutic use – ACE inhibitors are commonly used to treat cardiovascular disorders such as hypertension and chronic heart failure. Both of these disorders are associated with increased activity of the renin-angiotensin system, which through angiotensin II action has detrimental effects on the cardiovascular system. In addition to increasing blood pressure, angiotensin II causes both cardiovascular remodeling (changes in cardiovascular tissue distribution) and cardiovascular hypertrophy (increased growth of cardiovascular tissue). In hypertensive patients, ACE inhibitors are used to reduce blood pressure. In patients with left ventricular systolic dysfunction, ACE inhibitors are particularly effective in delaying or preventing congestive heart failure. Finally, in patients with myocardial infarctions, ACE inhibitors reduce overall mortality.

ACE inhibition and aldosterone breakthrough – ACE inhibitors chronically reduce circulating angiotensin II; however, they may only acutely reduce circulating aldosterone. In a phenomenon known as aldosterone breakthrough, aldosterone, a downstream hormone activated by the renin-angiotensin system, rises after chronic ACE inhibition. Aldosterone breakthrough occurs in hypertensive and heart failure patients receiving ACE inhibitors. Aldosterone has detrimental effects on the cardiovascular system and synergizes with angiotensin II to increase blood pressure and produce cardiovascular remodeling and hypertrophy. Thus, combined treatment of aldosterone synthesis blockers and ACE inhibitors is reported to be more successful in treating cardiovascular disorders (Jackson, 2010; Sato & Saruta, 2003).

Psychological effects of ACE administration – Consistent with the idea that angiotensin II contributes to lowered mood (see ▶ Angiotensin, this

volume), ACE inhibitors are reported to enhance mood in samples with cardiovascular disorders. One study reported a greater incidence of depressed mood associated with anxiety and decreased cognitive function in patients with hypertension. These deficits were absent in hypertensives taking ACE inhibitors (Braszko, Karwowska-Polecka, Halicka, & Gard, 2003). Interestingly, patients taking ACE inhibitors or angiotensin II receptor antagonists are more likely to continue drug therapy compared to patients taking other antihypertensive medications (Elliott, Plauschinat, Skrepnek, & Gause, 2007). However, it is unclear if this is caused by mood-elevating effects of ACE inhibitors or less severe side effects of ACE inhibitors relative to other cardiovascular disorder medications.

Cross-References

- ▶ Angiotensin
- ▶ Heart Failure
- ▶ Hypertension

References and Readings

Braszko, J., Karwowska-Polecka, W., Halicka, D., & Gard, P. (2003). Captopril and enalapril improve cognition and depressed mood in hypertensive patients. *Journal of basic and clinical physiology and pharmacology, 14*(4), 323.

Elliott, W., Plauschinat, C., Skrepnek, G., & Gause, D. (2007). Persistence, adherence, and risk of discontinuation associated with commonly prescribed antihypertensive drug monotherapies. *The Journal of the American Board of Family Medicine, 20*(1), 72.

Jackson, E. (2010). Renin and Angiotensin. In J. G. Hardman & L. E. Limbird (Eds.), *Goodman and Gilman's the pharmacological basis of therapeutics* (Chap. 30, 11th ed.). New York: McGraw-Hill. Retrieved from http://www.accessmedicine.com/content.aspx?aID=944099

Sato, A., & Saruta, T. (2003). Aldosterone breakthrough during angiotensin-converting enzyme inhibitor therapy. *American journal of hypertension, 16*(9), 781–788.

Anorexia Nervosa

Anna Maria Patino-Fernandez
Department of Pediatrics, University of Miami, Miami, FL, USA

Definition

The *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition, text revision (APA, 2000) includes as the essential features of anorexia nervosa the following: (1) refusal to maintain body weight: weight at least 15% below what is considered normal for others of the same height and age; (2) intense fear of gaining weight or becoming fat; (3) disturbance in body image or denial of seriousness of current low body weight; belief that the one is overweight though in reality they are underweight; and (4) amenorrhea: missing at least three consecutive menstrual cycles (if a female of childbearing age).

There are two types of anorexia nervosa: restricting and binge-eating/purging (APA, 2000). Many individuals with anorexia will severely restrict their calories sometimes taking in only a few hundred calories a day or just water. This is called the restricting type. Other individuals who eat and then fear weight gain may vomit or exercise; this type of anorexia is called the binge eating/purging type. The majority of patients with this disorder are female. This is a potentially very dangerous psychiatric disorder as fatal health complications may occur if untreated.

Warning signs for anorexia nervosa include the following (NIMH, 2011):

(a) Intense, persistent fear of putting on weight.
(b) Low self-esteem related to appearance.
(c) Desire to lose weight.
(d) Body dissatisfaction.
(e) Food preoccupation.
(f) Deliberate self-starvation with weight loss.
(g) Refusal to eat or highly restrictive eating.
(h) Excessive facial/body hair because of inadequate protein in the diet.
(i) Compulsive exercise.

(j) Abnormal weight loss.
(k) Sensitivity to cold: People with anorexia nervosa often complain of feeling cold (hypothermia) because their body temperature drops.
(l) Absent or irregular menstruation.
(m) Hair loss: Individual may develop lanugo (a term used to describe the fine hair on a newborn) on their body.

Treatment of Anorexia Nervosa

The first goal for treatment is to ensure the person's physical health, which involves restoring a healthy weight. Reaching this goal may require hospitalization. Once a person's physical condition is stable, treatment usually involves individual psychotherapy and family therapy. Supportive group therapy may follow, and self-help groups within communities may provide ongoing support. Based on existing, limited evidence, it appears that behavioral family therapy may be considered a reasonable first-line approach for treatment of anorexia in adolescents (Lock et al., 2010).

Another positive development in the treatment of anorexic patients who have regained their weight is in the area of psychopharmacology. While drug treatments such as antidepressants have had little effect combating the symptoms of anorexia, researchers are now finding that medication can help if the patient's weight has returned to normal. In one study (Couturier & Lock, 2007), two thirds of anorexics who took Prozac after they had recovered their weight did not relapse, compared with 16% who took a placebo. Atypical antipsychotics, especially olanzapine, have been tried in open-label non-randomized single-case studies and suggest a possible benefit in increasing weight and decreasing weight obsession. At present, there are no randomized trials using psychopharmacological interventions in children.

Cross-References

▶ Eating Disorders: Anorexia and Bulimia Nervosa

References and Readings

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author. Text Revision.
Couturier, J., & Lock, J. (2007). A review of medication use for children and adolescents with eating disorders. *Journal of Canadian Academy of Child and Adolescent Psychiatry, 16*, 173–176.
Lock, J., Le Grange, D., Agras, W. S., Moye, A., Bryson, S. W., & Jo, B. (2010). Randomized clinical trial comparing family-based treatment to adolescent focused individual therapy for adolescents with anorexia nervosa. *Archives of General Psychiatry, 67*, 1025–1032. doi:10.1001/archgenpsychiatry.2010.128.
National Institute of Mental Health (NIMH). (2011). *Eating disorders*. Retrieved from http://www.nimh.nih.gov/health/publications/eating-disorders/complete-index.shtml

Antagonism

▶ Hostility, Psychophysiological Responses

Anterior Hypothalamic Area

▶ Hypothalamus

Anthropometric

▶ Body Mass Index

Anthropometrics

▶ Body Composition

Antianxiety Drug

▶ Anxiolytic

Antibodies

Anna C. Phillips
Sport & Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Immunoglobulins

Definition

Antibodies are proteins secreted by white blood cells (B lymphocytes). Their task is to circulate in the body and tag, destroy, or neutralize bacteria, viruses, or other harmful or foreign materials (antigens). They do this by opsonizing or coating foreign materials which marks them for destruction or neutralization.

Antibodies are also called immunoglobulins (Ig), which are present on the surface of B cells and act as receptors for foreign materials. Immunoglobulins can be classified into several classes: IgA, IgE, IgM, IgG, and IgD, some of which have further subtypes. Each type of antibody has a range of different immune functions. IgA is found at the mucosal surfaces (e.g., mouth, nose, gastrointestinal tract) and can be measured in saliva. IgM is the first type of antibody produced in response to a novel foreign material. IgG is the most common antibody found in the body. Vaccines work by presenting a dead or altered form of an antigen to the immune system in order to provoke an antibody response.

Cross-References

- ▶ Antigens
- ▶ Immunoglobulins

References and Readings

Goldsby, R. A., Kindt, T. J., Osborne, B. A., & Kuby, J. (2003). *Immunology* (5th ed.). New York: W.H. Freeman.

Antibody Generators

- ▶ Antigens

Antidepressant Medications

Tatsuo Akechi
Department of Psychiatry and Cognitive-Behavioral Medicine, Graduate School of Medical Sciences, Nagoya City University, Nagoya, Aichi, Japan

Synonyms

Antidepressants

Definition

Effective drugs for treatment of patients with depressive disorders

Description

History of antidepressants began in the 1950s when imipramine, currently known as one of the tricyclic antidepressants (TCA), appeared. Imipramine was initially developed as a derivative of chlorpromazine (antipsychotic agent), and it was hoped that imipramine would be an effective antipsychotic drug (Hales & Yudofsky, 2003; Sadock & Sadock, 2003). Interestingly, imipramine did not have antipsychotic efficacy; however, it was shown to be effective in the treatment of depression. Subsequently, many other antidepressants have been developed, and there are approximately 30 different antidepressants now, although some antidepressants are not available in some countries.

Antidepressant medications are most popular treatments for patients with depressive disorders, especially patients with mild to moderate major depression (Association American

Psychiatric, 2010). Although there are several different types of antidepressants including TCA, tetracyclic antidepressants, selective serotonin reuptake inhibitors (SSRI), serotonin noradrenaline reuptake inhibitors (SNRI), monoamine oxidase inhibitors (MAOIs), and other antidepressants, the effectiveness of these antidepressant medications is generally comparable between classes and within classes of medications (Association American Psychiatric, 2010; Hales & Yudofsky, 2003; Sadock & Sadock, 2003). On the other hand, side effect profiles clearly differ among the different classes of antidepressants.

Antidepressants affect the serotonergic and/or catecholaminergic systems in the central nervous system, and these changes are considered to be associated with treatment effect for patients with depressive disorders. The effect of the antidepressants will be gradually observed after a couple of weeks since implementation of the pharmacotherapy.

Most TCAs inhibit the reuptake of norepinephrine, serotonin, and, to a lesser extent, dopamine. Furthermore, TCAs also block muscarinic cholinergic receptors, H1 histamine receptors, and α1-adrenergic receptors. Most side effect profiles of the TCAs are mainly produced by these mechanisms. Potential side effects of TCAs include arrhythmias, orthostatic hypotension, sedation, constipation, dry mouth, urinary hesitancy, and so on. Currently available TCAs are imipramine, amitriptyline, nortriptyline, clomipramine, etc. Tetracyclic antidepressants are developed to reduce side effect profiles of TCAs; however, these drugs have similar side effect profiles. Tetracyclic antidepressants include maprotiline and mianserin.

In 1980s, SSRIs, specifically inhibiting the reuptake of serotonin, were developed as antidepressants. First, SSRI is fluoxetine and other SSRIs, including paroxetine, sertraline, citalopram, escitalopram, and so on, are also available now. Side effects of SSRIs are nausea, vomiting, insomnia, activation (e.g., restlessness, agitation), sexual dysfunction, gastrointestinal bleeding, etc. We can also use some SNRIs which specifically inhibit the reuptake of serotonin and norepinephrine. Usable SNRIs are venlafaxine, milnacipran, duloxetine, and so on. In general, side effect profiles of SNRIs are similar to the ones of SSRIs; however, some SNRIs can cause hypertension and urinary retention.

MAOIs increases concentration of monoamines through inhibiting monoamine oxidase. MAOIs currently used as antidepressants include phenelzine, moclobemide, and so on. MAOIs have unique pharmacological property, and this have been characterized by showing to be effective in treating depressed patients, with atypical features (e.g., reactive mood, sensitivity to rejection).

Other types of antidepressants include bupropion (dopamine norepinephrine reuptake inhibitor), mirtazapine (norepinephrine-serotonin modulator), trazodone (serotonin modulator), nefazodone (serotonin modulator), and so on. Bupropion is classified as a dopamine norepinephrine reuptake inhibitor, the effect of dopamine, however, is relatively weak. Mirtazapine is thought to work through noradrenergic and serotonergic mechanisms despite of not being a reuptake inhibitor.

In addition to efficacy for depressive disorders, antidepressants are also effective for treatment of patients with anxiety disorders (Hales & Yudofsky, 2003; Sadock & Sadock, 2003). Recent studies suggest that antidepressants are also useful for treating other medical condition such as neuropathic pain and depressive and negative symptoms of schizophrenia. Some antidepressants have efficacy for other medical conditions. For example, SSRIs are used for treatment of bulimia nervosa. Bupropion can be a choice for patients who would like to quit smoking. Thus, although antidepressants were initially developed for treatment of depression, these medications have been known to be useful for broader medical conditions.

Cross-References

▶ Neurotransmitter

References and Readings

Association American Psychiatric. (2010). Practice guideline for the treatment of patients with major depressive disorder, 3 edition. *The American Journal of Psychiatry, 167*(Suppl), 1–118.

Hales, R. E., & Yudofsky, S. C. (2003). *Textbook of clinical psychiatry* (4th ed.). Arlington, VA: The American psychiatric Publishing.

Sadock, B. J., & Sadock, V. A. (2003). *Kaplan & Sadock's synopsis of psychiatry* (9th ed.). Philadelphia: Lippincott, Williams & Wilkins.

Antidepressants

► Antidepressant Medications

Antidiuretic Hormone

► Vasopressin

Antigens

Anna C. Phillips
Sport & Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Antibody generators

Definition

Antigens are antibody generators, any foreign material which is recognized by the body as foreign and causes the production of antibodies.

Antigens can be bacteria, viruses, toxins, or foreign nonself materials. A molecular part of a microorganism or foreign material that is recognized by immune cells is the part which is called antigen. Certain cells of the immune system (B and T lymphocytes) recognize antigen by their specific receptors. B cells (matured in the bone marrow) are the immune cells that recognize antigen via their surface receptors or antibody. T cells (matured in the thymus) recognize antigen via their T cell receptor, but only after antigens have been processed into smaller peptides by antigen-presenting immune cells. Vaccines work by presenting a dead or altered form of an antigen to the immune system in order to provoke a response. For further information, see Goldsby, Kindt, Osborne, & Kuby, 2003.

Cross-References

► Antibodies
► Immunoglobulins

References and Readings

Goldsby, R. A., Kindt, T. J., Osborne, B. A., & Kuby, J. (2003). *Immunology* (5th ed.). New York: W.H. Freeman.

Antihypertensive

► Antihypertensive Medications

Antihypertensive Drugs

► Antihypertensive Medications

Antihypertensive Medications

Amy Jo Marcano-Reik
Department of Bioethics, Cleveland Clinic, Cleveland, OH, USA
Center for Genetic Research Ethics and Law, Case Western Reserve University, Cleveland, OH, USA

Synonyms

Antihypertensive; Antihypertensive drugs; High blood pressure medications

Definition

Antihypertensive medications are a class of medicines/drugs that are used to treat hypertension or high blood pressure. High blood pressure has been associated with kidney malfunction, heart disease, stroke, and other conditions. By decreasing blood pressure, antihypertensive medications may help to alleviate, if not prevent, these conditions from developing and/or progressing. As with most medical conditions, lifestyle changes tend to be the first direction to take when a person suffers from hypertension. These changes may include decreased salt intake, altered diet, increased physical activity, and stress reduction techniques. If the initial approaches and methods are not effective in reducing blood pressure, then the medicinal route is often the next step in treatment. There are many classes of antihypertensive medications. Some examples include thiazide diuretics, which decrease the amount of fluid in blood vessels and help to dilate blood vessels, increase blood flow, and decrease blood pressure. Another type of antihypertensive medication includes angiotensin-converting enzyme (ACE) inhibitors. ACE inhibitors act on the renin/kidney-angiotensin-aldosterone system to reduce blood pressure by blocking the conversion of certain proteins, which ultimately increases cardiac output, decreases circulation of sodium throughout the system, and increases sodium excretion. Beta-adrenergic blocking agents, or beta-blockers, are also commonly administered to lower blood pressure by blocking the effects of epinephrine, or adrenaline, and other stress-related hormones; however, beta-blockers have also been associated with adverse effects in other bodily systems and, for this reason, are not commonly the first antihypertensive medication prescribed. Calcium channel blockers (CCBs) may also be administered to treat hypertension. CCBs decrease cardiac contractions, which results in a decrease in cardiac output, and, ultimately, a decrease in blood pressure throughout the entire body. There are other antihypertensive medications that, depending on the patient, severity of hypertension, and potential secondary bodily system consequences, may be more effective. Although antihypertensive medications are effective in reducing high blood pressure, it is important to consider each patient's medical history, lifestyle, and potential complications when prescribing antihypertensive medications, such as cardiac disease, history of stroke or epilepsy, or other serious medical conditions.

Cross-References

- ▶ Cardiac Output
- ▶ Hypertension
- ▶ Renin
- ▶ Salt, Intake
- ▶ Sodium, Sodium Sensitivity

References and Readings

Beers, M., Berkow, R. (2000). Cardiovascular disorders: Hypertension (p. 833). The Merck Manual of Geriatrics. (3rd edition). Whitehouse Station, NJ: Merck and Company.

Chobanian, A. V., Bakris, G. L., Black, H. R., Cushman, W. C., Green, L. A., Izzo, J. L., Jr., et al. (2003). The seventh report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure. *Journal of the American Medical Association, 289*(19), 2560–2572.

Fuchs, L. C., Landas, S. K., & Johnson, A. K. (1997). Behavioral stress alters coronary vascular reactivity in borderline hypertensive rats. *Journal of Hypertension, 15*, 301–307.

Kirby, R. F., & Johnson, A. K. (1992). Regulation of sodium and body fluid homeostasis during development: Implications for the pathogenesis of hypertension. *Experentia, 48*, 345–351.

Nelson, M. (2010). Drug treatment of elevated blood pressure. *Australian Prescriber, 33*, 108–112.

Anti-inflammatory Medications

Nicole Brandt[1] and Rachel Flurie[2]
[1]School of Pharmacy, University of Maryland, Baltimore, MD, USA
[2]University of Maryland, Baltimore, MD, USA

Synonyms

Nonsteroidal Anti-inflammatory Medications (NSAIDs)

Definition

Anti-inflammatory medications work by suppressing the inflammatory process in the body caused by certain disease states, immune reactions, or any type of noxious agent. There are several clinical symptoms that present when the body's inflammatory process is stimulated: warmth, pain, redness, and swelling. The process of inflammation is a sequence of events that lead to local vasodilation and increased capillary permeability, recruitment of leukocytes and phagocytic cells to the inflamed area, and tissue degeneration and fibrosis.

There are many classes of drugs that affect different cells involved in the inflammatory process (e.g., antihistamines, leukotriene modifiers, glucocorticoids) but the classic anti-inflammatory agents are those that inhibit an enzyme called cyclooxygenase. Cyclooxygenase converts arachidonic acid to prostaglandins (PG) which are essential to the inflammatory process. There are two cyclooxygenases: COX-1 and COX-2. COX-1 is responsible for creating PG involved in gastrointestinal protection, platelet aggregation, vasoconstriction, and renal function. COX-2 is responsible for creating PG involved in renal function, vasodilation, platelet aggregation, inflammation, pain, and fever. The primary target of anti-inflammatory drugs is COX-2, but selectivity is difficult to achieve due to the conformation of the active sites on the enzymes.

The biggest class of anti-inflammatory drugs are the nonsteroidal anti-inflammatory drugs (NSAID). As a class, they are competitive, reversible, active site inhibitors of the COX enzymes. Within the class, they can be further divided based on their chemical makeup and their selectivity of the COX enzymes (i.e., selective, nonselective). Inhibition of COX-2 provides the basis for the therapeutic effect of NSAIDs (antipyretic, analgesic, anti-inflammatory) while inhibition of COX-1 leads to a majority of the adverse effects (gastrointestinal). Aspirin is an irreversible inhibitor of COX-1 and COX-2, and therefore, the duration of action of aspirin depends on the lifetime of the COX enzyme at different target tissues. Of note, the inhibition of COX-1 that leads to platelet disaggregation lasts for the lifetime of the platelet (8–12 days). This is why aspirin is used for cardiovascular and stroke prevention.

Anti-inflammatory medications can be used for a variety of disorders and disease states. They provide symptomatic relief from pain and inflammation associated with musculoskeletal disorders such as rheumatoid arthritis and osteoarthritis. Their analgesic effects are generally only effective for mild to moderate intensity pains and especially when inflammation is the underlying cause of the pain.

Cross-References

▶ Aspirin
▶ Inflammation

References and Readings

Barrett, K. E., Barman, S. M., Boitano, S., & Brooks, H. (2010). *Ganong's review of medical physiology* (23rd ed.). New York: McGraw-Hill.

Brunton, L. L., Chabner, B. A., & Knollmann, B. C. (2011). *Goodman and Gilman's the pharmacological basis of therapeutics* (12th ed.). New York: McGraw-Hill.

Antioxidant

Sarah Aldred
School of Sport and Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

Definition

An antioxidant is a substance that has the ability to prevent oxidation. An antioxidant can act to inhibit an oxidant or reactions promoted by reactive oxygen species. Reactive oxygen species (ROS) comprise free radicals (pro-oxidant molecules such as superoxide) and non-radical species (such as hydrogen peroxide) and are produced in normal bodily processes such as metabolism or respiration. ROS are aptly named as they are indeed very reactive, and will oxidize proteins,

lipids, or DNA that they come into contact with, causing adducts or altering the function of these bodily molecules. Antioxidants serve to prevent damage or dysfunction by balancing ROS production and effectively neutralizing them. Examples of antioxidants in the body may be endogenous (produced by the body) or exogenous (taken in via the diet). Endogenous antioxidants, including the enzymes superoxide dismutase and catalase, may be upregulated, or increased, in response to ROS release. Examples of exogenous antioxidants include vitamins A, C, and E.

References and Readings

Gutteridge, J. M. C., & Halliwell, B. (1995). *Antioxidants in nutrition, health, and disease*. Oxford: Oxford University Press.

Antiplatelet Therapy

► Aspirin

Antiserum

► Serum

Anxiety

Kim Lavoie
Department of Psychology, University of Québec at Montreal (UQAM); Montreal Behavioural Medicine Centre, Montréal, Québec, Canada
Division of Chest Medicine, Hôpital du Sacré-Coeur de Montréal; Research Centre, Montreal Heart Institute, Montréal, Québec, Canada

Synonyms

Anxiety disorder; Fear; Performance anxiety; State anxiety; Stranger anxiety

Definition

Anxiety may be defined as an "apprehensive anticipation of future danger or misfortune accompanied by a feeling of dysphoria or somatic symptoms of tension," (American Psychiatric Association, 2000, p. 820) (American Psychiatric Association, 2000). The origin of the word anxiety is "to vex or trouble," and is often associated with feelings of fear, worry, discomfort, and dread (Antony & Barlow, 1996). Anxiety is often considered synonymous with *fear*, and though related, they are, in fact, conceptually and clinically distinct. Whereas anxiety may be conceptualized as a negative mood state that may occur in the absence of a specific trigger, fear is better conceptualized as an emotional response to a real or perceived threat. Fear is also more closely related to escape and avoidance behaviors, where anxiety may be experienced in situations that are perceived as either uncontrollable or unavoidable (Cannon, 1929).

Description

Anxiety has many dimensions, including emotional, cognitive, behavioral, and somatic, which characterize many of the responses seen in anxious individuals (Dugas & Ladouceur, 2000). For example, anxiety-related emotional responses include feelings of fear, worry, and apprehension; anxiety-related cognitive responses include anticipation of negative outcomes, biases in information processing, and distorted beliefs; anxiety-related behavioral responses include distraction, procrastination, avoidance, compulsions, and distraction; and anxiety-related somatic or physiological responses include those that signal increases in autonomic (i.e., sympathetic) responses such as increased heart rate, blood pressure and respiration rate, sweating, dizziness, and trembling. Anxiety also has some well-established *biological bases* beyond being associated with increased sympathetic arousal. For example, neural circuits involving the amygdala (emotion-processing center of the brain) and hippocampus (memory center of the

brain) have been strongly implicated in various manifestations of anxiety. Moreover, low levels of at least two neurotransmitters, *gamma*-aminobutyric acid (GABA), which reduces activity in the central nervous system, and serotonin, a neurotransmitter implicated in mood regulation, have both been associated with increased anxiety (Cannon, 1929).

Normal Versus Abnormal Anxiety

Like other negative mood states (e.g., depression, anger), anxiety may be experienced only briefly, and may often be considered a normal or adaptive reaction to situational demands or stress by promoting effective coping. One example of adaptive anxiety relates to performance. Optimal performance on various behavioral tasks (e.g., playing a musical instrument, sharp shooting) may require experiencing at least moderate levels of anxiety (known as the *Yerkes-Dodson law*) (Selye, 1956), which dictates that performance generally increases with moderate increases in mental and physiological arousal. However, performance starts to decline when anxiety (and mental and physiological arousal) becomes too intense. This relationship is often illustrated graphically by an inverted U-curve. Other adaptive forms of anxiety may also be closely tied to the "*fight or flight*" response, originally proposed by Walter Cannon (1929) (Ohman, 2000). Cannon's theory postulated that animals react to stressful situations, particularly those involving threats of bodily harm or injury, with a sudden activation of the sympathetic nervous system, which is said to prime the animal for fighting or fleeing the threat. This theory was later extended to humans and recognized as the initial stage of the *general adaptation syndrome* (described by Hans Selye) (Riggs & Keane, 2006) that regulates stress responses among humans and other organisms.

On the other hand, when anxiety becomes excessive or exaggerated, which may be determined by its intensity and rationale in relation to a particular event or situation, it may be classified as an *anxiety disorder* (discussed in greater detail in the next section). Anxiety disorders represent a group of syndromes that are described in the Diagnostic and Statistical Manual of Mental Disorders-4th Edition Revised (DSM-IV-R) (American Psychiatric Association, 2000), that characterize various types of abnormal or pathological anxiety. The DSM-IV-R has defined six anxiety disorders, including generalized anxiety disorder, panic disorder (with or without agoraphobia), obsessive-compulsive disorder, phobias (including social anxiety disorder), posttraumatic stress disorder, and childhood anxiety disorders (including separation anxiety disorder). Anxiety disorders are very common, and are estimated to affect approximately 18% of Americans (American Psychiatric Association, 2000).

Anxiety Subtypes

Aside from pathological or "psychiatric" levels of anxiety that generally characterized anxiety disorders, there are several other forms of anxiety that tend to fall somewhere between normal and pathological, and will probably be experienced by most people at some point in their lives.

1. Performance anxiety

   As mentioned above, Yerkes and Dodson (Selye, 1956) described a phenomenon linking anxiety-related arousal to performance on various behavioral tasks. They discovered that a moderate level of mental and physiological arousal was necessary to ensure optimal performance on various tasks, and that performance decreased as anxiety either increased or decreased from this minimal or moderate level. This is best depicted by an inverted U-shaped curve, and became known as the Yerkes-Dodson law (Selye, 1956). This phenomenon is what best describes modern day test anxiety and its associated performance.
2. Stranger and social anxiety

   Although "social anxiety disorder" represents one of the anxiety disorders described in the DSM-IV-R, there exist milder, less debilitating forms of stranger or social anxiety. This type of anxiety is normally experienced in childhood when introduced to new or unfamiliar people and may be adaptive (i.e., prevent abduction or abuse by strangers); however, this type of anxiety may persist into adulthood

with little to no purpose (American Psychiatric Association, 2000).

3. Choice or decision anxiety

   Anxiety induced by the need to choose between similar options is increasingly being recognized as a problem for individuals and for organizations, as it has been related to increased procrastination and lost productivity. This type of anxiety, when it becomes exaggerated, closely resembles one of the manifestations of "generalized anxiety disorder," which characterizes individuals with a high intolerance to uncertainty and a high tendency to worry about the negative consequences of making a wrong decision (Yerkes & Dodson, 1908, Dugas & Ladouceur, 2000).

4. State versus trait anxiety

   Anxiety can either be experienced acutely and briefly, or represent a more stable and enduring underlying personality trait. The term "state" anxiety has been used to describe anxiety experienced "in the moment" in response to a particular event or situation. It is typically brief and short lived. In behavioral medicine, this can be manifested, for example, by a person experiencing state anxiety related to a medical procedure such as a blood test or undergoing brain scanning. On the other hand, the term "trait" anxiety has been used to describe a more stable tendency to respond with state anxiety when anticipating or faced with potentially threatening situations. Historically, "trait anxiety" has been closely linked to the personality trait of "neuroticism." Trait anxiety has also been related mainly to self-reported negative health outcomes, but also to objectively defined outcomes.

Cross-References

- ▶ Anxiety Disorder
- ▶ Coffee Drinking, Effects of Caffeine
- ▶ Mental Stress
- ▶ Negative Thoughts
- ▶ Nocebo and Nocebo Effect
- ▶ Stress

References and Readings

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Arlington, TX: American Psychiatric Press.

Antony, M. M., & Barlow, D. H. (1996). Emotion theory as a framework for explaining panic attacks and panic disorder. In R. M. Rapee (Ed.), *Current Controversies in the Anxiety Disorder* (pp. 55–76). New York: Kluwer Academic/Plenum.

Cannon, W. B. (1929). *Bodily changes in pain, hunger, fear, and rage*. New York: Appleton.

Dugas, M. J., & Ladouceur, R. (2000). Targeting intolerance of uncertainty in two types of worry. *Behavior Modification, 24*, 635–657.

Ohman, A. (2000). Fear and anxiety: Evolutionary, cognitive, and clinical perspectives. In M. Lewis & J. M. Haviland-Jones (Eds.), *Handbook of emotions* (pp. 573–593). New York: Guilford Press.

Riggs, D., & Keane, T. M. (2006). Assessment strategies in the anxiety disorders. In B. O. Rothbaum (Ed.), *Pathological Anxiety: Emotional Processing in Etiology and Treatment* (pp. 91–114). New York: Guilford Press.

Selye, H. (1956). *The stress of life*. New York: McGraw-Hill.

Yerkes, R. M., & Dodson, J. D. (1908). The relation of strength of stimulus to rapidity of habit-formation. *Journal of Comparative Neurology and Psychology, 18*, 459–482.

Anxiety and Cardiovascular Disease

▶ Anxiety and Heart Disease

Anxiety and Heart Disease

Manjunath Harlapur[1] and Daichi Shimbo[2]

[1]Center of Behavioral Cardiovascular Health, Division of General Medicine, Columbia University, New York, NY, USA

[2]Center for Behavioral Cardiovascular Health, Columbia University, New York, NY, USA

Synonyms

Anxiety and cardiovascular disease

Definition

Anxiety is an emotional response to a situation, which has both psychological and physiological consequences. Anxiety may be a normal response to daily life situations. However, a heightened or an inappropriate level of anxiety may lead to several deleterious consequences to overall health. There is mounting research about the role of anxiety in the pathophysiology of heart disease.

Description

In the past decade, there has been increasing interest in the relationship between anxiety and heart disease. As reviewed by Rozanski, Blumenthal, and Kaplan (1999) prior to 1999, there were a limited number of prospective studies demonstrating a relationship between anxiety and subsequent cardiovascular disease (CVD) outcomes in healthy populations and in patients with prevalent CVD.

After these initial studies, additional prospective studies examining the relationship between anxiety and CVD outcomes in participants without prior CVD history have been published. For example, Albert, Chae, Rexrode, Manson, and Kawachi (2005) investigated the relationship between anxiety and CVD events in women without a history of CVD and found that anxiety was associated with a higher risk of sudden cardiac death and fatal coronary heart disease. However, these relationships were attenuated after adjustment for medical factors. On the other hand, Shen et al. (2008) showed that in older men without CVD history, anxiety was associated with incident myocardial infarction even after adjustment for medical factors, medication use, adverse health behaviors, and other psychosocial factors including depression. More recently, Janszky, Ahnve, Lundberg, and Hemmingsson (2010), showed that in young Swedish men, anxiety was associated with an increased risk of incident coronary heart disease events including myocardial infarction during long-term follow-up (mean = 37-year follow-up) in unadjusted and adjusted multivariable models. In 2010, Roest, Martens, de Jonge, & Denollet (2010) published a meta-analysis of 20 studies showing that anxiety was associated with incident coronary heart disease events and cardiac death, independent of medical factors and health behaviors. No relationship was found between anxiety and nonfatal myocardial infarction in five studies that examined myocardial infarction as a separate outcome.

Additional prospective studies have also examined the relationship between anxiety and CVD outcomes in participants with prevalent CVD. For instance, Shibeshi, Young-Zu, and Blatt (2007) showed in patients with coronary artery disease that a high level of anxiety over time predicted an increased risk of nonfatal myocardial infarction or death after adjustment for possible confounders. Huffman, Smith, Blais, Jannuzzi and Fricchione (2008) showed that anxiety was associated with an increased risk of CVD complications during a hospitalization for a myocardial infarction in adjusted models. More recently, Martins, de Jonge, Beeya, Cohen, and Whooley (2010) demonstrated that anxiety was independently associated with CVD events in men with stable coronary artery disease. In 2010, Roest, Martens, Denollet and Jonge (2010) published a meta-analysis examining the relationship between anxiety and CVD outcomes in post-myocardial infarction patients. Anxiety was associated with an increased risk of CVD events independent of other prognostic factors.

Although the evidence base is growing in the area of anxiety and heart disease, several questions remain.

First, given that depression is highly comorbid with anxiety, it has not been determined with a high level of certainty whether the relationship of anxiety with CVD events is independent of depression. In the meta-analysis by Roest et al. (2010), which included participants without CVD history, only 5 of the 20 studies adjusted for depression, although the associations in 4 of the 5 studies remained significant. In patients with preexisting CVD, this issue may be more complex. Few studies have ascertained the independent contributions of anxiety and depression

on CVD outcomes. Strik, Denollet, Lousberg, and Honig (2003) found that although both anxiety and depression were separately associated with increased CVD events in post-myocardial infarction patients, the association between anxiety (and not depression) and CVD events remained significant when both psychosocial factors were placed into the same multivariable model. Doering et al. (2010) showed that in adjusted models, the combined presence of persistent anxiety and depression over 3 months was associated with mortality in patients with ischemic heart disease, whereas persistent anxiety only and persistent depression only were not. In addition to depression, some evidence suggests that the combination of anxiety and Type D personality may be cardiotoxic. van den Broek et al. (2009) showed that anxiety was associated with ventricular arrhythmias in patients with implantable cardioverter-defibrillators but only in the presence of Type D personality. Therefore, the CVD risk associated with anxiety may depend on comorbid depression and/or Type D personality. The contributions of other psychosocial/personality factors remain unknown.

Second, the mechanisms underlying the association between anxiety and CVD events also are unknown. Possible candidates include accelerated subclinical atherosclerosis, autonomic dysregulation, ventricular electrical instability, unhealthy lifestyles, and reduced treatment adherence.

Third, the construct of anxiety is broad, and it is unclear what constitutes the main "ingredients" of anxiety-associated CVD risk. In the prospective studies of non-CVD and CVD participants, anxiety has been assessed using self-report measures and also by interviewer assessment. Further, phobic anxiety, generalized anxiety, neurotic anxiety, somatic symptoms of anxiety, social introversion, manifest anxiety, and psychasthenia are among the different manifestations of anxiety that have been associated with increased CVD risk.

Lastly, it is currently not known whether treating anxiety using pharmacologic or non-pharmacologic strategies reduces the risk of CVD events.

Cross-References

- ▶ Anxiety and Its Measurement
- ▶ Anxiety Disorder
- ▶ Coronary Artery Disease

References and Readings

Albert, C. M., Chae, C. U., Rexrode, K. M., Manson, J. E., & Kawachi, I. (2005). Phobic anxiety and risk of coronary heart disease and sudden cardiac death among women. *Circulation, 11*, 480–487.

Doering, L. V., Moser, D. K., Riegel, B., McKinley, S., Davidson, P., Baker, H., Meischke, H., Dracup, K. (2010). Persistent comorbid symptoms of depression and anxiety predict mortality in heart disease. *International Journal of Cardiology*, 145, 188–192.

Huffman, J. C., Smith, F. A., Blais, M. A., Jannuzzi, J. L., & Fricchione, G. L. (2008). Anxiety, independent of depressive symptoms, is associated with in-hospital cardiac complications after acute myocardial infarction. *Journal of Psychosomatic Research, 65*, 557–563.

Janszky, I., Ahnve, S., Lundberg, I., & Hemmingsson, T. (2010). Early-onset depression, anxiety, and risk of subsequent coronary heart disease: 37-year follow-up of 49,321 young Swedish men. *Journal of the American College of Cardiology, 56*, 31–37.

Martins, E. J., de Jonge, P., Beeya, N. A., Cohen, B. E., & Whooley, M. A. (2010). Scared to death? Generalized anxiety disorder and cardiovascular events in patients with stable coronary heart disease: The heart and soul study. *Archives of General Psychiatry, 67*(7), 750–758.

Roest, A. M., Martens, E. J., de Jonge, P., & Denollet, J. (2010). Anxiety and risk of incident coronary heart disease: A meta-analysis. *Journal of the American College of Cardiology, 56*, 38–46.

Roest, A. M., Martens, E. J., Denollet, J., & Jonge, P. (2010). Prognostic asssociation of anxiety post myocardial infarction with mortality and new cardiac events: A meta-analysis. *Psychosomatic Medicine, 72*, 563–569.

Rozanski, A., Blumenthal, J. A., & Kaplan, J. (1999). Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation, 99*, 2192–2217.

Shen, B. J., Avivi, Y. E., Todaro, J. F., Spiro, A., III, Laurenceau, J.-P., Ward, K. D., et al. (2008). Anxiety characteristics independently and prospectively predict myocardial infarction in men: The unique contribution of anxiety among psychologic factors. *Journal of the American College of Cardiology, 51*, 113–119.

Shibeshi, W. A., Young-Zu, Y., & Blatt, C. M. (2007). Anxiety worsens prognosis in patients with coronary artery disease. *Journal of the American College of Cardiology, 49*, 2021–2027.

Strik, J. M. H., Denollet, J. K. L., Lousberg, R., & Honig, A. (2003). Comparing symptoms of depression and anxiety as predictors of cardiac events and increased health care consumption after myocardial infarction. *Journal of the American College of Cardiology, 42*(10), 1801–1807.

van den Broek, K. C., Nyklicek, I., van der Voort, P. H., Alings, M., Meijer, A., & Denollet, J. (2009). Risk of ventricular arrhythmia after implantable defibrillator treatment in anxious type D patients. *Journal of the American College of Cardiology, 54*(6), 531–537.

Anxiety and Its Measurement

Kate L. Jansen[1], Katherine T. Fortenberry[2] and Molly S. Clark[1]
[1]Department of Family Medicine, University of Mississippi Medical Center, Jackson, MS, USA
[2]Department of Family and Preventative Medicine, The University of Utah, Salt Lake City, UT, USA

Synonyms

Anxiousness; Worry

Definition

Anxiety is a psychological and physiological state characterized by cognitive, physiological, and behavioral components. It is described as the "apprehensive anticipation of future danger or misfortune accompanied by a feeling of dysphoria or somatic symptoms of tension" (American Psychiatric Association [APA], 1994, p. 764). The ICD-10 defines anxiety as feelings of apprehension, motor tension such as fidgeting or muscle tension, and autonomic overactivity such as lightheadedness or sweating (World Health Organization [WHO], 1993). These components can be measured through self-report instruments, clinical interview, and behavioral observations.

Description

Several types of assessment measures can be used in the evaluation and treatment of anxiety. Screening questions and instruments are often used first when anxiety is suspected in a clinical setting. These can include single-item questions or brief screening instruments to determine whether further evaluation is warranted. Self-report measures are commonly used in both research and clinical settings. Individuals complete these measures individually and report their perspective of the symptoms they are experiencing. This type of measure allows the individual to complete the information independently, minimizing assessor's time investment. However, this format does not allow individuals to elaborate on their answers and decreases qualitative information obtained regarding the symptoms. Clinical interviews may consist of structured or unstructured series of questions asked by the administrator regarding the client's symptomatology. This format allows for the administrator to gather more information from the client regarding their answers, and structured interviews can be formatted in such a way that symptoms are unlikely to be missed. One disadvantage of clinical interviews is a greater time investment from the administrator to conduct the interview. Finally, anxiety may be assessed by behavioral observation, particularly via observation of a client in an anxiety-provoking situation.

Selection of an appropriate measure depends largely on what information the assessor wishes to obtain and what purpose the information will serve. Functions of anxiety measurement include data collection, differential diagnosis, clinical description, case formulation, treatment planning, and evaluating outcome. If differential diagnosis is the purpose of assessment, it is particularly important to ensure that the measure or technique chosen addresses the suspected disorder. Some measures (i.e., the structured clinical interview Anxiety Disorder Interview Schedule or ADIS), are designed to assess all diagnoses classified by the DSM-IV as anxiety disorders whereas many self-report measures address only specific disorders or symptoms (i.e., the Yale-Brown Obsessive Compulsive Scale). Measures used to aid clinical description can help to identify particular symptoms of concern, as well as provide information to help the assessor better understand the individual's experience. For example, the Beck Anxiety Inventory measures

specific physical symptoms related to an individual's anxiety, as well as the degree of severity or frequency with which the anxiety is experienced. Although these measures are not sufficient unto themselves for a diagnosis of an anxiety disorder, they provide a better understanding of the individual's perception of the problem. Similarly, assessment measures may be used to clarify case conceptualization and treatment plan. Use of a measure such as the Trimodal Anxiety Questionnaire can help focus treatment onto the aspect of anxiety most distressing to the client: somatic, behavioral, or cognitive. Anxiety measures may also be used to address treatment outcome. This may be done in one of several ways. Measures with a particular diagnostic cutoff score (such as the ADIS) can be used to help determine if the individual continues to meet criteria for diagnosis. Other measures, like the Beck Anxiety Inventory, which measures symptoms along a continuum, may be used to assess improvement of certain symptoms over time.

There are several important factors to consider when selecting a measure of anxiety, including the age, culture, and education level of the individual or group of individuals (Derogatis & Lynn, 2000). This factor is particularly important when assessing children/adolescents or older adults. In both groups there are measures developed to specifically address the presentation of anxiety unique to that age. Language and cultural considerations are also important. Anxiety is often expressed differently across cultures; for example, an individual from a culture that expresses anxiety primarily somatically would score inappropriately low on a measure of anxiety that focuses on cognitive components. Intellectual ability and reading level are other factors to consider in choosing an assessment measure. Effort should be made to match the measure to the individual's ability, particularly on self-report measures that require the individual to interpret and respond to question without the assessor's assistance. Finally, time commitment and cost of the measure should be considered. A screening questionnaire may be appropriate for a large-scale research study, whereas a lengthy clinical interview would be impractical.

There are numerous measures of anxiety that utilize different formats and address different symptoms of anxiety disorders. Though not exhaustive of all available measures of anxiety, listed below are samples of the measures available that have been empirically validated (Table 1) (Roemer, 2001). Included in the table is the main reference for the measure, the type of measurement (specifically self-report or clinician administered), a brief description of what the measure intended to address, and the appropriate age range for administration.

The measurement of anxiety serves one of several functions and can encompass one or more aspects of its presentation. Peter Lange, a prominent theorist in the field of anxiety, suggested that anxiety consists of three components: cognitive, physiological, and behavioral (Antony, 2001). Cognitive aspects of anxiety consist of thoughts and beliefs that are irrational or otherwise unhelpful to the situation. Examples may include the thought that if something can go wrong it will, or the belief that it is best to prepare for the worst possible outcome of a situation. These thoughts are related to physiological aspects of anxiety such as sweating, accelerated heart rate, or muscle tension. Physiological components of anxiety may be misinterpreted by the individual, as well as by health care providers, as symptoms of physical illness. For example, chronic gastrointestinal issues may be the result of underlying anxiety or, panic-induced heart palpitations and shortness of breath may be misinterpreted as a heart condition. Behavioral responses to anxiety include the methods an individual uses to cope with distressing thoughts and physical reactions such as avoiding triggers to anxiety or engaging in behaviors designed to prevent negative events from occurring. Different anxiety disorders have specific behavioral responses to anxiety, such as compulsive behaviors present in obsessive compulsive disorder, or avoidance of social interactions in social phobia. In addition to the three components identified by Lange, an emotional aspect of anxiety has been recognized. Similarly to depression and fear, anxiety has been conceptualized as the experience of high negative affect and low positive

Anxiety and Its Measurement, Table 1 Selected measures of anxiety

Measure name	References	Items	Description	Age
Acceptance and action questionnaire	Hayes et al. (2002)	9 item self-report	Measures emotional acceptance and avoidance	Adult
Affective control scale	Williams (1992)	42 item self-report	Measures fear of loss of control	Adult
Anxiety attitude and belief scale	Brown et al. (2000)	36 item self-report	Measures vulnerability to an anxiety disorder	Adult
Anxiety control questionnaire	Rapee et al. (1996)	30 item self-report	Measures perceived control over events and situations	Adult
Anxiety disorder interview schedule revised	Brown, Di Nardo, and Barlow (1994)	Clinician administered	Measures anxiety disorder symptoms	Adult
Anxiety screening questionnaire	Wittchen and Boyer (1998)	15 item self-report	Measures anxiety, intended for primary care settings	Adult
Anxiety sensitivity index	Reiss et al. (1986)	16 item self-report	Measures the fear of anxiety symptoms	Adult
Anxious self-statements questionnaire	Kendall and Hollon (1989)	32 item self-report	Measures anxiety-related thoughts	Adult
Anxious thoughts and tendencies scale	Ganellen et al. (1986)	19 item self-report	Measures anxious thought styles	Adult
Anxious thoughts inventory	Wells (1994)	22 item self-report	Measures dimensions of worry: social health and meta	Adult
Beck anxiety inventory	Beck et al. (1988)	21 item self-report	Measures symptoms of anxiety, used specifically to differentiate anxiety from depression	Adult
Beck anxiety inventory for youth	Beck, Beck, and Jolly (2001)	20 item self-report	Measures symptoms of anxiety separate from depression in youth	Age 7–14
Cardiac anxiety questionnaire	Eifert et al. (2000)	18 item self-report	Measures heart-related anxiety for individuals with and without heart disease	Adult
Children's Yale-brown obsessive-compulsive scale	Goodman et al. (1986)	Clinician administered	Measures children's obsessions and compulsions	Age 6–14
Cognition checklist	Beck et al. (1987)	26 item self-report	Measures frequency of depression and anxiety-related thoughts	Adult
Cognitive somatic anxiety questionnaire	Schwartz, Davidson, and Goleman (1978)	14 item self-report	Measures cognitive and somatic aspects of anxiety	Adult
Depression and anxiety in youth scales	Newcomer, Barenbaum, and Bryant (1994)	45 to 30 parent, teacher, and child report	Measures children's symptoms of depression and anxiety	Age 6–19
Depression anxiety stress scale	Lovibond, Lovibond (1995)	42 item self-report	Measures anxiety, depression, and stress	Adult
Discomfort intolerance scale	Schmidt, Richey, and Fitzpatrick (2006)	5 item self-report	Measures how individual tolerate bodily sensations	Adult
Endler multidimensional anxiety scales	Endler, Edwards, and Vitelli (1991)	60 item self-report	Measures state, trait, and perception of anxiety	Age 12-adult

(*continued*)

Anxiety and Its Measurement, Table 1 (continued)

Measure name	References	Items	Description	Age
Fear of pain questionnaire III	McNeil and Rainwater (1998)	30 item self-report	Measures fear of pain for chronic pain, general medical and nonmedical populations	Adult
Fear questionnaire	Marks and Matthews (1979)	24 item self-report	Measure phobias and associated anxiety and depression	Adult
Four-dimensional anxiety and depression scale	Bystritsky, Waikar, and Vapnik (1996)	24 item self-report	Measures four dimensions of anxiety and depression: emotional, physical, cognitive, and behavioral	Adult
Four systems anxiety questionnaire	Koksal and Power (1990)	60 item self-report	Measures four components of anxiety: cognitive, feelings, behavioral, and somatic	Adult
Frost multidimensional perfectionism scale	Frost et al. (1990)	35 item self-report	Measures perfectionism	Adult
Hamilton anxiety rating scale	Hamilton (1959)	Clinician administered	Measure generalized anxiety symptoms	Adult
Health anxiety questionnaire	Lucock and Morley (1996)	21 item self-report	Measures health-related concerns	Adult
Hospital anxiety and depression scale	Zigmond and Snaith (1983)	14 item self-report	Measures depression and anxiety in medical patients	Adult
Liebowitz social anxiety scale	Fresco et al. (2001)	24 item self-report	Measures social discomfort	Adult
Looming maladaptive style questionnaire – revised	Riskind (1997)	6 vignettes with 8 questions each	Measures general cognitive style	Adult
Metacognitions questionnaire	Cartwright-Hatton and Wells (1997)	65 item self-report	Measures beliefs about worries and thoughts	Adult
Mood anxiety symptom questionnaire	Watson and Walker (1996)	90 item self-report	Measures tripartite model of anxiety and depression	Adult
Multidimensional anxiety questionnaire	Reynolds (1999)	40 item self-report	Measures total anxiety symptoms over the past month	Adult
Multidimensional anxiety scale for children	March (1998)	39 item self-report	Measures symptoms of anxiety disorders	Age 8–19
Multidimensional perfectionism scale	Hewitt and Flett (1991)	45 item self-report	Measures three subscales of perfectionism: self-oriented, other-oriented, and socially prescribed	Adult
Obsessive-compulsive inventory – revised	Foa et al. (2002)	18 item self-report	Measures symptoms of obsessive compulsive disorder	Adult
Pain anxiety symptoms scale	McCracken, Zayfert, and Gross (1992)	40 item self-report	Measures fear of pain	Adult
Penn state worry questionnaire	Meyer et al. (1990)	16 item self-report	Measures degree of worry	Adult
Positive and negative affect scales	Watson, Clark, and Tellegen, 1988	20 item self-report	Measures positive and negative affect	Adult
Reactions to tests	Sarason, 1984	40 item self-report	Measures test-taking anxiety	Adult

(*continued*)

Anxiety and Its Measurement, Table 1 (continued)

Measure name	References	Items	Description	Age
Revised children's manifest anxiety scale	Reynold and Richmond (1979)	37 item self-report	Measures children and adolescent symptoms of anxiety	Age 6–19
Self-rating anxiety scale	Zung (1971)	20 item self-report	Measures symptoms of anxiety	Adult
Self-report for childhood anxiety-related disorders	Birmaher et al. (1997)	41 item parent and self-report	Measures general anxiety, separation anxiety, social phobia, school phobia, and physical symptoms of anxiety.	Age 8+
Social interaction anxiety scale	Mattick and Clark (1998)	19 item self-report	Measures social interaction fears	Adult
Spence children's anxiety scale	Spence, Barrett, and Turner (2003)	45 item self and parent report	Measures children's symptoms of anxiety disorders	Age 2.5–6.5 and 8–12
State-trait anxiety inventory (form y)	Spielberger et al. (1983)	20 item self-report	Measures state and trait anxiety	Adult
State-trait anxiety inventory for children	Spielberger (1983)	Clinician administered	Measures both state and trait anxiety symptoms	Age 9–12
Taylor manifest anxiety scale	Taylor (1953)	50 item self-report	Measures anxiety symptoms	Adult
Test anxiety inventory	Spielberger (1980)	20 item self-report	Measures level of test-taking anxiety	Adult
Thought control questionnaire	Wells and Davies (1994)	30 item self-report	Measures strategies for controlling unpleasant thoughts	Adult
Trimodal anxiety questionnaire	Lehrer and Woolfolk (1982)	36 item self-report	Measures somatic, behavioral, and cognitive aspects of anxiety	Adult
White bear suppression inventory	Wegner and Zanakos (1994)	15 item self-report	Measures thought suppression	Adult
Worry-emotionality scale – revised	Morris, Davis, and Hutchings (1981)	10 item self-report	Measures test taking anxiety	Adult
Yale-brown obsessive-compulsive scale	Goodman et al. (1989)	Clinician administered	Measures obsessions and compulsions	Adult

affect (Barlow, 2004). Anxiety measures are designed to assess one or more of these anxiety components.

Cross-References

- ▶ Anxiety
- ▶ Anxiety and Heart Disease
- ▶ Anxiety Disorder
- ▶ Posttraumatic Stress Disorder
- ▶ Trait Anxiety
- ▶ Worry

References and Readings

American Psychiatric Association. (1994). *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.). Washington, DC: Author.

Antony, M. (2001). Assessment of anxiety and the anxiety disorders: An overview. In M. M. Antony & S. M. Orsillo (Eds.), *Practioner's guide to empirically based measures of anxiety* (AABT clinical assessment series, pp. 9–17). New York: Kluwer Academic/Plenum Press.

Barlow, D. H. (2004). *Anxiety and its disorders: The nature and treatment of anxiety and panic*. New York: Guilford Press.

Beck, J. S., Beck, A. T., Jolly, J., & Steer, R. A. (2001). *Manual for the Beck Youth Inventories of emotional*

and social impairment (2nd ed.). San Antonio: Harcourt Assessment.

Beck, A. T., Brown, G., Steer, R. A., Eidelson, J. I., & Riskind, J. H. (1987). Differentiating anxiety and depression: A test of the cognitive content-specicity hypothesis. *Journal of Abnormal Psychology, 96*, 179–185.

Beck, A. T., Epstein, N., Brown, G., & Steer, R. A. (1988). An inventory for measuring clinical anxiety: Psychometric properties. *Journal of Consulting and Clinical Psychology, 56*, 893–897.

Birmaher, B., Khetarpal, S., Brent, D., Cully, M., Balach, L., Kaufman, J., et al. (1997). The Screen for Child Anxiety Related Emotional Disorders (SCARED): Scale construction and psychometric characteristics. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*, 545–553.

Brown, G. P., Craske, M. G., Tata, P., Rassovsky, Y., & Tsao, J. C. I. (2000). The anxiety attitude and belief scale: Initial psychometric properties in an undergraduate sample. *Clinical Psychology & Psychotherapy, 7*, 230–239.

Brown, T. A., DiNardo, P. A., & Barlow, D. H. (1994). *Anxiety Disorders Interview Schedule for DSM-IV: Lifetime Version (ADIS-IV-L)*. Albany, NY: Graywind Publications.

Bystritsky, A., Waikar, S., & Vapnik, T. (1995). The four-dimensional anxiety and depression scale. *Anxiety, 2*, 47–50.

Cartwright-Hatton, S., & Wells, A. (1997). Beliefs about worry and intrusions: The MetaCognitions Questionnaire and its correlates. *Journal of Anxiety Disorders, 11*, 279–296.

Derogatis, L. R., & Lynn, L. L. I. I. (2000). Screening and monitoring psychiatric disorder in primary care populations. In M. E. Maruish (Ed.), *Handbook of psychological assessment in primary care settings* (pp. 115–152). Mahwah: Lawrence Erlbaum Associates.

Eifert, G. H., Thompson, R. N., Zvolensky, M. J., Edwards, K., Haddad, J. H., Frazer, N. L., et al. (2000). The Cardiac Anxiety Questionnaire: Development and preliminary validity. *Behaviour Research and Therapy, 38*, 1039–1053.

Endler, N. S., Edwards, J. M., & Vitelli, R. (1991). *Endler Multidimensional Anxiety Scales (EMAS): Manual*. Los Angeles: Western Psychological Services.

Foa, E. B., Huppert, J. D., Leiberg, S., Langner, R., Kichic, R., Hajcak, G., et al. (2002). The obsessive-compulsive inventory: Development and validation of a short version. *Psychological Assessment, 10*, 206–214.

Fresco, D. M., Coles, M. E., Heimberg, R. G., Liebowitz, M. R., Hami, S., Stein, M. B., et al. (2001). The Liebowitz Social Anxiety Scale: A comparison of the psychometric properties of self-report and clinician-administered formats. *Psychological Medicine, 31*, 1025–1035.

Frost, R. O., Marten, P., Lahart, C., & Rosenblate, R. (1990). The dimensions of perfectionism. *Cognitive Therapy and Research, 14*(5), 449–468.

Ganellen, R. J., Matuzas, W., Uhlenhuth, E. H., Glass, R., & Easton, C. R. (1986). Panic disorder, agoraphobia, and anxiety-relevant cognitive style. *Journal of Affective Disorders, 11*, 219–225.

Goodman, W., Price, L., Rasmussen, S., Mazure, C., Fleischmann, R., Hill, C., et al. (1989). The Yale–Brown Obsessive Compulsive Scale: I. Development, use, and reliability. *Archives of General Psychiatry, 46*, 1006–1011.

Goodman, W. K., Rasmussen, S. A., Price, L. H., & Rapaport, J. L. (1986). *Children's Yale Brown Obsessive-Compulsive Scale (CY-BOCS)*. Unpublished manuscript.

Hamilton, M. (1959). The assessment of anxiety states by rating. *The British Journal of Medical Psychology, 32*, 50–55.

Hayes, S. C., Bissett, R. T., Strosahl, K., Follette, W. C., Polusney, M. A., Pistorello, J., et al. (2002). *Psychometric properties of the Acceptance and Action Questionnaire (AAQ)*. Unpublished manuscript.

Hazlett-Stevens, H. (2009). Assessment and treatment of anxiety in primary care. In L. C. James & W. T. O'Donohue (Eds.), *The primary care toolkit: Practical resources for the integrated behavioral care provider* (pp. 169–182). New York: Springer.

Hewitt, P. L., Flett, G. L., Turnbull-Donovan, W., & Mikail, S. (1991). The Multidimensional Perfectionism Scale: Reliability, validity, and psychometric properties in psychiatric sample. *Psychological Assessment, 3*, 464–468.

Hunter, C. L., Goodie, J. L., Oordt, M. S., & Dobmyer, A. C. (2009). *Integrated behavioral health in primary care: Step-by step guidance for assessment and intervention*. Washington, DC: American Psychological Association.

Kendall, P. C., & Hollon, S. D. (1989). Anxious self-talk: Development of the Anxious Self-Statements Questionnaire (ASSQ). *Cognitive Therapy and Research, 13*, 81–93.

Köksal, F., & Power, K. (1990). Four Systems Anxiety Questionnaire (FSAQ). A self-report measure of somatic, cognitive, behavioral and feeling components. *Journal of Personality Assessment, 54*, 534–544.

Lehrer, P. M., & Woolfolk, R. L. (1982). Self-report assessment of anxiety: Somatic, cognitive, and behavioral modalities. *Behavioral Assessment, 4*, 167–177.

Lovibond, S. H., & Lovibond, P. F. (1995). *Manual for the Depression Anxiety Stress Scales* (2nd ed.). Sydney, Australia: Psychological Foundation of Australia.

Lucock, M., & Morley, S. (1996). The Health Anxiety Questionnaire. *British Journal of Health Psychology, 1*, 137–150.

March, J. S. (1997). *Manual for the Multidimensional Anxiety Scale for Children (MASC)*. Toronto: Multi-Health Systems.

Marks, I. M., & Mathews, A. M. (1979). Brief standard self-rating for phobic patients. *Behaviour Research and Therapy, 17*, 263–267.

Mattick, R. P., & Clarke, J. C. (1998). Development and validation of measures of social phobia scrutiny fear and social interaction anxiety. *Behaviour Research and Therapy, 36*, 455–470.

McCracken, L. M., Zayfert, C., & Gross, R. T. (1992). The Pain Anxiety Symptoms Scale: Development and validation of a scale to measure fear of pain. *Pain, 50*, 67–73.
McNeil, D. W., & Rainwater, A. J. (1998). Development of the Fear of Pain Questionnaire – III. *Journal of Behavioral Medicine, 21*, 389–410.
Meyer, T., Miller, M., Metzger, R., & Borkovec, T. D. (1990). Development and validation of the Penn State Worry Questionnaire. *Behaviour Research and Therapy, 28*, 487–495.
Morris, L. W., Davis, M. A., & Hutchings, C. H. (1981). Cognitive and emotional components of anxiety: Literature review and a revised worry-emotionality scale. *Journal of Educational Psychology, 73*, 541–555.
Newcomer, P. L., Berenbaum, E. M., & Bryant, B. R. (1994). *Depression and Anxiety in Youth Scale: Examiner's manual*. Austin, TX: PRO-ED.
Rapee, R. M., Craske, M. G., Brown, T. A., & Barlow, D. H. (1996). Measurement of perceived controlover anxiety-related events. *Behavior Therapy, 27*, 279–293.
Reiss, S., Peterson, R. A., Gursky, D. M., & McNally, R. J. (1986). Anxiety sensitivity, anxiety frequency, and the prediction of fearfulness. *Behavior Research and Therapy, 24*, 1–8.
Reynolds, W. M. (1999). *Multidimensional Anxiety Questionnaire*. Odessa, FL: Psychological Assessment Resources.
Reynolds, C. R., & Richmond, O. B. (1979). What I think and feel: A revised measure of children's manifest anxiety. *Journal of Personality Assessment, 43*, 281–283.
Riskind, J. H. (1997). Looming vulnerability to threat: A cognitive paradigm for anxiety. *Behaviour Research and Therapy, 35*, 386–404.
Roemer, L. (2001). Measures for anxiety and related constructs. In M. M. Antony & S. M. Orsillo (Eds.), *Practioner's guide to empirically based measures of anxiety* (AABT clinical assessment series, pp. 49–83). New York: Kluwer Academic/Plenum Press.
Sarason, I. G. (1984). Stress, anxiety, and cognitive interference: Reactions to tests. *Journal of Personality and Social Psychology, 46*, 929–938.
Schmidt, N. B., Richey, J. A., & Fitzpatrick, K. K. (2006). Discomfort intolerance: Development of a construct and measure relevant to panic disorder. *Journal of Anxiety Disorders, 20*, 263–280.
Schwartz, G. E., Davidson, R. J., & Goleman, D. J. (1978). Patterning of cognitive and somatic processes in the self-regulation of anxiety: Effects of meditation versus exercise. *Psychosomatic Medicine, 40*, 321–328.
Seligman, M. E. P., Walker, E. F., & Rosenhan, D. L. (2001). *Abnormal psychology* (4th ed.). New York: W.W. Norton.
Spence, S. H., Barrett, P. M., & Turner, C. M. (2003). Psychometric properties of the Spence Children's Anxiety Scale with young adolescents. *Journal of Anxiety Disorders, 17*(6), 605–625.
Spielberger, C. D. (1980). *Test Anxiety Inventory. Preliminary professional manual*. Palo Alto, CA: Consulting Psychologists Press.
Spielberger, C. D., Gorsuch, R. L., Lushene, R., Vagg, P. R., & Jacobs, G. A. (1983). *Manual for the State-Trait Anxiety Inventory*. Palo Alto, CA: Consulting Psychologists Press.
Taylor, J. A. (1953). A personality scale of manifest anxiety. *Journal of Abnormal and Social Psychology, 48* (2), 285–290.
Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*, 1063–1070.
Watson, D., & Walker, L. M. (1996). The long-term stability and predictive validity of trait measures of affect. *Journal of Personality and Social Psychology, 70*, 567–577.
Wegner, D. M., & Zanakos, S. (1994). *Chronic thought suppression. Journal of Personality., 62*, 615–640.
Wells, A. (1994). A multi-dimensional measure of worry: Development and preliminary validation of the Anxious Thoughts Inventory. *Anxiety, Stress and Coping, 6*, 289–299.
Wells, A., & Davies, M. I. (1994). The Thought Control Questionnaire: A measure of individual differences in the control of unwanted thoughts. *Behaviour Research and Therapy, 32*, 871–878.
Williams, K. E. (1992). *An analogue study of panic onset*. Unpublished doctoral dissertation, Department of Psychology, American University, Washington, DC.
Wittchen, H. U., & Boyer, P. (1998). Screening for anxiety: Sensitivity and specificity of the Anxiety Screening Questionnaire (ASQ-15). *The British Journal of Psychiatry, 173*(Suppl. 34), 10–17.
World Health Organization. (1993). *The ICD-10 classification of mental and behavioral disorders: Diagnostic criteria for research*. Geneva: Author.
Zigmond, A. S., & Snaith, R. P. (1983). The Hospital Anxiety and Depression Scale. *Acta Psychiatrica Scandinavica, 67*, 361–370.
Zung, W. W. K. (1971). A rating instrument for anxiety disorders. *Psychosomatics, 12*, 371–379.

Anxiety Disorder

Rachel Millstein
SDSU/UCSD Joint Doctoral Program in Clinical Psychology, University of California, San Diego/San Diego State University, San Diego, CA, USA

Synonyms

Anxiety; Stress disorder

Definition

Anxiety disorders are a group of disorders characterized by intense or excessive fear, nervousness, dread, or worry. While normal anxiety can create feelings of stress or worry, it typically abates once a stressor is eliminated. Pathological anxiety, of the type experienced in an anxiety disorder, is of a high intensity, lasts for a long duration (typically over 6 months), and interferes with daily life. It is estimated that about 8–18% of the U.S. adult population experiences at least one anxiety disorder each year.

Description

There are eight types of anxiety disorders recognized by the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (4th edition; DSM-IV-TR). Each has different specific symptoms, but the symptoms all involve elements of excessive fear or worry. Cognitive Behavioral Therapy and medication are commonly used treatment approaches that have been empirically supported for a variety of anxiety disorders.

Panic Disorder With or Without Agoraphobia

Description and Symptoms

Panic disorder occurs when a person experiences repeated, unexpected, and feared panic attacks. A panic attack is a sudden, discrete (approximately 5–30 min), and intense anxiety reaction that typically includes several of the following symptoms: pounding heart or accelerated heart rate, sweating, trembling or shaking, feelings of smothering or shortness of breath, feelings of choking, chest pain, nausea, feeling dizzy or faint, feeling detached from reality or oneself, fear of losing control or going crazy, fear of dying, chills or hot spells, and numbness or tingling. Panic disorder is diagnosed by the presence of panic attacks, plus the accompanying fear of having attacks in the future or their implications. A commonly held fear of a person suffering from panic disorder is experiencing an embarrassing or noticeable attack while in public. Thus, agoraphobia is often comorbid with panic disorder. Agoraphobia is the fear and avoidance of being in public places, crowds, or places from which escape may be difficult.

Treatment

Treatment is inherently individualized, and includes analyzing the triggers of panic attacks, restructuring distorted anxious and catastrophic thoughts, relaxation training, habituation to the somatic symptoms of panic, lifestyle changes, and medication usually (SSRI antidepressants or benzodiazepines).

Generalized Anxiety Disorder (GAD)

Description and Symptoms

GAD is characterized by persistent worry on most days for at least 6 months. The worry may focus on two or more stressful situations, or it may be generalized to most life domains. The worry involved with GAD is out of proportion of the actual stressors and causes distress or impairment in daily life. Symptoms of GAD include feeling restless or on edge, being easily fatigued, difficulty concentrating or remembering, irritability, muscle tension, and sleep disturbance.

Treatment

Common therapeutic techniques for GAD include relaxation training, cognitive restructuring of dysfunctional thoughts, problem solving, lifestyle changes and distraction, and medication.

Social Phobia

Description and Symptoms

Social phobia (or social anxiety disorder) is the excessive or irrational fear of public embarrassment or perceived scrutiny. Social phobia can cause people to avoid certain social situations or endure them with distress, to the point where the fear negatively impacts quality of life. People with social phobia recognize that their fear is excessive. Common social phobias involve public speaking, eating in public, blushing, writing in public, and using public restrooms.

Treatment

Social phobia typically responds well to Cognitive Behavioral Therapy to examine and replace distorted thoughts that perpetuate the fears. Relaxation training, exposure to feared situations, social skills assertiveness training, and medications are also empirically supported treatments. Group therapy can he helpful for people with social phobia.

Specific Phobia

Description and Symptoms

Specific phobias are excessive and irrational fear or avoidance of one or more objects or situations. The types recognized by the DSM-IV-TR include animals (e.g., snakes, spiders), natural environment (e.g., heights, storms), blood or injections, situational (e.g., flying, elevators), and others (e.g., vomiting, loud sounds). Criteria for diagnosis of a specific phobia, like social phobia, include persistent anxiety and fear of the object or situation, recognition that the fear is excessive, avoidance of the feared stimulus or enduring it with distress, and interference with daily life.

Treatment

Like social phobia, specific phobias respond well to Cognitive Behavioral Therapy and relaxation training. Graded exposures are very common and effective for phobias. People suffering from a phobia are asked to develop a hierarchy of imaginal and real stimuli that evoke the fear. Clients are then exposed to each step of the hierarchy gradually, with the support of a therapist and relaxation practice, until they can face the highest or most feared object or situation.

Obsessive-Compulsive Disorder (OCD)

Description and Symptoms

Obsessions are anxiety-inducing repeated, intrusive, unwanted, and uncontrollable recurrent thoughts or ideas. Compulsions are excessive, ritualistic, repetitive, intentional behaviors, rules, or thoughts done to allay the anxiety associated with obsessions. OCD involves having obsessions and/or compulsions that are time consuming (more than 1 hour a day), recognition that they are irrational, and cause a person significant distress and impairment of daily functioning. Common obsessions are contamination/germs, symmetry, and hoarding. Common compulsions are washing, checking, and counting.

Treatment

OCD can respond well to Cognitive Behavioral Therapy and/or medication, depending on a variety of factors including severity. Psychological treatments include relaxation training, cognitive restructuring of catastrophic or maladaptive thoughts, and exposure and response prevention (ERP). ERP involves exposure to the obsessional object or thought, and then enforced (by a therapist or companion) prevention from performing the compulsion or gradually reducing the number of times it is performed, until the behavior is extinguished and the obsession becomes less anxiety provoking. ERP is an empirically supported treatment for OCD.

Posttraumatic Stress Disorder (PTSD)

Description and Symptoms

PTSD can occur after witnessing or having experienced a severe trauma that involved the threat of or actual violence and/or death and feelings of horror or helplessness. Not everyone who experiences a traumatic event will develop PTSD, and the precise mechanisms of etiology are unknown but likely involve a complex interplay of genetic predisposition, environmental, personality, and other psychological factors. The symptoms of PTSD fall into three groups: re-experiencing the trauma in dreams or intrusive thoughts (flashbacks), emotional numbing, detachment, and avoidance, and hyperarousal that includes irritability and excessive startle responses to stimuli. PTSD may be acute or chronic, depending on the duration: (whether or not symptoms last longer than 3 months).

Treatment

PTSD responds to several of the cognitive and medication methods described for the previous anxiety disorders. Exposure therapy is used heavily, to assist the person with re-experiencing the feared experience, with the goal of ultimately reducing anxiety and arousal

through habituation and cognitive restructuring. Support groups for PTSD may also be helpful. Eye-movement desensitization and reprocessing therapy (EMDR) is a newer empirically supported treatment for PTSD. It involves having the victim hold the feared memory in his or her mind, and then watching the therapist's fingers move rapidly in a diagonal, back-and-forth motion, to alter the vividness and anxiety provoked by the images.

Acute Stress Disorder

Acute stress disorder is a newer diagnosis that may be a precursor to PTSD. It involves developing anxiety responses (similar to those in PTSD) following a stressful event. However, for this diagnosis, the symptoms must appear and retreat within a month.

Anxiety Disorder due to a General Medical Condition and Substance-Induced Anxiety Disorder

Description and Symptoms

Both of these types of anxiety disorders draw on externally identifiable stimuli as the primary source of the anxiety; the anxiety displayed is not better attributable to one of the other, previously described disorders.

Treatment

Treatment of the anxiety in both of these situations involves treating the underlying cause: medical or drug related.

Several of the above-mentioned anxiety disorders can be associated with health conditions and may also have medical consequences of relevance to behavioral medicine. For example, PTSD has been found to result in some people from myocardial infarctions, and PTSD predicts future cardiac events.

Cross-References

- ▶ Adrenocorticotropin
- ▶ Alcohol Abuse and Dependence
- ▶ Antidepressant Medications
- ▶ Anxiety
- ▶ Anxiety and Heart Disease
- ▶ Anxiety and Its Measurement
- ▶ Anxiolytic
- ▶ Cognitive Behavioral Therapy (CBT)
- ▶ Coping
- ▶ Coping Strategies
- ▶ Coping Styles
- ▶ Cortisol
- ▶ Daily Stress
- ▶ Depression: Symptoms
- ▶ Epinephrine
- ▶ Fear and Fear Avoidance
- ▶ Mental Illness
- ▶ Norepinephrine/Noradrenaline
- ▶ Panic Attack
- ▶ Panic Disorder
- ▶ Posttraumatic Stress Disorder
- ▶ Psychiatric Disorder
- ▶ Psychiatric Illness
- ▶ Psychological Disorder
- ▶ Psychological Pathology
- ▶ Psychological Stress
- ▶ Stress
- ▶ Stress: Appraisal and Coping
- ▶ Stressor
- ▶ Substance Abuse: Treatment
- ▶ Worry

References and Readings

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.

Andreasen, N. C., & Black, D. W. (2006). *Introductory textbook of psychiatry* (4th ed.). Washington, DC: American Psychiatric Publishing.

Barlow, D. H. (Ed.). (2008). *Clinical handbook of psychological disorders* (4th ed.). New York: Guilford Press.

Bourne, E. J. (2000). *The anxiety & phobia workbook* (3rd ed.). Oakland, CA: New Harbinger.

Kessler, R. C., Chiu, W. T., Demler, O., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of twelve-month DSM-IV disorders in the National Comorbidity Survey Replication (NCS-R). *Archives of General Psychiatry, 62*(6), 617–627.

National Alliance on Mental Illness (NAMI). Retrieved January 10, 2011, from http://www.nami.org

National Institute of Mental Health (NIMH). *Anxiety disorders*. Retrieved January 10, 2011, from http://www.nimh.nih.gov/health/publications/anxiety-disorders/introduction.shtml

Anxiolytic

Toru Okuyama
Division of Psycho-oncology and Palliative Care, Nagoya City University Hospital, Nagoya, Aichi, Japan

Synonyms

Antianxiety drug; Minor tranquilizer

Definition

Anxiolytics can be defined as a drug used to treat anxiety. In current medicine, however, anxiety is treated using a variety of drugs including antidepressants, antipsychotics, and anticonvulsants. Recent evidence indicates that even the first choice for the treatment of anxiety disorders such as panic disorder and social anxiety disorder is antidepressants. Therefore, the term anxiolytics is recently defined more appropriately as a drug whose main target symptom is anxiety. Anxiolytics are originally referred to as minor tranquilizers to be distinguished from antipsychotics as major tranquilizers.

Description

Main examples of anxiolytics are barbiturates, benzodiazepines, azapirones, and hydroxyzine. In the historical point of view, barbiturates were the first drugs used to treat anxiety. But later on, they have been prescribed rarely and were replaced by benzodiazepines because they have strong physical and psychological addiction potential. On the other hand, barbiturates are still used for the treatment of epilepsy and general anesthesia, and for the short-term treatment of severe insomnia, especially in case of benzodiazepine-refractory insomnia.

In 1960, chlordiazepoxide, the first benzodiazepine anxiolytic, was made available, followed by diazepam in 1963. The mechanism of action of benzodiazepines includes their bindings to gamma amino butyric acids (GABA)-A receptors, which increase the affinity of GABA and their receptors, resulting in the increase of opening frequency of GABA-A receptors and therefore of potentiating GABAergic neurotransmission. Benzodiazepines have been used widely in clinical practice for the treatment of, for example, anxiety associated with psychiatric disorders including anxiety disorders, mood disorders, insomnia, seizures, alcohol withdrawal syndrome, and agitation. Most of the side effects caused by benzodiazepines are related to their sedating and muscle relaxing effects, such as drowsiness, lightheadedness, lack of coordination, and amnesia. Apart from these side effects, benzodiazepines are basically safe and effective in the short term. But in the long term, they may cause tolerance, dependence, and withdrawal. Risk factors of benzodiazepine dependence include a longer duration of use and a history of substance (alcohol or drug) abuse. Short-half-life benzodiazepine agents are more likely to develop withdrawal symptoms. Therefore, when long-term users of short-half-life benzodiazepine agents stop the use, gradual decrease or once switching with long-half-life benzodiazepine agents may be necessary to prevent withdrawal symptoms.

Azapirones are one of the alternatives to benzodiazepines. They exert their antianxiety effects by potentiating postsynaptic serotonin1A receptors. Only three agents, buspirone, gepirone, and tandospirone, are currently available for clinical use. Effectiveness of azapirones on anxiety associated with general anxiety disorder was shown by meta-analysis. Efficacy of azapirones on anxiety associated with other anxiety disorders has not been established yet. The advantage of azapirones over benzodiazepines is that they do not cause sedation, cognitive dysfunction, or addictive/dependence potential. But azapirones may not be superior to benzodiazepines in the antianxiety effects. Common side effects are headaches, dizziness, nausea, and diarrhea.

Hydroxyzine, an antihistamine, also possesses anxiolytic properties. Although little evidence indicates the usefulness of hydroxyzine in clinical anxiety, it was shown that it is as effective as benzodiazepines in the treatment of generalized anxiety disorder while causing fewer side effects.

Cross-References

- ▶ Anxiety Disorder
- ▶ Dependence, Drug
- ▶ Insomnia
- ▶ Panic Disorder
- ▶ Substance Abuse

References and Readings

Chessick, C. A., Allen, M. H., Thase, M., Batista Miralha da Cunha, A. B., Kapczinski, F. F., de Lima, M. S., et al. (2006). Azapirones for generalized anxiety disorder. *Cochrane Database of Systematic Reviews, 3*, CD006115.

Nemeroff, C. B. (2003). Anxiolytics: Past, present, and future agents. *The Journal of Clinical Psychiatry, 64* (Suppl. 3), 3–6.

Ravindran, L. N., & Stein, M. B. (2010). The pharmacologic treatment of anxiety disorders: A review of progress. *The Journal of Clinical Psychiatry, 71*(7), 839–854.

Anxiousness

- ▶ Anxiety and Its Measurement

Apolipoproteins: APOA-I, APOA-IV, APOE

William Whang
Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Definition

Apolipoproteins are proteins that bind to lipids and are required for the assembly and function of lipoproteins, which transport lipids through the blood and lymph systems. ApoA-I is synthesized in the liver and intestine and is found on virtually all high-density lipoprotein particles. ApoA-I Milano, a naturally occurring variant identified in rural Italy, is associated with very low high-density lipoprotein levels and lower-than-expected risk of coronary artery disease (Nissen et al., 2003). A randomized trial of recombinant ApoA-I Milano administered intravenously produced significant regression of coronary atherosclerosis as measured by intravascular ultrasound. ApoE, synthesized in the liver, is present in chylomicrons, very low-density lipoproteins, and intermediate-density lipoproteins and is important in the metabolism of triglyceride-rich particles (Corder et al., 1993). The isoform ApoE4 has been associated with increased risk of Alzheimer's disease and coronary artery disease. A meta-analysis of ApoE genotypes found relationships to low-density lipoprotein cholesterol and coronary artery disease risk (Bennet et al., 2007). APOA-IV, synthesized in the intestine, is present in chylomicron remnants, intermediate-density lipoproteins, and high-density lipoproteins (Rader and Hobbs, 2008). Its function in lipoprotein metabolism is currently unknown. Please also refer to the "▶ lipoprotein" entry in this encyclopedia for related information.

References and Readings

Bennet, A. M., Di Angelantonio, E., Ye, Z., Wensley, F., Dahlin, A., Ahlbom, A., et al. (2007). Association of apolipoprotein E genotypes with lipid levels and coronary risk. *Journal of the American Medical Association, 298*(11), 1300–1311.

Corder, E. H., Saunders, A. M., Strittmatter, W. J., Schmechel, D. E., Gaskell, P. C., Small, G. W., et al. (1993). Gene dose of apolipoprotein E type 4 allele and the risk of Alzheimer's disease in late onset families. *Science, 261*(5123), 921–923.

Nissen, S. E., Tsunoda, T., Tuzcu, E. M., Schoenhagen, P., Cooper, C. J., Yasin, M., et al. (2003). Effect of recombinant ApoA-I Milano on coronary atherosclerosis in patients with acute coronary syndromes: A randomized controlled trial. *Journal of the American Medical Association, 290*(17), 2292–2300.

Rader, D. J., & Hobbs, H. H. (2008). Disorders of lipoprotein metabolism (Chap. 350). In A. S. Fauci, E. Braunwald, D. L. Kasper, S. L. Hauser, D. L. Longo, J. L. Jameson, & J. Loscalzo (Eds.), *Harrison's principles of internal medicine, 17e*. New York: McGraw-Hill.

Appearance Evaluation

- ▶ Body Image

Appetite

► Leptin

Appetite and Appetite Regulation

Mustafa al'Absi and Bingshuo Li
University of Minnesota Medical School, University of Minnesota, 235 School of Medicine, Duluth, MN, USA

Definition

Appetite is the desire for food intake that may be produced by normal metabolic and energy needs or be other cues that may increase desire for food intake, including appearance, taste, and smell.

Description

The desire for food intake is important in addressing energy and metabolic needs for survival of the organism. The process is regulated by several central brain and peripheral mechanisms. These mechanisms are governed by both homeostatic needs and external cues that may influence desire for food consumption. Several well-studied factors, which regulate food desirability, directly influence appetite, including biological, behavioral, cognitive, and hedonic factors. For example, sensory information and memorial representations of food and associated emotions and motivation state may increase appetite for certain food items.

The involvement of central nervous system in appetite occurs at different levels. For example, signaling related to metabolic needs are received by the brain from different parts of the body that are involved in metabolic activities. One of the receiving structures is the arcuate nucleus (ARC) of the hypothalamus. The ARC coordinate with other nuclei within the hypothalamus information related to the metabolic-homeostatic needs. Experimental research has demonstrated the role of the ARC as a feeding control center. Research has shown that lesion of the ARC leads to overeating and obesity.

The ARC integrates hormonal signals and contains populations of neurons that express neuropeptide Y (NPY) which is involved in regulating energy balance. In addition to NPY, neurons within the ARC express agouti-related protein and proopiomelanocortin. The latter produces α-melanocyte-stimulating hormone, an important appetite regulating peptide and energy regulation; evidence suggests that animals and humans who genetically lack this hormone are likely to be obese. In addition to the ARC, the ventromedial hypothalamus (VMH), the paraventricular nucleus (PVN), and lateral hypothalamic (LH) are also involved in energy balance. Lesions to the PVN, for example, lead to increased feeding behavior and weight gain. These central structures receive information from other parts of the brain through both neuronal and hormonal signaling systems. Vagal afferent pathways and peripheral peptides carry information related to energy needs and metabolic status to the ARC and other structures where additional sensory information is also integrated to influence appetite.

There are several hormones that are directly involved in regulating appetite and energy balance. These are briefly described here, but will also be presented elsewhere in this encyclopedia. Leptin is a hormone involved in regulating energy homeostasis and is released from adipose tissue. Administering leptin reduces food intake and may result in loss of body weight. Ghrelin is released mostly by the stomach and is involved in meal initiation and termination. In contrast with leptin, there is inverse association between ghrelin and adiposity. Administering ghrelin increases food intake in humans. Glucagon-like peptide 1 (GLP-I) is a hormone produced primarily by L cells of the small intestine and is usually increased following a meal. This hormone reduces appetite and food intake. Insulin has a direct impact on metabolic status and dietary intake. It is produced by the pancreas, and similar to leptin, it is associated with energy balance and total body fat stores and fat distribution.

Its secretion increases immediately following a meal. Animal studies have shown that administering insulin reduces food intake and body weight.

Cognitive-hedonic processes, including sensory perceptions of food and food-related memory representations and emotions, have potent effects on appetite regulation. Experiments have shown that perceptions or memory representations of food or food-related cues modulate not only neural activity in specific brain areas involved in cognitive control of eating behaviors, but also eating-related physiological responses such as saliva, gastric acid, and insulin secretion. Recent findings suggest that neural circuits in the nucleus accumbens and ventral pallidum play key roles in the neuromechanism of liking palatable food. In addition, dopaminergic system from the ventral tegmental area of midbrain to the nucleus accumbens is critical to the neuromechanism of wanting of food.

Appetite and Obesity

Dysregulation of metabolic homeostatic and cognitive-hedonic appetite regulatory processes dispose people to obesity. In this case, increased appetite leads to excessive energy intake, and this energy excess is readily stored as fat by the body for later use. Several possible causes of appetite dysregulation have been identified and are currently under active investigation. These causes include abnormal nutrient sensing, overstimulation of food-related reward mechanisms, emotion dysregulation, and negative environmental influences. Pharmacological interventions for obesity and targeting at the dysregulation endocrine and neuronal appetite regulatory processes are also under development. Although many signaling chemicals and brain areas involved in appetite regulation have been identified. The therapeutic potential of this knowledge and individual differences relevant to how they increase risk for obesity remain to be elucidated. Psychotherapeutic interventions, aiming at enhancing self-control over excessive appetite and promoting healthy food-related choices and lifestyles, are believed to be more effective than pharmacological interventions in the fight against obesity.

Cross-References

- ▶ Hormones
- ▶ Leptin
- ▶ Obesity

References and Readings

He, W., Lam, T. K. T., Obici, S., & Rossetti, L. (2006). Molecular disruption of hypothalamic nutrient sensing induces obesity. *Nature Neuroscience, 9*(2), 227–233. doi:10.1038/nn1626.

Saper, C. B., Chou, T. C., & Elmquist, J. K. (2002). The need to feed: Homeostatic and hedonic control of eating. *Neuron, 36*(2), 199–211. doi: S0896627302009698.

Woods, S. C., Schwartz, M. W., Baskin, D. G., & Seeley, R. J. (2000). Food intake and the regulation of body weight. *Annual Review of Psychology, 51*, 255–277. doi:10.1146/annurev.psych.51.1.255.

Apple Shaped

▶ Central Adiposity

Applied Behavior Analysis

Yori Gidron
Faculty of Medicine and Pharmacy,
Free University of Brussels (VUB), Jette,
Belgium

Definition

Applied behavior analysis (ABA) reflects a systematic description and implementation of a therapeutic intervention to change a given behavior, based on the principles of the science of learning and behavior (Sulzer-Azaroff & Mayer, 1991). This process involves an analytic investigation of various triggers that determine and modulate behavior. The determinants are usually environmental and include reinforcements or punishments, and these are time-linked to observed behavioral responses. This is often

done in clinical psychopathological studies but has also been used to investigate behavior at the workplace.

One area where ABA has been extensively used is the study and treatment of autism (Vismara & Rogers, 2010). Interventions in developmental disorders would, for example, include an initial analysis of existing behavior as a function of its environmental modulators (e.g., aggression in a child with developmental disorders, in response to a material reinforcement). The child then learns self-control as modulated by an intervention (signaling delayed rewards). The ABA enables to observe systematic changes in behavior due to existing environmental determinants or due to therapeutically manipulated interventions, using multiple observations and a within-subject detailed observation and recording of behavior.

ABA is also useful to analyze and treat behaviors which may result from or that occur in the context of medical procedures. Surgery for removal of a chraniopharygioma often results in physiological and behavioral changes. In one case study, a 6-year-old girl exhibited severe aggression following surgery for this rare tumor. The aggression was negatively reinforced by escaping certain task demands and positively reinforced by preferred food. Following a highly structured behavioral intervention with extinction, her aggression declined to 88% from baseline levels (Hammond & Hall, 2011). These results exemplify how ABA is very useful for analyzing the determinants of aberrant behavior and how it may be successful as a therapeutic intervention in various clinical settings.

Cross-References

- ▶ Behavior Modification
- ▶ Behavioral Intervention

References and Readings

Hammond, J. L., & Hall, S. S. (2011). Functional analysis and treatment of aggressive behavior following resection of a craniopharyngioma. *Developmental Medicine and Child Neurology, 53*, 369–374.

Sulzer-Azaroff, B., & Mayer, R. (1991). *Behavior analysis for lasting change*. Fort Worth, TX: Holt, Reinhart & Winston.

Vismara, L. A., & Rogers, S. J. (2010). Behavioral treatments in autism spectrum disorder: What do we know? *Annual Review of Clinical Psychology, 27*, 447–468.

Applied Cognitive Psychology

- ▶ Human Factors/Ergonomics

Applied Experimental Psychology

- ▶ Human Factors/Ergonomics

Aptitude Testing

- ▶ Functional Versus Vocational Assessment

Arbitrary Inference

- ▶ Catastrophizing/Catastrophic Thinking

Arcuate Nucleus

- ▶ Hypothalamus

Area Under the Curve (AUC)

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Area under the curve across all time

Definition

A common use of the term "area under the curve" (AUC) is found in pharmacokinetic literature. It represents the area under the plasma concentration curve, also called the plasma concentration-time profile. It is of interest to know the area under the curve, i.e., the area defined by the plasma concentration curve at the top and the x-axis (time) at the bottom. The AUC is a measure of total systemic exposure to the drug.

AUC is one of several important pharmacokinetic terms that are used to describe and quantify aspects of the plasma concentration-time profile of an administered drug (and/or its metabolites, which may or may not be pharmacologically active themselves). These include:

- C_{max}: The maximum concentration or maximum systemic exposure
- T_{max}: The time of maximum concentration or maximum systemic exposure
- $t_{1/2}$ or half-life: The time required to reduce the plasma concentration to one-half of its initial value

Integral calculus is used to calculate AUC, as can be done for other areas defined by a curve. This calculation requires a starting point and an ending point. The starting point is typically the time of administration, represented as time zero, to any subsequent time point, represented as t. This is denoted as $AUC_{(0–t).}$ It is also common to calculate AUC across all time, which is a measure of total systemic exposure. This is denoted as $AUC_{(0–infinity).}$

Cross-References

▶ Pharmaceutical Industry: Research and Development

References and Readings

Dhillon, S., & Kostrzewski, A. (Eds.). (2006). *Clinical pharmacokinetics*. London: Pharmaceutical Press.

Mulder, G. J., & Powers, W. J. (Eds.). (2006). *Pharmaceutical toxicology*. London: Pharmaceutical Press.

Area Under the Curve Across All Time

▶ Area Under the Curve (AUC)

Arithmetic Mean

▶ Mean (Average)

Arousal

▶ Affect Arousal

Arrhythmia

Elizabeth R. Pulgaron and Diana Wile
Department of Pediatrics, University of Miami, Miami, FL, USA

Synonyms

Dysrhythmia

Definition

A normal heart beat in a healthy individual consists of electrical impulses, which engage all four ventricles of the heart, producing a smooth ebb and flow of electrical impulses and contractions to pump blood throughout the human body. An arrhythmia is an aberrant heart rhythm, which is either a change in the speed or pattern of the heart. Palpitations, near syncope and syncope (suddenly feeling light-headed or losing consciousness), chest pain, and shortness of breath are symptoms commonly associated with arrhythmias (American Heart Association, 2011). Arrhythmias can result in tachycardia (increase in heart rate) or bradycardia (decrease in heart rate) which in severe cases could lead to sudden death.

There are four main types of arrhythmias: premature beats, supraventricular arrhythmias, ventricular arrhythmias, and bradyarrhythmias. Premature beats are the most common type of arrhythmia. They are often asymptomatic and do not require treatment. They can occur in the atria (the two upper chambers of the heart; premature atrial contractions) or in the ventricles (the two lower chambers of the heart; premature ventricular contractions). Premature beats often occur in healthy adults, but certain heart diseases can cause premature beats. Stress, exercise, or excessive caffeine intake or nicotine use can trigger premature beats. Supraventricular arrhythmias are tachycardias that start in the atria or the atrioventricular node. Types of supraventricular arrhythmias include atrial fibrillation, atrial flutter, paroxysmal supraventricular tachycardia, and Wolff-Parkinson-White syndrome. Supraventricular tachycardia is the most common abnormal tachycardia in children. Of the more severe arrhythmias, atrial fibrillation is the most common. It occurs when the heart's electrical signal does not travel as it should through the chambers, and as a result, blood is not pumped into the lower two chambers of the heart. Ventricular arrhythmias can be life threatening and need immediate medical attention. Bradyarrhythmias cause the heart to beat slower than normal which may result in decreased blood flow to the brain and loss of consciousness (National Heart Lung and Blood Institute, 2009).

In an otherwise healthy adult heart, arrhythmias can be caused by scar tissue as a result of a heart attack, heart disease, high blood pressure, diabetes, hyperthyroidism, smoking, and excessive alcohol and/or caffeine intake, illegal drug use, stress, or prescription medications as well as dietary supplements. Arrhythmias can also occur as a comorbid condition due to a diseased or deformed heart, which is typically the case with pediatric patients. Some arrhythmias occur without significant effects, but others can result in fainting, cardiac arrest, severe organ damage, and/or stroke. In the most severe cases, cardiac arrhythmias may result in sudden death.

Treatment of arrhythmias depends on severity. More mild cases may be treated solely with medication. The disadvantage of being treated with medication is that pills must be taken daily and indefinitely. Common side effects include nausea, fatigue, headaches, dizziness, palpitations, and skin rash (Horovitz, 1997). One of the more concerning side effects is proarrythmia, which results in recurrent subsequent arrhythmias which can be more intense than the original arrhythmias. Medication side effects often result in a lack of adherence. Other cases may require surgery to either permanently stop tachycardia or implant a pacemaker to regulate the heart beat. The most severe cases may require radiofrequency catheter ablation, an invasive procedure which requires several catheters to be inserted into the heart through a vessel in the groin or arm. The catheter is moved to the site of the arrhythmia, and radiofrequency ablation (very-high-frequency radio waves are used to heat the tissue) or cryoablation (an extremely cold substance is used to freeze the tissue) is used to destroy the site.

Cross-References

- ▶ Coronary Heart Disease
- ▶ Heart

References and Readings

American Heart Association. (2011). *Symptoms, diagnosis & monitoring of arrhythmia*. Retrieved January 26, 2011, from http://www.heart.org/HEARTORG/Conditions/Arrhythmia/SymptomsDiagnosisMonitoringofArrhythmia/Symptoms-Diagnosis-Monitoring-of-Arrhythmia_UCM_002025_Article.jsp

Horovitz, E. (1997). *Arrhythmias, a patient's guide*. Menlo Park, CA: Health Trend Publishing.

National Heart Lung and Blood Institute. (2009). *Arrhythmia*. Retrieved January 12, 2011, from http://www.nhlbi.nih.gov/health/dci/Diseases/arr/arr_all.html

ART

▶ Infertility and Assisted Reproduction: Psychosocial Aspects

▶ In Vitro Fertilization, Assisted Reproductive Technology

Arteries

Linda C. Baumann and Alyssa Karel
School of Nursing, University of Wisconsin-Madison, Madison, WI, USA

Definition

Arteries are blood vessels that carry blood away from the heart. In most cases, arteries carry oxygenated blood. The exception is pulmonary arteries which carry deoxygenated blood from the heart to the lungs to become oxygenated. Because the arterial system is a high-pressure system due to the pressure created by ventricular contraction, arterial walls are generally thick in structure. The two main arteries branching from the heart are the pulmonary artery, which carries blood to the pulmonary circulation, and the aorta, which carries blood into systemic circulation.

Arteries contain smooth muscle and elastic fibers to allow arterial walls to stretch with ventricular contraction and then recoil pushing blood forward. Large arteries such as the aorta and pulmonary artery are composed mainly of elastic tissue and a smaller proportion of smooth muscle, while smaller arteries or arterioles are composed mostly of smooth muscle with little elastic tissue. The contraction and relaxation of smooth muscle dilates or constricts the arterioles and controls blood pressure and blood flow distribution.

Structurally, arteries have three layers. The outermost layer is called the tunica adventitia. It mostly consists of fibrous connective tissue and provides support and prevents tearing of the vessel walls. The middle layer is the tunica media, and is composed of a layer of smooth muscle and a layer of elastic tissue. This is the thickest layer and is responsible for the changes in diameter when the artery contracts and dilates. The innermost layer is referred to as the tunica intima and is made up of endothelium, which form a smooth lining.

Atherosclerosis is a common disorder of the arteries in which plaque – the accumulation of fatty acids, cholesterol, calcium, and other cellular waste products – forms in the arteries and can block blood flow. Although the exact cause of atherosclerosis is unknown, there are many behavioral risk factors. These include excessive alcohol use, high-fat diets, obesity, and smoking.

References and Readings

Atherosclerosis. (2010). *American Heart Association*. Retrieved August 13, from http://www.americanheart.org/presenter.jhtml?identifier=4440

Atherosclerosis. (2010). *Medline Plus*. Retrieved March 20, from http://www.nlm.nih.gov/medlineplus/ency/article/000171.htm

Jarvis, C. (2008). *Physical examination and health assessment* (5th ed.). St. Louis, MO: Mosby Elsevier.

Lewis, S. L., Heitkemper, M. M., Dirksen, S. R., O'Brien, P. G., & Bucher, L. (2007). *Medical surgical nursing: Assessment and management of clinical problems* (7th ed.). St. Louis, MO: Mosby Elsevier.

Thibodeau, G. A., & Patton, K. T. (2007). *Anatomy and physiology* (6th ed.). St. Louis, MO: Mosby Elsevier.

Arteriography

▶ Angiography/Angioplasty

Arteriosclerosis

▶ Atherosclerosis

Arthritis

Beth Schroeder
University of Delaware, Newark, DE, USA

Synonyms

Joint inflammation; Joint pain

Definition

Arthritis is a general term used to describe inflammation of one or more joints of the body and/or

their surrounding connective tissues. This is can be a progressive condition in which the articular cartilage covering the bones at the joint surfaces begins deteriorating, leading to a loss of smooth and friction-free movement. The cartilage is also responsible for absorbing some of the mechanical forces transmitted through the joint. As the cartilage is lost, the joint space between the articulating bones begins to narrow, altering the distribution of mechanical forces through the joint. As the arthritis progresses, it may lead to bone cyst or osteophyte formation, as well as exposure of the subchondral bone surfaces.

The symptoms of arthritis often include pain, swelling, and stiffness of the involved joints, which all may lead to limited range of motion and mobility. Treatment of arthritis is based upon its cause, but it is initially directed toward reducing pain and swelling and restoring mobility. Exercise is known to be beneficial for bone and joint health, as it helps to maintain range of motion as well as to increase bone and muscle strength. Continual activity and movement of joints is also necessary to provide nutrients and remove wastes, since articular cartilage lacks its own blood supply.

While it is known that trauma, bacteria, or infections may cause arthritis, often the trigger for the pathology is unknown. Some of the most common forms of arthritis include osteoarthritis, rheumatoid arthritis, juvenile idiopathic arthritis, psoriatic arthritis, infectious arthritis, and reactive arthritis.

Osteoarthritis (OA), also known as degenerative joint disease, commonly affects the major weight-bearing joints of the knee and hip, but it can affect any joint in the body. It is a result of an imbalance in the remodeling process of joints, as connective tissue and bone destruction outweighs its repair. This imbalance affects the joint capsule, and many individuals with OA may complain of joint instability.

Rheumatoid arthritis (RA) is an autoimmune condition in which the synovial lining of a joint is affected. It is characterized by periods of exacerbation and remission, and it also includes systemic symptoms, such as fatigue, fever, and impaired cardiopulmonary function.

Juvenile idiopathic arthritis (JIA) is a catch-all term for arthritides that begin before the age of 16, with each type having unique characteristics and impairments.

Psoriatic arthritis is a type of arthritis that occurs in patients with the skin condition psoriasis. Most often, the joints in the spine and at the ends of the fingers and toes are affected.

Sometimes bacteria, a fungus, or a virus may infect a joint and lead to arthritis. This is known as septic or infectious arthritis. The effects from this type of arthritis are local, affecting the specific joint in the body that the foreign organism attacks. It also typically has an acute onset, and it may be accompanied by other symptoms, such as fever or chills.

Reactive arthritis is similar to infectious arthritis. It is caused by an infection; however, the arthritic symptoms will occur at a site other than the one actually infected. This type of arthritis is often associated with Reiter syndrome, a condition which includes arthritis, urethritis, and conjunctivitis.

Cross-References

▶ Arthritis: Psychosocial Aspects

References and Readings

A.D.A.M. Medical Encyclopedia [Internet]. Atlanta (GA): A.D.A.M., Inc.; ©2010. Arthritis; [last reviewed 2010 Feb 05; cited 2011 April 18]; [about 7 p.]. Available from: http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0002223

A.D.A.M. Medical Encyclopedia [Internet]. Atlanta (GA): A.D.A.M., Inc.; ©2010. Psoriatic arthritis; [last reviewed 2010 May 13; cited 2011 April 18]; [about 2 p.]. Available from: http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0001450/

A.D.A.M. Medical Encyclopedia [Internet]. Atlanta (GA): A.D.A.M., Inc.; ©2010. Rheumatoid arthritis; [last reviewed 2010 Feb 07; cited 2011 April 18]; [about 6 p.]. Available from: http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0001467/

Goodman, C. C., & Fuller, K. S. (2009). *Pathology: Implications for the physical therapist* (3rd ed.). St. Louis, MO: Saunders Elsevier.

Hansen, J. T. (2010). *Netter's clinical anatomy*. Philadelphia: Saunders Elsevier.

Kisner, C., & Colby, L. A. (2002). *Therapeutic exercise*. Philadelphia: F.A. Davis Company.

Arthritis: Psychosocial Aspects

Mary C. Davis, Alex Zautra and
Shannon L. Stark
Department of Psychology, Arizona State University, Tempe, AZ, USA

Synonyms

Arthritis; Chronic pain; Musculoskeletal pain

Definition

The psychosocial aspects of arthritis encompass both stable and dynamic psychological and social factors that play a role in the etiology of and adaptation to musculoskeletal pain conditions.

Description

Psychosocial Factors in Arthritis

Because the course of arthritis varies considerably across individuals, investigators have attempted to identify factors that predict the extent to which patients with arthritis are able to minimize symptoms and maximize well-being. Somewhat surprisingly, radiographic and other objective assessments of disease activity do not reliably predict the experience symptoms and disability among patients. In contrast, psychosocial factors play an important role in the etiology and course of rheumatic diseases, over and above objective disease markers.

In this chapter, stress and stress responses are considered central to understanding how psychosocial factors contribute to adaptation in rheumatic disease. A stressor is defined as an event that is perceived as threatening or beyond one's ability to cope and that evokes physiological, affective, cognitive, and behavioral responses. Stressors may be brief or long term and range in magnitude from minor events to major or traumatic events. In this framework, the pain, disability, and other demands imposed by the pain condition itself can act as a stressor. Stress responses vary between individuals, depend on current context, and may be key mediators in the link between stress and mental and physical health outcomes in rheumatic illness. Thus, it is useful to ask not only *who* is most affected by illness and other stressors but also *when* are individuals most vulnerable.

Much of the literature on stress and adaptation has focused on factors that place individuals at greater risk of poor functioning. More recently, though, research efforts have broadened to include not only risk but also "resilience factors," that is, factors that help individuals recover from episodes of stress and preserve their well-being despite pain and other stressors. In the sections that follow, affective, cognitive, and social factors that moderate responses to stress in patients with arthritis are first considered, followed by discussion of interventions that target these factors to promote better adaptation among individuals with rheumatic conditions.

Stress

The association between stressful life events and the etiology and course of rheumatic disease depends on the timing and magnitude of the events. For example, traumatic events experienced in childhood, including abuse, neglect, and parental loss, significantly increase the risk of developing chronic inflammation and chronic pain and have been linked to the severity of pain in adulthood (Von Korff et al., 2010). Traumatic events in adulthood also increase disease severity, particularly for individuals who experience post-traumatic stress disorder (PTSD) symptoms. Several mechanisms may account for the trauma-disease severity link. Trauma exposure may trigger PTSD and other affective symptoms, which in turn increase symptom severity. Additionally, traumatic events may disrupt the hypothalamic-pituitary-adrenal-cortical axis (HPAC), an important component of physiological stress response system. For example, childhood maltreatment reported by women with fibromyalgia and osteoarthritis (OA) have been linked to elevated cortisol, a key stress hormone associated with the HPAC (Nicolson, Davis, Kruszewski, & Zautra, 2010). HPAC dysregulation, reflected in

both hypo- and hypercortisol reactivity, has been associated with traumatic experiences and PTSD and with vulnerability to disease activity.

Major and minor life events also predict symptom severity in rheumatic illness. For example, patients with rheumatoid arthritis (RA) experienced reduced symptoms in the weeks following a major life event, but increased symptoms in the weeks following minor stressors (Potter & Zautra, 1997). One possible explanation for this finding is that a major event can elicit an increase in cortisol, which acts to dampen immune functioning. Minor life events, on the other hand, may enhance immune stimulating hormones and make cortisol less effective in dampening immune function (Davis et al., 2008). Clearly, stressors themselves are important factors in the experience of chronic pain, but psychological and social factors that influence coping responses are also likely to play a significant role. Coping factors can be categorized as primarily affective, cognitive, or social.

Affective Components

The stable trait-like aspects as well as the fluctuating state aspects of affect have both been linked to adaptation in rheumatic disease. Although much of the literature has focused on the detrimental role of negative affect, positive affect also plays a role in determining how individuals respond to stress. One of the most frequently studied affective disorders in rheumatic disease patients is depression. Depression is so highly prevalent among chronic pain patients that some investigators have suggested that depressive symptoms should be considered part of the experience of pain. In fact, neuroimaging studies have revealed that pain activates brain regions associated with both sensory and affective components of pain (Tolle et al., 1999). Current depression as well as a history of recurrent depression predict greater pain during episodes of increased stress and more distress during episodes of increased pain among RA patients (Zautra et al., 2007). Because depression and stress are both associated with inflammatory activity in RA, each of these factors may increase disease activity. Thus, depression may increase vulnerability to increased pain and inflammation during episodes of stress among patients with rheumatic disease.

Individual differences in negative and positive affectivity have also been linked to vulnerability to the detrimental effects of stress among pain patients. For example, neuroticism is a personality trait characterized by elevated negative affect and has been linked to elevated pain in both cross-sectional and longitudinal studies of pain patients (Charles, Gatz, Pedersen, & Dahlberg, 1999). Conversely, greater positive affectivity is related to decreased pain and increased functional ability in patients with rheumatic disease (Villanueva, Cornett, Yocum, & Castro, 1999).

Within-person changes in affect may also have implications for illness severity and course. Studying the day-to-day variations in mood can yield information about times *when* patients are most or least vulnerable to the effects of stress. Because both negative and positive emotions make unique contributions to quality of life and adaptation, it is important to consider how variations in both affective states over time relate to the course of rheumatic disease. Increases in negative affect have been linked to increased pain and greater sensitization to pain, as well as to increases in stress (Janssen, 2002). Thus, negative affect may be not only a part of the experience of pain itself but also a response to stress in pain patients. Positive affect, in contrast, helps to decrease vulnerability to both pain and stress in rheumatic patients (Zautra, Johnson, & Davis, 2005).

Cognitive Components

Cognitive stress responses reflect patients' appraisals of the stressor and of their ability to manage it and typically have been characterized as dimensions of coping. A key aspect of this appraisal process is the extent to which individuals perceive a sense of control or lack thereof. In the case of rheumatic illness, an important sense of control centers on an individual's confidence in her or his ability to manage pain and other symptoms that are often unpredictable. One widely used instrument developed to quantify these control beliefs, the Arthritis Self-Efficacy

Scale, yields scores that reflect a sense of control over pain, function, and other arthritis symptoms (Lorig, Mazonson, & Holman, 1993). High arthritis self-efficacy scores consistently relate to better functional health in arthritis patients. For example, higher levels of arthritis self-efficacy relate to higher pain thresholds and increased tolerance to standardized pain stimuli in RA (Keefe et al., 1997). Similarly, arthritis self-efficacy predicts lower pain and better physical functioning in OA.

In contrast to arthritis self-efficacy, pain catastrophizing is characterized by beliefs about a lack of control of symptoms. Among arthritis patients, catastrophic pain beliefs relate to higher ratings of pain intensity, more frequent pain behaviors, and greater pain-related disability, disease activity, and health-care utilization (Keefe, Lumley, Anderson, Lunch, & Carson, 2001). Neuroimaging studies have linked pain catastrophizing with greater activation of brain regions associated with anticipation of pain, emotional aspects of pain, and attention to pain (Gracely et al., 2004). Pain catastrophizing is also related to decreased noxious inhibitory control of pain, indicating less effective modulation of pain signaling at the level of the spinal cord. Thus, pain catastrophizing may impact pain via a number of mechanisms, including directly by amplifying the central nervous system's processing of pain and indirectly by hampering the endogenous descending inhibitory pathway.

Arthritis self-efficacy and pain catastrophizing are among the most frequently studied cognitive factors in rheumatic disease, but others have received some empirical attention as well. More recently, interest has been directed toward evaluating the contribution of pain acceptance to adaptation in rheumatic disease. The capacity to accept pain without trying to alter or avoid it has emerged as a moderator of pain-related disability and distress among arthritis patients. For example, OA and fibromyalgia patients who were more versus less able to accept their pain showed smaller increases in negative affect during weeks of elevated pain and reported higher overall levels of positive affect (Kratz, Davis, & Zautra, 2007). In instances of uncontrollable pain in particular, pain acceptance may be a valuable cognitive resource to preserve affective and physical health. Beyond cognitive factors that are specific to pain or stress management, those that reflect broader beliefs regarding one's own resilience are emerging as key to health in rheumatic disease. For example, recent evidence suggests that a sense of a purpose in life and belief in one's own capacity to bounce back from difficulty relate to faster habituation to thermal pain stimuli (Smith et al., 2009). Thus, "resilience" factors are gaining traction as important predictors of successful coping.

Social Factors

Social relationships are ever-present sources of both stress and fulfillment in everyday life and play an important role in adaptation to rheumatic disease. Social pain is recognized as a concept that focuses on the interplay between social relationships and physical pain. Social pain is an emotional response to perceived exclusion from desired social relationships or perceived devaluation or rejection from significant members of an individual's social network. Just as physical pain is adaptive in signaling a threat of physical harm, social pain is adaptive because it signals a need for social connectedness. Findings from neuroimaging studies suggest that the neural circuitry underlying physical and social pain overlap; the affectively distressing components of both activate the anterior cingulate cortex. Social pain may have special relevance for patients with rheumatic conditions for several reasons. Patients may experience social pain related to their condition, when they perceive that important others do not understand their pain. The resulting sense of stigma and estrangement can further exacerbate their physical and social pain. In addition, physical pain episodes themselves may make patients more vulnerable to social pain, potentially creating a downward spiral of increasing pain, isolation, and disability.

On the positive side of social relations, social connectedness can reduce pain intensity, increase pain tolerance, and dampen stress-related changes in mood and symptoms. One proposed pathway through which social connectedness

affects health outcomes is by buffering individuals from the negative effects of increased pain and stress. For example, an investigation of fibromyalgia patients revealed that when a significant other was present, patients reported less sensitivity to thermal pain and showed diminished pain-related brain activity, compared to when they were alone (Montoya, Larbig, Braum, Preissl, & Niels, 2004). Thus, social connections are double-edged; they can both help and hinder rheumatic patients' capacity to respond effectively to pain and stress. To sustain health and well-being, individuals with rheumatic conditions must have the ability not only to sustain strong social connections but also to draw on them during difficulty.

Psychosocial Interventions for Arthritis

At present, cognitive-behavioral treatment (CBT) for pain is widely considered to be among the most efficacious behavioral interventions available (Morley, Eccleston, & William, 1999). CBT explicitly targets maladaptive ways of thinking, feeling, and behaving in response to the illness and yields improvements in pain, coping, and social role functioning compared to other psychosocial treatments and standard medical care. However, it yields less substantial improvements in mood disturbance, possibly because it does not target the emotion regulation problems that are common among rheumatic disease patients.

Accruing evidence highlights the potential value of a mindful-acceptance-based approach to enhance the capacity of patients to manage the physical, emotional, and social demands of their illness. Rather than encouraging control of pain and dysfunctional thoughts, an approach grounded in mindful-acceptance targets enhanced awareness and acceptance of current experiences, including pain and other stresses. An expanded awareness of current experience also incorporates attention to the positive features of the moment. Some treatments go further and explicitly include an emphasis on engagement in rewarding activity to bolster social connectedness and positive affect. The goal of these approaches is to build both psychological and social resources and the capacity to access and utilize those resources when needed. Further, the benefits of an acceptance-based approach may be most apparent for those patients who are most vulnerable to emotion dysregulation. For example, in RA patients with a history of recurrent depression, mindful-acceptance treatment yielded greater improvements in pain, fatigue, positive and negative affect, and inflammation at posttreatment and 6 month follow-up compared to CBT and an education control (Zautra et al., 2008). Thus, the capacity to attend to both positive and negative experiences in an intentional way and to build greater social connectedness may be key to promoting functional health in arthritis patients.

Existing evidence points to the key roles of psychosocial risk and resilience factors in adaptation to rheumatic disease and encourages continued efforts to advance understanding of how biological, psychological, and social factors interact to promote health and well-being in patients with rheumatic conditions.

Cross-References

- Arthritis
- Pain Management/Control
- Psychosocial Characteristics
- Psychosocial Factors
- Stress

References and Readings

Charles, S. T., Gatz, M., Pedersen, N. L., & Dahlberg, L. (1999). Genetic and behavioral risk factors for self-reported joint pain among a population-based sample of Swedish twins. *Health Psychology, 186*, 644–654.

Davis, M. C., Zautra, A. J., Younger, J., Motivila, S., Attrep, J., & Irwin, M. (2008). Chronic stress and regulation of cellular markers of inflammation in rheumatoid arthritis. *Brain, Behavior, and Immunity, 22*, 24–32.

Gracely, R. H., Geisser, M. E., Giesecke, M. A. B., Petzke, F., Williams, D. A., & Clauw, D. J. (2004). Pain catastrophizing and neural responses to pain among persons with fibromyalgia. *Brain, 127*, 835–843.

Janssen, S. A. (2002). Negative affect and sensitization to pain. *Scandinavian Journal of Psychology, 73*, 212–220.

Keefe, F. J., Affleck, G., Lefebvre, J. C., Starr, K., Caldwell, D. S., & Tennen, H. (1997). Coping strategies in rheumatoid arthritis: A daily process analysis. *Pain, 69*, 43–48.

Keefe, F. J., Lumley, M., Anderson, T., Lunch, T., & Carson, K. L. (2001). Pain and emotion: New research directions. *Journal of Clinical Psychology, 587*, 587–607.

Kratz, A. L., Davis, M. C., & Zautra, A. J. (2007). Pain acceptance moderates the relation between pain and negative affect in female osteoarthritis and fibromyalgia patients. *Annals of Behavioral Medicine, 33*, 291–301.

Lorig, K. R., Mazonson, P. D., & Holman, H. R. (1993). Evidence suggesting that health education for self-management in patients with chronic arthritis has sustained health benefits while reducing health care costs. *Arthritis and Rheumatism, 36*, 439–446.

Montoya, P., Larbig, W., Braum, C., Preissl, H., & Niels, B. (2004). Influence of social support and emotional context on pain processing and magnetic brain responses in fibromyalgia. *Arthritis & Rheumatism, 50*, 4035–4044.

Morley, S., Eccleston, C., & William, A. (1999). Systematic review and meta-analysis of randomized controlled trials of cognitive behavior therapy and behavior therapy for chronic pain in adults, excluding headache. *Pain, 80*, 1–13.

Nicolson, N. A., Davis, M. C., Kruszewski, D., & Zautra, A. J. (2010). Childhood maltreatment and diurnal cortisol patterns in women with chronic pain. *Psychosomatic Medicine, 72*, 471–480.

Potter, P. T., & Zautra, A. J. (1997). Stressful life events' effects on rheumatoid arthritis disease activity. *Journal of Consulting and Clinical Psychology, 65*, 319–323.

Smith, B. W., Tooley, E. M., Montague, E. Q., Robinson, A. E., Cosper, C. J., & Mullins, P. G. (2009). The role of resilience and purpose in life in habituation to heat and cold pain. *The Journal of Pain, 10*, 493–500.

Tolle, T. R., Kaufmann, T., Siessmeir, T., Lautenbacher, S., Berthele, A., Munz, F., et al. (1999). Region-specific encoding of sensory and affective components of pain in the human brain: A positron emission tomography correlation analysis. *Annals of Neurology, 45*, 40–47.

Villanueva, I., Cornett, M., Yocum, D., & Castro, W. L. (1999). Living healthy with arthritis: Individual's positive affect predicts outcomes of a community based multidisciplinary interventional program focusing on wellness and preventive care in arthritis. *Arthritis and Rheumatism, 42*, S1244.

Von Korff, M., Alonso, J., Ormel, J., Angermeyer, M., Bruffaerts, R., Fleiz, C., et al. (2010). Childhood psychosocial stressors and adult onset arthritis: Broad spectrum risk factors and allostatic load. *Pain, 143*, 76–83.

Zautra, A. J., Davis, M. C., Reich, J. W., Nicassio, P., Tennen, H., Finan, P., et al. (2008). Comparison of cognitive behavioral and mindfulness meditation interventions on adaptation to rheumatoid arthritis for patients with and without history of recurrent depression. *Journal of Consulting and Clinical Psychology, 3*, 408–421.

Zautra, A. J., Johnson, L. M., & Davis, M. C. (2005). Positive affect as a source of resilience for women in chronic pain. *Journal of Consulting and Clinical Psychology, 73*, 212–220.

Zautra, A. J., Parrish, B. P., Van Puymbroeck, C. M., Tennen, H., Davis, M. C., Reich, J. W., et al. (2007). Depression history, stress, and pain in rheumatoid arthritis patients. *Journal of Behavioral Medicine, 30*, 187–197.

Aspirin

William Whang[1] and Ana Victoria Soto[2]
[1]Division of Cardiology, Columbia University Medical Center, New York, NY, USA
[2]Medicine – Residency Program, Columbia University Medical Center, New York, NY, USA

Synonyms

Antiplatelet therapy

Definition

Drug that inhibits platelet aggregation.

Aspirin inhibition of the enzyme cyclooxygenase-1 (COX-1) in platelets prevents arachidonic acid-induced production of thromboxane A2, a potent mediator of platelet aggregation, as well as vasoconstriction. The half-life of aspirin is only 20 min in the plasma, but due to its irreversible effects on COX-1, the therapeutic effects can last the lifetime of the platelet, up to 1 week following administration (Patrono et al., 1985). Aspirin has been shown to reduce cardiovascular events in the setting of acute myocardial infarction and acute stroke (Antithrombotic Trialists' Collaboration, 2002). It is also recommended that aspirin be administered (300–350 mg) to patients undergoing stent placement (Schwartz et al., 1988). Higher doses of aspirin ($\geq$300 mg/day) have been shown to

produce a greater effect on COX-2, thereby achieving anti-inflammatory and analgesic properties as well. Aspirin at those higher doses, though, has been shown to have gastrointestinal side effects such as bleeding by eroding the protective prostaglandins in the gut mucosa (Roderick, Wilkes, & Meade, 1993).

For primary prevention of cardiovascular events, meta-analyses have indicated a reduction in myocardial infarction risk that is offset by an increase in risk of major gastrointestinal and extracranial bleeding. However, no reduction in cardiovascular events has been shown in a meta-analysis of studies involving patients with diabetes (Antithrombotic Trialists' (ATT) Collaboration, 2009; Stavrakis, Stoner, Azar, Wayangankar, & Thadani, 2011).

Cross-References

▶ Coagulation of Blood

References and Readings

Antithrombotic Trialists' (ATT) Collaboration. (2009). Aspirin in the primary and secondary prevention of vascular disease: collaborative meta-analysis of individual participant data from randomised trials. *Lancet, 373*, 1849–1860.

Antithrombotic Trialists' Collaboration. (2002). Collaborative meta-analysis of randomized trials of antiplatelet therapy for prevention of death, myocardial infarction, and stroke in high risk patients. *British Medical Journal, 324*, 71–86.

King, S. B., 3rd, Smith, S. C., Jr., Hirshfeld, J. W., Jr., Jacobs, A. K., Morrison, D. A., Williams, D. O., et al. (2008). 2007 focused update of the ACC/AHA/SCAI 2005 guideline update for percutaneous coronary intervention: A report of the American College of Cardiology/American Heart Association Task Force on practice guidelines. *Journal of the American College of Cardiology, 51*, 172–209.

Patrono, C., Ciabattoni, G., Patrignani, P., Pugliese, F., Filabozzi, P., Catella, F., et al. (1985). Clinical pharmacology of platelet cyclooxygenase inhibition. *Circulation, 72*, 1177–1184.

Roderick, J. P., Wilkes, H. C., & Meade, T. W. (1993). The gastrointestinal toxicity of aspirin: An overview of randomized controlled trials. *British Journal of Clinical Pharmacology, 35*, 219–226.

Schwartz, L., Bouassa, M. G., Lesperance, J., Aldridge, H. E., Kazim, F., Salvatori, V. A., et al. (1988). Aspirin and dipyridamole in the prevention of restenosis after percutaneous transluminal coronary angioplasty. *The New England Journal of Medicine, 318*, 1714–1719.

Stavrakis, S., Stoner, J. A., Azar, M., Wayangankar, S., & Thadani, U. (2011). Low-dose aspirin for primary prevention of cardiovascular events in patients with diabetes: A meta-analysis. *The American Journal of the Medical Sciences, 341*, 1–9.

Assertiveness Training

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

Assertiveness training (AT) is a behavioral intervention, often part of cognitive behavior therapy (CBT), stress management, or anger management. In AT, people learn to express themselves in social contexts, despite having barriers. These include learning to express socially "unaccepted" emotions such as anger, disliking others' behavior, and requests for someone to change their behavior. In an assertive statement, there are often: a problem in another person's behavior, a request to change, and, if needed, a statement of the consequences if one's request is not met. Such a statement can also include one's attitude or experienced emotions due to the other person's behavior. Yet, all these need to be done while respecting the other person.

AT is often taught in a group format and is usually done in a gradual manner. People first learn to state simple requests such as "I cannot see, can you please move a bit?" until they are able to state difficult statements such as "yesterday, you made me angry and humiliated me in front of everyone. I ask you please not to say those things again in public, otherwise, I will need to consider our friendship." In an intervention done on hostility reduction, Gidron and Davidson (1996) and Gidron, Davidson, and

Bata (1999) used AT as part of a CBT intervention. Their CBT yielded greater reduction in hostility in healthy and cardiac patients and greater reductions in blood pressure in the latter study, compared to a minimal attention education-control group. Furthermore, in a reanalysis pertinent to AT, Davidson, Macgregor, Stuhr, and Gidron (1999) found that greater constructive anger expression mediated the effects of hostility reduction on blood pressure changes. Constructive anger expression was the aim of the AT element of their program. In another study, hypertensive patients were found to exhibit less assertiveness than normotensive patients. Additionally, especially during confrontation, overt anger expression led to elevated physiological responses in hypertensive patients (Larkin & Zayfert, 2004).

In other domains in behavior medicine, AT can be of great importance. For example, training patients to communicate their concerns and symptoms more adequately to physicians could be an important application of AT in medicine. This can be important in empowering patients to take more control over their health care. AT could be especially important for patients with an introverted or type D personality, who were found not to communicate their symptoms to doctors (Schiffer, Denollet, Widdershoven, Hendriks, & Smith, 2007). AT may be of benefit for such patients. Another example is the use of AT in communication between health professionals working in teams, often under immense psychosocial pressures such as in the operating room (Buback, 2004). The domain of AT exemplifies the multiple roles of behavior medicine – increasing the knowledge of the effects of behavior on the body and implementing such knowledge in patient interventions and in medical teams as well.

Cross-References

- ▶ Anger Management
- ▶ Anger-In
- ▶ Anger-Out
- ▶ Stress Management

References and Readings

Buback, D. (2004). Assertiveness training to prevent verbal abuse in the OR. *AORN Journal, 79*, 148–150, 153–158, 161–164.

Davidson, K., Macgregor, M. W., Stuhr, J., & Gidron, Y. (1999). Increasing constructive anger verbal behavior decreases resting blood pressure: A secondary analysis of a randomized controlled hostility intervention. *International Journal of Behavioral Medicine, 6*, 268–278.

Gidron, Y., & Davidson, K. (1996). Development and preliminary testing of a brief intervention for modifying CHD-predictive hostility components. *Journal of Behavioral Medicine, 19*, 203–220.

Gidron, Y., Davidson, K., & Bata, I. (1999). The short-term effects of a hostility-reduction intervention on male coronary heart disease patients. *Health Psychology, 18*, 416–420.

Larkin, K. T., & Zayfert, C. (2004). Anger expression and essential hypertension: Behavioral response to confrontation. *Journal of Psychosomatic Research, 56*, 113–118.

Schiffer, A. A., Denollet, J., Widdershoven, J. W., Hendriks, E. H., & Smith, O. R. (2007). Failure to consult for symptoms of heart failure in patients with a type-D personality. *Heart, 93*, 814–818.

Assessment

Anthony J. Wheeler and Scott DeBerard
Department of Psychology, Utah State University, Logan, UT, USA

Synonyms

Intellectual testing; Psychological testing

Definition

Assessment in behavioral medicine aims to evaluate the biological, psychological, and social functioning of a patient. This may involve assessing physiological, mood, cognitive, and social functioning and integrating these findings to inform appropriate research and practice.

Description

The overall purpose of assessment is to examine characteristics of patients or research

participants. Assessment in behavioral medicine can include a wide variety of possible domains (e.g., physical, cognitive, mood, quality of life, health behaviors, etc.). Assessment can also take on different forms, such as a patient-reported survey, a rating scale filled out by a health-care practitioner, or a device which measures physiological functioning. Users of such assessments are tasked with using and administering assessment instruments that have demonstrated reliability and validity. Regardless of the manner in which assessment data are gathered, they can be used to identify health problems, guide practitioners in making treatment decisions, and measure progress of health interventions.

Assessing physical functioning is a common practice in behavioral medicine as it can identify physical health problems or risk factors for future problems and provide benchmarks for treatment (Vingerhoets, 2001). Common examples of physical assessment are biofeedback, electrocardiography, and endocrine system function. These assessments may be carried out by physicians, nurses, or other health-care providers. In each of these cases, the assessment tool can yield important information about a patient's current physical status, which can then be used to recommend appropriate interventions, if necessary.

Cognitive assessments may be used when deficits of learning, memory, and problem-solving capabilities are suspected. These instruments frequently involve hands-on tasks such assembling figures and solving visual puzzles as well as more verbal types of tasks that require basic problem-solving abilities and general knowledge. The Wechsler Adult Intelligence Scale-IV is a good example of one such cognitive test (Lichtenberger & Kaufman, 2009). Cognitive assessments are often administered by psychologists, and the findings may used to guide psychological, medical, or occupational interventions.

Assessing psychological characteristics relative to mood states and personality is another useful assessment activity conducted by behavioral medicine practitioners. These tools are most often paper and pencil surveys which assess various symptom constellations. Psychological assessments range in length and time required to complete the inventory. The Beck Depression Inventory-II, for example, has 21 multiple-choice items and takes about 5 min to complete. The Minnesota Multiphasic Personality Inventory-II has several 100 items and takes at least an hour to complete (Butcher, 2011). Psychologists are the most frequent administrators and interpreters of these types of tests.

Identifying and measuring health behaviors is yet another type of assessment conducted in behavioral medicine. This might involve measuring diet, alcohol intake, exercise activity, or sleep habits, among others. Such behaviors are often the focus of interventions in behavioral medicine and are often associated with both psychological and physical health status (Vingerhoets, 2001). Further, health behavior assessment can be used to identify risk factors for diseases such as diabetes, substance dependence, or hypertension.

An increasingly popular area of assessment is in patient quality of life (Ware & Gandek, 1998). These assessments are patient-reported surveys that include measures of physical disability, pain and suffering, and self-efficacy. A common example is the Short Form-36 health survey, a 36-item survey of daily functioning and health status. These survey data are commonly used in research and practice to evaluate the effectiveness of pharmacological or surgical treatments and establish functional benefit for patients (O'Connor et al., 2009).

Finally, it should be noted that in most areas of medicine, especially in clinical trials, a typical approach for assessing patient outcomes is to include both disease-specific and overall quality of life measures. This approach to assessment ensures that both changes to specific disease processes and also patient-relevant outcomes in terms of functional abilities are equally considered.

Assessment can take on many forms and purposes in behavioral medicine. Assessment is a commonplace and useful tool for the behavioral medicine practitioner. It can be used to identify risk for pathology, inform interventions, and mark progress to change.

Cross-References

- ▶ Ambulatory Blood Pressure
- ▶ Anxiety and Its Measurement
- ▶ Beck Depression Inventory (BDI)
- ▶ Depression: Measurement
- ▶ Functional Versus Vocational Assessment
- ▶ Health Assessment Questionnaire
- ▶ Health Behaviors
- ▶ Health Outcomes Research
- ▶ Health Survey Questionnaire
- ▶ Health-Related Quality of Life
- ▶ Hospital Anxiety Depression Scale
- ▶ McGill Pain Questionnaire
- ▶ Measures of Quality of Life
- ▶ Medical Outcomes Study
- ▶ Patient-Reported Outcome
- ▶ Physical Activity and Health
- ▶ Quality of Life: Measurement
- ▶ Reliability and Validity
- ▶ SF-36
- ▶ Validity

References and Readings

Butcher, J. N. (2011). *A beginner's guide to the MMPI-2* (3rd ed.). Washington, DC: American Psychological Association.

Gregory, R. J. (2010). *Psychological testing: History, principles, and applications*. Saddle River, NJ: Prentice Hall.

Lichtenberger, E. O., & Kaufman, A. S. (2009). *Essentials of WAIS-IV Assessment (Essentials of Psychological Assessment)*. Hoboken, NJ: Wiley.

O'Connor, A.M., Bennett, C.L., Stacey, D., Barry, M., Col, N. F., Eden, K. B., et al. (2009). Decision aids for people facing health treatment or screening decisions. *Cochrane Database of Systematic Reviews, 2009*(3), CD001431.

Vingerhoets, A. J. J. M. (2001). *Assessment in behavioral medicine*. New York: Brunner-Routledge.

Ware, J. E., & Gandek, B. (1998). Overview of the SF-36 health survey and International Quality of Life Assessment (IQOLA) Project. *Journal of Clinical Epidemiology, 51*, 903–912.

Assessment of Functions

- ▶ Functional Versus Vocational Assessment

Assessments of Work Functions

- ▶ Functional Versus Vocational Assessment

Assisted Living

Josh Allen
Care and Compliance Group, Inc. American Assisted Living Nurses Association, Wildomar, CA, USA

Definition

Assisted living is a state regulated and monitored residential long-term care option. Assisted living provides or coordinates oversight and services to meet the residents' individualized scheduled needs, based on the residents' assessments and service plans and their unscheduled needs as they arise.

Services

Services allowed are typically outlined in state law and regulation, and typically include:

- 24-hour awake staff to provide oversight and meet scheduled and unscheduled needs
- Provision and oversight of personal and supportive services (assistance with activities of daily living and instrumental activities of daily living)
- Medication management
- Health-related services (e.g., coordination of nursing services, hospice, home health, etc.)
- Social services
- Recreational activities
- Meals and snacks
- Housekeeping and laundry
- Transportation

A resident has the right to make choices and receive services in a way that will promote the

resident's dignity, autonomy, independence, and quality of life (Adapted from the Assisted Living Workgroup report, 2003).

Environment

Assisted living has a look and feel that is distinctly different from the physical plant in more institutional long-term care settings. The assisted living environment emphasizes the creation of a homelike atmosphere, and while health services are often provided or directed onsite, they are carried out in a manner that encourages privacy. For example, most institutional nursing facilities feature a large "nurses station" that houses charts, medical equipment, and personnel. While many assisted living communities have a similar space, it is usually held behind closed doors so as not to dominate the environment.

References and Readings

Assisted Living Workgroup. (2003). The Assisted Living Workgroup: A report to the U.S. Senate Special Committee on aging. 2003. http://www.theceal.org/assets/PDF/ALWReportIntro.pdf. Accessed 3 Apr, 2012

Assisted Reproductive Technology

► Infertility and Assisted Reproduction: Psychosocial Aspects
► In Vitro Fertilization, Assisted Reproductive Technology

Assisted Suicide

► Euthanasia

Associate

► Co-workers

Asthma

Akihisa Mitani
Department of Respiratory Medicine, Mitsui Memorial Hospital, Chiyoda-ku, Tokyo, Japan

Synonyms

Bronchial asthma

Definition

Asthma is one of the most common chronic diseases and may develop at any age, although new-onset asthma is less frequent in the elderly. Many adolescents experience a remission of childhood asthma symptoms before they have fully matured, with recurrence several years later.

Asthma is characterized by a chronic inflammatory disorder of the airways, airflow obstruction which could be at least partly reversed, and bronchial hyperresponsiveness. In not a few cases, asthma can be diagnosed on the basis of a patient's symptoms and medical history. The patient has history of any of the following: cough, recurrent wheeze, recurrent chest tightness, and recurrent difficult breathing. These symptoms are usually associated with airflow limitation and occur or worsen in the presence of various stimuli, including animals, changes in temperature, drugs (aspirin), respiratory infections, smoke, exercise, and emotional stress. The patient also might have atopic diseases and a family history of asthma. The airway obstruction measured by lung function test may help confirm the diagnosis of asthma (Weiss & Speizer, 1993).

When asthma is well controlled, the patient can avoid various unpleasant symptoms, risk of exacerbations is reduced, and decline in lung function slows down. In order to achieve this goal, various components of treatment are required (Global Initiative for Asthma [GINA], 2009; National Heart & Blood Institute, 2007).

First of all, the development of a partnership between the patients and their health-care team is required. The shared vision of the goal is essential. The patients also have to acquire a certain level of knowledge about asthma, which enables them to avoid risk factors, take medications correctly, monitor their status, and seek medical help appropriately.

Next, the patients should avoid the risk factors that make their asthma control worse, including smoke, drugs, food, house dust, and animals. For example, up to 28% of adult patients with asthma response to aspirin, resulting in asthma exacerbations. The drugs that cause symptoms should be completely avoided. Influenza vaccination is also recommended for the patients because infection itself worsens asthma control.

Medications take a starring role in controlling asthma, which are divided into two categories, reliever medication and controller medications. Reliever medication (preferably a short-acting beta agonist (SABA)) provides the patient a quick relief from acute symptoms. The patient should be encouraged to take it as needed. However, SABA does not treat the airway inflammation underlying asthma, although useful for symptom control. No patient with persistent asthma should be treated by SABA alone. They need to take regularly one or more controller medications. These medications prevent symptoms or attacks from occurring. Inhaled glucocorticoids (often called inhaled corticosteroids (ICS)) are recommended as the initial and primary therapy in all patients with moderate persistent asthma. Other controller medications include a long-acting beta agonist (LABA) (combination inhaler with ICS is in widespread use), theophylline, and leukotriene-modifying agents. The difficult-to-treat asthma patient might be introduced of oral glucocorticoids and/or anti-IgE treatment.

Cross-References

- ▶ Asthma and Stress
- ▶ Asthma: Behavioral Treatment
- ▶ Lung Function

References and Readings

Global Initiative for Asthma (GINA). (2009). *Global strategy for asthma management and prevention*. Full text available online at www.ginasthma.org

National Heart, Lung, and Blood Institute. (2007). *Expert panel report 3: Guidelines for the diagnosis and management of asthma* (Item No. 08–4051). Full text available online at www.nhlbi.nih.gov/guidelines/asthma/asthgdln.htm

Weiss, S. T., & Speizer, F. E. (1993). Epidemiology and natural history. In E. B. Weiss & M. Stein (Eds.), *Bronchial asthma mechanisms and therapeutics* (Vol. 3). Boston: Little, Brown.

Asthma and Stress

Akihisa Mitani
Department of Respiratory Medicine, Mitsui Memorial Hospital, Chiyoda-ku, Tokyo, Japan

Synonyms

Bronchial asthma; Tension

Definition

It is well accepted that stress is a modulator increasing the frequency, duration, and severity of the asthma symptoms. However, little is known about the underlying mechanisms, although there are some reasonable models, such as decreased corticosteroid signals. Further investigations are needed.

Description

Asthma had been long considered as primarily psychogenic, often called asthma nervosa, until the inflammatory basis of the disease was revealed in the latter half of the twentieth century. In recent years, it is widely accepted that asthma is caused by chronic inflammation in the airway, and inhaled glucocorticoids which can inhibit the inflammation are recommended as the initial and

primary therapy. Furthermore, because asthma itself in turn produces stress, the correlation between asthma and stress might be arise only from asthma-induced stress. Still, various observational studies indicate that asthma is greatly influenced by psychosocial factors and stress. Between 20% and 30% of patients with asthma experience acute exacerbations when they are really feeling the stress. Nowadays, stress is seen as a modulator that accentuates the airway inflammatory response to environmental triggers, increasing the frequency, duration, and severity of the symptoms.

Stress is considered the common state that occurs when demands from environmental challenge an individual's adaptive capacity, or ability to cope. It is still under investigation through which mechanism stress worsens asthma control. However, there are some popular hypotheses (Chen & Miller, 2007; Haczku & Panettieri, 2010; Vig & Forsythe, 2006).

When the body is challenged physically or psychologically, short-term activation of neuroendocrine and autonomic nervous systems adapts to the stress for surviving during the period of challenge. Acute stress, which you have to adapt to quickly, causes the immediate activation of hypothalamic-pituitary-adrenal (HPA) axis and sympathetic-adrenal-medullary (SAM) axis, which induce the release of various hormones. It might be said that the HPA axis and the SAM axis can convert the stress detected by brain to the physiological signal.

It is well known that the activation of HPA axis increases the secretion of corticosteroids from the adrenal cortex, and the activation of SAM axis causes the increased release of epinephrine and norepinephrine. This fact evokes a certain paradox. Corticosteroids inhibit the inflammation in the airways, and beta-stimulants such as epinephrine might work as a bronchodilator. These hormones seem to be beneficial in controlling asthma.

Explanations that could resolve this paradox are mainly based on the hormone depletion and the resistance of receptors under chronic stress, matters more than acute stress in the issue of clinical practice. There is some evidence that the prolonged stress continues the releases of various stress hormones, which is exhausted at last. When chronic stress first begins, there is an initial elevation of the corticosteroids level. But as time passes, this elevation diminishes. It is also argued that receptors for stress hormones become downregulated after prolonged exposure, making immune cells less sensitive to not only endogenous signaling but also medications.

Stress could worsen asthma in other ways. Stress can have an influence on self-management of asthma, including drug adherence and avoidance of risk factors, which might make the control of asthma difficult (Alvarez & Fitzgerald, 2007). Stress also can change a perception of asthma symptoms and make the patients believe that their condition is getting worse. In extreme cases, an occurrence of hyperventilation caused by a panic disorder makes a medical treatment for asthma exacerbation complicated, and increases the frequency of hospitalization. Others suggest that psychological stress might be associated with increased risk of respiratory infection, which is an exacerbating factor of asthma.

It is true that the above models are reasonable enough, but there remain many problems to be solved. Does stress worsen asthma symptoms eventually by increasing inflammatory responses? To what extent is decreased sensitivity to glucocorticoids and epinephrine responsible for excessive inflammation in the patients with asthma? To answer these questions, well-organized prospective investigations in human clinical research are needed, in which clinical, immunological, and psychological variables are correctly and frequently evaluated (Busse & Kiecolt-Glaser, 1995).

Cross-References

► Asthma: Behavioral Treatment

References and Readings

Alvarez, G. G., & Fitzgerald, J. M. (2007). A systematic review of the psychological risk factors associated with near fatal asthma or fatal asthma. *Respiration, 74*(2), 228.

Busse, W. W., & Kiecolt-Glaser, J. K. (1995). NHLBI Workshop summary. Stress and asthma. *American Journal of Respiratory and Critical Care Medicine, 151*(1), 249.

Chen, E., & Miller, G. E. (2007). Stress and inflammation in exacerbations of asthma. *Brain, Behavior, and Immunity, 21*(8), 993.

Haczku, A., & Panettieri, R. A. (2010). Social stress and asthma: The role of corticosteroid insensitivity. *The Journal of Allergy and Clinical Immunology, 125*(3), 550.

Vig, R. S., & Forsythe, P. (2006). The role of stress in asthma: Insight from studies on the effect of acute and chronic stressors in models of airway inflammation. *Annals of the New York Academy of Sciences, 1088*, 65.

Asthma Education and Prevention Program

Elizabeth R. Pulgaron
Department of Pediatrics, University of Miami, Miami, FL, USA

Synonyms

Asthma interventions

Definition

The National Asthma Education and Prevention Program (NAEPP) was initiated in March 1989 by the National Heart Lung and Blood Institute (NHLBI) to address the needs of those affected by asthma in the United States. The goals of the NAEPP include raising awareness of asthma as a serious chronic illness, teaching how to recognize symptoms of asthma and facilitating appropriate diagnosis, promoting effective control of asthma via treatment, and conducting education programs (National Heart Lung and Blood Institute [NHLBI], n.d.). Some of the recommendations for education programs provided by the NAEPP include educating patients from the time of diagnosis and integrating education into every phase of asthma care, education being provided by all members of the health care team, teaching asthma self-management while being culturally sensitive, developing treatment goals with the patient rather than for the patient, having a written action plan (especially for those with moderate-to-severe asthma), and promoting adherence through communication and family involvement.

Description

Various asthma education programs are in existence, and some have been empirically tested. Gibson and colleagues (2009) reviewed 36 trials that compared self-management education for adults with asthma to usual care. Overall, results indicated that self-management education reduced complications, emergency care visits, unscheduled visits to the doctors, decreased number of days off from work or school, and improved quality of life. Changes in lung function as a result of participating in these interventions were minimal. The conclusions from this meta-analysis stated that asthma self-management programs that include self-monitoring of symptoms or peak flow readings in conjunction with regular medical visits and written action plans are the most effective at improving health outcomes for adults. One factor to consider when creating and assessing these types of programs is the audience they are intended for. Inadequate health literacy has been identified as an area of concern for many adults and parents of children with chronic conditions. In adults with asthma, health literacy has been strongly correlated with poorer disease knowledge and improper metered-dose inhaler (MDI) use (Williams, Baker, Honig, Lee, & Nowlan, 1998).

In the pediatric asthma literature, Bernard-Bonnin and colleagues (1995) did not report findings nearly as positive as those seen in the adult literature. From their meta-analysis of 11 randomized controlled trials of self-management programs for children with asthma, they concluded that self-management teaching did not reduce the number of days missed from school, asthma attacks, hospitalizations, or the number of emergency room visits. The authors recognized the

limitations of their review, including the small number of studies included, the pooling of studies without regard for sociodemographic factors or disease severity, and the broad study criteria for being included in the analysis. Yet at the present time, this is the only pediatric asthma education program meta-analysis currently available. Other, more recent, pediatric programs in the literature have exhibited success across morbidity, knowledge, and psychosocial outcomes. For example, Krishna and colleagues (2003) examined the effectiveness of a self-management education program delivered through interactive multimedia sessions conducted during a family's standard clinic visit compared to just receiving standard-of-care asthma education. Compared to those children who only received standard education, those who participated in the interactive multimedia program displayed increased asthma knowledge, decreased number of days asthma symptoms were experienced, and decreased number of emergency department visits and used lower daily doses of inhaled corticosteroids at a follow-up visit.

Cross-References

- ▶ Asthma
- ▶ Asthma and Stress
- ▶ Asthma: Behavioral Treatment
- ▶ Lung Function

References and Readings

Bernard-Bonnin, A. C., Stachenko, S., Bonin, D., Charette, C., & Rousseau, E. (1995). Self-management teaching programs and morbidity of pediatric asthma: A meta-analysis. *The Journal of Allergy and Clinical Immunology, 95*, 34–41.

Brewin, A. M., & Hughes, J. A. (1995). Effect of patient education on asthma management. *British Journal of Nursing, 4*, 81–101.

Gibson, P. G., Powell, H., Wilson, A., Abramson, M. J., Haywood, P., Bauman, A., et al. (2009). Self management education and regular practitioner review for adults with asthma. *Cochrane Database of Systematic Reviews, 3*, 1–81. doi:10.1002/14651858.CD001117.CD001117.

Krishna, S., Francisco, B. D., Balas, E. A., König, P., Graff, G. R., & Madsen, R. W. (2003). Internet-enabled interactive multimedia asthma education program: A randomized trial. *Pediatrics, 111*, 503–510. doi:10.1542/peds.111.3.503.

National Heart Lung and Blood Institute. (n.d.). *National asthma education and prevention program*. Retrieved February 1, 2011, from http://www.nhlbi.nih.gov/about/naepp/naep_pd.htm

Williams, M. V., Baker, D. W., Honig, E. G., Lee, T. M., & Nowlan, A. (1998). Inadequate literacy is a barrier to asthma knowledge and self-care. *Chest, 114*(4), 1008–1015.

Asthma Interventions

▶ Asthma Education and Prevention Program

Asthma: Behavioral Treatment

Akihisa Mitani
Department of Respiratory Medicine, Mitsui Memorial Hospital, Chiyoda-ku, Tokyo, Japan

Synonyms

Behavior modification program; Behavior therapy; Bronchial asthma

Definition

It is well accepted that psychogenic factor has a close relationship with asthma symptoms. Some patients with asthma experience acute exacerbations preceded by emotional stress. This fact has made us expect the use of psychological in addition to conventional physical and pharmacological interventions, in order to achieve the successful management of asthma. Relaxation training, systematic desensitization, and assertive training were tested with enthusiasm especially in the 1970s, but later experiments and analyses have thrown doubt on them. Application of biofeedback techniques to asthma treatment has been still discussed (Ritz & Dahme, 2004).

Biofeedback techniques consist of direct and indirect techniques. In direct techniques, including respiratory resistance feedback, spirometric biofeedback, and trachea-noise biofeedback, lung function itself is measured and modified. Most of indirect techniques have targeted facial muscle EMG, heart rate, and heart rate variability. However, it has not been proved that they can contribute as adjunctive treatments of asthma. Furthermore, these techniques are technically difficult in practice, and the procedure, especially in the case of direct techniques, might worsen asthma symptoms.

The self-management of asthma plays a central role in asthma treatment, and psychological factors may influence not only the symptoms but also the management of asthma in many ways (Clark & Mitchell, 2009). The patients have to acquire a certain level of knowledge about asthma, which enables them to avoid risk factors, take medications correctly, monitor their status, and seek medical help appropriately. It is clear that self-management encompasses very much more than patient education. It implies involving patients in the care they receive and encouraging them to become active partners in managing their illness. Behavioral and cognitive process, such as operant behavior, in the self-management has also been eagerly investigated. The successful performance of self-management skills to avoid an asthma attack is, conversely, an example of negative reinforcement. Adequate coping can facilitate the self-management. Conditioned fear and anxiety to asthma derived from previous traumatic experiences such as acute exacerbation sometimes alters the way of recognition of asthmatic symptoms (Creer, 2008). Inadequate symptom perception might be associated with an overuse of reliever medication, irrespective of lung function, causing the unnecessary side effects. It is, therefore, suggested that psychological interventions may be appropriate for patients who are unable to achieve the self-management of asthma because they might have behavioral or cognitive problems.

There is not enough evidence that behavioral treatment can contribute substantially to the treatment of asthma (Dahl & Gustafsson, 1990; King, 1980). This is due to the poor methodology of the studies as well as the inherent problems of conducting such trials. It is recommended that larger and well-conducted randomized trials use valid outcome measures to evaluate the effectiveness of psychological interventions for adults with asthma.

Cross-References

- ▶ Asthma and Stress
- ▶ Asthma, General Section Followed by Subcategories
- ▶ Biofeedback

References and Readings

Clark, N. M., & Mitchell, H. E. (2009). Effectiveness of educational and behavioral asthma interventions. *Pediatrics, 123*(Suppl 3), S185.

Creer, T. L. (2008). Behavioral and cognitive processes in the self-management of asthma. *The Journal of Asthma, 45*(2), 81.

Dahl, J., & Gustafsson, D. (1990). Effects of a behavioral treatment program on children with asthma. *The Journal of Asthma, 27*(1), 41.

King, N. J. (1980). The behavioral management of asthma and asthma-related problems in children: A critical review of the literature. *Journal of Behavioral Medicine, 3*(2), 169.

Ritz, T., & Dahme, B. (2004). Behavioral interventions in asthma: Biofeedback techniques. *Journal of Psychosomatic Research, 56*(6), 711.

Atherogenesis

- ▶ Atherosclerosis

Atherosclerosis

Jennifer Carter
The University of Iowa, Iowa City, IA, USA

Synonyms

Arteriosclerosis; Atherogenesis; Atherosclerotic plaque

Definition

Atherosclerosis is the thickening and hardening of artery walls that occurs as the immune system responds to injuries to the single-layer endothelial wall of an artery. These injuries can be caused by a variety of insults, including cigarette smoke derivatives, toxins, infectious agents, elevated circulating lipids or glucose, and increased blood pressure. Monocytes and platelets are recruited to the site of injury. Monocytes cross the endothelial layer into the subintimal space where they become macrophages and phagocytose oxidized lipoproteins, forming foam cells. These foam cells accumulate in the subintimal space forming fatty streaks on the endothelial wall. Smooth muscle cells wall off the lipid plaque, forming a layer of collagen known as a fibrous cap between the plaque in the subintima and the endothelial wall. Eventually, the plaque may bulge into the arterial lumen, partially occluding the artery. If the artery becomes so occluded that blood flow is critically decreased, an infarct occurs. If this occurs on a coronary artery, a myocardial infarct, or heart attack, occurs. Health behavior change is a critical component in atherosclerosis prevention. Helping patients stop smoking and prevent or treat hypertension, hyperlipidemia, and impaired glucose tolerance are effective methods for preventing the progression of atherosclerosis.

References and Readings

Gropper, S., Smith, J., & Groff, J. (2005). *The role of lipids and lipoproteins in atherogenesis advanced nutrition and human metabolism* (4th ed., pp. 166–167). Belmont, CA: Thomson Wadsworth.

Ignarro, L. J., Balestrieri, M. L., & Napoli, C. (2007). Nutrition, physical activity, and cardiovascular disease: An update [Review]. *Cardiovascular Research, 73*(2), 326–340. doi:10.1016/j.cardiores.2006.06.030.

Kumar, V., Robbins, S. L., & Cotran, R. S. (2003). *Arteriosclerosis Robbins basic pathology* (7th ed., pp. 328–338). Philadelphia/London: WB Saunders.

Ross, R. (1999). Atherosclerosis – An Inflammatory Disease. *New England Journal of Medicine, 340*(2), 115–126. doi:10.1056/NEJM199901143400207.

Atherosclerotic Plaque

► Atherosclerosis

Atrial Fibrillation

William Whang
Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Definition

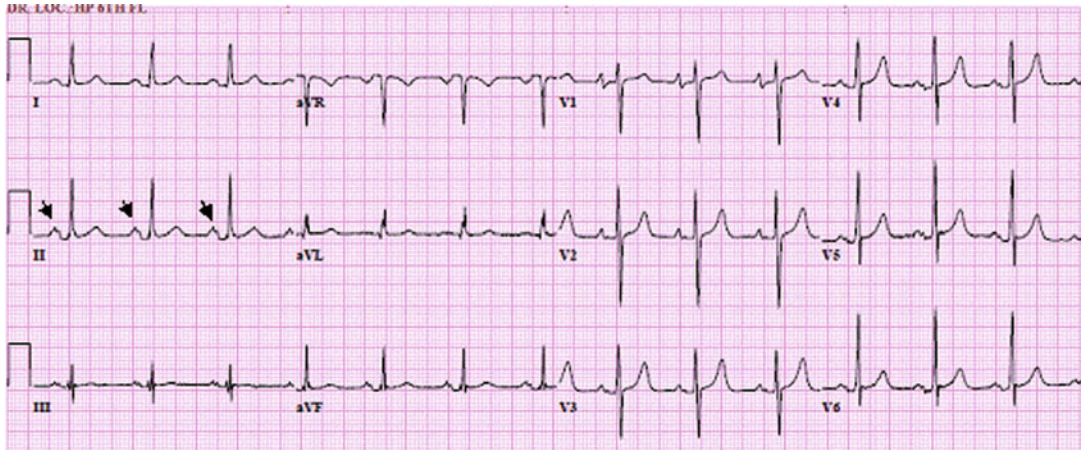
Atrial fibrillation is a cardiac rhythm disorder characterized by chaotic atrial electrical activation, resulting in an irregular heartbeat.

Description

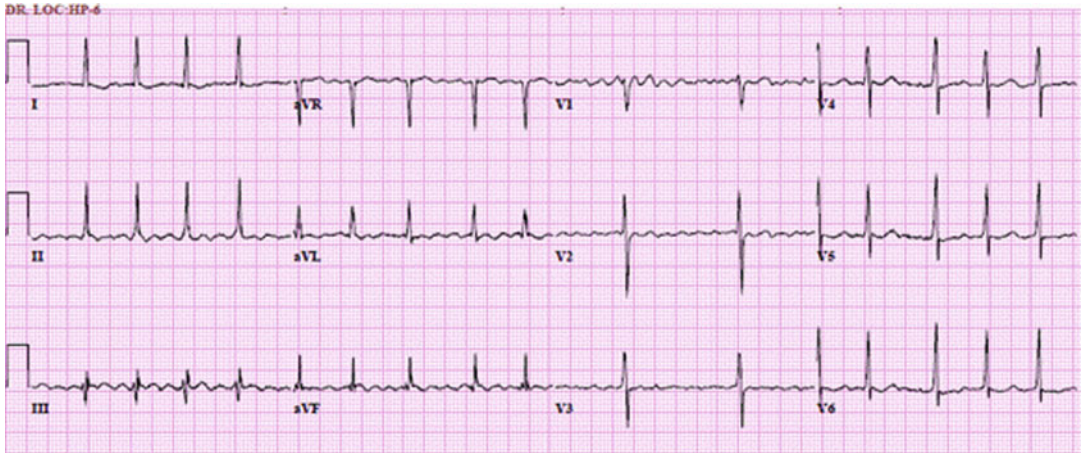
Atrial fibrillation (AF) is the most common type of cardiac rhythm disorder and affects 1–2% of the general population (Camm et al., 2010). It is characterized by chaotic electrical activation in the left and right atria, usually at rates of 200–300 beats per minute. Normally, cardiac activation begins with depolarization in the sinus node in the right atrium. The right and left atria are activated via intercellular gap junctions, and atrial depolarization is represented on the surface electrocardiogram (ECG) by the P wave (Fig. 1). The difference between normal rhythm and atrial fibrillation is exemplified by the ECGs seen in Fig. 2. AF is usually diagnosed on ECG by the presence of an irregularly irregular cardiac rhythm, without visible P waves.

Numerous factors are associated with the development of AF, including cardiovascular conditions such as hypertension, heart failure, and coronary artery disease and valvular heart disease (Camm et al., 2010). Aging, hyperthyroid, obesity, and diabetes mellitus have also been related to risk of AF.

Individuals with AF usually present with symptoms including palpitations, fatigue, shortness of



Atrial Fibrillation, Fig. 1 12-lead electrocardiogram during normal sinus rhythm, with arrows pointing to P waves that indicate organized atrial activity



Atrial Fibrillation, Fig. 2 12-lead electrocardiogram during atrial fibrillation, with lack of P waves and an irregularly irregular rhythm

breath, and/or light-headedness. However, a significant proportion of patients with AF are asymptomatic, especially older patients. In observational studies, AF is associated with worse cardiovascular prognosis, including increased risk of stroke, heart failure, cardiac hospitalizations, and mortality. However, it is not yet clear whether mitigation of AF itself improves these risks or whether it is more a marker of other pathology.

The most established health risk for most patients with AF is the risk of stroke, and this risk is related to several possible mechanisms (Camm et al., 2010). For instance, patients with AF have been shown to have relative stasis of blood flow in the left atrium, which is thought to lead to greater risk of clot formation. Abnormalities of the inner surface of the heart, the endocardium, and clotting and platelet activation have also been described. The decision to treat with anticlotting medication (anticoagulants) to prevent stroke is usually based on the number of stroke risk factors.

The CHADS2 score is often used to estimate clinical suspicion for stroke in the setting of AF

(Camm et al., 2010). The components of the CHADS2 score include congestive heart failure, hypertension, age >75, diabetes, and history of stroke (counted twice). The estimated risk of stroke for someone with CHADS2 score of zero is about 1.9% per year, and this risk increases to about 18.2% per year for someone with the maximum CHADS2 score of 6. Meta-analyses of stroke prevention trials have estimated that a relative risk reduction of 64% versus placebo from anticoagulant medication such as warfarin.

Over time, long-lasting atrial fibrillation leads to changes in atrial size including dilatation and scarring. The typical pattern of AF involves a progression from short, infrequent episodes to longer frequent attacks, to sustained AF. AF is categorized by the length of time it lasts with each episode. Paroxysmal AF terminates spontaneously and lasts as long as 7 days at a time (Camm et al., 2010). Persistent AF lasts longer than 7 days or requires termination through antiarrhythmic medication or with DC cardioversion. Permanent AF occurs when the constant presence of AF is accepted by both patient and physician, and efforts are directed at controlling the heart rate despite continued presence of an irregular rhythm.

Independent of treatment for stroke prevention, strategies for therapy of AF are broadly divided into rate control and rhythm control. Rate control consists of an emphasis on preventing sustained episodes of fast heart rate (>100 beats per minute), usually through medications that can slow conduction from the atria to the ventricles such as beta blockers, calcium channel blockers, and digoxin. Rhythm control involves a focus on maintaining normal sinus rhythm usually through antiarrhythmic medications or through surgical or catheter-based procedures to treat AF. The Atrial Fibrillation Follow-Up Investigation of Rhythm Management (AFFIRM trial) showed in a group of 4,060 patients with AF who were 65 or older or who had other risk factors for stroke, rate control and rhythm control (which consisted mainly of treatment with amiodarone) were equivalent in terms of 5-year mortality (Wyse et al., 2002). In a number of patients, however, symptoms from AF are detrimental to quality of life such that this becomes the main reason to pursue a rhythm control strategy.

Ablative therapy for AF is a growing treatment that involves creating barriers to electrical conduction in atrial tissue, typically with radiofrequency energy often delivered via catheters. In particular, tissue at the junction of the left atrium and the pulmonary veins has been noted to be a frequent trigger of AF, and electrical isolation of the pulmonary veins is especially effective at reducing AF in patients with paroxysmal episodes.

There is some evidence that psychosocial symptoms may be related to atrial fibrillation. Lange and colleagues showed in a group of 54 patients with persistent AF that depressive mood was associated with greater risk of recurrence after DC cardioversion (Lange, 2007). In a triggering analysis that used event monitoring and electronic diaries, Lampert and colleagues observed in 75 patients with paroxysmal or persistent AF that arrhythmia episodes were more likely preceded by negative emotions and less likely by happiness (Lampert et al., 2008). Analyses of the Framingham Offspring Study have found that baseline levels of tension, anger, and hostility predicted increased 10-year risk of AF in men, but not in women (Eaker, Sullivan, Kelly-Hayes, D'Agostino, & Benjamin, 2004, 2005).

References and Readings

Camm, A. J., Kirchhof, P., Lip, G. Y., Schotten, U., Savelieva, I., Ernst, S., et al. (2010). Guidelines for the management of atrial fibrillation: The task force for the management of atrial fibrillation of the European Society of Cardiology (ESC). *European Heart Journal, 31*, 2369–2429.

Eaker, E. D., Sullivan, L. M., Kelly-Hayes, M., D'Agostino, R. B., Sr., & Benjamin, E. J. (2004). Anger and hostility predict the development of atrial fibrillation in men in the Framingham offspring study. *Circulation, 109*, 1267–1271.

Eaker, E. D., Sullivan, L. M., Kelly-Hayes, M., D'Agostino, R. B., Sr., & Benjamin, E. J. (2005). Tension and anxiety and the prediction of the 10-year incidence of coronary heart disease, atrial fibrillation,

and total mortality: The Framingham offspring study. *Psychosomatic Medicine, 67*, 692–696.
Lampert, R. B. M., Brandt, C., Dziura, J., Liu, H., Donovan, T., Soufer, R., et al. (2008). Impact of emotions on triggering of atrial fibrillation. *Circulation, 118*, S640.
Lange, H. W., & Herrmann-Lingen, C. (2007). Depressive symptoms predict recurrence of atrial fibrillation after cardioversion. *Journal of Psychosomatic Research, 63*(5), 509–513.
Wyse, D. G., Waldo, A. L., DiMarco, J. P., Domanski, M. J., Rosenberg, Y., Schron, E. B., et al. (2002). A comparison of rate control and rhythm control in patients with atrial fibrillation. *The New England Journal of Medicine, 347*, 1825–1833.

Atrophy

Beth Schroeder
University of Delaware, Newark, DE, USA

Synonyms

Muscle wasting

Definition

Atrophy in the simplest sense is a type of cellular adaptation in which a cell, tissue, or organ of the body reduces in size. It is often the result of chronic stressors being applied to the body. Chronic stressors are things such as a physical injury, disease pathology, or immobilization and disuse. It is commonly seen as a symptom in neuromuscular and musculoskeletal conditions or injury, but atrophy may affect any body system or structure.

Description

When cells of the body are under stressors such as those listed above, there is a potential for cellular injury and cell damage. These cellular injuries may include lack of blood flow to the area, which is known as ischemia, infection, immune system responses, lack of adequate nutrition, or physical trauma. If the stressors persist, cells will attempt to make cellular adaptations in order to maintain homeostasis to withstand them. Examples of cellular adaptations include atrophy, hypertrophy, hyperplasia, metaplasia, or dysplasia may occur. By adapting, the cells are able to avoid injury or cell death.

In the human body atrophy may occur for physiologic or pathologic reasons. Physiologic atrophy is associated with the natural aging process, and it usually involves things such as general muscle wasting and bone loss. Pathologic atrophy is the result of some form of cellular injury, such as a neuromuscular condition, cancer, peripheral vascular disorders resulting in inefficient blood flow, or spinal cord injury.

The musculoskeletal system can become atrophied for two general reasons: disuse atrophy or neurogenic atrophy. With disuse atrophy, skeletal muscles may atrophy due to immobilization and disuse, which often occurs following an injury, such as an ankle sprain, or surgery, such as a joint replacement procedure. Individuals may also immobilize themselves if movement causes significant pain or discomfort. In neurogenic atrophy, the nerve supply to the affected muscle is disrupted in some way. This type of muscle atrophy may also occur with a lower motor neuron injury, such as a cut peripheral nerve or spinal cord injury. After this type of injury, the involved muscles become partially or completed denervated, meaning that the nerve supply to the muscle is disrupted. This results in less voluntary movement and control of the involved muscles. It is thought that this lack of use is responsible for the physiological changes that occur in muscles that ultimately result in their atrophy.

Several morphological changes occur in the muscles when they atrophy. A loss in the contractile proteins actin and myosin occurs, as well as changes in the blood supply, as the capillary density of these muscles decrease. When these skeletal muscle proteins are lost, a decrease in muscle fiber length and diameter will occur, and ultimately atrophy of the muscle as a whole. This loss of muscle mass also leads to weakness, as the muscles are no longer capable of producing the same amount of force. This is an important factor

for patients undergoing surgery. Postsurgical patients are a population of individuals that often have disuse atrophy of their muscles. For example, patients undergoing an anterior cruciate ligament (ACL) repair surgery tend to have weak, atrophied quadriceps after the surgery. For this reason, patients often participate in strengthening programs before the surgery.

Atrophy does not need to be caused by an injury as described above. It can also be due to a genetic condition. Spinal muscular atrophy (SMA) is a genetic condition resulting from loss of ventral horn cells in the spinal cord that are responsible for motor function. Individuals with this condition suffer from skeletal muscle atrophy, weakness, and hypotonia. Complaints of fatigue are also common in this population. Those with SMA may experience respiratory problems as well because the diaphragm is a skeletal muscle often affected. As the condition progresses, the use of assistive devices, such as a wheelchair, for mobility may become necessary.

The treatment of atrophy depends upon its cause as well as its impact on the individual. Those with disuse atrophy may reverse the effects of atrophy from physical therapy or a simple exercise program. If the cause of the atrophy is more genetic or permanent in nature, like that occurs with SMA or spinal cord injury patients, exercise will still be beneficial, but the treatments may need to be augmented with the use of adaptive equipment, such as splints or braces.

Cross-References

► Stressor

References and Readings

Drake, R. L., Wayne Vogl, A., & Mitchell, A. W. M. (2010). *Gray's anatomy for students* (2nd ed.). Philadelphia: Churchill Livingstone Elsevier.

Goodman, C. C., & Fuller, K. S. (2009). *Pathology: Implications for the physical therapist* (3rd ed.). St. Louis, MO: Saunders.

Kisner, C., & Colby, L. A. (2007). *Therapeutic exercise* (5th ed.). Philadelphia: F.A. Davis Company.

MedlinePlus [Internet]. Muscle atrophy, Bethesda (MD): National Library of Medicine (US), (updated 2010 Feb 6; cited 2011 April 4), (about 2 p.). Available from http://www.nlm.nih.gov/medlineplus/ency/article/003188.htm

Purves, D., Augustine, G. J., Fitzpatrick, D., Hall, W. C., LaMantia, A., McNamara, J. O., & White, L. E. (2008). *Neuroscience* (4th ed.). Sunderland, MA: Sinauer Associates.

Robinson, A. J., & Snyder-Mackler, L. (2008). *Clinical electrophysiology* (3rd ed.). Philadelphia, PA: Lippincott.

Attachment Theory

Angela M. Hicks[1] and Carolyn Korbel[2]
[1]Department of Psychology, Westminster College, Salt Lake City, UT, USA
[2]The Neurobehavioral Clinic and Counseling Center, Lake Forest, CA, USA

Definition

Bowlby (1969, 1988) described an attachment as an emotional bond that is characterized by the tendency to seek out and maintain proximity to a specific attachment figure, particularly during times of distress.

Description

Overview of Attachment Theory

Normative processes. Bowlby's attachment theory (e.g., Bowlby, 1969, 1988) suggests that humans' most intimate relationship partners serve important functions related to distress alleviation. More specifically, he theorized that *people* rely on *their* primary caregivers, or *attachment figures*, for feelings of comfort and security, especially during times of distress. According to Bowlby, the attachment system evolved in order to keep vulnerable human infants within close proximity to their caregivers. Normatively, when an infant experiences distress, the attachment system will be activated. The infant will then signal its distress to the

caregiver who will respond in an appropriate manner and the infant's distress will be alleviated. Over time, the infant develops an emotionally primary bond with its caregiver such that caregiver proximity in and of itself provides feelings of comfort/security. Bowlby suggested that four specific attachment behaviors are present in such relationships. First, the infant relies on the attachment figure as a source of comfort, or *safe haven* to turn to when distressed. Once a sense of comfort and security is established, the infant uses the caregiver as a *secure base* from which to explore the environment. Because the attached person relies on the caregiver for feelings of comfort and safety, infants *seek proximity* to their attachment figures and experience significant distress when separated from them (*separation distress*).

With increasing age, individuals rely less on actual proximity to attachment figures and more on *internalized representations* of these individuals (Bowlby, 1969, 1988; Brethereton, 1985). Specifically, through repeated emotionally relevant interactions with attachment figures, people are theorized to develop unconscious representations of those relationships, termed *internal working models* (IWMs). Under conditions of distress, in which the attachment system is activated, individuals derive feelings of emotional security through the IWM when their attachment figures are not physically present.

While Bowlby (1969, 1988) initially conceptualized attachment theory with regard to relationship processes during infancy and childhood, such bonds are thought to be influential during adolescence and adulthood as well. In adulthood, attachment bonds are formed primarily within the context of romantic relationships (e.g., Hazan & Shaver, 1987; Weiss, 1988). Adult romantic relationships have increasingly been conceptualized as attachment bonds characterized by the same central components as attachment bonds in infancy: heightened proximity maintenance, resistance to separation, and utilization of the partner as a preferred target for comfort- and security-seeking (Hazan & Zeifman, 1999). As noted earlier, attachment theory proposes that internal working models are representations of the caregiving, distress-alleviating functions of attachment figures. If, as Bowlby argued, the attachment system operates in this fashion "from the cradle to the grave," then it follows that internalized representations of *adult* attachment figures – romantic partners – should function to promote distress alleviation among adults in the same way that internalized representations of caregivers are hypothesized to do in infancy and childhood. A growing body of research suggests that romantic partners do, in fact, seek one another out during times of distress and, alternatively, offer comfort and support to their partners (e.g., Collins & Feeney, 2000).

Internal working models were originally conceptualized as a set of expectations about the self and close others, based in previous experiences, which guide cognitive, emotional, and behavioral responses to current experiences. More recently, however, such models are thought to reflect an individual's capacity and typical strategies for managing positive and negative emotional arousal, processes that are collectively known as *emotion regulation* (Mikulincer & Shaver, 2007). The increasing attention paid to the emotion regulating functions of the internal working model is important in light of the concurrent focus on emotion processing as one mechanism through which close relationships influence health (Diamond, 2001; Diamond & Fagundes, 2010; Diamond & Hicks, 2004; Ryff, Singer, Wing, & Love, 2001).

Individual differences. The normative perspective contends that all humans are innately predisposed to form attachment bonds. Yet, attachment theory suggests that not all attachment relationships are of similar *quality*. Specifically, individuals whose caregivers provided consistent and responsive distress alleviation are theorized to develop secure working models of attachment (Ainsworth, Blehar, Waters, & Wall, 1978; Bowlby, 1969, 1988). Conversely, those who did not experience consistent or responsive caregiving develop insecure models. A student of Bowlby's, Mary Ainsworth, and her colleagues identified three patterns. Each pattern is thought to reflect a specific history of caregiver interaction and emotion processing. Specifically, *secure*

persons are described as having experienced consistent and responsive caregiving. As a result, they associate proximity to caregivers with effective distress alleviation, see others as willing to provide responsive care, and themselves as worthy of it. *Anxious* persons are described as having experienced inconsistently responsive caregiving, resulting in uncertainty about whether proximity to caregivers will result in distress alleviation. They therefore develop an internal working model in which they are unworthy of love and comfort and in which attachment figures are unreliable. *Avoidant* persons are described as having experienced consistently unresponsive caregiving. Hence, they do not associate proximity to caregivers with feelings of comfort or distress alleviation, and therefore develop an interpersonal style that emphasizes self-reliance and involves distancing themselves from others during times of stress. Such "attachment styles" were initially conceptualized as a relatively stable trait-like individual difference dimension. Individual differences in patterns of attachment are thought to shape emotional processing, relational cognition, and relationship behavior over the life course.

Much like the normative aspects of attachment, there are also parallels between infant caregiver and adult romantic relationships on the individual difference dimension. In their groundbreaking study, Hazan and Shaver (1987) found evidence for three similar patterns of difference in adults with respect to their romantic partners. For example, adults that were identified as *secure* reported feeling comfortable with closeness and experiencing reciprocal support provision in relationships. Those identified as *anxious* reported wanting more closeness than relationship partners were willing to provide, and feeling uncertain about their partners' devotion. Adults classified as *avoidant* reported feeling uncomfortable with closeness and preferring more emotional distance from their partners.

Measurement. Across the lifecourse, measures of attachment assess the extent to which one is comfortable relying on their attachment figure when under distress. Such measures aim to tap into the content of the internal working model and differentiate between those with a secure, anxious, or avoidant "attachment style." The primary measure to assess attachment in infancy is the *strange situation*, a laboratory procedure during which the infant experiences brief separations and reunions with the primary caregiver providing the opportunity for researchers to observe the infants' tendency to seek proximity to and derive comfort from the caregiver when under distress (Ainsworth, Blehar, Waters, & Wall, 1978). Methods for assessing attachment from middle childhood through adolescence have been slower to emerge. As noted earlier, with increasing cognitive capabilities, children come to rely less and less on actual physical proximity to the caregiver and more on representations of the attachment relationship that are contained within the internal working model. From middle to late childhood three different measurement approaches have been utilized to assess the child's attachment representation/s. These methods include projective measures of attachment (e.g., Separation Anxiety Test), structured interviews (e.g., Child Attachment Interview), and questionnaires (e.g., Security Scale). During adolescence, methods which mirror those used with adults are utilized. A slightly modified version of the Adult Attachment Interview assesses adolescents' states of mind with regard to attachment. Other studies use a revised version of the Experiences in Close Relationships (described below) to identify attachment-related anxiety and avoidance in primary attachment relationships.

When considering issues of measurement in adulthood, it is important to note that adult attachment research has grown almost independently within two research traditions (reviewed by Crowell, Fraley, & Shaver, 2008). The measures used by the two traditions reflect differences of opinion regarding the extent to which the content of the internal working model is consciously accessible. The first line of work uses measures that tap into nonconscious perceptions and beliefs about close relationships. These measures assess either (1) adults' current state of mind with respect to early attachment relationships, as assessed by the Adult Attachment Interview, or (2) the coherence of adults' descriptions of their current romantic relationships, as

assessed by the Current Relationships Index. The second line of research uses the Experiences in Close Relationships (ECR) inventory; a paper and pencil measure assessing consciously accessible levels of comfort with closeness to and heightened vigilance regarding the availability of romantic partners. Finally, although attachment patterns have historically been viewed as discrete prototypes (i.e., secure, anxious, and avoidant), more current research measures these dimensions as continuous, rather than categorical, attributes (i.e., Fraley, Waller, & Brennan, 2000). The current entry will make reference secure, anxious, and avoidant "types" for the sake of clarity and to reflect the categorical approach pervasive in prior literature.

Implications for Behavioral Medicine

The last 15 years have seen a dramatic proliferation of research evidence pointing to the influence of attachment experiences and relationships on important markers of physical health. One important theoretical framework that helps ground this work emphasizes the influence of attachment histories and relationships on emotion regulation as one important mechanism linking attachment to health outcomes (Diamond & Hicks, 2004). This perspective aligns nicely with a growing body of research implicating emotions as a central mechanism in links between relationships and health (Ryff et al., 2001). Thus, it is important to integrate this discussion of the health implications of attachment with a brief review of the impacts of attachment processes on emotional experience. Research evidence examining links between relationships and health suggests that both the normative and individual differences components of attachment relationships are important.

Attachment security conveys multiple emotion regulation benefits during childhood and adolescence. Securely attached children are more likely to view themselves as worthy of love and caring as noted above. As such, they value their health and well-being. Perceiving attachment figures as available and helpful in times of distress allows securely attached children to acknowledge their discomfort and seek support when needed. The adaptive emotion regulation strategies that secure children use promote healthy social (i.e., increased social competence, positive peer and family relationships), emotional (i.e., lower levels of depression, anxiety, and social anxiety, and fewer concerns about loneliness), and behavioral functioning (i.e., fewer somatic complaints, fewer behavioral concerns or conduct problems).

Unlike their securely attached counterparts, insecurely attached children develop strategies that are less adaptive. Anxiously attached children tend to utilize hyperactivating strategies, and display heightened distress to promote proximity to attachment figures. Because they are so sensitive to distress and use such ineffective support-seeking strategies, they are unable to effectively regulate their negative affect. Anxious children therefore more often display frequent and intense expressions of negative affect (i.e., tantrums), behavioral dysregulation, anxiety, and depression. Anxiously attached children and adolescents also tend to be clingy toward attachment figures, display a high need for approval, and report having lower self-esteem. In contrast, avoidant children have learned to use deactivating emotion regulation strategies that minimize negative affect and support-seeking behaviors. However, their deactivating strategies may not be very effective at regulating negative emotion, as avoidantly attached adolescents are more likely to display conduct disordered and criminal behavior than those with other attachment organizations.

Attachment and emotion regulation. As with attachment patterns in infancy and childhood, adult attachment styles are associated with robust individual differences in emotion regulation processes (e.g., Mikulincer & Shaver, 2007). Attachment theory maintains that internal working models impact attention and processing of affectively relevant information. For example, secure individuals are more likely to interpret benign stimuli in more neutral or even positive ways, and they are more likely to offer the benefit of the doubt when a relationship partner engages in ambiguous, yet potentially threatening behaviors. Individuals with more secure working models are also more comfortable relying on others for

support during times of distress. They also report engaging in more constructive intra- and interpersonal regulatory strategies. It follows, then, that secure individuals report more frequent and intense bouts of positive emotion, and less frequent and less intense negative emotion.

While secure attachment is conceptualized as a resource that promotes effective regulation, attachment insecurity appears to interfere with the ability to effectively modulate emotional experience. Specifically, those characterized as *anxious* are likely to interpret neutral stimuli as more hostile and negative. They are also more likely to make negative, blaming attributions of partners' ambiguous behaviors. They tend to engage in indirect and ineffective support seeking and less constructive regulatory strategies. Not surprisingly, then, anxious individuals report more intense and more frequent negative emotional experiences. They report less positive emotions as well, and do not always derive the same benefits from positive events or experiences. Attachment avoidance is conceptualized as involving minimization and suppression of emotional experience. Supporting this view, persons endorsing high levels of avoidance report low levels of both negative *and* positive emotions, and high levels of emotional control. Rather than seeking support, avoidant individuals tend to distance themselves from others especially when levels of distress appear to be high. Recent research suggests that avoidant individuals report such low levels of emotional activation because they use a preemptive strategy of focusing attention away from affectively relevant stimuli and events.

Attachment and health. In general, research demonstrating associations between attachment and physical health across the life span takes three different approaches. The first describes a developmental neuroscience-based approach for understanding how early experiences with primary caregivers "tune" developing neurobiological systems' sensitivity to stress, and in essence predispose individuals toward regulatory strategies that are highly influenced by early attachment experiences. The second emphasizes the moderating effects of attachment on "microlevel" physiological processes, such as autonomic nervous system and hypothalamic pituitary adrenocortical (HPA) axis functioning. The third takes a more macrolevel approach, investigating the incidence and management of specific diseases and conditions. The most robust findings from each of these literatures are described below.

An emerging neuroscience literature has begun to demonstrate compelling links between affectively rich, synchronous mother-infant interactions with the development and functioning of the relational right brain and the orbitofrontal system. These neuroanatomical structures are directly responsive to the infant's affective and relational environment; in this way, the affective tone of repeated interactions with caregivers influences the "hardwiring" of pathways that are implicated in emotion regulation (Schore, 2000). Thus early experiences serve to create a template for emotion regulation at the neurophysiological level. This compelling research suggests that one of the ways relationships "get under our skin" is that they shape the development of stress responses through psychoneurobiologically mediated pathways.

Studies linking adult attachment HPA axis functioning typically assess salivary cortisol, while those examining sympathetic and parasympathetic branches of the autonomic nervous system typically assess heart rate, blood pressure, respiratory sinus arrhythmia, and electrodermal activity. Each of these systems is important from a behavioral medicine perspective, as dysregulation of each system has been linked to potentially deleterious health outcomes. This research primarily emphasizes differences in baseline, or resting levels as well as short-term reactivity to laboratory stressors (though at least one study had examined reactivity to daily relationship events). In general, insecurely attached individuals show heightened HPA and ANS stress reactivity. For example, anxious individuals are found to experience heightened electrodermal, heart rate, and blood pressure reactivity to general laboratory stressors (such as difficult mathematical tasks with frustrating interruptions). They also demonstrated greater electrodermal and HPA reactivity during laboratory conflict with their

romantic partners. Consistent with their heightened concerns around attachment figure availability, anxious persons have also demonstrated heightened cortisol to stimuli priming thoughts of abandonment in a laboratory, as well as heighted daily cortisol output during a brief travel-related separation from their romantic partner. In terms of basal levels, anxious persons have been found to have suppressed resting parasympathetic and HPA activity, both presumed to indicate a dysregulation in those systems. All of these findings are consistent with the evidence demonstrating anxious individuals' heightened emotional reactivity as well (discussed earlier).

Avoidant individuals, on the other hand, tend to report blunted emotional experiences; they report neither high negative nor positive emotions generally, and demonstrate little emotional reactivity to laboratory stressors. Yet, they have been found to demonstrate heightened physiological reactivity to lab stressors. For example, avoidant individuals showed heightened HPA reactivity to an in-lab conflict with their romantic partners, as well as to exposure to stimuli priming thoughts of abandonment. In other studies, they experienced heightened sympathetic and electrodermal reactivity to nonrelationship laboratory stress tasks (i.e., stressful mathematical tasks). Further, they have been found to show a blunted basal parasympathetic activity. Thus, some researchers suggest that avoidant individuals' defensive regulatory strategy, while effective at suppressing intense affective states, may come at a physiological cost.

Another line of research examines empirical associations between attachment and health across a range of health-related domains. While some studies find that having a chronic illness itself does not increase one's odds of developing an insecure attachment, other researchers find a greater frequency of insecure attachment classifications, particularly anxious attachment, among pediatric populations (i.e., among premature infants, infants with congenital heart disease, and in pediatric patients with cerebral palsy, epilepsy, cleft lip, and cystic fibrosis; Feeney, 2000; Minde, 1999). Although the influence of child and disease-specific factors is likely to exert some influence on attachment, poor maternal and family relationship quality are thought to provide a more robust influence on children's attachment organizations. Insecure attachments among adolescents and adults have also been associated with psychosomatic illnesses, physical complaints, symptom reporting, anxiety, and depression (Maunder & Hunter, 2001). Attachment insecurity has also been linked to greater emotion-focused coping among children with asthma and also among healthy university students. Among adults with diabetes mellitus, avoidant attachment has been associated with poor metabolic control, adherence, and less health care utilization. Diabetic adults with anxious attachments had high health care utilization costs and, surprisingly more optimal metabolic control levels (Ciechanowski et al., 2004).

A growing and compelling literature suggests that attachment-related processes broadly predict personal well-being, coping, physiological responses to stress, and health-related behaviors and illness management across the life span. Additional research that examines the emotion regulation pathways by which these associations are mediated will increase the field's understanding of how primary attachment relationships interact with critical psychobiological mechanisms to influence health across the life span.

Cross-References

- ▶ Anxiety and Its Measurement
- ▶ Avoidance
- ▶ Child Development
- ▶ Emotional Control
- ▶ Emotional Expression
- ▶ Emotional Responses
- ▶ Emotions: Positive and Negative
- ▶ Family, Relationships

References and Readings

Ainsworth, M. D. S., Blehar, M. C., Waters, E., & Wall, S. (1978). *Patterns of attachment: A psychological study of the Strange Situation*. Hillsdale, NJ: Erlbaum.

Bowlby, J. (1969). *Attachment*. New York: Basic Books.
Bowlby, J. (1988). *A secure base: Parent-child attachment and healthy human development*. London: Basic Books.
Bretherton, I. (1985). Attachment theory: Retrospect and prospect. In I. Bretherton and E. Waters (Eds.), *Growing points of attachment theory and research. Monographs of the Society for Research in Child Development, 209*(1–2), 3–35.
Cassidy, J., & Shaver, P. R. (2008). *Handbook of attachment: Theory, research, and clinical applications*. New York: Guilford Press.
Ciechanowski, P., Russo, J., Katon, W., Von Korff, M., Ludman, E., Lin, E., et al. (2004). Influence of patient attachment style on self-care and outcomes in diabetes. *Psychosomatic Medicine, 66*, 720–728.
Collins, N. L., & Feeney, B. C. (2000). A safe haven: An attachment theory perspective on support seeking and caregiving in intimate relationships. *Journal of Personality and Social Psychology, 78*(6), 1053–1073.
Crowell, J. A., Fraley, R. C., & Shaver, P. R. (2008). Measurement of individual differences in adolescent and adult attachment. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (2nd ed., pp. 599–634). New York: Guilford Press.
Diamond, L. M. (2001). Contributions of psychophysiology to research on adult attachment: Review and recommendations. *Personality and Social Psychology Review, 5*, 276–295.
Diamond, L. M., & Fagundes, C. P. (2008). Developmental perspectives on links between attachment and affect regulation over the lifespan. *Advances in Child Behavior and Development, 36*, 83–134.
Diamond, L. M., & Fagundes, C. P. (2010). Psychobiological research on attachment. *Journal of Social and Personal Relationships, 27*, 218–225.
Diamond, L. M., & Hicks, A. M. (2004). Psychobiological perspectives on attachment: Implications for mental and physical health. In S. R. R. J. Simpson (Ed.), *Adult attachment: Emerging issues and new directions*. New York: Guilford Press.
Feeney, J. A. (2000). Implications of attachment style for patterns of health and illness. *Child: Care, Health and Development, 26*, 277–288.
Fraley, R. C., Waller, N. G., & Brennan, K. A. (2000). An item response theory analysis of self-report measures of adult attachment. *Journal of Personality and Social Psychology, 78*(2), 350–365.
Hazan, C., & Shaver, P. R. (1987). Romantic love conceptualized as an attachment process. *Journal of Personality and Social Psychology, 52*(3), 511–524.
Hazan, C., & Zeifman, D. (1999). Pair bonds as attachments: Evaluating the evidence. In J. Cassidy, & P.R. Shaver (Eds.), *Handbook of Attachment Theory and Research*.
Maunder, R. G., & Hunter, J. J. (2001). Attachment and psychosomatic medicine: Developmental contributions to stress and disease. *Psychosomatic Medicine, 63*, 556–567.
Mikulincer, M., & Shaver, P. R. (2007). *Attachment in adulthood: Structure, dynamics, and change*. New York: Guilford Press.
Minde, K. (1999). Mediating attachment patterns during a serious medical illness. *Infant Mental Health Journal, 20*(1), 105–122.
Ryff, C. D., Singer, B. H., Wing, E., & Love, G. D. (2001). Elective affinities and uninvited agonies. In B. H. Singer & C. D. Ryff (Eds.), *Emotions, social relationships, and health* (pp. 133–175). New York: Oxford University Press.
Schore, A. N. (2000). Attachment and the regulation of the right brain. *Attachment and Humand Development, 2*, 23–47.
Weiss, R. S. (1988). Loss and recovery. *Journal of Social Issues, 44*, 37–52.

Attention

▶ Coffee Drinking, Effects of Caffeine

Attention Training

▶ Meditation
▶ Transcendental Meditation

Attitudes

Austin S. Baldwin
Department of Psychology, Southern Methodist University, Dallas, TX, USA

Synonyms

Beliefs; Evaluations

Definition

An attitude is broadly defined as a tendency to evaluate a particular entity with some degree of favor or disfavor (Eagly & Chaiken, 1993).

Ajzen and Fishbein (2005), two preeminent attitude researchers, have distinguished between two general types of attitudes: attitudes toward objects and attitudes toward behavior. Attitudes toward behavior, specifically health behavior, are more relevant to behavioral medicine.

Description

Attitudes and Health Behavior

Current understanding of attitudes is based in an expectancy-value framework in which an attitude toward a behavior is a function of (a) beliefs that engaging in the behavior will result in certain outcomes (positive and negative), and (b) evaluations associated with outcomes that result from engaging in the behavior (positive and negative). For example, a person's attitude toward engaging in physical activity is a function of his or her beliefs about the consequences of exercising (e.g., improved health and appearance, time taken from other activities) and evaluations associated with the outcomes of exercise (e.g., increased energy, body soreness). The belief and evaluation components can also be conceptualized as cognitive (beliefs) and affective (evaluations) components.

Attitudes toward engaging in health behaviors are central to understanding how people make health-related decisions. A person's belief about whether taking his or her hypertension medication is beneficial to his or her health, jointly with an evaluation of whether the effects of taking the medication are positive or aversive, will to some degree, influence his or her decision to take the medication. The centrality of attitudes to health decision making is evidenced in the fact that (a) attitudes are found in all prominent theories of health behavior, and (b) health message campaigns are largely based on the assumption that changing attitudes will result in behavior change. In the next section, the most prominent health behavior theories will be described, including how attitudes are conceptualized in the models and the evidence for the influence of attitudes on behavior. In the following section, different approaches to changing health behavior through changing attitudes will be described. Finally, a brief overview on the limits of the influence of attitudes on behavior will be provided.

Health Behavior Theories

Health Belief Model. The Health Belief Model (HBM) includes six constructs that are proposed to guide decisions to engage in health-related behaviors. The constructs are (1) perceived susceptibility to the health threat, (2) perceived severity of the health threat, (3) perceived benefits of engaging in the behavior, (4) perceived barriers to engaging in the behavior, (5) cues to action, and (6) self-efficacy to engage in the behavior. Although not explicitly described as attitudes, the constructs of perceived benefits and perceived barriers to engaging in the behavior fit the definition of attitudes well. The measurement of these two constructs captures people's positive (benefits) and negative (barriers) beliefs and evaluations of engaging in the behavior. According to the HBM, both constructs have a direct influence on behavior.

Across a variety of health behaviors, evidence suggests that higher benefits and lower barriers (i.e., more favorable attitudes) are associated with a greater likelihood to engage in health behaviors. For example, higher benefits and lower barriers have been shown to be associated with higher levels of mammogram adherence (Friedman, Neff, Webb, & Latham, 1998) and condom use (Volk & Koopman, 2001). The strength of the influence of benefits and barriers on behavior, as well as the influence of other constructs in the model, varies across different domains. Perceived barriers, however, have been shown to be the strongest predictor of behavior across all behavioral domains (Champion & Skinner, 2008).

Theory of Planned Behavior. The Theory of Planned Behavior (TPB) suggests that health behaviors are influenced most proximally by intentions to engage in the behavior. Intentions are influenced by attitudes toward the behavior, perceived norms to engage in the behavior, and perceived control over the behavior. In the model, attitudes are conceptualized and measured as both beliefs about engaging in the behavior and

evaluations of the associated outcomes. Because attitudes are thought to be proximal predictors of intentions rather than behaviors, attitudes have an indirect influence on behavior through intentions.

Evidence across a variety of health behavior domains, including exercise, smoking cessation, cancer screenings, substance use, and safe sex practices indicate that attitudes are an important predictor of behavior. As with the HBM, however, the strength of the influence of attitudes varies across different behavioral domains (Ajzen, Albarracin, & Hornik, 2007).

Transtheoretical Model of Behavior Change. The Transtheoretical Model (TTM) of behavior change assumes that behavior change is best understood as a process through which people progress through a series of discrete stages. Specifically, people go from not thinking about the behavior (precontemplation), to thinking about the behavior (contemplation), to considering the behavior (preparation), to engaging in the behavior (action). There is also a stage that considers continued behavior over time (maintenance). An important factor in the progression of stages is the weighing of the pros and cons for engaging in the relevant behavior. The TTM labels this factor decisional balance. As with the HBM, decisional balance is not explicitly described as an attitude but it captures people's beliefs and evaluations of the benefits (pros) and costs (cons) of engaging in the behavior. Thus, it fits the definition of attitudes well. According to the TTM, as people progress through the stages toward engaging in the behavior, decisional balance systematically changes such that pros increase and cons decrease.

The systematic shifting in pros and cons is characteristic of stage progression, and there is evidence for this shifting across many health behavior domains (e.g., smoking cessation, exercise, sunscreen use, safer sex practices; Prochaska, Redding, & Evers, 2008). In fact, evidence across many health domains suggests that moving from precontemplation to action requires a 1 standard deviation increase in perceived pros of engaging in the behavior (pros) and a .50 standard decrease in the perceived cons. Thus, people's evaluations of the benefits and costs of engaging in the behavior are central to behavior change.

Precaution Adoption Process Model. The Precaution Adoption Process Model (PAPM) is also a stage-based model of behavior change in which it is assumed that people progress from being unaware of the health issue to acting through a series of discrete stages. Unlike the TTM, the PAPM specifies the processes that are thought to guide movement between specific stages. For example, when people progress from being undecided about acting (Stage 3) to deciding to act (Stage 5) or not (Stage 4), beliefs about the effectiveness and difficulty of engaging in the behavior are thought to be critical at that stage (Weinstein, Sandman, & Blalock, 2008). These particular beliefs are thought to be less important, however, at other stages of the change process. As with the HBM and TTM, beliefs about the effectiveness and difficulty of engaging in the behavior are not explicitly described as attitudes. Yet, as with the other models, these beliefs capture people's evaluations of the behavior that fits the description of attitudes well.

Unlike the other models, there is much less evidence from research on the PAPM on the specific role of attitudes in influencing behavior. There is evidence that PAPM-based interventions are effective (Weinstein et al., 2008), but little attention has been paid specifically to the influence of beliefs about effectiveness and difficulty on behavior.

Attitude Change to Change Health Behavior

Evidence from persuasion research suggests that persuasive messages can change attitudes through two processes: one in which people process message content deeply and deliberately, the other in which peripheral aspects of the message (e.g., credibility of the source, people's mood) influence attitudes (Chaiken, Liberman, & Eagly, 1989; Petty & Cacioppo, 1986). Depending on the circumstances in which the communication occurs, attitudes can be influenced through one process or the other, or they can operate jointly. Attitudes are more likely to change and persist, however, when the message content is processed deeply and

deliberately. Message content is more likely to be processed deeply and deliberately when it is personally relevant.

Health campaigns are largely based on the assumption that changing attitudes will result in behavior change. This is evidenced by the fact that many health campaigns focus on making people aware of the costs and benefits of engaging (or not engaging) in behaviors. Different types of health communications and messages have been shown to be effective in changing attitudes and behavior. Three types of health messages are described below.

Message Framing. The effect of a health message on attitudes and health behaviors can differ depending on whether the message content is framed in terms of what one stands to gain (gain frame) or what one stands to lose (loss frame) by engaging (or not engaging) in a behavior. Whether a gain- or loss-framed message is more effective depends on the level of risk or uncertainty associated with engaging in the behavior. When risk or uncertainty is high, loss-frame messages should be more effective; when risk or uncertainty is low, gain-frame messages should be more effective. For example, loss-frame messages (i.e., what one stands to lose by not engaging in the behavior) are more effective in promoting illness-detecting behaviors (e.g., cancer screening) because there is some uncertainty about whether one will detect an unwanted outcome (e.g., a lump in a breast). In contrast, gain-frame messages (i.e., what one stands to gain by engaging in the behavior) are more effective in promoting prevention behaviors (e.g., sunscreen use) because there is little or no uncertainty associated with engaging in the recommended behavior (Rothman & Salovey, 1997).

Message Tailoring. Messages that are individually tailored to a person's attributes, interests, and/or concerns tend to be more effective in changing people's attitudes and health behaviors than standardized messages (Noar, Benac, & Harris, 2007). For example, a person who is not yet convinced of the health benefits of regular exercise should find a message that focuses on the benefits of exercise (i.e., tailored to current concerns) to be more relevant and convincing than a message that focuses on the variety of ways one can exercise (i.e., not tailored to concerns). The reason that tailored messages are more effective than standardized messages is because they are more personally relevant to the recipients, and thus people are more likely to process the message content (Kreuter, Bull, Clark, & Oswald, 1999).

Fear Appeals. Fear appeals are messages that are designed to evoke fear and worry about a health threat as a means to change attitudes and behavior. The rationale underlying fear appeals is that if people are made to feel anxious or worried about a health threat, they will develop a more favorable attitude about taking preventive action and will be more likely to behave accordingly. Thus, fear appeals target the affective component of attitudes. The effectiveness of fear appeals in changing behavior is mixed. Evidence suggests that fear appeals that contain clear and simple recommendation about how to take preventive action are more effective than appeals lacking behavioral recommendations (Witte & Allen, 2000)

Limits of the Influence of Attitudes on Behavior

It seems intuitive that prior to engaging in any health behavior, a sufficiently favorable attitude toward the behavior is needed. For example, it is quite unlikely that a person would get a colonoscopy without the belief that doing so would be beneficial to his or her health. But even when people hold favorable attitudes toward the behavior, they may not engage in it. In other words, attitudes are best thought of as a necessary, but not sufficient, influence on behavior.

There are various reasons why attitudes might not be sufficient to influence behavior change, or why messages aimed at changing attitudes are not effective. First, factors other than attitudes also influence behavior. Drawing from the Theory of Planned Behavior, social norms, perceived behavioral control, and behavioral intentions are other constructs known to influence behavior. In some contexts, people may hold favorable

attitudes toward engaging in a behavior, but because of normative influences or a lack of control over the behavior, they will be unlikely to engage in the behavior. For example, young adult women may hold a favorable attitude about getting the HPV vaccine, but because they believe their parents would not approve (social norms), or they lack the proper insurance coverage (control), they would be unlikely to receive the vaccine.

Second, health communications and messages typically target the cognitive component of people's attitudes (e.g., beliefs that engaging in the behavior will result in beneficial outcomes), often ignoring the affective component (i.e., evaluation of the outcomes of the behavior). For example, women may believe that having a mammogram is a good thing for their health (cognitive component), but also may feel discomfort or embarrassment about the procedure (affective component). To the extent that health messages focus only on the health benefits, and fail to address aspects of engaging in the behavior that are affective in nature, their effectiveness in changing attitudes and behavior may be limited.

Third, attitudes and behavior must be assessed at the same level of specificity in order for a strong relation between the two to exist. For example, a general attitude about overall health behaviors (e.g., "How do you feel about engaging in healthy habits?") is unlikely to have a strong relation with exercising regularly, eating sufficient amounts of fruits and vegetables, seeing a physician for regular medical screenings, *and* engaging in safe sex practices. Instead, attitudes that are as specific as the behaviors (e.g., "How do you feel about exercising regularly?") will have a stronger relation to the behaviors than a more general attitude. Thus, attitudes toward a behavior need to be targeted (e.g., in health communications) at the same level of specificity as the target behavior.

Cross-References

- ▶ Health Beliefs/Health Belief Model
- ▶ Tailored Communications
- ▶ Theory of Planned Behavior
- ▶ Transtheoretical Model of Behavior Change

References and Readings

Ajzen, I., Albarracín, D., & Hornik, R. (Eds.). (2007). *Prediction and change of health behavior: Applying the reasoned action approach*. Mahwah, NJ: Erlbaum.

Ajzen, I., & Fishbein, M. (2005). The influence of attitudes on behavior. In D. Albarracín, B. T. Johnson, & M. P. Zanna (Eds.), *The handbook of attitudes* (pp. 173–221). Mahwah, NJ: Erlbaum.

Chaiken, S., Liberman, A., & Eagly, A. H. (1989). Heuristic and systematic information processing within and beyond the persuasion context. In J. S. Uleman & J. A. Bargh (Eds.), *Unintended thought* (pp. 212–252). New York: Guilford Press.

Champion, V. L., & Skinner, C. S. (2008). The health belief model. In K. Glanz, B. K. Rimer, & K. Viswanath (Eds.), *Health behavior and health education: Theory, research, and practice* (4th ed., pp. 45–65). San Francisco: Jossey-Bass.

Eagly, A. H., & Chaiken, S. (1993). *The psychology of attitudes*. Fort Worth, TX: Harcourt.

Friedman, L. C., Neff, N. E., Webb, J. A., & Latham, C. K. (1998). Age-related differences in mammography use and in breast cancer knowledge, attitudes, and behaviors. *Journal of Cancer Education, 13*, 26–30.

Kreuter, M. W., Bull, F. C., Clark, E. M., & Oswald, D. L. (1999). Understanding how people process health information: A comparison of tailored and nontailored weight-loss materials. *Health Psychology, 18*, 487–494.

Noar, S. M., Benac, C. N., & Harris, M. S. (2007). Does tailoring matter? Meta-analytic review of tailored print health behavior change interventions. *Psychological Bulletin, 133*, 673–693.

Petty, R. E., & Cacioppo, J. T. (1986). *Communication and persuasion: Central and peripheral routes to attitude change*. New York: Springer.

Prochaska, J. O., Redding, C. A., & Evers, K. E. (2008). The transtheoretical model and stages of change. In K. Glanz, B. K. Rimer, & K. Viswanath (Eds.), *Health behavior and health education: Theory, research, and practice* (4th ed., pp. 97–121). San Francisco: Jossey-Bass.

Rothman, A. J., & Salovey, P. (1997). Shaping perceptions to motivate healthy behavior: The role of message framing. *Psychological Bulletin, 121*, 3–19.

Volk, J. E., & Koopman, C. (2001). Factors associated with condom use in Kenya: A test of the health belief model. *The AIDS Education and Prevention Journal, 13*, 495–508.

Weinstein, N. D., Sandman, P. M., & Blalock, S. J. (2008). The precaution adoption process model. In K. Glanz, B. K. Rimer, & K. Viswanath (Eds.), *Health behavior and health education: Theory, research, and practice* (4th ed., pp. 123–147). San Francisco: Jossey-Bass.

Witte, K., & Allen, M. (2000). A meta-analysis of fear appeals: Implications for effective public health campaigns. *Health Education & Behavior, 27*, 591–615.

Attribution Theory

Kevin S. Spink[1] and Darren Nickel[2]
[1]College of Kinesiology, University of Saskatchewan, Saskatoon, SK, Canada
[2]Physical Medicine & Rehabilitation, University of Saskatchewan, Saskatoon, SK, Canada

Synonyms

Causes; Explanations; Failure; Reasons; Success

Definition

Attribution theory is concerned with the conventions that individuals use in attempting to explain their behavior (Weiner, 1986).

Description

According to Bernard Weiner, one of the main contributors in this area, there is no one single attribution theory. Rather, there are a number of attribution-based theories, and attribution is better described as a field of study than as a single encompassing theory (Weiner, 2008). Notwithstanding this clarification, the idea of an attribution-based theory of motivation has been around for years. The premise is simple enough – interpretation of what caused an outcome is proposed to direct future behavior. In terms of motivation, it has been suggested that individuals seek the causes of outcomes because they want to understand and explain those outcomes and predict future behavior. As causes are sought for most important outcomes, especially those where the outcome was not expected (e.g., individuals might ask why they did not get a job or why they only got a B on the test), it should come as no surprise that seeking an explanation for health outcomes is no exception. As an example, individuals who ask why they did not finish all of their cardiac rehabilitation classes and then generate responses such as too busy, lazy, tired, or the instructor was poor are using an attribution-based theory of motivation.

It is important to recognize that one's explanations are perceptions and may or may not capture the actual cause. In the previous example, one's failure to attend all the cardiac classes may have actually been caused by poor time management skills (too busy), but the individual may attribute it to laziness. An individual's perceptions of what caused an outcome can influence expectations for future behavior, emotions, persistence, and ultimately future behavior. Using this example, it might be assumed that the individual who attributed not finishing all of the classes to the cause of being lazy, while feeling some shame, would likely put little effort into being active in the future because the perception might be that laziness is a character trait that is not going to change! On the other hand, an explanation that time management skills were not effective provides some hope as these skills could be improved, which could translate into actually being more active. So, it would appear that attributions might matter.

This was certainly the stance adopted by Fritz Heider, who is known as the "father" of attribution. In his 1958 book, *The Psychology of Interpersonal Relations*, Heider laid out his common-sense approach that captured the beliefs of the "person in the street." In his naïve action of analysis, Heider (1958) reasoned that individuals endeavor to structure and control at least part of their actions by understanding the causes of outcomes thereby improving the prediction of future events. In terms of perceived causes of success and failure for an outcome, Heider identified two internal causes (ability and effort) and one external cause (task difficulty). While the contributions of a number of other researchers helped to deliver the idea of attributions into main stream social psychology, arguably, it was the publication of the book *Attribution: Perceiving the causes of behavior* (Jones et al., 1972) that served to solidify the study of attribution as a legitimate form of inquiry that endures today.

One of the editors of that seminal book, Weiner, extended Heider's common-sense approach to postulate that individuals search for the causes of important outcomes because it was

reasoned that the interpretation of the past (perceived causes of past events) determined what will be done in the future. Weiner (2010) suggested that individuals often use four factors to explain outcomes – ability, effort, task difficulty, and luck. For instance, one's failure to resist eating that very tasty, but calorie-rich, bowl of chocolate ice cream while on a diet could be ascribed to a lack of willpower (ability as a type of personal trait), not trying hard enough to resist (lack of effort), the appeal of the ice cream (task too difficult), or the fact that it was served during a state of hunger (bad luck), or some combination of these causes.

While identifying causes for outcomes is important, Weiner (1986) argued that the properties underlying the specific causes may be of greater significance, as they are believed to influence emotions, future expectations, and motivation. Although Weiner (2010) suggested the possibility of other causal properties, his attribution-based theory affords the classification of causal ascriptions along three property dimensions – locus, stability, and controllability. First, a causal locus denotes that we tend to attribute causes to factors either within ourselves or within the environment (i.e., internal or external to ourselves). Second, as some causes are relatively constant while others may be variable, stability of attributions also is important (stable vs. unstable). For example, while ability is typically perceived as stable, effort may fluctuate. Third, while some attributions are under volitional control, others are not (controllable vs. uncontrollable). For example, failure to comply completely with a physician's prescription to lose weight that is ascribed to the cause of low effort may be controllable, whereas failure because of illness may not.

Beyond qualification of attributions along dimensions, one of the main tenets of Weiner's model of attribution theory is that these dimensions lead to predictable psychological consequences (cognitive and affective). While the dimensions of locus of causality and controllability are believed to interact with perceived outcomes in determining affective reactions, the stability dimension is believed to result in cognitive consequences in terms of expectations regarding future outcomes (Weiner, 2010).

In terms of affective consequences, Weiner (1986) makes it clear that both outcomes and attribution dimensions are believed to be important precursors. The important contribution of an attribution-based theory is the assertion that how we explain the outcomes (i.e., attributions), and not just the outcomes themselves, may influence affective experiences. For example, Weiner (2010) notes that feelings of pride and self-esteem following an outcome are expected to be influenced by locus. Increases in pride and self-esteem are expected when a positive outcome is attributed internally (e.g., high aptitude). Also, guilt and shame are believed to be influenced chiefly by the controllability dimension. Guilt is expected when a negative outcome is seen as caused by something personally controllable (e.g., lack of effort), while shame is expected when a negative outcome is caused by a personal attribute about which one can do nothing (e.g., low aptitude). Further, all of these emotional responses are believed to influence future decisions and actions (Weiner, 1986).

In terms of specific predictions for future expectations, it would be posited that one would expect a similar future outcome when an outcome is attributed to a stable cause (e.g., task was too difficult). It is less clear, however, whether expected future outcomes will be similar or not when an outcome is attributed to an unstable cause that could change (e.g., lack of effort). Future expectations, in turn, are believed to play an important role in determining intentions and future behavior (Weiner, 1986).

Attributions have certainly played out in the health area. For instance, an examination of the wellness end of health behavior has revealed that using attributions that are stable (e.g., an explanation that one is good at managing time around exercise) to explain typical exercise levels (health-enhancing behavior) predicted intention to maintain those levels during a forthcoming time period (Spink & Nickel, 2010). Those who felt that the causes of their typical levels of moderate and mild exercise were stable also intended to maintain those levels throughout a subsequent period. In addition to relationships with health-enhancing behaviors, attributions also have been

associated with self-efficacy, which has been identified as an important cognition that has been consistently associated with an array of health behaviors. Self-efficacy is defined as beliefs in one's capabilities to successfully execute a course of action (Bandura, 1997). As one example of the attribution/self-efficacy relationship, it has been demonstrated that the interpretation of one's past activity behavior (as reflected in attribution dimensions) improved the prediction of self-efficacy over and above that predicted by past behavior only (Nickel & Spink, 2010).

Attributions also appear to be associated with the illness end of the health continuum. In a meta-analysis examining psychological adjustment to disease, Roesch and Weiner (2001) reported that, for the most part, individuals who explained their disease as being caused by more internal, unstable, and controllable causes (e.g., overweight) also reported that they used more adaptive forms of coping (e.g., coping self-efficacy) and were ultimately more well adjusted than those who used more external, stable, and uncontrollable causes (e.g., exposure from the environment). In contrast, those who experienced negative psychological adjustment tended to use stable and uncontrollable attributions (e.g., it is in the genes) to explain their illness. These patterns would appear consistent with Weiner's (2010) contention that attributing failure to stable causes impedes hope and motivation, whereas ascribing failure to unstable causes creates hope and facilitates motivation.

The fact that attributions are perceptions suggests that interventions could be designed that alter unhealthy behavior by changing maladaptive attributions. One study examining the activity of older adults underscores this point (Sarkisian, Prochaska, Davis, & Weiner, 2009). Consistent with other research, it was assumed that older adults would report the cause of a failure to be active as "old age." As theory would suggest that stable and uncontrollable attributions for failure are especially detrimental to motivation, these individuals were retrained to attribute failure to be active to controllable factors. After the attribution retraining, it was revealed that the older adults increased their walking by over four kilometers per week. Results such as these are encouraging and suggest that attribution retraining programs may provide an effective method to improve health-type behavior when perceived causes for failure are maladaptive and, at the very least, deserve future research attention.

Cross-References

▶ Self-Efficacy

References and Readings

Bandura, A. (1997). *Self-efficacy: The exercise of control*. New York: Freeman.

Heider, F. (1958). *The psychology of interpersonal relations*. New York: Wiley.

Jones, E. E., Kanouse, D. E., Kelley, H. H., Nisbett, R. E., Valins, S., & Weiner, B. (Eds.). (1972). *Attribution: Perceiving the causes of behavior*. Morristown, NJ: General Learning Press.

Nickel, D., & Spink, K. S. (2010). Attributions and self-regulatory efficacy for health-related physical activity. *Journal of Health Psychology, 15*, 53–63.

Roesch, S. C., & Weiner, B. (2001). A meta-analytic review of coping with illness: Do causal attributions matter? *Journal of Psychosomatic Research, 50*, 205–219.

Sarkisian, C. A., Prohaska, T. R., Davis, C., & Weiner, B. (2009). Pilot test of an attributional retraining intervention to raise walking levels in sedentary older adults. *Journal of the American Geriatric Society, 55*, 1842–1846.

Spink, K. S., & Nickel, D. (2010). Self-regulatory efficacy as a mediator between attributions and intention for health-related physical activity. *Journal of Health Psychology, 15*, 75–84.

Weiner, B. (1986). *An attributional theory of motivation and emotion*. New York: Springer.

Weiner, B. (2008). Reflections on the history of attribution theory and research: People, personalities, publications, problems. *Social Psychology, 39*, 151–156.

Weiner, B. (2010). The development of an attribution-based theory of motivation: A history of ideas. *Educational Psychologist, 45*, 28–36.

Attributional Style

▶ Locus of Control

Attributional Style Questionnaire (ASQ)

► Optimism and Pessimism: Measurement

Autism Spectrum Disorders

► Developmental Disabilities

Autoimmune Diabetes

► Insulin-Dependent Diabetes Mellitus (IDDM)

Autoimmune Diabetes Mellitus

► Type 1 Diabetes Mellitus

Autonomic

► Heart Rate Variability

Autonomic Activation

Michael Richter[1] and Rex A. Wright[2]
[1]Department of Psychology, University of Geneva, Geneva, Switzerland
[2]College of Arts and Sciences, Department of Psychology, University of North Texas, Denton, TX, USA

Synonyms

Autonomic arousal; Autonomic reactivity

Definition

Autonomic activation refers to an increase in the activity of the autonomic nervous system, the physical system responsible for nonconsciously maintaining bodily homeostasis and coordinating bodily responses. It is assessed by comparing autonomic values obtained during a test period to those obtained during a rest or baseline period. Baseline measures commonly are taken shortly before test periods. However, they can be taken well in advance of test periods or after them. Autonomic activation can pertain to neuronal activity or activity of visceral structures affected by it, such as the ones involved in circulation, respiration, and digestion. The distinction between neuronal activation and visceral structure activation is not trivial given that an increase in the activity of a visceral structure may be caused by a decrease in neuronal activity. For instance, increases in the frequency of the heart beat – which are often interpreted as signals of autonomic activation – can be due to reduced activity in the parasympathetic branch of the autonomic nervous system.

References and Readings

Berne, R. M., Levy, M. N., Koeppen, B. M., & Stanton, B. A. (2004). *Physiology* (5th ed.). St. Louis, MO: Mosby.
Cacioppo, J. T., & Tassinary, L. G. (1990). *Principles of psychophysiology: Physical, social, and inferential elements*. New York: Cambridge University Press.
Cacioppo, J. T., Tassinary, L. G., & Berntson, G. G. (2000). *Handbook of psychophysiology* (2nd ed.). New York: Cambridge University Press.
Ganong, W. F. (2005). *Review of medical physiology* (22nd ed.). New York: McGraw-Hill.
Levick, J. R. (2009). *An introduction to cardiovascular physiology* (5th ed.). London, UK: Hodder.

Autonomic Arousal

► Autonomic Activation

Autonomic Balance

Julian F. Thayer
Department of Psychology, The Ohio State University, Columbus, OH, USA

Synonyms

Inflammation; Parasympathetic; Sympathetic

Definition

There is growing evidence for the role of the autonomic nervous system (ANS) in a wide range of somatic and mental diseases. The ANS is generally conceived to have two major branches: the sympathetic system, associated with energy mobilization, and the parasympathetic system, associated with vegetative and restorative functions. Normally, the activity of these branches is in dynamic balance. When this changes into a static imbalance, for example, under environmental pressures, the organism becomes vulnerable to pathology.

Resting heart rate (HR), by virtue of its dominant control via parasympathetic mechanisms (Levy, 1997; Uijtdehagge & Thayer, 2000), can be used as a rough indicator of autonomic balance, and several large studies have shown a largely linear, positive dose-response relationship between resting HR and all-cause mortality (see Habib, 1999, for a review). This association was independent of gender and ethnicity, and showed a threefold increase in mortality in persons with resting HR over 90 beats per minute (bpm) compared to those with resting HRs of less than 60 bpm.

Brook and Julius (2000) have detailed how autonomic imbalance in the sympathetic direction is associated with a range of metabolic, hemodynamic, trophic, and rheologic abnormalities that contribute to elevated cardiac morbidity and mortality. Autonomic balance has been shown to be associated with diabetes mellitus, and decreased HRV has been shown to precede evidence of disease provided by standard clinical tests (Ziegler, Laude, Akila, & Elgwhozi, 2001). In addition, autonomic balance and decreased parasympathetic activity is also associated with immune dysfunction and inflammation, which have been implicated in a wide range of conditions including cardiovascular disease, diabetes, osteoporosis, arthritis, Alzheimer's disease, periodontal disease, and certain types of cancers as well as declines in muscle strength and increased frailty and disability (Ershler and Keller, 2000; Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002). The common mechanism seems to involve excess pro-inflammatory cytokines such as interleukin 1 and 6 and tumor necrosis factor. Importantly, increased parasympathetic activity and acetylcholine (the primary parasympathetic neurotransmitter) have been shown to attenuate release of these pro-inflammatory cytokines, and sympathetic hyperactivity is associated with their increased production (Tracey, 2002; Thayer & Sternberg, 2010). Thus, autonomic imbalance may be a final common pathway to increased morbidity and mortality from a host of conditions and diseases.

Although the idea is not new (Sternberg, 1997), several recent reviews have provided strong evidence linking negative affective states and dispositions to disease and ill health (Friedman & Thayer, 1998; Kiecolt-Glaser et al., 2002; Thayer, Yamamoto, and Brosschot, 2010). All of these reviews implicate altered ANS function and decreased parasympathetic activity as a possible mediator in this link. An additional pathway between psychosocial stressors and ill health is an indirect one, in which psychosocial factors lead to poor lifestyle choices, including a lack of physical activity and the abuse of tobacco, alcohol, and drugs. Both sedentary lifestyle and substance abuse are associated with autonomic imbalance and decreased parasympathetic activity (Ingjaldsson, Laberg, & Thayer, 2003; Thayer & Lane, 2007; Thayer et al., 2010). In fact, the therapeutic effectiveness of smoking cessation, reduced alcohol consumption, and increased physical

activity rest in part on their ability to restore autonomic balance and increase parasympathetic activity.

Cross-References

► Heart Rate Variability

References and Readings

Brook, R. D., & Julius, S. (2000). Autonomic imbalance, hypertension, and cardiovascular risk. *American Journal of Hypertension, 13*, 112S–122S.

Ershler, W., & Keller, E. (2000). Age-associated increased interleukin-6 gene expression, late life diseases, and frailty. *Annual Review of Medicine, 51*, 245–270.

Friedman, B. H., & Thayer, J. F. (1998). Autonomic balance revisited: Panic anxiety and heart rate variability. *Journal of Psychosomatic Research, 44*, 133–151.

Habib, G. B. (1999). Reappraisal of heart rate as a risk factor in the general population. *European Heart Journal Supplements, 1*(H), H2–H10.

Ingjaldsson, J. T., Laberg, J. C., & Thayer, J. F. (2003). Reduced heart rate variability in chronic alcohol abuse: Relationship with negative mood, chronic thought suppression, and compulsive drinking. *Biological Psychiatry, 54*, 1427–1436.

Kiecolt-Glaser, J. K., McGuire, L., Robles, T. F., & Glaser, R. (2002). Emotions, morbidity, and mortality: New perspectives from psychoneuroimmunology. *Annual Review of Psychology, 53*, 83–107.

Levy, M. N. (1997). Neural control of cardiac function. *Baillière's Clinical Neurology, 6*, 227–244.

Sternberg, E. M. (1997). Emotions and disease: From balance of humors to balance of molecules. *Nature Medicine, 3*, 264–267.

Thayer, J. F., & Lane, R. D. (2007). The role of vagal function in the risk for cardiovascular disease and mortality. *Biological Psychology, 74*, 224–242.

Thayer, J. F., & Sternberg, E. M. (2010). Neural aspects of immunomodulation: Focus on the vagus nerve. *Brain, Behavior, and Immunity, 24*, 1223–1228.

Thayer, J. F., Yamamoto, S. S., & Brosschot, J. F. (2010). The relationship of autonomic imbalance, heart rate variability and cardiovascular disease risk factors. *International Journal of Cardiology, 141*, 122–131.

Tracey, K. J. (2002). The inflammatory reflex. *Nature, 420*, 853–859.

Uijtdehaage, S. B. H., & Thayer, J. F. (2000). Accentuated antagonism in the control of human heart rate. *Clinical Autonomic Research, 10*, 107–110.

Ziegler, D., Laude, D., Akila, F., & Elghozi, J. L. (2001). Time and frequency domain estimation of early diabetic cardiovascular autonomic neuropathy. *Clinical Autonomic Research, 11*(6), 369–376.

Autonomic Nervous System (ANS)

Michael Richter[1] and Rex A. Wright[2]
[1]Department of Psychology, University of Geneva, Geneva, Switzerland
[2]College of Arts and Sciences, Department of Psychology, University of North Texas, Denton, TX, USA

Synonyms

Vegetative nervous system; Visceral nervous system

Definition

The autonomic nervous system (ANS) is a part of the efferent (i.e., outgoing) division of the peripheral nervous system. It adapts the organism to internal and external changes, maintaining bodily homeostasis and coordinating bodily responses.

Description

The autonomic nervous system (also known as the visceral nervous system and vegetative nervous system) combines with the somatic nervous system to form the efferent (i.e., outgoing) division of the peripheral nervous system. It innervates glands, the heart, and smooth muscles of all visceral structures and adapts the organism to internal and external changes by regulating a wide range of bodily functions such as blood circulation, body temperature, respiration, and

digestion. The basic tasks of the autonomic nervous system are to maintain bodily homeostasis and coordinate bodily responses. In contrast to regulatory processes of the somatic nervous system, regulatory processes of the autonomic nervous system do not require conscious or voluntary control.

Anatomical Structure

The autonomic nervous system is comprised of two main branches or subsystems, (1) the sympathetic nervous system and (2) the parasympathetic nervous system. A third nervous system – the enteric system – is considered by some physiologists to be a part of the autonomic nervous system and by others to be independent of that system. The enteric nervous system consists of two large nerve networks located in the walls of the digestive tract, identified as the submucosal plexus and the myenteric plexus. It innervates the smooth muscle cells of the digestive tract as well as exocrine and endocrine cells, controlling local activity within the digestive tract (e.g., secretion of digestive juices and digestive motility). The enteric system can act autonomously, but also in response to sympathetic and parasympathetic input.

Basic functional units of the sympathetic and the parasympathetic nervous systems are preganglionic and postganglionic neurons. Preganglionic neurons have cell bodies in the spinal cord or brainstem and axons that extend to cell bodies of postganglionic neurons. Postganglionic neurons have cell bodies that are clustered in so-called ganglia and axons that innervate target visceral structures. Notably, preganglionic neurons typically synapse with more than one postganglionic neuron. Similarly, postganglionic neurons typically synapse with visceral structures in multiple locations, allowing pervasive structural influence. An anatomical exception to the above is seen in the adrenal medulla. Although the adrenal medulla is a part of the adrenal gland, its cells are modified postganglionic neurons directly innervated by preganglionic neurons.

The major anatomical difference between the sympathetic nervous system and the parasympathetic nervous system is the location of neuronal cell bodies. Sympathetic preganglionic neurons are located in the thoracic and upper lumbar segment of the spinal cord, whereas parasympathetic preganglionic neurons lie in the brainstem and the sacral spinal cord. Postganglionic neurons of the sympathetic system are located either in one of the sympathetic ganglion chains (sympathetic trunk, also called paravertebral ganglia) along the spinal cord or in the prevertebral ganglia in front of the spinal cord. Parasympathetic postganglionic neurons are located either in terminal ganglia that lie near the target organ or directly in the organ wall. Given the difference in the position of the ganglia, sympathetic preganglionic fibers are usually shorter than parasympathetic preganglionic fibers and sympathetic postganglionic fibers are usually longer than parasympathetic postganglionic fibers.

Sympathetic and Parasympathetic Innervations of Visceral Structures and Functioning

Most visceral structures have both sympathetic and parasympathetic innervations. Exceptions are the skin, most blood vessels and most sweat glands, which are only sympathetically innervated. In visceral structures with dual innervations, the sympathetic and parasympathetic systems work together to regulate bodily function. It is common for the sympathetic and parasympathetic systems to exert complementary influences on visceral structures, with sympathetic arousal leading to adjustments suitable for high activity ("fight and flight") and parasympathetic arousal leading to adjustments suitable for low activity and bodily restoration ("rest and digest"). Examples of high activity adjustments are constriction of blood vessels in the gastrointestinal (GI) tract, dilation of blood vessels in the skeletal muscles and lungs, and improved heart rate and contraction force. Examples of low activity and restorative adjustments are the reverse: dilation of blood vessels in the GI tract, constriction of blood vessels in the

skeletal muscles and lungs, and decreased heart rate and contraction force. However, there are multiple exceptions to this complementary influence rule. Consider, for example, sympathetic and parasympathetic influence on salivation. Both sympathetic arousal and parasympathetic arousal increase salivary flow, although to different degrees and yielding different compositions of saliva. It also is noteworthy that the systems may exert an activating or an inhibiting effect depending on the innervated structure. For instance, increased sympathetic arousal increases heart rate but decreases motility in the digestive tract. Parasympathetic activity activates digestion, but slows heart rate.

In working together, the sympathetic and parasympathetic nervous systems typically do not function in an all-or-none fashion, but rather activate to different degrees. Depending on the affected visceral structure and situation, one of the two systems may be more active than the other. For instance, at rest heart rate is mainly under parasympathetic nervous system control, subject to a negligible sympathetic influence. By contrast, at high levels of physical activity, it is mainly under sympathetic nervous system control. Shifts in sympathetic and parasympathetic influence can occur locally within a single visceral structure (e.g., the eye) or across visceral structures. Shifts in local influence occur to meet highly specialized demands (e.g., the change in pupil size to adapt to a change in ambient light). Global shifts adapt the body to large-scale environmental changes (e.g., the appearance of a substantial physical threat).

Neurotransmitters and Receptors

In addition to differing anatomically, the sympathetic and the parasympathetic nervous systems differ with respect to their neurotransmitters and the receptors that mediate their effects on visceral structures. The most important receptors are (1) cholinergic receptors stimulated by the neurotransmitter acetylcholine and (2) adrenergic receptors stimulated by the neurotransmitters norepinephrine and epinephrine. Acetylcholine is the neurotransmitter between all pre- and postganglionic neurons as well as between parasympathetic postganglionic neurons and visceral structures. Acetylcholine is also the neurotransmitter of the sympathetic postganglionic neurons that innervate the eccrine sweat glands and of sympathetic postganglionic neurons that innervate skeletal muscle vessels and cause vasodilation. All other sympathetic postganglionic neurons release norepinephrine. The adrenal medulla constitutes an exception. Despite the fact that cells of the adrenal medulla are modified sympathetic postganglionic cells, they release epinephrine and norepinephrine directly into the blood stream. It is noteworthy that acetylcholine and norepinephrine are the major neurotransmitters of the sympathetic and the parasympathetic nervous system, but cotransmitters like vasoactive intestinal polypeptide (VIP), adenosine triphosphate (ATP), or neuropeptide Y are frequent.

Central Control

An afferent (i.e., incoming) nervous system conveys information about the current state of the organism to structures in the central nervous system. These structures exert a regulatory impact by way of autonomic efferents. Central nervous system structures that control autonomic nervous system activity vary depending on afferent information that is received. The hypothalamus plays a central role in regulating activity of the autonomic nervous system by integrating autonomic, somatic, and endocrine responses that accompany different organism states. This central nervous system structure receives afferent input from visceral sensory neurons and is subject to the modulating impact of other central nervous system structures such as the amygdala and insular cortex. It influences autonomic centers located in the brainstem and can directly affect preganglionic neurons.

Summary

The autonomic nervous system is a part of the efferent (outgoing) division of the peripheral

nervous system. It innervates glands, the heart, and smooth muscles of all visceral structures and adapts the organism to internal and external changes, maintaining bodily homeostasis and coordinating bodily responses without requiring conscious or voluntary control. Two branches or subsystems of the autonomic nervous system are the sympathetic nervous system and the parasympathetic nervous system. These commonly – but not always – work in a complementary fashion to regulate bodily function, with sympathetic arousal leading to adjustments suitable for high activity ("fight and flight") and parasympathetic arousal leading to adjustments suitable for low activity and bodily restoration ("rest and digest"). In working together, the sympathetic and parasympathetic branches do not function in an all-or-none fashion, but rather activate to different degrees. Shifts in sympathetic and parasympathetic influence can occur locally within a single visceral structure or across visceral structures, with local shifts occurring to meet highly specialized demands and global shifts adapting the body to large-scale environmental changes. The sympathetic and the parasympathetic nervous systems differ anatomically and with respect to their neurotransmitters and the receptors that mediate their effects on visceral structures. Autonomic control is maintained by structures in the central nervous system that receive visceral information by way of an afferent (incoming) nervous system. A key central nervous system structure is the hypothalamus, which integrates autonomic, somatic, and endocrine responses that accompany different organism states.

Cross-References

- ▶ Acetylcholine
- ▶ Adrenaline
- ▶ Autonomic Activation
- ▶ Autonomic Balance
- ▶ Autonomic Nervous System (ANS)
- ▶ Epinephrine
- ▶ Parasympathetic Nervous System (PNS)
- ▶ Sympathetic Nervous System (SNS)

References and Readings

Berne, R. M., Levy, M. N., Koeppen, B. M., & Stanton, B. A. (2004). *Physiology* (5th ed.). St. Louis, MO: Mosby.

Cacioppo, J. T., & Tassinary, L. G. (1990). *Principles of psychophysiology: Physical, social, and inferential elements*. New York: Cambridge University Press.

Cacioppo, J. T., Tassinary, L. G., & Berntson, G. G. (2000). *Handbook of psychophysiology* (2nd ed.). New York: Cambridge University Press.

Ganong, W. F. (2005). *Review of medical physiology* (22nd ed.). New York: McGraw-Hill.

Levick, J. R. (2009). *An introduction to cardiovascular physiology* (5th ed.). London, UK: Hodder.

Autonomic Reactivity

▶ Autonomic Activation

AVE

▶ Abstinence Violation Effect

Average

▶ Mean (Average)

Avoidance

Deborah J. Wiebe
Division of Psychology, Department of Psychiatry, Southwestern Medical Center, University of Texas, Dallas, TX, USA

Synonyms

Avoidance coping; Avoidance goals; Avoidance motivation

Definition

Approach and avoidance represent two fundamental organizing principles underlying human behavior. Avoidance involves movement away from threats or negative outcomes, while approach involves movement toward rewards or positive outcomes. These avoidance and approach tendencies can be expressed in cognitive, affective, and behavioral domains. In the context of behavioral medicine, avoidance has been commonly examined in the context of avoidance coping and through avoidance-based self-regulation activities. Generally speaking, avoidance coping and self-regulation are associated with poorer psychological and physical health outcomes than are approach-based activities, although there are contexts when avoidance may be relatively adaptive (e.g., in the early stages of coping with stress; when there is little that can be done to alter the stress).

Description

Introduction

Human behavior is widely believed to be regulated by an approach system that orients appetitive behavior toward potential rewards and an avoidance system that orients behavior away from potential threats and punishment. These two systems have distinct underlying neurobiological substrates, where approach behaviors characterized by heightened sensitivity to reward are organized by the behavioral activation system (BAS) and avoidance behaviors characterized by heightened sensitivity to threat are organized by the behavioral inhibition system (BIS) (Gray, 1990). Avoidance can thus be defined as a system that regulates behavior around escaping or distancing oneself away from threats or punishments. Within behavioral medicine, avoidance has been most commonly examined in the context of avoidance coping and avoidance motivation.

Avoidance Coping

Coping refers to behavioral, cognitive, and affective strategies one engages in to reduce the experience and impact of stressful events. Avoidance coping represents one of several broad dimensions of coping strategies characterized by efforts to escape or distance oneself from stressful events and associated feelings of distress. This contrasts with approach coping strategies which are characterized by efforts to engage with and directly alter the demands of the stressor. Avoidance coping strategies can take different forms. Folkman and Moskowitz (2004) distinguished between distancing strategies where one recognizes the stressor but makes efforts to put it out of mind and strategies to escape the stressor or its implications such as using alcohol or other substances. Avoidance coping shares features with emotion-focused coping strategies, which involve efforts to minimize the emotional consequences of stressful events rather than to alter the stressor directly. However, the recent coping literature suggests that emotion-focused coping strategies can include both emotion-approach and emotion-avoidance strategies.

The use of avoidance coping is likely to reflect both individual and contextual factors. Individuals who have personality traits that are linked to the behavioral inhibition system (e.g., neuroticism) have been found to be more likely to utilize avoidance coping strategies than those who have traits that are linked to the behavioral activation system (e.g., extraversion). One's personal and environmental resources also influence the likelihood of using avoidance coping strategies. Holahan and Moos (1987), for example, found that avoidance coping was more common among individuals who had fewer personal (e.g., self-efficacy beliefs; internal locus of control), economic, and social resources. Finally, avoidance coping is partially determined by the demands of the stressor. Most people utilize multiple types of coping strategies in the course of dealing with the changing demands of stressful events. Avoidance coping is more common when one is dealing with situations that cannot be actively altered. For example, Lazarus and Folkman examined the coping strategies

of students before and after an important exam. Most students utilized more active approach-based coping strategies before the exam when their efforts would be effective at enhancing achievement. After the exam, however, when students could do little more than simply wait for their results, avoidance strategies increased.

As a general rule, avoidance coping is less adaptive than approach coping, as evidenced by its associations with poorer subjective well-being, psychological adjustment, and physical health. These associations may occur for several reasons. First, avoidance coping by definition does not alter the stressful situation. Thus, to the extent that one's coping efforts may be beneficial to health by minimizing the intensity, duration, or recurrence of a stressful event, avoidance coping may be harmful because it increases exposure the health-damaging consequences of stress. Second, avoidance coping requires effort and may consume resources that are not then available for other more adaptive coping strategies. Finally, some avoidance strategies may be damaging because they involve unhealthy behaviors that have fairly direct adverse health effects. For example, escape-avoidance strategies that involve trying to minimize distress through alcohol or substance use and poor dietary practices may have direct adverse health consequences.

Although avoidance coping is generally related to poorer health outcomes, there are some exceptions when avoidance coping may not be harmful and may even be adaptive. Avoidance strategies that occur in the early stages of dealing with a stressful event appear to be adaptive (Suls & Fletcher, 1985), potentially because one needs time to develop resources and skills to manage effectively. Similarly, avoidance strategies such as distancing may be adaptive when dealing with uncontrollable stressful events such as those associated with loss and bereavement (Carver, 2006; Folkman & Moskowitz, 2004).

Avoidance Motivation, Goals, and Self-Regulation

Self-regulation models argue that human behavior is motivated by a set of hierarchically arranged goals. The highest and most abstract goals reveal self-defining principles (e.g., to be of service to others), while the lowest and most concrete goals reflect behaviors that can be taken to reach the higher level goal (e.g., donate time and money to the food bank). Although multiple goals are likely to be active simultaneously, and may even conflict with each other, behavior is organized around movement toward accomplishing salient goals. Through positive and negative feedback loops analogous to a thermostat or a homeostatic process, individuals are believed to feel distress when their movement toward goal achievement is thwarted. From this perspective, stress can be conceptualized as the disruption of one's goal pursuits.

There are individual differences in the extent to which one has a predominantly approach or avoidance motivational orientation, and these differences have important implications for motivating health behavior and health behavior change. A given health behavior can be framed as an approach goal by focusing on the positive outcomes the behavior may achieve (e.g., flossing my teeth will result in healthy gums and fresh breath) or an avoidance goal by focusing on the negative outcomes the behavior may avoid (e.g., flossing my teeth will prevent gum disease). Mann and colleagues (Mann, Sherman, & Updegraff, 2004; Sherman, Mann, & Updegraff, 2006) demonstrated that health behavior messages were more likely to motivate behavior change if the frame of the message was congruent with an individual's predominant motivation. That is, individuals with a predominant avoidance orientation were more likely to change their behavior when the message frame emphasized an avoidance goal.

Cross-References

- ▶ Behavioral Inhibition
- ▶ Coping
- ▶ Coping Styles
- ▶ Escape-Avoidance Coping
- ▶ Negative Thoughts
- ▶ Passive Coping Strategies
- ▶ Self-regulation Model

References and Readings

Carver, C. S. (2006). Approach, avoidance, and the self-regulation of affect and action. *Motivation and Emotion, 30*, 105–110.

Folkman, S., & Moskowitz, J. T. (2004). Coping: Pitfalls and promise. *Annual Review of Psychology, 55*, 745–774.

Gray, J. A. (1990). Brain systems that mediate both emotion and cognition. *Cognition & Emotion, 4*, 269–288.

Holahan, C. J., & Moos, R. H. (1987). Personal and contextual determinants of coping strategies. *Journal of Personality and Social Psychology*, *52*, 946–955.

Mann, T., Sherman, D., & Updegraff, J. (2004). Dispositional motivations and message framing: A test of the congruency hypothesis in college students. *Health Psychology*, *23*, 330–334.

Penley, J. A., Tomaka, J., & Wiebe, J. S. (2002). The association of coping to physical and psychological health outcomes: A meta-analytic review. *Journal of Behavioral Medicine, 25*, 551–603.

Sherman, D. K., Mann, T., & Updegraff, J. A. (2006). Approach/avoidance motivation, message framing, and health behavior: Understanding the congruency effect. *Motivation and Emotion, 30*, 165–169.

Suls, J., & Fletcher, B. (1985). The relative efficacy of avoidant and nonavoidant coping strategies: A meta-analysis. *Health Psychology, 4*, 249–288.

Avoidance Coping

► Avoidance

Avoidance Goals

► Avoidance

Avoidance Motivation

► Avoidance

Avoidant Coping

► Defensiveness

B

Back Pain

Timothy H. Wideman and Michael J. L. Sullivan
Department of Psychology, McGill University, Montreal, QC, Canada

Synonyms

Backache; Dorsalgia; Lumbago

Definition

Pain located between the base of the neck and the gluteal folds that can also be associated with radiating pain in the lower extremities.

Description

Back pain is one of the most prevalent and costly conditions in the industrialized world. An estimated 80% of individuals will experience back pain at some point in their life. The vast majority of back pain episodes have an unknown etiology and are self-limiting. Approximately 80–90% of cases resolve within the first 6 weeks of onset. The relatively small percentage of individuals who develop chronic back pain (symptoms lasting longer than 3 months), however, account for the majority of the disability expenditures that are associated with this condition.

Criteria used to classify back pain are numerous and, in certain instances, divergent. For example, the classification of back pain has been based on the duration and location of symptoms, the underlying spinal pathology, the presence of spinal instability, and the quality of spinal movements. Current biopsychosocial models emphasize the importance of classifying back pain based on the presence of risk factors for severe illness or prolonged disability.

A detailed history and physical exam are used to screen for the presence of *Red Flags* and *Yellow Flags* in individuals presenting with acute back pain (less than 6 weeks since the onset of symptoms). Red Flags are signs and symptoms that suggest the possibility of serious pathology, such as carcinoma, immunodeficiency, damage to the spinal cord or cauda equina, and inflammatory disorders. Red Flags are identified via the following signs and symptoms: a history of severe trauma, significant weight loss, neurological signs and symptoms, severe worsening of pain, and/or systemic illness. Emergency medical attention is required in the relatively rare incidence in which a patient with back pain presents with a Red Flag. In the absence of Red Flags, screening for Yellow Flags is indicated. Yellow Flags are psychosocial signs and symptoms that suggest an elevated risk for prolonged pain and disability. Yellow Flags include: depressed mood, belief that physical activity and pain are damaging, job dissatisfaction, and a history of back pain or work disability. The presence of Yellow Flags calls for

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

risk-factor-targeted interventions that aim to prevent long-term disability.

In the absence of Red or Yellow Flags, clinical practice guidelines recommend minimal treatment for acute back pain. Recommendations emphasize the importance of reassuring patients that back pain is not commonly associated with serious pathology, that back pain typically resolves in the first 6 weeks following symptom onset, and that a prompt return to regular physical activities is indicated. Simple analgesic medication, such as acetaminophen (paracetamol) or Nonsteroidal Anti-Inflammatory Drugs (NSAIDs), and a brief course of manual therapy have been shown to be effective for symptom management. Imaging investigations are not warranted in these instances.

Cross-References

- ▶ Chronic Pain
- ▶ Pain Management/Control
- ▶ Pain, Psychosocial Aspects

References and Readings

Airaksinen, O., Brox, J. I., Cedraschi, C., Hildebrandt, J., Klaber-Moffett, J., Kovacs, F., Mannion, A. F., Reis, S., Staal, J. B., Ursin, H., Zanoli, G. (2006, March 1). Chapter 4: European guidelines for the management of chronic nonspecific low back pain. European spine journal: Official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society, *15*(Suppl 2): S192–S300.

The New Zealand Acute Low Back Pain Guide. Prepared by the New Zealand Accident Compensation Corporation (ACC), and endorsed by the New Zealand Guidelines group; available online at www.acc.co.nz

Waddell, G. (2004). *The back pain revolution* (2nd ed.). London: Churchill Livingstone.

Backache

- ▶ Back Pain

Bariatric Surgery

Shuji Inada
Department of Stress Science and Psychosomatic Medicine, Graduate School of Medicine, The University of Tokyo, Tokyo, Japan

Synonyms

Weight loss surgery

Definition

Bariatric surgery is a surgery for weight loss.

Since original bariatric surgery, jejunocolic bypass, was introduced in 1954, safer procedures have been developed.

Weight loss is thought to be achieved by reducing gastric volume and/or by causing intestinal malabsorption. It is also implied that changes in the entero-endocrine axis might affect body weight (Tadross & le Roux, 2009). Only bariatric surgery can produce durable long-term weight loss (Colquitt, Picot, Loveman, & Glegg, 2009).

Procedures commonly performed are as follows (Bult, van Dalen, & Muller, 2008; Jaunoo & Southall, 2010):

1. Adjustable gastric banding. This procedure involves placing a silicon band horizontally around the proximal part of stomach. The band can be inflated via a subcutaneous port and be adjusted to an extent of restriction without an operation.
2. Roux-en-Y gastric bypass. This procedure includes division of the upper stomach to the small pouch and connection of intestine to the pouch. This procedure achieves both reduction of gastric volume and malabsorption.
3. Biliopancreatic diversion (with duodenal switching). This procedure is developed as a malabsorptive procedure. Biliopancreatic duct and duodenum are removed from the tract and anastomosed to a more distal part of the ileum.

4. Sleeve gastrectomy. Though this procedure has been performed as the first stage of biliopancreatic diversion with duodenal switching in high-risk patients, it has been proved to achieve significant weight loss in some patients. If patients fail to lose weight with this procedure, the last part of biliopancreatic diversion is performed.

The mean percentage of excess weight loss was 61.2% for all patients undergoing bariatric surgery (Buchwald et al., 2004). Mortality at 30 or less days after bariatric surgery was 0.1% for purely restrictive procedure, 0.5% for gastric bypass procedures, and 1.1% for biliopancreatic diversion (Buchwald et al.).

Major complications of bariatric surgery are vomiting and leakage. Malabsorptive procedures may cause anemia and protein malnutrition.

Bariatric surgery is recommended for obese patients with a BMI over 40 kg/m^2 or with a BMI between 35 and 39.9 and a serious obesity-related health problem such as type 2 diabetes, coronary heart disease, or severe sleep apnea (National Heart Lung and Blood Institute, 2000; NICE clinical guideline 43, 2006).

Preoperative comprehensive assessments including psychosocial assessments are necessary for a risk-benefit analysis. While there are no uniform guidelines for psychosocial assessments, psychosocial assessments are usually focused on knowledge about bariatric surgery, weight and diet history, social status, and psychiatric comorbidity such as depression, eating disorders, and personality disorders (Bauchowitz et al., 2005; Wadden & Sarwer, 2006).

Contraindications to bariatric surgery include poor myocardial reserve, significant chronic obstructive airways disease or respiratory dysfunction, noncompliance with medical treatment, and psychological disorders of a significant degree (Jaunoo & Southall, 2010; Walfish, Vance, & Fabricatore, 2007).

Cross-References

► Obesity: Prevention and Treatment

References and Readings

Bauchowitz, A. U., Gonder-Frederick, L. A., Olbrisch, M., Azarbad, L., Ryee, M., Woodson, M., et al. (2005). Psychosocial evaluation of bariatric surgery candidates: A survey of present practices. *Psychosomatic Medicine, 67*, 825–832.

Buchwald, H., Avidor, Y., Braunwald, E., Jensen, M. D., Pories, W., Fahrbach, K., et al. (2004). Bariatric surgery a systematic review and meta-analysis. *Journal of the American Medical Association, 292*, 1724–1737.

Bult, M. J. F., van Dalen, T., & Muller, A. F. (2008). Surgical treatment of obesity. *European Journal of Endocrinology, 158*, 135–145.

Colquitt, J. L., Picot, J., Loveman, E., & Glegg, A. J. (2009). Surgery for obesity. *Cochrane Database of Systematic Reviews* , Issue 2

Jaunoo, S. S., & Southall, P. J. (2010). Bariatric surgery. *International Journal of Surgery, 8*, 86–89.

National Heart Lung and Blood Institute. (2000). The practical guide to identification, education and treatment of overweight and obesity in adults. NIH Pub no. 00-4084.

National Institute for Health and Clinical Excellence. (2006). Obesity: The prevention, identification, assessment and management of overweight and obesity in adults and children. NICE clinical guideline 43. http://guidance.nice.org.uk/CG43

Tadross, J. A., & le Roux, C. W. (2009). The mechanisms of weight loss after bariatric surgery. *International Journal of Obesity, 33*, S28–S32.

Wadden, T. A., & Sarwer, D. B. (2006). Behavioral assessment of candidates for bariatric surgery: A patient-oriented approach. *Obesity, 14*, 53S–62S.

Walfish, S., Vance, D., & Fabricatore, A. N. (2007). Psychological evaluation of bariatric surgery applicants: Procedures and reasons for delay or denial of surgery. *Obesity Surgery, 17*, 1578–1583.

Baroreceptors

David McIntyre
School of Sport and Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

Definition

Baroreceptors are mechanoreceptors that contribute to the autonomic regulation of blood pressure. Baroreceptors can be divided into the arterial

baroreceptors, primarily involved in the short-term regulation of blood pressure, and the cardiopulmonary baroreceptors, which react to changes in blood volume or central venous pressure.

Arterial baroreceptors, situated in the aortic arch and carotid sinus, increase their afferent output in response to distension of the arterial wall caused by increases in blood pressure within the vessel. They are sensitive to absolute pressure and rate of change of pressure, both of which vary over the cardiac cycle. The arrival of the pulse pressure wave at the baroreceptors causes distension of the vessel wall and generates pulse synchronous afferent firing that is maximal during early systole (Eckberg & Sleight, 1992).

The afferent traffic from the arterial baroreceptors provides the primary input to the baroreflex mechanism, which maintains short-term blood pressure homeostasis primarily by regulating heart rate and peripheral resistance via parasympathetic and sympathetic pathways. Increased baroreceptor activation depresses heart rate via vagal parasympathetic motor neurons and leads to dilation of blood vessels within the musculature through decreased sympathetic outflow. Conversely, decreased activation of the arterial baroreceptors leads to increased heart rate and constriction of blood vessels within the musculature (Jordan, 1995).

Behavioral interactions with arterial baroreceptor activation and the baroreflex are well documented. For example, physiological arousal is associated with an inhibition of the baroreflex (Marshall, 1995) and increased activation of the arterial baroreceptors has been associated with a dampening of cortical and behavioral activity (Berntson & Cacioppo, 2007; Eckberg & Sleight, 1992).

Cardiopulmonary baroreceptors are mechanoreceptors situated in the walls of the heart chambers and in the large blood vessels leading to the heart. Their primary role is in the regulation of blood volume such that increases in blood volume or central venous pressure result in reflex forearm vasodilation together with increased salt and water excretion (Eckberg & Sleight, 1992). There is evidence that cardiopulmonary baroreceptors also have an interactive effect on central arterial baroreflex interneurons (Eckberg & Sleight, 1992).

Cross-References

▶ Blood Pressure

References and Readings

Berntson, G. G., & Cacioppo, J. T. (2007). Integrative physiology: Homeostasis, allostasis, and the orchestration of systemic physiology. In J. T. Cacioppo, L. G. Tassinary, & G. G. Berntson (Eds.), *Handbook of psychophysiology* (pp. 433–451). Cambridge: Cambridge University Press.

Eckberg, D. L., & Sleight, P. (1992). *Human baroreflexes in health and disease*. Oxford: Clarendon Press.

Jordan, D. (1995). Central nervous integration of cardiovascular regulation. In D. Jordan & J. M. Marshall (Eds.), *Cardiovascular regulation* (pp. 1–14). London: The Physiological Society.

Marshall, J. M. (1995). Cardiovascular changes associated with behavioural alerting. In D. Jordan & J. M. Marshall (Eds.), *Cardiovascular regulation* (pp. 37–59). London: The Physiological Society.

Barrier Method of Protection

▶ Condom Use

Basal Metabolic Rate

Masayoshi Kumagai and Naoya Yahagi
Department of Metabolic Diseases, Graduate School of Medicine The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Definition

Basal metabolic rate (BMR) is the minimum level of energy required to sustain vital functions of organs such as the heart, lungs, liver, kidneys, intestine, nervous system, sex organs, muscles, and skin. It is measured at complete rest, in

a neutrally temperate environment, in a fasting state, and measured by the heat production or oxygen consumption per unit time, and expressed as the calories released per kilogram of body weight or per square of body surface per hour. Although there are several equations to estimate BMR, it is affected by a variety of factors such as age, hormones, exercise, body temperature, nutritional status, climate, or pregnancy.

Total energy expenditure (TEE) is the amount of energy needed by a person to meet the overall physical demands, which is the sum of basal metabolic rate (BMR), dietary-induced thermogenesis, and energy consumption during activity (Ravussin, Lillioja, Anderson, Christin, & Bogardus, 1986). BMR is the largest component of TEE, accounting for about 60–80% of TEE in ordinary people (Ravussin & Bogardus, 1989). Energy consumed by muscles accounts for up to 20% of BMR, and the brain, heart, liver, and kidneys also account for a large proportion in BMR (Elia, 1992a). BMR is the concept that was born in an attempt to evaluate minimum energy required for human to survive, and the word "basal metabolism" was first described by Magnus-Levy in 1899. It represents the integration of minimal activity of all the tissues or organs in a body under a steady-state condition.

Although accurate measurements of BMR require strict conditions and equipments, approximate estimation can be acquired through an equation. There are several predictive equations for BMR. In the early twentieth century, DuBois produced an equation using "surface area law" (DuBois & DuBois, 1915). It was later superseded by Harris-Benedict equation, which is now widely used (Harris & Benedict, 1919):

$$\text{BMR for males (kcal/day)}: \\ 66.4730 + 13.7516W + 5.0033S - 6.7750A$$

$$\text{BMR for females (kcal/day)}: \\ 665.0955 + 9.5634W + 1.8496S - 4.6756A$$

(W = weight in kilograms, S = stature in centimeters, A = ages in years)

Although Harris-Benedict equation might overestimate BMR in some cases (Daly et al., 1985), it is widely used because of its simplicity and usability.

Cross-References

► Energy: Expenditure, Intake, Lack of

References and Readings

Daly, J. M., Heymsfield, S. B., Head, C. A., Harvey, L. P., Nixon, D. W., Katzeff, H., et al. (1985). Human energy requirements: Overestimation by widely used prediction equation. *The American Journal of Clinical Nutrition*, *42*(6), 1170–1174.

DuBois, D., & DuBois, E. F. (1915). The measurements of the surface area of man. *Archives of Internal Medicine, 15*, 868–875.

Elia, M. (1992). Energy expenditure in the whole body. In J. M. Kinney & H. N. Tucker (Eds.), *Energy metabolism: Tissue determinants and cellular corollaries* (pp. 19–59). New York: Raven.

Harris, J. A., & Benedict, F. G. (1919). *A biometric study of basal metabolism in man*. Washington, DC: Carnegie Institute of Washington Publication. Publication No. 279.

Ravussin, E., & Bogardus, C. (1989). Relationship of genetics, age, and physical fitness to daily energy expenditure and fuel utilization. *American Journal of Clinical Nutrition, 49*(Suppl. 5), 968–975.

Ravussin, E., Lillioja, S., Anderson, T. E., Christin, L., & Bogardus, C. (1986). Determinants of 24-hour energy expenditure in man. Methods and results using a respiratory chamber. *Journal of Clinical Investigation, 78*(6), 1568–1578.

Baseline

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

The determination of an intervention's efficacy requires a comparison between a measure of the biological characteristic of interest (including psychological parameters) before the intervention is administered and at the end of its administration.

Data collected before its administration are called Baseline data.

Calculation of efficacy requires comparison of the Baseline and end-of-treatment data, usually presented in the form of a change score(s) of some type. If the characteristic of interest is a continuous variable, e.g., blood pressure (BP), a subject's end-of-treatment BP can be subtracted from his or her Baseline BP. In a study of an intervention to lower blood pressure, it may be seen that, overall, subjects' BPs are lower at the end of treatment than at Baseline. This reduction can be calculated in absolute terms as the mean reduction in millimeters of mercury (mmHg), e.g., 15 mmHg, or in percentage terms, e.g., 8%. Analyses can then be conducted to determine if the treatment has led to a statistically significant reduction in BP as compared with similar data collected from a control treatment group.

For other characteristics, other analyses are appropriate. For example, subjects may be rated at Baseline as mildly depressed, moderately depressed, and severely depressed based on their score(s) from an appropriate questionnaire/assessment tool. In this case, a shift analysis can be conducted to determine the numbers (and percentages) of subjects who moved category. In the case of a behavioral intervention for depression (perhaps cognitive behavioral therapy given for a certain number of weeks/sessions), it may be seen that a certain percent of the subjects "shifted" from the moderately depressed category at Baseline to the mildly depressed category at the end of the treatment period, and a different percentage shifted from the severely depressed category to the mildly depressed category.

Cross-References

- ▶ Cognitive Behavioral Therapy (CBT)
- ▶ Efficacy

B-Cell Stimulatory Factor 2

- ▶ Interleukins, -1 (IL-1), -6 (IL-6), -18 (IL-18)

Beck Depression Inventory (BDI)

Jane Upton
School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Depression

Definition

The Beck Depression Inventory is a 21-item multiple choice self-report inventory widely used measure of the presence and degree of depression in adolescents and adults.

The most recent version is the Beck Depression Inventory®-II (BDI®-II), constructed by Aaron T. Beck and colleagues (Beck, Steer, Ball, & Ranieri, 1996). The BDI®-II can be self-administered or verbally by a trained administrator, is validated for completion by 13- to 80-year-old individuals, and is available from http://www.pearsonassessments.com/HAIWEB/Cultures/en-us/Productdetail.htm?Pid=015-8018-370&Mode=summary. It has high test-retest reliability (Pearson $r = 0.93$) (Beck, Steer, & Brown, 1996). It also has high internal consistency (Alpha 0.91) (Beck, Steer, Ball, et al., 1996).

The BDI includes both cognitive and somatic symptoms of depression, unlike the Hospital Anxiety and Depression Scale which was developed for use with somatic illness patients, and therefore excludes somatic symptoms of depression to reduce confounding. The inclusion of somatic symptoms in the BDI enables the user to identify different types of depression in individuals (Canals, Blade, Carbajo & Domenech-Llaberia, 2001).

Cross-References

- ▶ Cognitive Function
- ▶ Depression: Symptoms

- ▶ HADS
- ▶ Somatic Symptoms

References and Readings

Beck, A. T., Steer, R. A., Ball, R., & Ranieri, W. (1996). Comparison of Beck depression inventories – IA and II in psychiatric outpatients. *Journal of personality assessment, 67*(3), 588–597. doi:10.1207/s15327752jpa6703_13. Accessed April 14, 2011, from doi:dx.doi.org.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for the Beck depression inventory-II*. San Antonio, TX: Psychological Corporation.

Canals, J., Blade, J., Carbajo, G., & Domenech-Llaberia, E. (2001). The Beck depression inventory: Psychometric characteristics and usefulness in non clinical adolescents. *European Journal of Psychological Assessment, 17*, 63–68.

Behavior

- ▶ Behavior Change
- ▶ Behavior Change Techniques
- ▶ Behavior Modification
- ▶ Behavioral Inhibition
- ▶ Behavioral Medicine

Behavior Change

Rachel J. Burns[1] and Alexander J. Rothman[2]
[1]Department of Psychology, University of Minnesota, Minneapolis, MN, USA
[2]Department of Psychology, University of Minnesota, Minneapolis, MN, USA

Synonyms

Health behavior change

Definition

Behavior change is the process of modifying a behavior, often to produce a desired outcome. Behavior change involves the substitution of one pattern of behavior for another.

Description

Many health conditions can be prevented or delayed if preventive actions are performed. Accordingly, national and international health agencies encourage people to engage in a range of behavioral strategies, including attending cancer screenings, being physically active, refraining from tobacco use, and using methods that protect against sexually transmitted diseases and infections (U.S. Department of Health and Human Services, 2010; World Health Organization, 2010). Moreover, behavior change is often a central component in the treatment of various health conditions, especially the management of chronic disease. For instance, dietary changes and restrictions are essential in the management of diabetes mellitus and celiac disease (American Diabetes Association, 2008; Green & Cellier, 2007). Behavior change is thus a cornerstone of preventive measures and treatments that aim to promote, protect, and restore health and well-being.

The significance of behavior change has prompted the development of several theoretical models that delineate the processes underlying behavior change and elucidate conditions that inhibit and facilitate behavior change. These models focus predominantly on individual-level factors (e.g., attitudes, perceived norms, intentions) and fall into one of two categories. Continuum-based models rely on linear combinations of specified variables to predict the likelihood of behavior (e.g., theory of planned behavior (Ajzen, 1991); social cognitive theory (Bandura, 1986)). In contrast, stage-based models assume that behavior change involves movement through a series of qualitatively distinct stages and that the factors that facilitate transitions between stages are unique (Weinstein, Rothman, & Sutton, 1998; e.g., transtheoretical model of behavior change (Prochaska & Velicer, 1997) precaution adoption process model (Weinstein, 1988)).

Behavior change is a process that unfolds over time. The behavior must first be initiated and

then, depending on the nature of the behavior, must be maintained over time (Rothman, 2000). For instance, simple preventive behaviors, such as vaccinations, typically require a single performance of the target behavior to achieve the desired health benefit. In contrast, more complex behaviors, such as physical activity or taking antiretroviral drugs, must be sustained over time in order to achieve the desired health benefit. However, extant models of behavior change have tended to focus on elucidating factors that predict the initiation of the behavior change process and given relatively less consideration to factors that predict maintenance of the target behavior. Indeed, most models of behavior change fail to distinguish between initiation and maintenance phases of the behavior change process.

Initiation and maintenance of behavior have been conceptualized as distinct phases in the behavior change process, and distinct factors are thought to influence the behavioral decisions that are made during initiation and maintenance (Rothman, 2000). The distinction between the initiation and maintenance of behavior has been further refined into a four-phase process model (Rothman, Baldwin, Hertel, & Fuglestad, 2011). Each phase is qualitatively distinct, and transition between stages is determined by a distinct set of decision criteria. The initial response phase encompasses the initial effort put forth by an individual seeking to make a behavioral change. For instance, a person may decide to become physically active and begin attending exercise classes. If one has strong efficacy beliefs and positive expectations about the outcomes associated with the target behavior, the target behavior is likely to be enacted reliably. Consistent performance of the target behavior demarcates the beginning of the continued response phase, in which one continues to expend effort in order to establish the target behavior. During this phase, the individual struggles to remain motivated to engage in the behavior and to manage the conflict between continuing to enact the new behavior and the challenges and unpleasantness associated with the new behavior. For example, a person who has recently started an exercise program may struggle to continue attending exercise classes after encountering barriers, such as sore joints or financial constraints.

The realization of initial rewards, sustained self-efficacy beliefs, sustained outcome expectations, and the ability to overcome obstacles facilitate movement from the continued response phase to the maintenance phase. During the maintenance phase, individuals no longer struggle to engage in the behavior; however, enactment of the behavior still requires effort. Individuals also remain sensitive to the costs and benefits associated with the behavior and are particularly attuned to the value of achieved outcomes (Rothman et al., 2011). Thus, someone who has been exercising regularly may be conscious of the physiological changes that have resulted from exercising and will compare the benefits of these outcomes to the costs of exercising. Satisfaction with the outcomes of the new pattern of behavior becomes a key determinant of maintenance. If the perceived costs of the behavior exceed the perceived benefits of the outcomes, then the behavior will be discontinued. Finally, the transition from maintenance to the habit phase, in which the behavioral pattern becomes self-perpetuating and automatic, occurs when people cease to regularly assess the perceived value of the behavior and its associated outcomes.

Models of behavior change are particularly useful in that they permit the identification of precise constructs to target when seeking to change behavior. Thus, theoretical models guide the design and implementation of interventions that promote behavior change (Michie & Prestwich, 2010). By identifying candidate targets for interventions, theoretical models also provide insight into the relative effectiveness of intervention strategies because intervention strategies are differentially suited to target particular constructs. For instance, a theoretical model may suggest that skill acquisition is a chief antecedent of behavior change and, thus, identifies skill development as a central goal of an intervention.

Models of health behavior change that focus on individual-level factors (e.g., attitudes, perceived norms, intentions) guide the design of

interventions that seek to change how people think and feel about particular behaviors. However, some models adopt an ecological perspective and focus on the structural and environmental factors that promote and inhibit behavior change (e.g., Stokols, 1992). These types of models are useful in guiding the development of structural interventions, such as policy changes. Several attempts have been made to integrate individual-level and ecological models (e.g., Kremers, 2010). Such integrative frameworks are useful in designing multilevel interventions because they suggest how specific combinations of individual-level (e.g., attitudes, perceived norms, intentions) and structural-level factors (e.g., laws, access to resources) interact.

When thinking about behavior change, it is also important to consider how the many properties of the behavior itself may influence the change process. For example, behavior change may involve the adoption or cessation of a behavior. Adoption requires the performance of a new behavior, such as beginning a new exercise routine (e.g., going to the gym three times a week), whereas cessation involves discontinuing a behavior, such as quitting smoking. Some behavior change may involve concurrent adoption and cessation behaviors. For instance, changing one's eating behavior can involve beginning to eat vegetables with dinner and ceasing to eat fried foods at dinner. The nature of the target behavior offers insight into the theoretical model that is best suited to guide behavior change. For instance, operant conditioning theory distinguishes between reinforcement and punishment (Skinner, 1938). Reinforcement involves the use of strategies that increase the frequency of the target behavior. In contrast, punishment involves the use of strategies that decrease the frequency of the target behavior. Accordingly, reinforcement-based models might be best suited for thinking about how to promote adoption behaviors, whereas punishment-based models might be best suited for thinking about how to promote cessation behaviors.

It is also important to recall that there are particular challenges associated with changes in specific behavioral domains. Simple preventive behaviors, such as getting a vaccine, require that the behavior in question be enacted once, or very infrequently, to achieve the desired outcome. Conversely, complex preventive health behaviors, such as exercising regularly, require that a behavior be enacted repeatedly before the desired outcome is obtained. Moving toward the maintenance and habit phases of the behavior change process is thus particularly important for complex preventive health behaviors.

In conclusion, behavior change is an important process in the prevention and treatment of illness. Several theoretical models have been developed to elucidate the processes involved in behavior change. Many prominent models focus on predictors of behavior change at the individual level; however, it is important to also consider environmental and structural influences on behavior change. Although many theoretical models fail to distinguish between the initiation and maintenance of behavior, these phases are qualitatively distinct and are driven by distinct factors. Theoretical models are essential in the development of effective behavior change interventions. The properties of the target behavior can also have implications for the intervention strategy that is adopted.

Cross-References

- ▶ Behavior
- ▶ Ex-Smokers
- ▶ Health Behavior Change
- ▶ Health Behaviors
- ▶ Lifestyle, Modification
- ▶ Risk Factors and Their Management
- ▶ Smoking and Health
- ▶ Smoking Behavior

References and Readings

Ajzen, I. (1991). The theory of planned behavior. *Organizational Behavior and Human Decision Processes, 50*, 179–211.

American Diabetes Association. (2008). Nutrition recommendations and interventions for diabetes. *Diabetes Care, 31*, S61–S78.

Bandura, A. (1986). *Social foundations of thought and action*. Englewood Cliffs, NJ: Prentice-Hall.
Green, P. H., & Cellier, C. (2007). Celiac disease. *New England Journal of Medicine, 357*, 1731–1743.
Kremers, S. P. J. (2010). Theory and practice in the study of influence on energy balance-related behaviors. *Patient Education and Counselling, 79*, 291–298.
Michie, S., & Prestwich, A. (2010). Are interventions theory-based? Development of a theory coding scheme. *Health Psychology, 29*, 1–8.
Prochaska, J. O., & Velicer, W. F. (1997). The transtheoretical model of health behavior change. *American Journal of Health Promotion, 12*, 38–48.
Rothman, A. J. (2000). Toward a theory-based analysis of behavioral maintenance. *Health Psychology, 19*, 64–69.
Rothman, A. J., Baldwin, A. J., Hertel, A. W., & Fuglestad, P. (2011). Self-regulation and behavior change: Disentangling behavioral initiation and behavioral maintenance. In K. D. Vohs & R. F. Baumeister (Eds.), *Handbook of self-regulation: Research, theory and applications* (pp. 106–124). New York: Guilford Press.
Skinner, B. F. (1938). *The behavior of organisms: An experimental analysis*. New York: Appleton-Century-Crofts.
Stokols, D. (1992). Establishing and maintaining health environments: Toward a social ecology of health promotion. *American Psychologist, 47*, 6–22.
U.S. Department of Health and Human Services. (2010). Office of Disease Prevention and Health Promotion. (n.d.). *Healthy people 2020 objectives*. Washington, DC. Retrieved October 3, 2011, from http://www.healthypeople.gov/2020/topicsobjectives2020/pdfs/HP2020objectives.pdf
Weinstein, N. D. (1988). The precaution adoption process. *Health Psychology, 7*, 355–386.
Weinstein, N. D., Rothman, A. J., & Sutton, S. R. (1998). Stage theories of health behavior. *Health Psychology, 17*, 290–299.
World Health Organization. (2010). Guidelines for the management of sexually transmitted infections. Retrieved October 2, 2011, from http://whqlibdoc.who.int/publications/2010/9789241599979_eng.pdf.

Behavior Change Techniques

Susan Michie[1] and Marie Johnston[2]
[1]University College London, London, UK
[2]School of Medicine and Dentistry, University of Aberdeen, Aberdeen, Scotland, UK

Definition

A behavior change technique (BCT) is a systematic procedure included as an active component of an intervention designed to change behavior.

The defining characteristics of a BCT (Michie, Abraham et al., 2011) are that it is:

- Observable
- Replicable
- Irreducible
- A component of an intervention designed to change behavior
- A postulated active ingredient within the intervention

In this context, a definition of behavior, agreed across disciplines of psychology, sociology, anthropology, and economics, is "anything a person does in response to internal or external events. Actions may be overt (motor or verbal) and directly measurable, or covert (activities not viewable e.g., physiological responses) and indirectly measurable; behaviours are physical events that occur in the body and are controlled by the brain" (Hobbs, Campbell, Hildon, & Michie, 2011). This was arrived at via a Delphi exercise of 14 members of a multidisciplinary advisory group, starting with a shortlist of definitions of behavior compiled through library catalogue searching and using key reference sources such as the *American Psychological Association Dictionary* and the *Oxford Concise Dictionary of Sociology*. The definition was synthesized from constructs that were included in at least 50% of the definitions reaching an agreed threshold of perceived usefulness.

A BCT is thus the smallest component compatible with retaining the postulated active ingredients, that is, the proposed mechanisms of change, and can be used alone or in combination with other BCTs. A BCT should be well specified so that effectiveness of the BCT can be evaluated (e.g., in randomized controlled trials, in factorial experimental designs (Collins et al., 2011), or N-of-1 studies). However, the evidence base for effectiveness may or may not have been established. Examples of BCTs are as follows: "Prompts/cues," "Information about health consequences," "Incentive," "Goal setting," "Self-monitoring," "Action planning," "Behavioral rehearsal/practice," "Graded tasks," "Social support/encouragement," "Persuasive communication," and "Habit formation."

BCTs specify the minimum content of *what* must be delivered, that is, minimum content that would allow identification of that technique (e.g., feedback must involve providing the target audience with information about their specific behavior). A BCT does not specify the *how*, that is, the mode of delivery, and it is possible for a given BCT to be delivered in many different ways. For example, feedback may be delivered by letter or face to face, to groups or to an individual, on one occasion or frequently, by a therapist or by an automatic electronic message.

Description

Behavior change interventions include one or more BCTs. Some well-recognized behavior change interventions contain reliable combinations of BCTs, for example, relapse prevention includes both *problem solving* and *action planning*, whereas more general labels may contain variable combinations of BCTs, for example, the contents of "cognitive behavior therapy" are very variable (Gatchel, Peng, Peters, Fuchs, & Turk, 2007). For full specification of a behavior change intervention, both the active content, that is, the BCTs, and the mode of delivery need to be described (Davidson et al., 2003).

Behavior change interventions may influence behavior in several ways: behavior can be initiated or terminated, or increased or decreased in frequency, duration, or intensity. For most behaviors, there is variation between people and within individuals over time in all of these dimensions, influenced by environmental, social, cognitive, and emotional variables. Studies of how behavior varies within and between people have led to an understanding of how to use external factors to modify behavior. Technologies of behavior change have been developed within disciplines of applied psychology (e.g., clinical, educational) and adopted and extended in a wide variety of intervention functions and policies, such as commercial advertising and social marketing (Michie, van Stralen, & West, 2011). These technologies are made up of individual BCTs.

Why Are Behavior Change Techniques Important?

The importance of behavior change in improving health is illustrated by the increasing evidence that behavior influences health outcomes, and increasing investment by funding governments and scientific bodies in the development and evaluation of interventions to change population, patient, and practitioner behaviors. An example is the US National Institutes of Health's Office of Behavioral and Social Sciences Research (OBSSR) which was founded in 1995 with a budget of $27 million a year, in recognition of the key role that behavioral and social factors often play in illness and health.

Interventions to change behavior are typically complex, involving many interacting components (Craig et al., 2008). BCTs are the active ingredients of these interventions but are often poorly described in research protocols and published reports (Michie, Fixsen, Grimshaw, & Eccles, 2009). Components may be described in terms that are vague, general, and/or ambiguous and with labels, for example, "behavioral counseling," that can mean different things to different researchers or practitioners, thus acting as a barrier to replication, the essential cornerstone for scientific progress. In contrast, biomedical interventions are precisely specified (e.g., the pharmacological "ingredients" of prescribed drugs, their dose, and frequency of administration).

This lack of precision and lack of consensually agreed terms lead to problems in replication in primary research, in evidence synthesis in systematic reviews, and in implementation in practical applications. It also undermines the task of establishing those that are effective in changing behavior and understanding the causal mechanisms underlying behavior change. If intervention descriptions are idiosyncratic or ambiguous, and cannot therefore be interpreted reliably, it is impossible to aggregate the evidence to ascertain their effectiveness. Additionally, there is no value in evaluating an intervention if one cannot accurately identify and describe what is being evaluated and how competently it was delivered; it

would be impossible to implement if shown to be effective. The absence of an internationally agreed method to specify and report the content of behavior change interventions hampers the development of effective interventions.

Although the CONSORT statement for randomized trials of "nonpharmacologic" interventions calls for precise details of interventions in research, including a description of the different intervention components (Boutron, Moher, Altman, Schulz, & Ravaud, 2008), it gives no guidance as to what details. The UK Medical Research Council's guidance (Craig et al., 2008) for developing and evaluating complex interventions acknowledges this problem and also the problem of lack of consistency and consensus in use of terminology (Michie, Johnston, Francis, Hardeman, & Eccles, 2008).

The Development of a Method of Specifying BCTs

These problems have been addressed by the development of systematically generated and applied collections or "taxonomies" of BCTs. These have been constructed by identifying BCTs within written reports of the interventions, or texts describing interventions, in a bottom-up, inductive fashion and, to date, their hierarchical structures have not been established. They have been developed in relation to different behavior types: physical activity and healthy eating (Abraham & Michie, 2008; Michie, Ashford et al., 2011), smoking (Michie, Hyder, Walia, & West, 2011; West, Evans, & Michie, 2011), excessive alcohol use (Michie et al., 2012), and condom use (Abraham, Good, Warren, Huedo-Medina, & Johnson, 2011).

Using such taxonomies with standardized labels and definitions will improve current practice by ensuring that, when the taxonomy is used, a technique is always described by the same label and that a label is always used for the same technique. In current practice, the same component techniques within behavioral interventions may be described in protocols and published reports with different labels (e.g., "self-monitoring" may be labeled "daily diaries"). Conversely, the same labels may be applied to different techniques (e.g., "behavioral counseling" may involve "educating patients" or "feedback, self-monitoring, and reinforcement."

Specifying interventions by BCTs allows multivariate statistical analysis to identify specific BCTs associated with effective interventions, the "active ingredients." Heterogeneous, complex interventions have been synthesized to identify effective component BCTs in systematic reviews using the statistical technique of meta-regression. This has been conducted for physical activity and healthy eating (Dombrowski et al., 2012; Michie, Abraham, Whittington, McAteer, & Gupta, 2009), alcohol (Michie et al., 2012), smoking cessation (West et al., 2010), and HIV prevention behaviors (Albaraccin et al., 2005; de Bruin, Viechtbauer, Hospers, Schaalma, & Kok, 2009; Good, Warren, Huedo-Medina, Abraham, & Johnson, 2011). "Active ingredients" have also been identified in the English Stop Smoking Services by analyzing protocols for behavioral support for smoking cessation in terms of BCTs and investigating associations with a national database of carbon monoxide verified quit rates (West, Walia, Hyder, Shahab, & Michie, 2010).

Since the first taxonomy with demonstrated reliability was published in 2008 (Abraham & Michie, 2008), this method has been widely used internationally, for example, to specify interventions (Araujo-Soares, McIntyre, MacLennan, & Sniehotta, 2009), synthesize evidence (see above), and design interventions (McKenzie et al., 2008). However, further development is necessary for maximal usefulness. The number of BCTs included, the BCTs relevant for changing a wider range of behaviors, the precise and unambiguous specification of each BCT, the acceptability and feasibility of using the methods, as well as the need for a structure that goes beyond a list to develop a true taxonomy are required. Therefore, this work is being taken forward using consensus methods on an international basis (Michie, Abraham et al., 2011).

In addition to specifying the BCTs, it will be important to develop shared methods of reporting on both the methods of delivery (Gatchel et al., 2007) and the competence with which they are delivered. Frameworks for the specification of professional competences for the delivery of

BCTs are being developed and have been used to advise national governments (Dixon & Johnston, 2010) and as a basis for a national training program (NHS Centre for Smoking Cessation and Training (NCSCT), 2011).

The Benefits of the BCT Approach

1. *Developing behavior change interventions*: Intervention developers will be able to use a comprehensive list of BCTs (rather than relying on the limited set they are aware of) to design interventions and will be able to report the intervention content in well-defined and detailed ways.
2. *Reporting interventions*: Specifying intervention content by BCT will facilitate the accurate, replicable description of behavior change interventions. Both intervention and control conditions can be specified using BCTs in randomized controlled trials.
3. *Implementing effective interventions in practice*: BCT specification will facilitate faithful implementation of interventions found to be effective.
4. *Replicating interventions and control conditions*: Specifying interventions by BCTs will help the replication of both intervention and control conditions.
5. *Synthesizing evidence*: Systematic reviewers will be able to use a reliable method for extracting information about intervention content, thus identifying and synthesizing discrete, replicable, potentially active ingredients associated with effectiveness.
6. *Linking to theory*: Linking BCTs with theories of behavior change allows reviewers to investigate possible mechanisms of action (Michie et al., 2009; Dombrowski et al., 2012).
7. *Accumulating scientific knowledge about behavior change*: A shared terminology for specifying behavior change interventions allows the more efficient accumulation of knowledge and investigations of generalization across behaviors, populations, and settings.

Advancing the Science of Behavior Change

A well-developed system of defining and labeling BCTs would allow the science of behavior change to accumulate evidence and advance theory of behavior change. The BCT approach is already providing a method for doing this. Early versions of BCT taxonomies have allowed reviewers to synthesize heterogeneous interventions to identify effective component BCTs. Evaluation of the effectiveness of combinations of BCTs can help test theories of behavior change. So, for example, the finding that interventions with a combination of self-monitoring and feedback were effective supports the mechanisms of change proposed by Carver and Scheier's Control Theory (Carver & Scheier, 1982). Further evidence is needed to develop theory. If we are to better understand both the effects and processes of behavior change interventions, we need to develop a method to link BCTs to mechanisms of action.

For these methods to maximize scientific advance, a shared system for describing behavior change interventions is necessary, and this will require collaborative work to develop agreed labels and definitions and reliable procedures for their application across disciplines and countries. Even the "best" taxonomy is inevitably a work-in-progress as new BCTs are likely to continue to emerge from ongoing research and practice, in the same way that the labeling of peptides and botanical taxonomies continue to be developed.

Acknowledgments We thank Ronan O'Carroll for helpful comments on an earlier draft.

Cross-References

▶ Behavior Change
▶ Behavior Modification
▶ Cognitive Behavioral Therapy (CBT)
▶ Population Health
▶ Randomized Clinical Trial

References and Readings

Abraham, C., Good, A., Warren, M. R., Huedo-Medina, T., & Johnson, B. (2011). Developing and testing a SHARP taxonomy of behaviour change techniques included in condom promotion interventions. *Psychology & Health, 26*(Suppl. 2), 299.

Abraham, C., & Michie, S. (2008). A taxonomy of behaviour change techniques used in interventions. *Health Psychology, 27*, 379–387.

Albaraccin, D., Gillette, J. C., Earl, A. N., Glasman, L. R., Durantini, M. R., & Ho, M. H. (2005). A test of major assumptions about behaviour change: A comprehensive look at the effects of passive and active HIV-prevention interventions since the beginning of the epidemic. *Psychological Bulletin, 131*(6), 856–897.

Araujo-Soares, V., McIntyre, T., MacLennan, G., & Sniehotta, F. F. (2009). Development and exploratory cluster-randomised opportunistic trial of a theory-based intervention to enhance physical activity among adolescents. *Psychology & Health, 24*(7), 805–822.

Boutron, I., Moher, D., Altman, D. G., Schulz, K. F., & Ravaud, P. (2008). Extending the CONSORT statement to randomized trials of nonpharmacologic treatment: Explanation and elaboration. *Annals of Internal Medicine, 148*(4), 295–309.

Carver, C. S., & Scheier, M. F. (1982). Control theory: A useful conceptual framework for personality-social, clinical, and health psychology. *Psychological Bulletin, 92*(1), 111–135.

Collins, L. M., Baker, T. B., Mermelstein, R. J., Piper, M. E., Jorenby, D. E., Smith, S. S., et al. (2011). The multiphase optimization strategy for engineering effective tobacco use interventions. *Annals of Behavioral Medicine, 41*(2), 208–226.

Craig, P., Dieppe, P. A., Macintyre, S., Michie, S., Nazareth, I., & Petticrew, M. (2008). Developing and evaluating complex interventions: The new Medical Research Council guidance. *BMJ, 337*, a1655.

Davidson, K. W., Goldstein, M., Kaplan, R. M., Kaufmann, P. G., Knatterud, G. L., Orleans, C. T., et al. (2003). Evidence-based behavioral medicine: What is it and how do we achieve it? *Annals of Behavioral Medicine, 26*(3), 161–171.

de Bruin, M., Viechtbauer, W., Hospers, H. J., Schaalma, H. P., & Kok, G. (2009). Standard care quality determines treatment outcomes in control groups of HAART-adherence intervention studies: Implications for the interpretation and comparison of intervention effects. *Health Psychology, 28*(6), 668–674.

Dixon, D., & Johnston, M. (2010). *Health behaviour change competency framework: Competences to deliver interventions to change lifestyle behaviours that affect health (monograph on the Internet)*. Edinburgh: The Scottish Government, (cited 2011 Dec 8). Retrieved 2012, from www.healthscotland.com/documents/4877.aspx

Dombrowski, S. U., Sniehotta, F. F., Avenell, A., Johnston, M., MacLennon, G., & Araujo-Soares, V. (2012). Identifying active ingredients in complex behavioral interventions for obese adults with obesity-related co-morbidities or additional risk factors for co-morbidities: A systematic review. *Health Psychology Review, 6*, 7–32.

Gatchel, R. J., Peng, Y. B., Peters, M. L., Fuchs, P. N., & Turk, D. C. (2007). The Biopsychosocial approach to chronic pain: Scientific advances and future directions. *Psychological Bulletin, 133*(4), 581–624.

Good, A., Warren, M., Huedo-Medina, T., Abraham, C., & Johnson, B. (2011). Identifying active ingredients in "efficacious" HIV prevention interventions: A meta-analysis. *Psychology & Health, 26*(Suppl. 2), 299–300.

Hobbs, L., Campbell, R., Hildon, Z., & Michie, S. (2011). Behaviour change theories across psychology, sociology, anthropology and economics: A systematic review. *Psychology & Health, 26*(Suppl. 2), 31.

McKenzie, J., French, S. D., O'Connor, D., Grimshaw, J., Mortimer, D., Michie, S., et al. (2008). IMPLEmenting a clinical practice guideline for acute low back pain evidence-based manageMENT in general practice (IMPLEMENT): Cluster randomiszed controlled trial study protocol. *Implementation Science, 3*, 11.

Michie, S., Abraham, C., Eccles, M. P., Francis, J. J., Hardeman, W., & Johnston, M. (2011). Strengthening evaluation and implementation by specifying components of behaviour change interventions: A study protocol. *Implementation Science, 6*, 10.

Michie, S., Abraham, C., Whittington, C., McAteer, J., & Gupta, S. (2009). Effective techniques in healthy eating and physical activity interventions: A meta-regression. *Health Psychology, 28*(6), 690–701.

Michie, S., Ashford, S., Sniehotta, F. F., Dombrowski, S. U., Bishop, A., & French, D. P. (2011). A refined taxonomy of behaviour change techniques to help people change their physical activity and healthy eating behaviours – the CALO-RE taxonomy. *Psychology & Health, 26*(11), 1479–1498.

Michie, S., Fixsen, D., Grimshaw, J., & Eccles, M. (2009). Specifying and reporting complex behaviour change interventions: The need for a scientific method. *Implementation Science, 4*, 40.

Michie, S., Hyder, N., Walia, A., & West, R. (2011). Development of a taxonomy of behaviour change techniques used in individual behavioural support for smoking cessation. *Addictive Behaviors, 36*(4), 315–319.

Michie, S., Johnston, M., Francis, J., Hardeman, W., & Eccles, M. (2008). From theory to intervention: Mapping theoretically derived behavioural determinants to behaviour change techniques. *Applied Psychology, 57*, 660–680.

Michie, S., van Stralen, M. M., & West, R. (2011). The behaviour change wheel: A new method for characterising and designing behaviour change interventions. *Implementation Science, 6*, 42.

Michie, S., Whittington, C., Hamoudi, Z., Zarnani, F., Tober, G., & West, R. (2012). Identification of behaviour change techniques to reduce excessive alcohol consumption. *Addiction*, 107. doi:10.1111/j.1360-0443.2012.03845.xs

NHS Centre for Smoking Cessation and Training (NCSCT) [homepage on the Internet]. UK: NCSCT; c2011 (cited 2011 Dec 8). Retrieved from http://www.ncsct.co.uk/

West, R., Evans, A., & Michie, S. (2011). Behavior change techniques used in group-based behavioral support by

the English Stop-Smoking Services and preliminary assessment of association with short-term quit outcomes. *Nicotine & Tobacco Research, 13*, 1316–1320.
West, R., Walia, A., Hyder, N., Shahab, L., & Michie, S. (2010). Behavior change techniques used by the English Stop Smoking Services and their associations with short-term quit outcomes. *Nicotine & Tobacco Research, 12*(7), 742–747.

Behavior Modification

Misuzu Nakashima
Hizen Psychiatric Center, Yoshinogari, Kanzaki, Saga, Japan

Synonyms

Behavior therapy

Definition

Behavior modification is to change behavior by techniques to improve behavior, such as altering behavior and reaction to stimuli through positive and negative reinforcement of adaptive behavior and/or the reduction of maladaptive behavior through positive and negative punishment. The techniques used in behavior modification are based on principle of learning.

Description

Behavior modification and behavior therapy have been used almost interchangeably in literature, although they have some very minor differences. Some people think behavior modification to be a part of behavior therapy; other people think that behavior modification contains behavior therapy. In addition, some people use term behavior therapy only in the context of the medical field.

The term behavior modification was created earlier than behavior therapy because the first use of the term behavior modification appears to have been by Edward Thorndike in 1911, and afterward, Skinner, B.F., continued to use the term behavior therapy. The principles to change behavior in the behavior modification or behavior therapy will be introduced. In the treatment of mental disorders, please refer to the page on behavior therapy.

The Representative Theory and Technique of the Behavior Therapy/ Behavior Modification

Applied Action Analytical Model

Skinner, B. F., paid attention to the action of an individual and its relationship to the environment, and he came up with operant conditioning. He proved through experiments that frequency of behavior freely changes by operating on its environment. Behavior analysis chooses a target behavior to become the object of the treatment and clarifies contingency as a result of this method. In behavior analysis, they investigate mutual relations of antecedent, behavior, and consequence and inspect the hypothesis on the causes which results to the target behavior. Reinforcement plays a key role for intervention, and it is called by techniques such as shaping or token economy. Refer to a page on operant conditioning for more details.

Neobehavioristic Mediational S–R Theory

This is a theory based on drive reduction theory proposed by Hull, C.L. In this theory, it is explained that the motivated behavior is roused by a drive, and the desire is the intervening variable, and the behavior that these drive and desire satisfies and reduced is reinforced. For Skinner, B.F. who aimed at predicting and controlling behavior (a dependent variable) only in an environmental condition (an independent variable), this model considers drive to be any factors in the living body (a parameter). With this model, Eysenk, H.J., and Wolpe, J., produced cures for neurotic behavior that focuses on fear as the drive among these neurotic behaviors. Systematic desensitization method or flooding is nominated for representative technique.

Social Learning Theory Model

This is the model that was proposed by Bandura, A. Observational learning plays a key role in social

learning, and the person takes action not only in reaction to the stimulation from the outside world but also by mediation of cognition. He raised external reinforcement, expectation, and self-efficacy as factors of behavior modification. Modeling and self-control are representative techniques.

Cognitive-Behavioral Therapy Model

This is a model I consider when I regard cognition as important as an intervening factor in modifying actions because cognition always influences behavior. A change of the cognition is indispensable since this is the goal of behavior change and treatment. Cognitive restructuring and self-instructional training are representative techniques.

Cross-References

- ▶ Behavior Therapy
- ▶ Behavioral Therapy
- ▶ Lifestyle, Modification
- ▶ Operant Conditioning

References and Readings

Bandura, A. (1963). *Social learning and personality development*. New York: International Thomson.
Bellack, A. S., Hersen, M., & Kadin, A. E. (1990). *International handbook of behavior modification and therapy*. New York: Plenum Press.
Eysenck, H. J. (1960). *Behavior therapy and the neuroses*. Oxford: Pergamon Press.
Gambrill, E. D. (1977). *Behavior modification – handbook of assessment, intervention, and evaluation*. San Francisco: Jossy-Bass.
Hull, C. L. (1943). *Principle of behavior*. New York: Appleton Century.
Skinner, B. F. (1959). *Cumulative record*. New York: Appleton Century.
Thorndike, E. L. (1911). *Animal intelligence*. New York: The Macmillan.
Wolpe, J. (1958). *Psychotherapy by reciprocal inhibition*. Stanford: Stanford University Press.

Behavior Modification Program

- ▶ Asthma: Behavioral Treatment

Behavior Therapy

- ▶ Asthma: Behavioral Treatment
- ▶ Behavior Modification

Behavioral Disengagement

- ▶ Distraction (Coping Strategy)

Behavioral Disorder

- ▶ Psychological Disorder

Behavioral Ecological Model

- ▶ Ecological Models: Application to Physical Activity

Behavioral Endocrinology

- ▶ Psychoneuroendocrinology

Behavioral Immunology

Kavita Vedhara[1] and Karen Dawe[2]
[1]Institute of Work, Health and Organisations, University of Nottingham, Nottingham, UK
[2]School of Social and Community Medicine, University of Bristol, Oakfield House, Oakfield Grove, Bristol, UK

Synonyms

Psychoneuroendocrinology; Psychoneuroimmunology

Definition

"Behavioral immunology" refers to the branch of behavioral medicine concerned with bidirectional interactions between behavior and the immune system.

Description

The field of psychoneuroimmunology is providing growing evidence that psychosocial factors can influence immunity and health through both direct and indirect routes, including behavioral modifiers.

The brain is considered an "immuno-privileged" organ, protected from normal inflammatory processes by the blood–brain barrier, a network of endothelial cells that form tight junctions with brain capillaries and prevent the entry of blood-borne substances to the brain. There are, however, a number of pathways of communication between the distributed elements of the immune system and the central nervous system that provide potential mechanisms of interaction.

Cytokines, chemical messengers used by the immune system, are able to signal to the brain. It has been shown that following infection by a pathogen (e.g., virus or bacteria) macrophages (a type of white blood cell) release the cytokine interleukin-1 (IL-1) into the bloodstream. Circulating IL-1 is known to induce alterations in brain activity and changes in the metabolism of central brain chemicals and neurotransmitters such as norepinephrine, serotonin, and dopamine in discrete brain areas.

Cytokines are large molecules and as such are not able to cross the blood–brain barrier. However IL-1, and possibly other cytokines too, are able to communicate with the brain via peripheral nerves such as the vagus nerve. The vagus nerve is a branch of the autonomic nervous system with both afferent and efferent fibers and is ideally situated to convey immune information. The peripheral IL-1 signal is transduced into neuronal information, which is transmitted to the brain by the vagus nerve. This signal is then retransduced into chemical information in the form of IL-1 synthesized centrally in the brain itself (Evans, Hucklebridge, & Clow, 2000).

In addition to neuronal transduction via the vagus nerve, immune information can be sent by afferent nerve fibers from the lymphoid organs themselves. These fibers have receptors for cytokines and other similar molecules produced by immune cells.

Once an immune signal is interpreted by the brain, it is able to influence monoamine activity. Monoamines (noradrenaline, serotonin, and dopamine) are neurotransmitters that operate in those parts of the brain involved in the regulation of mood, reward, appetite, sleep, and reproduction. A single administration of inflammatory cytokines induces changes in monoamine activity in a manner similar to changes induced by stress. There are therefore multiple potential mechanisms of connectivity between the brain and immune system.

Accordingly, the brain is capable of influencing immune processes. Long before the mechanisms of interaction between the brain and immune system were known, it was discovered that the immune system of rats could be trained by classical Pavlovian conditioning to respond to a neutral stimulus previously paired with a stimulus with direct immune-modulatory properties (Ader & Cohen, 1975 in Evans et al., 2000). Humans have since been shown to respond in the same way.

The effects of conditioning are varied, so it is likely that a number of mechanisms are involved. Some evidence suggests that T cells may play an important role in conditioning of the immune system. Other research has suggested that conditioning of the immune system requires the involvement of opioid-mediated circuits within the central nervous system. However, the exact mechanism is yet to be elucidated.

The field of behavioral immunology is also concerned with the effect of volitional behavior, such as sleep, physical activity, nutrition, and substance abuse on the workings of the immune system. Smoking, alcohol use, and cocaine have all been shown to affect the immune system.

Smoking cigarettes has effects on the immune system which may be direct, or may occur via

endocrine-mediated mechanisms. Nicotine is reported to affect both humoral and cellular immunity. Compared to nonsmokers, adult smokers have higher white blood cell counts and lower natural killer (NK) cell activity (Irwin & Cole in Vedhara & Irwin, 2007).

Alcohol use is also known to suppress immune system functioning and may act directly or indirectly via gonadal steroid hormones (Penedo & Dahn in Vedhara & Irwin, 2007). Further, alcohol use in the context of clinical depression acts in a synergistic manner to suppress the immune system; while alcohol and depression each have a suppressant effect on the immune system, the interaction of alcohol substance abuse and affective disorders may result in significantly greater immune impairment than either condition alone.

Cocaine is thought to negatively alter the responsiveness of the immune system via its effects on the functioning of NK cells, T cells, neutrophils, and macrophages, and by dysregulating the production of cytokines.

In diseases of the immune system, such as HIV, health behaviors that can directly influence immune functioning, such as substance abuse and adherence to medication regimes, have serious clinical implications (Pereira & Penedo in Vedhara & Irwin, 2007). Among HIV+ individuals cigarette smoking increases the risk of developing opportunistic respiratory infections, oropharyngeal candidiasis, and cervical and anal neoplasia. Alcohol consumption is associated with impaired immune and viral responses to antiretroviral treatment among HIV+ individuals, while cocaine use has been linked to impaired immune functioning, enhanced HIV infectivity, and replication.

Physical exercise has also been shown to influence immune parameters in a variety of populations; however the majority of studies researching this topic have been conducted with HIV+ individuals. These have shown positive effects of physical exercise on CD4 and CD8 cell number, NK cell number, NK cell count, and better immunological control of latent viruses (e.g., herpes). In healthy older adults, increased physical activity is associated with higher in vitro measures of immune functioning. However studies in this area have been flawed by small sample sizes, short follow-up periods, limited number of immune parameters, and uncontrolled confounding variables such as uncertainty regarding adherence to the exercise program.

Lack of sleep is another behavioral factor thought to be important for immune functioning. It has been suggested that loss of sleep, or disordered sleep, adversely affect resistance to infection, increases cancer risk, alters inflammatory disease progression, and reduces NK cell counts. Normal sleep is associated with redistribution of circulating lymphocyte subsets, increase of NK cell activity, increases in certain cytokines, and a relative shift toward Th1 cytokine expression. It has even been suggested that disordered sleep, a symptom of clinical depression, may be a crucial behavioral factor that mediates the relationship between depression and alterations in immune system functioning.

The relationship between sleep and immune functioning is a bidirectional one; animal studies have shown cytokines to have both sleep-promoting and inhibitory effects on sleep depending on the cytokine in question, plasma levels, and circadian phase. Less is known about the interaction between sleep and cytokines in humans. Human studies are necessarily limited by a lack of means to measure cytokine levels in the brain as systemic levels of cytokines may not be an accurate reflection of brain cytokine activity. However these basic findings have informed theories on the role of the cytokines in clinical contexts, including the inflammation theory of depression and daytime fatigue in conditions such as chronic fatigue and cancer.

In some clinical conditions, such as cancer or hepatitis C, large doses of cytokines are given therapeutically. Treatment such as this is often associated with depressed mood, anhedonia, fatigue, poor concentration, and disordered sleep. In the absence of disease, administration of inflammatory cytokines leads to depressed mood, increased somatic concern, cognitive impairment, and difficulties with flexible thinking. The effects are similar following physiological activation of the body's own cytokines; the experimental administration of bacterial

endotoxin results in activation of pro-inflammatory cytokines which leads to depressed mood, anxiety, and impaired performance on verbal and nonverbal memory functions.

Findings from experimental situations such as these can be extended to clinical contexts in which levels of pro-inflammatory cytokines are increased as the consequence of invasion by a pathogen. Replication of invading pathogens triggers a stereotypical immune response which is coordinated by inflammatory cytokines. Cytokines direct white blood cells to the site of infection, induce them to proliferate, differentiate, and activate mechanisms involved in pathogen destruction. The crucial cytokines to this process are IL-1b, IL-6, and tumor necrosis factor-a. Elimination of invading pathogens in this way results in a characteristic set of symptoms that are experienced as clinical illness. In addition to disease-specific symptoms that are dependent on the nature of the pathogen, infections are also associated with a host of nonspecific symptoms including fever, malaise, increased sleep, anorexia, anhedonia, reduced reproductive behavior, and social withdrawal. This coordinated behavioral response to infection has been termed "sickness behavior". The result of interactions between cytokines and the central nervous system, sickness behavior is considered an evolutionary strategy to maximize chances of survival after infection, and represents an attempt to conserve energy by limiting functions not essential to fighting the infection. Sickness behavior is coordinated by the brain with the cytokine IL-1 being the key molecule signaling between macrophages and the brain (Evans et al., 2000).

Cross-References

- ▶ Psychoneuroimmunology
- ▶ Sickness Behavior

References and Readings

Evans, P., Hucklebridge, F., & Clow, A. (2000). *Mind, immunity and health*. London: Free Association Books.

Kandel, E. R., Schwartz, J. H., & Jessell, T. M. (Eds.). (2000). *Principles of neural science*. New York: McGraw-Hill.

Vedhara, K., & Irwin, M. (Eds.). (2007). *Human psychoneuroimmunology*. New York: Oxford University Press.

Behavioral Inhibition

Cara Wong
School of Psychology, University of Sydney, Sydney, NSW, Australia

Synonyms

Cognitive control; Response inhibition; Self-control

Definition

Behavioral inhibition can be generally defined as an individual's ability to inhibit a desired behavior, or prepotent response. It belongs to a subset of "executive functions" which are the top-down self-regulatory functions that allow us to consciously control our actions afforded by the frontal cortex (Fuster, 1997).

Behavioral inhibition can be divided into three interrelated processes: (a) inhibition of an initial prepotent response to an event; (b) cessation of an ongoing response, which allows a delay in the decision to respond; and (c) the protection of this period of delay and the self-directed responses that occur within it from disruption by competing events and responses (interference control). Behavioral inhibition also involves overriding or inhibiting competing urges, and delaying gratification (Barkley, 1997; Muraven & Baumeister, 2000; Shallice & Burgess, 1993).

Characteristics

Refraining from performing an immediate desire often requires more effort than taking action, (for example, it is more difficult for a smoker to refrain from smoking, than to gratify their desire

to smoke) and involves more than just passive inaction. There are many situations where an individual may be required to use inhibition, where something is desired but not the right thing to do. Circumstances that involve temporal delays (i.e., delay of gratification), conflicts in temporally related consequences, or where there is a need to generate a novel response may rely heavily on behavioral inhibition. Consequently, inhibition is particularly relevant for addictive or impulsive behaviors for example drug use, smoking, or gambling, and health behaviors such as making healthy versus unhealthy food choices, exercising, etc. The Temporal Self-regulation Theory (Hall & Fong, 2007) is a relatively recent theory of behavior that includes behavioral inhibition and self-regulation as additional determinants of behavior. The theory predicts that the likelihood of performing a behavior is a function of both an individual's self-regulatory capacity (including behavioral inhibition), and the presence or absence of cues to that behavior in the environment.

Problems with behavioral inhibition are typically seen in children, individuals with attention deficit hyperactivity disorder (ADHD), or patients with frontal lobe damage/disorders (Barkley, 1997; Shallice & Burgess, 1991). Common symptoms of disinhibition in these groups of people include: acting out on impulses or desires, a strong inclination to seek immediate reinforcement or gratification, and difficulty inhibiting an action once it has begun. In normal children, behavioral inhibition (as well as other executive functions) improves with age, which coincides with the development and maturation of the frontal cortex (Russell, 1948).

Behavioral inhibition or self-control can be thought of as one of a subset of controlled or effortful cognitive processes (Hasher & Zacks, 1979). This is because the individual or self exerts control over its own responses rather than allowing them to proceed in their normal or automatic fashion (Muraven & Baumeister, 2000). In contrast, most behaviors occur automatically and do not require active participation or need the self to override the natural response and implement a different one (Bargh, 1994). The benefit of having automatic behaviors is that they are performed more efficiently; however, the disadvantage is that they are more rigid and difficult to change. Although controlled processes require more effort and resources, they are much more flexible.

Assessment of Behavioral Inhibition

Behavioral inhibition is assessed by performance on cognitive and behavioral tasks that require withholding of responding, delayed responding, cessation of ongoing responses, and resisting distraction or disruption by competing events. These include the Stroop color-word interference task, where the participant must override the prepotent response to read the word instead of say the color; and the stop-signal task where the participant is required to stop a just-begun or well-along-the-way motor response, and the delay of gratification paradigm where participants have a choice of a smaller immediate reward or a later, larger reward.

Cross-References

▶ Social Inhibition

References and Readings

Bargh, J. A. (1994). The four horsemen of automaticity: Awareness, intention, efficiency, and control in social cognition. In *Handbook of Social Cognition: Basic Processes* (Vol. 1, pp. 1–40). Hillsdale, NJ: Erlbaum.

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin, 121*(1), 65.

Fuster, J. M. (1997). *The prefrontal cortex*. Philadelphia: Lippincott-Raven.

Hall, P. A., & Fong, G. T. (2007). Temporal self-regulation theory: A model for individual health behavior. *Health Psychology Review, 1*(1), 6–52.

Hasher, L., & Zacks, R. T. (1979). Automatic and effortful processes in memory. *Journal of Experimental Psychology General, 108*(3), 356–388.

Muraven, M., & Baumeister, R. F. (2000). Self-regulation and depletion of limited resources: Does self-control resemble a muscle? *Psychological Bulletin, 126*(2), 247–259.

Russell, W. R. (1948). Functions of the frontal lobes. *Lancet, 1*(6497), 356.

Shallice, T., & Burgess, P. (1991). Higher-order cognitive impairments and frontal lobe lesions in man. In H. S. Levin & H. M. Eisenberg (Eds.), *Frontal lobe function and dysfunction* (pp. 125–138). New York: Oxford University Press.

Shallice, T., & Burgess, P. (1993). Supervisory control of action and thought selection. In A. Baddeley & L. Weiskranntz (Eds.), *Attention: Selection, Awareness, and Control* (pp. 171–187). Oxford: Oxford University Press.

Behavioral Intention

▶ Intention

Behavioral Intervention

▶ Behavior Modification
▶ Lifestyle, Modification

Behavioral Intervention Technologies

▶ eHealth and Behavioral Intervention Technologies

Behavioral Medicine

Marc D. Gellman
Behavioral Medicine Research Center, Department of Psychology, University of Miami, Miami, FL, USA

Definition

Behavioral medicine is an interdisciplinary field concerned with the development and integration of sociocultural, psychosocial, behavioral, and biomedical knowledge and techniques relevant to the understanding of health and illness and the application of this knowledge and these techniques to disease prevention, health promotion, etiology, diagnosis, treatment, and rehabilitation. The original definition of the field of behavioral medicine was developed at the Yale Conference on Behavioral Medicine and later published by Gary Schwartz and Stephen Weiss (1977). Since that time, there have been various refinements to the definition as reflected in the preceding definition.

Neal Miller (1909–2002), an American psychologist and recipient of the National Medal of Science (1964), is often credited as being the founder of behavioral medicine. He made significant contributions to our understanding of the relationship between reinforcement mechanisms and the control of autonomic behavior, and in pioneering the field of biofeedback, which is used successfully today to treat a variety of medical conditions.

Description

One of the earliest articles discussing issues that now fall within the discipline of behavioral medicine was published in 1956 in the *Journal of Medical Education*, and entitled "Premedical school education in the social and behavioral sciences" (Lidz & Pilot, 1956). The first book title to include the term was published in 1973 (Birk, 1973). The *Journal of Behavioral Medicine* was first published by Plenum in 1978 and is now published by Springer. A Medline search of the term behavioral medicine found over 27,000 references, clear evidence that the growth in this field has been enormous.

The discipline's first professional organization, the Society of Behavioral Medicine (SBM), was founded in 1979 and headquartered in the United States. In the following years, similar professional organizations were founded in Europe and Asia. The late 1980s saw the birth of the International Society of Behavioral Medicine (ISBM), which initially linked together six member societies. These organizations have grown substantially over the past two decades. The ISBM reports over 25 affiliated member societies, including those in Australia, Brazil, Chile, China, Denmark, Finland, Germany, Hungary,

Italy, Japan, Mexico, the Netherlands, Norway, Romania, Slovakia, South Africa, South Korea, Spain, Sweden, Thailand, the United Kingdom, and Venezuela. Additional societies are continuously being formed and joining the ISBM federation.

The SBM holds annual meetings, and the ISBM holds meetings every 2 years (the planning is considerably more complex given the very diverse membership and the international locations). The active participation by members in these meetings is reflected in the excellent attendance statistics. Approximately 1,300 attendees participate in the annual SBM meetings, with approximately 1,000 attendees participating in the ISBM's biannual International Congress of Behavioral Medicine. The first International Congress was held in Uppsala, Sweden, in 1990. Subsequent meetings have been held in Hamburg, Germany (1992); Amsterdam, the Netherlands (1994); Washington DC, USA (1996); Copenhagen, Denmark (1998); Brisbane, Australia (2000); Helsinki, Finland (2002); Mainz, Germany (2004); Bangkok, Thailand (2006); Tokyo, Japan (2008); and Washington DC, USA (2010).The 2012 meeting will be held in Budapest, Hungary.

Further evidence of the growth of the discipline of behavioral medicine is provided by the fact that training in this field can be found in universities around the world, ensuring that the next generation of researchers and practitioners will be trained by current experts. Before going on to specialize in behavioral medicine research or clinical practice, individuals often receive their terminal degrees in disciplines such as medicine, public health, nursing, and psychology.

In 2004, the Institute of Medicine's Committee on Behavioral and Social Sciences in Medical School Curricula (Institute of Medicine Report, 2004) issued the report "Improving Medical Education: Enhancing the Behavioral and Social Sciences Content of Medical School Curricula." This report identified six major domains of knowledge that should be represented in undergraduate medical education. They include: (1) mind-body interactions in health and disease, (2) patient behavior, (3) physician role and behavior, (4) physician-patient interactions, (5) social and cultural issues in health care, and (6) health policy and economics.

The Liaison Committee on Medical Education (LCME), the nationally recognized accrediting authority for medical education programs leading to the M.D. degree in United States and Canadian medical schools, now requires that the curriculum of a medical education program must include behavioral and socioeconomic topics in addition to basic science and clinical disciplines. Furthermore, the medical education program must demonstrate its ability to provide students with an understanding of the manner in which people of diverse cultures and belief systems perceive health and illness and respond to various symptoms, diseases, and treatments.

This diversity is a tremendous strength in this interdisciplinary field.

Cross-References

- ▶ Health Psychology
- ▶ International Society of Behavioral Medicine
- ▶ Society of Behavioral Medicine

References and Readings

Birk, L. (Ed.). (1973). *Biofeedback: Behavioral medicine*. New York: Grune and Stratton.

Institute of Medicine Report. (2004). *Improving medical education: Enhancing the behavioral and social science content of medical school curricula*. Washington, DC: National Academies Press.

Lidz, T., & Pilot, M. L. (1956). Premedical school education in the social and behavioral sciences. *Journal of Medical Education, 31*(10 Part 1), 692–696.

Schwartz, G., & Weiss, S. (1977). What is behavioral medicine? *Psychosomatic Medicine, 39*(6), 377–381.

Behavioral Oncology

- ▶ Cancer: Psychosocial Treatment

Behavioral Sciences at the Centers for Disease Control and Prevention

Dana Brimmer[1] and Emily Zielinski-Gutierrez[2]
[1]Division of High-Consequence Pathogens and Pathology, Centers for Disease Control and Prevention, McKing Consulting Corporation, Atlanta, GA, USA
[2]Division of Vector-Borne Diseases, Centers for Disease Control and Prevention, Ft. Collins, CO, USA

Synonyms

Centers for Disease Control and Prevention

Basic Information

Behavioral science is an integral part of the United States (US) Centers for Disease Control and Prevention (CDC), an agency under the Department of Health and Human Services, which is the lead public health organization for the United States. The Communicable Disease Center, as it was first known, was a unit of the Public Health Service founded in Atlanta, Georgia, on July 1, 1946 (Centers for Disease Control and Prevention [CDC], 1996). Its mission was to fight communicable diseases, in particular malaria. The mandate to eradicate malaria by eliminating mosquitoes stemmed from the Malaria Control in Wartime Areas agency during the Second World War, and thus the agency originally employed more engineers and entomologists than public health doctors (Centers for Disease Control and Prevention [CDC], 2011a). The agency mission eventually grew beyond communicable diseases to include the prevention of disease, injury, and disability, promotion of good health, and preparation for new public health threats. As a result, the name was changed in 1970 to the Centers for Disease Control, with the words "and Prevention" added in 1992 (CDC, 1996).

Today the agency is responsible for public health planning, research, and prevention of infectious and chronic diseases, occupational health, health statistics, and the health component of national emergencies, from hurricanes to natural outbreaks to bioterrorism. CDC is comprised of the Center for Global Health, National Institute for Occupational Safety and Health, and 10 different offices (Centers for Disease Control and Prevention [CDC], 2011b). Within each office, there are national centers, divisions, branches, and programs, for example, the Office of Infectious Diseases houses the National Center for Emerging and Zoonotic Infectious Diseases, to which the Division of High-Consequence Pathogens and Pathology, and Chronic Viral Disease Branch belongs (CDC, 2011b). While both CDC and the US National Institutes of Health (NIH) fall under the U.S. Department of Health and Human Services, CDC engages in disease investigation and epidemiology, public health service – such as diagnostic reference services and compilation of reportable disease statistics – and applied prevention and response, as compared to the medical research agency objectives of the NIH. While CDC does provide extramural funding to health departments and service organizations, and some research funding to universities and other organizations, CDC extramural funding is markedly lower than that provided via NIH.

Behavioral scientists at the CDC work in a variety of public health areas such as health communication, HIV, autism, injury, chronic and infectious diseases, and birth defects (Centers for Disease Control and Prevention [CDC], 2006). Although the behavioral sciences were not formally incorporated at the CDC until the 1980s, this branch of science has become increasingly important to control and prevent both chronic and infectious diseases (CDC, 2006). In 1995, social and behavioral scientists at the CDC established the Behavioral and Social Sciences Working Group (BSSWG) to bring awareness to the fields in which the behavioral sciences contribute (CDC, 2006). Today, the group has approximately 700 members with oversight by the Office of the Associate Director for Science (CDC, 2006).

Behavioral scientists bring with them the research methods from psychology, sociology,

anthropology, and communications, which allow scientists to look at the intersecting impact of the environment, culture, and sociodemographic factors on public health problems. As noted in a 2006 article in CDC's Morbidity and Mortality Weekly Report, behavioral scientists use "qualitative, quantitative, or multiple methods to explore the effects of behavioral, social, and cultural factors on public health problems." Using a mixed research approach that includes qualitative and quantitative methods, behavioral science provides insight into the depth and breadth of public health problems.

CDC behavioral scientists bring experience in survey construction and implementation, often working collaboratively with epidemiologists and other staff. The Behavioral Risk Factor Surveillance System (BRFSS), which monitors behavioral risk factors that influence health outcomes, is a good example of behavioral science at work in the world of survey design (Centers for Disease Control and Prevention [CDC], 2011c). This annual survey, conducted by CDC, collects behavioral health data in all 50 states, the District of Columbia, Puerto Rico, the US Virgin Islands, and Guam. Analysis of BRFSS data informs health policy and prioritizes resources for public health problems. Many behavioral scientists also specialize in the field of evaluation, which allows CDC to critically evaluate whether programs are reaching the targets set and permits a process of continual refinement to meet the community and programmatic needs.

Behavioral scientists at CDC combine biomedical knowledge with systematically gathered information about communities to construct appropriate, effective interventions and health messages. Behavioral scientists can identify how best to implement interventions and evaluate outcomes to allow for sustainable and cost-effective programs. The role of behavioral scientists at CDC can be illustrated through the public health issues of the human immunodeficiency virus (HIV), vaccine safety, and chronic fatigue syndrome.

The importance of the role played by professionals who study human behavior and who suggest ways to intervene in human practices was exemplified by CDC's response to the epidemic of HIV, which involved engaging populations who were at risk for HIV infection in a process of behavior change. The fact that behaviors such as sexual activity and drug use were sensitive and often covert emphasized the need for qualitative and quantitative research and creative methodologies. For example, Semaan et al., in a meta-analysis looked at the effects of HIV prevention in drug users and found interventions with this population significantly reduced risky sexual behaviors (Semaan, Des Jarlais, et al., 2002; Semaan, Kay, et al., 2002).

Conducting needs assessments through focus groups and individual interviews within target communities are examples of how behavioral scientists get involved in the formative phase of interventions, such as in the area of vaccine safety. Outbreaks of measles and pertussis in the USA attest to the public health impact of lowered vaccination rates and yet there are population groups in which resistance to childhood vaccination is prominent (Feiken et al., 2000). In the case of childhood vaccination, members of the public are often challenged to interpret potentially frightening information, and health professionals are at a loss to comprehend why people would reject lifesaving tools. Behavioral medicine methodologies can work between these views, seeking ways to translate concerns, information, and perspectives of the community so policy makers and education experts can develop effective health communication interventions that meet the goals of public health and constituents.

An example of integrating behavioral science and epidemiology at the CDC is the chronic fatigue syndrome (CFS) program. This program has conducted several population-based studies as well as a general clinic research study to assess the prevalence and incidence of CFS, risk factors, and biological aspects associated with the condition. From the behavioral science perspective, the program was the first to publish research in the USA evaluating healthcare providers' perceptions, knowledge, attitudes, and beliefs on CFS. For example, one study measured how physicians and healthcare providers perceive CFS and how their perceptions may affect the diagnosis and management of the illness (Brimmer, Fridinger, Lin, & Reeves, 2010). A separate study examining continuing medical education showed that

when targeting healthcare providers at conferences, the conference size, theme, and mode of education courses may increase education efforts, which help direct resources for future initiatives (Brimmer, McCleary, Lupton, Faryna, & Reeves, 2009).

CDC has a rich history of using behavioral scientists and behavioral science methods to prevent, control, and conduct surveillance on infectious and chronic diseases. The advent of the BSSWG has further augmented the role of behavioral science within the CDC while increasing communication and strengthening agency objectives. Whether behavioral scientists work to prevent infectious disease or promote healthy lifestyles to reduce chronic disease morbidity, they use the fundamentals of evidence-based research, and quantitative and qualitative methods to meet public health goals.

Cross-References

- Behavior
- Behavior Change
- Chronic Fatigue Syndrome
- Epidemiology
- Fatigue
- Health Communication
- Infectious Diseases
- Methodology
- Public Health
- Qualitative Research Methods
- Research Methodology
- Surveys

References and Readings

Brimmer, D. J., Fridinger, F., Lin, J. M., & Reeves, W. C. (2010). U.S. healthcare providers' knowledge, attitudes, and beliefs concerning chronic fatigue syndrome. *BMC Family Practice, 21*(11), 28.

Brimmer, D. J., McCleary, K. K., Lupton, T. A., Faryna, K. M., & Reeves, W. C. (2009). Continuing medical education challenges in chronic fatigue syndrome. *BMC Medical Education, 2*(9), 70. PMID:9954535.

Centers for Disease Control and Prevention. (1996). History of CDC. *Morbidity and Mortality Weekly Report, 45*, 526–528.

Centers for Disease Control and Prevention. (2006). Behavioral and social sciences and public health at CDC. *Morbidity and Mortality Weekly Report, 55* (Suppl. 2), 14–16.

Centers for Disease Control and Prevention. (2011a). *Our history, our story*. Retrieved from http://www.cdc.gov/about/history/ourstory.htm

Centers for Disease Control and Prevention. (2011b). *CDC organization*. Retrieved from http://cdc.gov/about/organization/cio.htm

Centers for Disease Control and Prevention. (2011c). *Behavioral risk factor surveillance system: Turning information into health*. Retrieved from http://www.cdc.gov/BRFSS/

Feiken, D. R., Lezotte, D. C., Hamman, R. F., Salmon, D. A., Chen, R. T., & Hoffman, D. E. (2000). Individual and community risk of measles and pertussis associated with personal exemptions to immunization. *Journal of the American Medical Association, 284*(24), 3145–3150.

National Institutes of Health. (2011). *About NIH*. Retrieved from http://www.nih.gov/about

Semaan, S., Des Jarlais, D. C., Sogolow, E., Johnson, W., Hedges, L., Ramirez, G., et al. (2002). A meta-analysis of the effect of HIV prevention interventions on the sex behaviors of drug users in the United States. *Journal of Acquired Immune Deficiency Syndromes, 30*(Suppl. 1), S73–S93.

Semaan, S., Kay, L., Strouse, D., Sogolow, E., Mullen, P., Neumann, M., et al. (2002). A profile of U.S.-based trials of behavioral and social interventions for HIV risk reduction. *Journal of Acquired Immune Deficiency Syndromes, 30*(Suppl. 1), S30–S50.

Behavioral Sleep Medicine

Wendy Troxel[1] and Michelle Drerup[2]
[1]Psychiatry and Psychology, University of Pittsburgh, Pittsburgh, PA, USA
[2]Clinical Assistant Professor of Medicine, Sleep Disorders Center Neurological Institute, Cleveland Clinic, Cleveland, OH, USA

Definition

The field of behavioral sleep medicine (BSM) is a burgeoning, multidisciplinary specialty that represents the integration of clinical sleep medicine and health psychology. Although the field of BSM was initiated by psychologists, individuals from other related disciplines, including nursing,

psychiatry, and general medicine, have also contributed to the growth and diversity of the field. In general, BSM specialists focus on the identification and treatment of the psychological or behavioral factors that contribute to the development and/or maintenance of sleep disorders, including factors that may influence adherence to prescribed sleep treatments. In particular, BSM techniques have been applied to the treatment of nightmares (i.e., Imagery Rehearsal Therapy; Hasler & Germain, 2009; Moore & Krakow, 2007), narcolepsy and idiopathic hypersomnias (Garma & Marchand, 1994; Mullington & Broughton, 1993), and adherence to continuous positive airway pressure treatment of obstructive sleep apnea (Aloia, Arnedt, Riggs, Hecht & Borrelli, 2004; Means & Edinger, 2007). This entry will focus on the principles of behavioral treatment of insomnia, as this is arguably the most well-known and rigorously tested application of BSM techniques.

Description

Cognitive behavioral therapy for insomnia (CBT-I) involves a diverse set of prescriptions designed to improve sleep consolidation and quality by modifying thoughts and behaviors that interfere with sleep. These treatments can be administered in individual or group formats, and typically range from 4 to 8 sessions in length. Evidence suggests that these treatments are preferred by patients and have consistent strong short-term and long-term efficacy (Irwin, Cole, & Nicassio, 2006; Morin et al., 2006) with few apparent side effects.

Techniques Utilized in CBT-I

Stimulus control therapy (Bootzin, Epstein, & Wood, 1991; Morin et al., 1999) aims to reinforce associations between sleepiness, sleep, and the sleep environment. The patient is instructed to go to bed only when feeling sleepy, and to use the bed and bedroom for sleep and sex only. If awake in bed for extended periods of time (e.g., 20 min or longer), the individual is instructed to get out of bed and leave the bedroom until feeling sleepy again.

Sleep restriction therapy is designed to increase sleep efficiency and consolidate sleep by restricting the time spent in bed, only to the amount of time the patient is actually sleeping (Spielman, Saskin, & Thorpy, 1983, 1987). Sleep restriction is often associated with slight-to-moderate sleep deprivation, which increases sleepiness, and enhances the ability to fall asleep and to maintain sleep. Given that sleep restriction may lead to increased daytime sleepiness (temporarily, usually), patients should be cautioned about operating machinery of performing duties that require high levels of alertness. This strategy is not a good option for patients that already report excessive daytime sleepiness symptoms, which may be due to untreated obstructive sleep apnea or other conditions, or patients that have a history of mania/hypomania.

Relaxation techniques aim to reduce physical and emotional tensions that are incompatible with sleep (Coursey, Frankel, Gaarder, & Mott, 1980; Hauri, 1991; Jacobs et al., 1993). Several specific relaxation techniques have been evaluated for insomnia, including autogenic training, progressive muscle relaxation, and biofeedback.

Cognitive therapy is based on the premise that maladaptive thoughts and beliefs about sleep and the consequences of sleep loss (e.g., I can't function without 8 h of sleep) increase tension and arousal, which perpetuates insomnia. In turn, cognitive techniques aim to help patients identify and correct these maladaptive thoughts and beliefs about sleep (Harvey, 2002; Harvey, Tang, & Browning, 2005).

Sleep hygiene refers to practices, habits, and environmental factors that facilitate getting good quality sleep. Exercising, having a pre-bedtime "wind-down" routine, avoiding stimulants and naps, and limiting alcohol intake are examples of behaviors that may enhance sleep quality. Sleep hygiene has limited efficacy as a stand-alone treatment for insomnia (Lacks & Morin, 1992); however, it is often useful in conjunction with other behavioral interventions.

Summary

Behavioral sleep medicine is a burgeoning, multidisciplinary field that has developed a diverse set

of psychological and behavioral treatments to treat sleep disorders. As a field, BSM has actively promoted the dissemination of empirically supported treatments, with the most solid evidence base in support of cognitive-behavioral interventions for insomnia. Challenges for this field will be to continue to train and accredit BSM providers, to ensure proper reimbursement for services, and to continue to develop empirical support for BSM techniques in diverse populations.

Cross-References

- ▶ Insomnia
- ▶ Sleep

References and Readings

Aloia, M. S., Arnedt, J. T., Riggs, R. L., Hecht, J., & Borrelli, B. (2004). Clinical management of poor adherence to CPAP: Motivational enhancement. *Behavioral Sleep Medicine, 2*, 205–222.

Bootzin, R. R., Epstein, D., & Wood, J. M. (1991). Stimulus control instructions. In P. J. Hauri (Ed.), *Case studies in insomnia* (pp. 19–28). New York: Plenum Publishing.

Coursey, R. D., Frankel, B. L., Gaarder, K. R., & Mott, D. E. (1980). A comparison of relaxation techniques with electrosleep therapy for chronic, sleep-onset insomnia a sleep-EEG study. *Biofeedback and Self-Regulation, 5*, 57–73.

Garma, L., & Marchand, F. (1994). Non-pharmacological approaches to the treatment of narcolepsy. *Sleep, 17*, S97–S102.

Harvey, A. G. (2002). A cognitive model of insomnia. *Behaviour Research and Therapy, 40*, 869–893.

Harvey, A. G., Tang, N. K., & Browning, L. (2005). Cognitive approaches to insomnia. *Clinical Psychology Review, 25*, 593–611.

Hasler, B. P., & Germain, A. (2009). Correlates and treatments of nightmares in adults. *Sleep Medicine Clinics, 4*, 507–517.

Hauri, P. J. (1991). Sleep hygiene, relaxation therapy, and cognitive interventions. In P. J. Hauri (Ed.), *Case studies in insomnia* (pp. 65–84). New York: Plenum Publishing.

Irwin, M. R., Cole, J. C., & Nicassio, P. M. (2006). Comparative meta-analysis of behavioral interventions for insomnia and their efficacy in middle-aged adults and in older adults 55+ years of age. *Health Psychology, 25*, 3–14.

Jacobs, G. D., Rosenberg, P. A., Friedman, R., Matheson, J., Peavy, G. M., Domar, A. D., et al. (1993). Multifactor behavioral treatment of chronic sleep-onset insomnia using stimulus control and the relaxation response. A preliminary study. *Behavior Modification, 17*, 498–509.

Lacks, P., & Morin, C. M. (1992). Recent advances in the assessment and treatment of insomnia. *Journal of Consulting and Clinical Psychology, 60*, 586–594.

Means, M. K., & Edinger, J. D. (2007). Graded exposure therapy for addressing claustrophobic reactions to continuous positive airway pressure: A case series report. *Behavioral Sleep Medicine, 5*, 105–116.

Moore, B. A., & Krakow, B. (2007). Imagery rehearsal therapy for acute posttraumatic nightmares among combat soldiers in Iraq. *The American Journal of Psychiatry, 164*, 683–684.

Morin, C. M., Bootzin, R. R., Buysse, D. J., Edinger, J. D., Espie, C. A., & Lichstein, K. L. (2006). Psychological and behavioral treatment of insomnia: An update of recent evidence (1998–2004). *Sleep, 29*, 1398–1414.

Morin, C. M., Hauri, P. J., Espie, C. A., Spielman, A. J., Buysse, D. J., & Bootzin, R. R. (1999). Nonpharmacologic treatment of chronic insomnia. An American Academy of Sleep Medicine review. *Sleep, 22*, 1134–1156.

Mullington, J., & Broughton, R. (1993). Scheduled naps in the management of daytime sleepiness in narcolepsy-cataplexy. *Sleep, 16*, 444–456.

Spielman, A. J., Saskin, P., & Thorpy, M. J. (1983). Sleep restriction treatment of insomnia. *Sleep Research, 12*, 286.

Spielman, A. J., Saskin, P., & Thorpy, M. J. (1987). Treatment of chronic insomnia by restriction of time in bed. *Sleep, 10*, 45–56.

Behavioral Therapy

Misuzu Nakashima
Hizen Psychiatric Center, Yoshinogari, Kanzaki, Saga, Japan

Synonyms

Behavior modification

Definition

The term behavior therapy was suggested for the first time as the treatment concept that unified all behavior modification of whose foundation was an experiment based on a learning theory by Eysenk, H.J., in 1959. Plural studies and theories

were accumulated, and behavior therapy expanded to a treatment theory, technique, and its coverage afterward. The definition of behavior therapy has become largely extended. Behavior therapy is psychotherapy aimed at behavior modification where focus was assigned to an action, and to the understanding of the problem of the person in structure and the function of the action and form called the context. The characteristic of the behavior therapy is a point which the learning theory arrived at after identifying the problem and making the hypothesis through an experimental study.

Eileen, D.G., indicates that there are seven characteristics of behavior modification:

- Assessment and intervention informed by behavioral principles
- Emphasis on identification of current controlling conditions
- Deemphasize on labeling
- Emphasis on observable, countable responses
- Emphasis on positive, not punitive change method
- Emphasis on measurement of effects
- Rejection of special causative factors related to "problematic" behavior

Description

History

When learning a theory and its techniques, it is important to understand the history of behavior therapy because it is in behavior therapy where various psychological studies and knowledge of the clinical field were interlaced. It is difficult to express it by a word or single thought and theory.

The origin of behavior therapy dates back to behaviorism in the 1920s whose "heart" was on objectivity and scientific analyzes. Watson, J.B., has already applied the principle of respondent conditioning of Pavlov, I., to behavior disorder those days. In the early 1950s, Skinner, B.F., in the USA treated mental patients with operant conditioning and had already used the term behavior therapy. At the same time, Wolpe, J., in South Africa developed a technique called systematic desensitization from a study of neurosis and its cure. In the UK, Eysenk, H.J., performed a case study on neurosis and behavioral disorder using the techniques of experimental psychology. These knowledge and methodologies whose methods and objects are different were integrated to encompass the basic foundations of behavior therapy in the 1950s. Also, applied behavior analysis and neobehavioristic mediational S-R theory which were the representative theories of behavior therapy were also developed by this time. In addition, based on these theories, the techniques such as operant reinforcement, token economy, systematic desensitization method, and flooding were developed. There were only a few theories that could be compared with the theories and the resulting theory and technique of behavior therapy was clear and strong. In the early days, behavior therapy emphasized to put foundations on objectivity and in the scientific theory, and it accomplished remarkable development. Social learning theory was proposed after this, and the current cognitive-behavioral therapy led it. Most of neobehaviorists of that time performed theorization and the inspection through animal experiment, but Bandura, A., brought about the social learning theory that could explain the learning and the modification of social behavior among human beings. By this theory, he suggested observational learning and technique called modeling where there was more focus on cognition than was given the past learning theory. He considered expectation or self-efficacy to be a factor of reinforcement operation in behavior modification.In the 1960s, the rise of criticisms that human cognitive processes cannot be fully grasped through observed data only brought attention to cognition as a factor that effects treatment.

It was during this time that putting together various theories and techniques and every clinical object and purpose to develop a single technique package emerged. Treatment packages became more complicated when techniques were collected from a number of theories. Furthermore, in the USA, Ellis, A., produced rational-emotive therapy, and Beck, S.J., came up with cognitive therapy. They aimed at the intervention on the cognition domain and made remarkable effect on

the treatment of patients with depression. It attracted more attention when patients who went through behavior therapy but did not benefit from it showed remarkable change when introduced to cognitive therapy. Both Ellis and Beck started as therapists in the field of psychoanalysis, and they were both influenced by psychoanalysis, philosophy, behavior psychology, cognitive psychology, and personal construct psychology of Kelly, G.A. They emphasized the importance of the cognition of the patient. Both of them assumed the model that most disorders occur from wrong cognition or wrong process of cognition. Currently, among the models of behavior therapy and the models of the cognitive therapy, there is a difference in positioning of the cognition and action in the treatment. However, researchers and therapists called this using the general term "the cognitive-behavioral therapy," and this has increased popularity worldwide. A lot of these experimental studies are performed and attracted attention as a treatment based on the evidences gathered. In addition, with the increasing social needs, its coverage continues to spread.

Procedure for Treatment

Behavior therapy has many procedures for treatment. I will indicate the basic common features here. The process consists of four parts. The treatment goes on from 1 to 4, but it is necessary to go back to 1 or 2 when therapist gets new data or when treatment is not effective.

1. Assessment: Therapists assess client without labeling or categorizing them as "wimpy" or with "low self-esteem." They concretely collect data about the behavior, thought, feeling, environment, and so on. The goal of intervention is clearly stated between therapist and clients because of the data gathered.
2. Case formulation: Once enough interviews or observations or tests are conducted, the gathered data are analyzed, and relations or patterns are identified. Target behavior (or thought or environment) that has a tendency to change or to affects client's life or has profound influence on other problem is chosen. Case formulation is made with clients and shared.
3. Intervention: Intervention is conducted using appropriate technique.
4. Evaluation: Therapists or/and clients collect quantitative data to examine the effects of intervention. The frequency, magnitude, or duration of behavior, thought, or feeling before and intervention is compared.

Application

Initially, behavior therapy was intended for use in psychiatry or clinical psychology, but it later became useful also for education and other fields such as psychosomatic medicine, physical disease, preventive medicine, public health, and living environment. In the field of psychiatry, behavior therapy is used for the treatment of anxiety disorder (exposure) such as obsessive-compulsive disorder, space phobia and social phobia or the single phobia, and schizophrenia (token economy, Social Skills Training, family behavior therapy). In addition, it is also used in behavior medicine, such as for muscle-contraction headache and hypertensive treatment (biofeedback and various relaxation training) and corpulence (stimulation control, self-control).

Cross-References

- ▶ Applied Behavior Analysis
- ▶ Behavior Change
- ▶ Behavior Modification
- ▶ Behavioral Intervention
- ▶ Behavioral Medicine
- ▶ Behavioral Therapy
- ▶ Classical Conditioning
- ▶ Cognitive Behavioral Therapy (CBT)
- ▶ Cognitive Distortions
- ▶ Cognitive Restructuring
- ▶ Operant Conditioning
- ▶ Self-efficacy
- ▶ Systematic Desensitization

References and Readings

Eysenck, H. J. (1960). *Behavior therapy and the neuroses 1960*. Oxford: The Pergamon Press.

Eysenck, H. J., & Martin, I. (1987). *Theoretical foundations of behavior therapy*. New York: Plenum Press.

Gambrill, E. D. (1977). *Behavior modification -handbook of assessment, intervention, and evaluation*. San Francisco: Jossy-Bass.
Wilson, G. T., & Frank, C. M. (1982). *Contemporary behavior therapy, conceptual and empirical foundations*. New York: Guilford Press.
Wolpe, J. (1969). *The practice of behavior therapy*. New York: Pergamon Press.

Beliefs

Chad Barrett
Department of Psychology, University of Colorado Denver, Denver, CO, USA

Synonyms

Attitudes; Cognitions; Health beliefs

Definition

Beliefs refer to a conviction, or an attitude, that affirms something to be true. Belief involves a mental state of having a particular attitude, stance, or opinion about something. Belief can refer to expectations and assumptions about mundane matters concerning rules in the physical, social, and/or spiritual worlds (e.g., assuming that a chair can support your weight, that it is improper behavior to laugh during a eulogy, or that supernatural beings cause diseases), or they may also refer to existential, ethical, political, philosophical, theological, or scientific matters, among others as well. Many beliefs are the result of past experience (e.g., if I smile when I meet people, then they are more likely to be friendly toward me), cultural influence (e.g., it is wrong to eat pork), and from deliberate and critical reflection. Beliefs are often constructed by observing the behavior of others and by observing the consequences of others' actions. For example, individuals may acquire the belief that seatbelts are not that important if, while growing up, their parents did not wear seatbelts and were never injured in a car accident. Alternatively, if one's parents were seriously injured as a result of not wearing their seatbelt, then one might likely conclude that seatbelts are indeed important. Similarly, beliefs can be transmitted through explicit and implicit instruction from a variety of agents including family, friends, community members, educational and religious institutions, and various forms of media. Further, beliefs are often constructed through a dynamic interaction with other members of the same culture. Some beliefs may receive greater reinforcement, while others may receive greater discouragement or punishment.

Description

Beliefs can potentially have important influences on individuals, groups, and societies. Cognitive-behavioral theory highlights the importance of beliefs in how they may influence a person's mental and physical health. A variety of unhealthy beliefs have been associated with elements of mental health problems. For example, perfectionist beliefs are often associated with anxiety, depression, and anger. Treatment for many mental health-related problems typically involves identifying and challenging unhealthy beliefs and replacing them with healthier and more adaptive beliefs.

Beliefs can affect people's health-related behaviors which in turn affect their health (Wilkinson, Vasudevan, Honn, Spitz, & Chaberlain, 2009). According to the Health Belief Model, people are more likely to engage in health-promoting behaviors if they believe they are susceptible to a certain disease or condition and if they believe that the benefits of engaging in health-promoting behaviors outweigh the challenges to engaging in those behaviors (Rosenstock, Strecher, & Becker, 1988). Bandura (1977, 1986) suggested that individuals' health-related behaviors are influenced by their beliefs about self-efficacy (i.e., the degree to which people believe they are capable of performing a certain behavior or making health-promoting changes in behavior) and whether they expect the positive benefits to outweigh any negative

aspects. According to the Theory of Reasoned Action (Ajzen & Fishbein, 1980), health-related behaviors are primarily influenced by a person's intentions to engage in any particular health-related behavior. These intentions are shaped by attitudes and subjective norms. Attitudes are derived from an individual's beliefs about the consequences of certain health-related behaviors. Subjective norms are derived from individuals' beliefs about how important others think they should behave and their motivations to comply with such beliefs. In addition, according to the Theory of Planned Behavior (Ajzen, 1991), persons' intentions to engage in a particular health-related behavior are affected by their beliefs regarding the extent to which various factors may impede or facilitate performing the health-related behavior. Each theory underscores the impact of belief on health-related behaviors.

Health beliefs often include beliefs about illness, treatment, adherence, self-efficacy, locus of control, and perceptions of one's relationship with health-care providers. A recent meta-analysis (Gherman et al., 2011) examined the association between health beliefs related to diabetes and adherence to treatment for diabetes. Beliefs about self-efficacy, perceiving a positive relationship with health-care providers, and beliefs about the personal consequences of treatment adherence strongly predicted greater adherence to treatment for diabetes among patients. The more adherent patients tended to have greater levels of confidence in their ability to follow medical recommendations, and they expected more meaningfully positive consequences from adhering to treatment. They also viewed their relationships with health-care providers as more positive.

People's beliefs can also affect the likelihood that they will seek medical help when needed and engage in preventative behaviors (Fischer & Farina, 1995; Godin & Conner, 2008). For example, people may be less likely to seek out mental health treatment if they have negative attitudes about mental health services and cultural beliefs that view mental illness as shameful for the individual and the individual's family (Jang, Chiriboga, Herrera, Martinez Tyson, & Schonfeld, 2011). People who have fatalistic views about health (i.e., believe certain health conditions such as cancer cannot be prevented or cured) are less likely to engage in preventative behaviors, seek medical help early, and adhere to treatment recommendations (e.g., Monteros & Gallo, 2011). Certain religious beliefs may also influence help-seeking behavior. People who see health providers as "doing God's work" are more likely to seek help and adhere to treatment. People who see a conflict between medical science and their religious beliefs are sometimes less likely to seek early treatment and/or comply with medical recommendations (Exline & Rose, 2005; Miller and Kelley, 2005).

Sociodemographic variables appear to influence health-related beliefs and behaviors. Generally, people of higher socioeconomic status (SES) typically have more accurate health beliefs and are more likely to engage in healthy behaviors (Wilkinson et al., 2009). This may reflect a variety of the advantages that come with higher SES such as increased access to health care and higher levels of education. Also, compared to men, women tend to report more accurate health beliefs and engage in more health-promoting behaviors and less risky health behaviors. There also appear to be differences between racial and ethnic groups. To summarize the results of one study, European Americans tended to report healthier beliefs and greater medical compliance relative to Asian Americans, Hispanics, and African Americans (Courtenay, Mccreary, & Merighi, 2002). These findings may be related to SES and may partly reflect the advantages of higher SES.

Cross-References

- ▶ Cognitions
- ▶ Cognitive Factors
- ▶ Cognitive Mediators
- ▶ Health Behaviors
- ▶ Health Risk (Behavior)
- ▶ Meaning (Purpose)
- ▶ Norms
- ▶ Religion/Spirituality

- ▶ Religious Social Support
- ▶ Religiousness/Religiosity
- ▶ Theory of Planned Behavior
- ▶ Theory of Reasoned Action

References and Readings

Ajzen, I. (1991). The theory of planned behavior. *Organizational Behavior & Human Decision Processes, 50*, 179–211.

Ajzen, I., & Fishbein, M. (1980). *Understanding attitudes and predicting social behavior*. Englewood Cliffs, NJ: Prentice-Hall.

Bandura, A. (1977). Self-efficacy: Toward a unifying theory of behavioral change. *Psychology Review, 84*, 191–215.

Bandura, A. (1986). *Social foundations of thought and action: A social cognitive theory*. New York: Prentice-Hall.

Courtenay, W. H., Mccreary, D. R., & Merighi, J. R. (2002). Gender and ethnic differences in health beliefs and behaviours. *Journal of Health Psychology, 7*, 219–231.

Exline, J. J., & Rose, E. (2005). Religious and spiritual struggles. In R. F. Paloutzian & C. L. Park (Eds.), *Handbook of the psychology of religion and spirituality* (pp. 435–459). New York: Guilford.

Fischer, E., & Farina, A. (1995). Attitudes toward seeking professional psychological help: A shortened form and consideration for research. *Journal of College Student Development, 36*, 368–373.

Gherman, A., Schnur, J., Montgomery, G., Sassu, R., Veresiu, I., & David, D. (2011). A meta-analysis of health beliefs and diabetes self-care. *The Diabetes Educator, 37*, 392–408.

Godin, G., & Conner, M. (2008). Intention-behavior relationship based on epidemiologic indices: An application to physical activity. *American Journal of Health Promotion, 22*, 180–182.

Jang, Y., Chiriboga, D. A., Herrera, J. R., Martinez Tyson, D., & Schonfeld, L. (2011). Attitudes toward mental health services in Hispanic older adults: The role of misconceptions and personal beliefs. *Community Mental Health Journal, 47*, 164–170.

Miller, L., & Kelley, B. S. (2005). Relationships of religiosity and spirituality with mental health and psychopathology. In R. F. Paloutzian & C. L. Park (Eds.), *Handbook of the psychology of religion and spirituality* (pp. 435–459). New York: Guilford.

Monteros, K. E., & Gallo, L. C. (2011). The relevance of fatalism in the study of Latina's cancer screening behavior: A systematic review of the literature. *International Journal of Behavioral Medicine, 18*, 310–318.

Rosenstock, I. M., Strecher, V. J., & Becker, M. H. (1988). Social learning theory and the health belief model. *Health Education Quarterly, 15*, 175–183.

Wilkinson, A. V., Vasudevan, V., Honn, S. E., Spitz, M. R., & Chaberlain, R. M. (2009). Sociodemographic characteristics, health beliefs, and the accuracy of cancer knowledge. *Journal of Cancer Education, 24*, 58–64.

Bender

- ▶ Binge Drinking

Benefit Evaluation in Health Economic Studies

Amiram Gafni[1] and Stephen Birch[2]
[1]Department of Clinical Epidemiology and Biostatistics, Centre for Health Economics and Policy Analysis, McMaster University, Hamilton, ON, Canada
[2]Clinical Epidemiology and Biostatistics (CHEPA), McMaster University, Hamilton, ON, Canada

Synonyms

Cost-benefit analysis (CBA); Cost-effectiveness analysis (CEA); Cost-utility analysis (CUA); Healthy-years equivalents (HYEs); Quality-adjusted life years (QALYs); Willingness-to-pay (WTP)

Definition

The rationale for economic evaluation of healthcare programs arises from the concepts of scarcity, choice, and opportunity cost. In the presence of scarcity, economic evaluation is about "...ensuring that the *value* of what is gained from an activity outweighs the *value* of what has to be sacrificed" (Williams, 1983).

Three techniques have been used for comparisons of consequences (what is gained) and costs (what is sacrificed) in economic evaluations of healthcare interventions: cost-benefit analysis (CBA), cost-effectiveness analysis (CEA), and cost-utility analysis (CUA). This entry describes

three outcome/consequences valuation techniques: quality-adjusted life-years (QALYs), Healthy-years equivalents (HYEs), and Willingness-to-pay (WTP). Comments on their appropriateness and validity from an economic perspective are provided.

Description

Economic Evaluation and Implications for Outcome Measurement

Where interest lies in solving resource allocation problems (i.e., *economic* evaluations) it is important that the methods used to measure consequences (what is gained) are consistent with the discipline of *economics*. The most commonly cited goal of economic evaluations is to maximize the health-related well-being of the population (i.e., the gains) from available resources. Thus, the methods used to measure health-related well-being must be consistent with the underlying welfare economic theory on which the analysis is based on. The "welfarist" approach is the one most commonly used. An extra-welfarist approach has been suggested, but there are many issues related to whether there is any "extra" in the extra-welfarist approach (Birch & Donaldson, 2003). The requirements of the measurement methods in order that these methods are valid ways of measuring outcomes for use in economic evaluations are briefly described. More details can be found in Gafni & Birch, 1995.

The Internal Structure of Preference Formulation

Under the welfarist approach to economics, an individual's preferences are embodied in that individual's utility function. Thus, for a measure of outcome to be consistent with the welfarist approach it must be consistent with a theory of utility. Users can choose between alternative theories based on how they would like individuals to behave (i.e., based on its normative appeal). Alternatively, they might choose to be guided by the approach that individuals are the best judges of their own welfare and hence choose a theory based on its accuracy in measuring the individual's true preferences, irrespective of how they feel about these preferences.

Attitudes Toward Risk

Preferences can be measured under conditions of certainty or uncertainty. When projects are approved from a societal perspective, it has been suggested that risk to the individual can be ignored and mean values can be used for a measure of outcome, but, health outcomes are intrinsic to individuals and cannot be redistributed among individuals. Hence, social decision-making concerning health outcomes should incorporate individuals' attitudes to risk if it is to reflect individuals' preferences (Ben-Zion & Gafni, 1983).

Aggregation of Individual Preferences

Different models exist for aggregating preferences, each of which is based on restrictions imposed on the set of preferences and/or the aggregation rules. The aggregation of individuals' utilities, necessarily involves attaching weights to the utilities of different individuals or groups (i.e., equity considerations are an intrinsic part of any social utility function). Hence, the calculation of a social utility function must reflect the equity criterion adopted in the analysis. If externalities (i.e., the effect of one person's health status on another person's utility) exist, they should be taken into account when constructing the social utility function.

The Meaning of Validity in Outcome Assessment

The basic concepts that are involved in determining the quality of a measurement are validity and reliability. In addition, measures should be tested to determine whether the measurement task is feasible and acceptable (e.g., clarity of the presentation, length of the interview, etc.). The difference between the "classical" psychometric approach and the economic approach is in the way that the validity of an instrument is determined. In economics, the validity of the instrument stems from the validity of the theory, which the instrument is derived from. It is often the case that researchers introduce additional assumptions, to those underlying the utility theory chosen (e.g., to simplify the measurement process). In this case, the validity of these additional assumptions should also be determined.

Quality-Adjusted Life-Years (QALYs)

The most commonly used measure of outcome in economic evaluations (CUA and CEA) is QALYs. QALYs are computed by adjusting each unit of time by a weight that reflects the quality of life in that unit of time and then discount it. By combining aspects of quality and quantity of life the QALY enables comparison of interventions that affect both of these dimensions. Moreover, it allows comparisons of effects on different dimensions of quality of life (e.g., pain versus hearing). In addition, the QALY is intuitively appealing to decision-makers as it can be "thought of as an equivalent number of years in full health – a number of quality adjusted life years" (Weinstein & Stason, 1977). It is presumably the lack of intuitive appeal that inhibited the use of the utility function directly (i.e., the meaning of a "util" (the unit of measurement) is not so easily understood by decision-makers). "The policy objective underlying the QALY literature is the maximization of the community's health. An individual's health is measured in terms of QALYs, and the community's health is measured as the sum of QALYs" (Wagstaff, 1991).

Although there is much agreement about the structure of the QALY measure (i.e., time durations weighted to reflect their quality of life and discounted), there is no agreement about the methods to be used to measure the weights. Unfortunately, different methods result in different numbers. This implies that the different methods cannot be measuring the same thing. The most commonly used approach is to assume that all individuals are expected utility maximizers (i.e., a theory of choice under uncertainty) and to use the standard gamble (SG) or time trade-off (TTO) approaches to measure these weights directly or indirectly (Drummond, Sculpher, Torrance, O'Brien, & Stoddart,, 2005). However, for QALYs to represent individuals' preferences for health requires additional conditions (i.e., in addition to those required by expected utility theory) to be satisfied (Gafni & Birch, 1995). Many studies have shown that expected utility is descriptively inaccurate and that people violate expected utility in systematic ways (Starmer, 2000). Furthermore, the additional conditions also lack empirical or normative support. Recognizing this fact, there are constant attempts in the literature to redefine QALYs (e.g., Weinstein, Torrance, & McGuire, 2009) or to use other utility theories as the foundation for the QALYs, which result in changing the way in which the weights should be measured (e.g., Bleichrodt & Pinto, 2006). Regardless of the recognition of the major problems associated with the QALY measure and some suggestions on how to deal with them, in empirical application it seems that nothing has changed. It might be that the ease of implementation might explain the lack of change. Researchers and practitioners seem to like simple solutions to complex problems (even if they might be wrong).

In terms of the implications for social preferences, as mentioned above, the health-related well-being of the community is calculated by summing individuals' QALY values. This implied the equity assumption that a QALY is a QALY regardless of who gains it and who loses it. This assumption has been widely criticized, and attempts are made to address this issue by developing a social weighing system (e.g., Dolan & Tsuchiya, 2006). The fact that the QALY metric is not likely to represent individuals' true preferences implies that a simple aggregation of QALYs is not likely to represent the community preference. A simple example is the case of a community of identical individuals. We need to ask only one individual for her preferences to know the community's preferences. But if the method used to measure the preferences does not represent the individual's true preferences, it cannot represent the community's preferences.

Healthy-Years Equivalent (HYE)

The HYE provides a user-friendly metric that is needed to improve communication as explained above. Unlike QALYs that mean different things to different people (and hence the need to distinguish the HYE from the QALY), the HYE means only one thing – it is a utility-based concept, derived from the individual's utility function by measuring the number of years in full health, holding other arguments in the utility function

constant, that produces the same level of utility to the individual as produced by the potential lifetime health profile following a given intervention (Gafni & Birch, 1997). The measurement of HYE requires that individuals be allowed to reveal their true preferences, which is consistent with the welfarist approach. It also seems reasonable when asking the public to assist in the determination of healthcare priorities, to choose measurement techniques that allow the public to reveal their true preferences. If not, why do we bother asking them at all?

Can an algorithm be developed to measure HYE that (a) does not require additional assumptions (i.e., in addition to the assumptions underlying the utility theory chosen) and (b) is feasible to use with the intended subjects (e.g., the number and complexity of questions asked is not too burdensome)? The concept of HYE does not require that an individual subscribe to expected utility theory. Any type of utility theory can be used as a basis for generating algorithms to measure HYE, and the choice of utility theory will determine the method of measurement. The only requirement is that preferences for health profiles are measured under uncertainty. For an individual who maximizes expected utility an algorithm that describes how to measure HYE without the additional assumptions of the QALY model is described in Johannesson (1995). In terms of feasibility of the HYE measure, "the jury is still out." Measuring HYE is likely to involve greater response burden, mainly in terms of the number of questions being asked. The need to simplify the assessment task (i.e., reduce the number of questions asked to generate HYE scores) is most evident in the case of large decision trees. This is because the number of different potential lifetime health profiles is likely to be large. Recently a method was suggested, which uses conjoint analysis that makes it feasible to generate the large number of HYE scores required (Johnson, Hauber, & Ozdemir, 2009).

Willingness-to-Pay (WTP)

The maximum amount that an individual is willing-to-pay is the measure typically used in cost-benefit analysis (CBA). WTP is appealing because it has its theoretical foundation in welfare economics and in particular, in the potential Pareto improvement criterion (Drummond et al., 2005). This criterion recognizes that often a policy, resulting in resource allocation, will create gainers and losers in welfare. But if the gainers could fully compensate the losers and remain better off themselves after the change, then society as a whole has benefited. Because compensation does not actually have to occur, it is called "potential" improvement. The measurement of benefit (gains) is the maximum that an individual is willing-to-pay for a good or service. The measurement of cost (losses) is the minimum amount that an individual is willing-to-accept (WTA) as compensation for the loss. A program is worth doing (i.e., cost beneficial) if the total WTP exceeds the total WTA.

Unlike CEA and CUA where the cost and consequences are measured using different units, in CBA the costs and consequences are measured using commensurate (typically monetary) units. This makes it easier to determine if a program is worth implementing or not. WTP is also appealing because it allows intersectoral comparisons, allows trade-offs between health and other goods, can capture externalities, the most sensitive outcome, and can be modified to capture the unique nature of health as a good (Gafni, 1997). The main objection to the WTP approach is that using a measure that is heavily influenced by ability to pay (i.e., WTP) will lead to evaluations favoring the rich. However, this objection was never tested empirically. Donaldson et al. (2002) show that the same income-distributional concerns apply to non-monetary valuations of health consequences, to measurement of costs, and to the decision rules of CUA/CEA. Hence, adopting CUA/CEA over CBA cannot be justified on the basis of avoiding distributional considerations.

A WTP instrument typically has two components: A description of the programs (or interventions) to be valued and a payment method to elicit an individual's WTP for the program in question. O'Brien and Gafni (1996) developed a set of questions to help researchers and practitioners

to determine how to design a proper WTP instrument for their evaluation. Examples of different approaches to elicit WTP values can be found in Gafni (1997), Drummond et al. (2005), and Donnaldson, Mason, and Shackley (2006).

References and Readings

Ben-Zion, U., & Gafni, A. (1983). Evaluation of public investment in health care: Is the risk irrelevant? *Journal of Health Economics, 2*, 161–165.

Birch, S., & Donaldson, C. (2003). Valuing the benefits and costs of health care programmes: Where's the 'extra' in extra-welfarism? *Social Science & Medicine, 56*, 1121–1133.

Bleichrodt, H., & Pinto, J. L. (2006). Conceptual foundations for health utility measurement. In A. M. Jones (Ed.), *The Elgar companion to health economics* (pp. 347–358). Cheltenham, UK: Edward Elgar.

Dolan, P., & Tsuchiya, A. (2006). The elicitation of distributional judgements in the context of economic evaluations. In A. M. Jones (Ed.), *The Elgar companion to health economics* (pp. 382–391). Cheltenham, UK: Edward Elgar.

Donaldson, C., Birah, S., & Gafni, A. (2002). The distribution problem in economic evalution: income and the valuation of costs and consequences of health care programmes. *Health Economics, 11*, 55–70.

Donaldson, C., Mason, H., & Shackley, P. (2006). Contingent valuation in health Care. In A. M. Jones (Ed.), *The Elgar companion to health economics* (pp. 392–404). Cheltenham, UK: Edward Elgar.

Drummond, M. F., Sculpher, M. J., Torrance, G. W., O'Brien, B. J., & Stoddart, G. L. (2005). *Methods for the economic evaluation of health care programmes* (3rd ed.). Oxford, UK: Oxford University Press.

Gafni, A. (1997). Willingness-to-pay in the context of an economic evaluation of healthcare programs: Theory and practice. *The American Journal of Managed Care, 3*(Supplement), S21–S32.

Gafni, A., & Birch, S. (1995). Preferences for outcomes in economic evaluations: An economic approach to addressing economic problems. *Social Science & Medicine, 40*, 767–776.

Gafni, A., & Birch, S. (1997). QALYs anf HYEs: Spotting the differences. *Journal of Health Economics, 16*, 601–608.

Johannesson, M. (1995). The ranking properties of healthy years equivalents and quality adjusted life years under certainty and uncertainty. *International Journal of Health Technology Assessment in Health Care, 11*, 40–48.

Johnson, F. R., Hauber, B., & Ozdemir, S. (2009). Using conjoint analysis to estimate healthy years equivalents for acute conditions: An application to vasomotor symptoms. *Value in Health, 12*, 146–152.

O'Brien, B., & Gafni, A. (1996). When do the 'dollars' make sense? Toward a conceptual framework for contingent valuation studies in health care. *Medical Decision Making, 16*, 288–299.

Starmer, C. (2000). Developments in non-expected utility theory: A hunt for descriptive theory of choice under uncertainty. *Journal of Economic Literature, 28*, 332–382.

Wagstaff, A. (1991). QALYs and the equity efficiency trade-off. *Journal of Health Economics, 10*, 21.

Weinstein, M. C., & Stason, W. B. (1977). Foundations of cost effectiveness analysis for health and medical practices. *The New England Journal of Medicine, 296*, 716–721.

Weinstein, M. C., Torrance, G. W., & McGuire, A. (2009). QALYs: The basics. *Value in Health, 12*(Supplement 1), S5–S9.

Williams, A. (1983). The economic role of 'health indicators'. In G. Teeling Smith (Ed.), *Measuring the social benefits of medicine* (pp. 63–67). London: Office of Health Economics.

Benefit Finding

Kristen Riley
Department of Psychology, University of Connecticut, Storrs, CT, USA

Synonyms

Adversarial growth; Posttraumatic growth; Stress-related growth

Definition

Benefit finding refers to a reported positive life change resulting from the struggle to cope with a challenging life event such as trauma, illness, or other negative experiences. The positive psychology movement has recently driven a shift toward an emphasis on the positive consequences of negative events. The discovery of benefits by individuals experiencing adversity is well documented and plays a prominent role in theories of cognitive adaptation to threatening circumstances and in emerging literature on posttraumatic growth and psychological thriving.

It is highly prevalent, has been studied in a variety of settings, has an association with personality and emotional well-being, and can predict health outcomes month and even years later. Benefit finding enhances emotional and physical adaptation in the face of adversity.

By definition, some view benefit finding as searching for benefits, that is, as a verb (Tennen & Affleck, 2009), while others measure it as a form of growth, as a noun. Additionally, benefit finding, the phenomenon of positive life changes that people report following their struggle to cope with negative life experiences, is often also referred to as stress-related growth, adversarial growth, or posttraumatic growth. While some have attempted to define these constructs as separate and distinct, others have used the terms interchangeably, and there is a consensus for the need to more narrowly define these terms for consistency across the field (Park, Lechner, Antoni, & Stanton, 2009).

Tennen and Affleck (2002) are careful to distinguish benefit finding from other terms as a perception of positive change rather than veridical change. Those who choose to view benefit finding as veridical change have measurement difficulties, as most measures used to assess growth are based on self-report, and therefore are inherently only perceptions of change.

Benefit finding manifests itself in a variety of ways. Individuals often report a newfound appreciation for their strength and resilience. Some benefit from negative experience in a social context, claiming that their relationships are stronger, that they feel more emotionally connected, and that they feel more compassionate or altruistic. Others emphasize developing the ability to recognize the important parts of life, redirecting priorities, and even openness to religion and spirituality. While there is a focus on positive psychological benefits, research suggests that benefit finding may also have a positive impact on physical health (Bower, Low, Moskowitz, Sepah, & Epel, 2008).

Benefit finding has also been conceptualized in myriad ways: as cognitive reappraisal, as a personality characteristic, and even as a coping mechanism. However, there has been an emphasis on the distinction between active efforts to recall benefits as a coping strategy during difficult times, deemed benefit reminding, and benefit finding (i.e., benefit-related cognitions as adaptive beliefs).

Benefit finding and stress-related growth have been studied mainly in the context of health and medical illness. Medical illness often induces feelings of uncertainty, fear, and loss. Growth and benefit finding is also widely reported as a result of illness. Research has proliferated from early studies on the impact of myocardial infarction to interest in the role of benefit finding and growth in cancer, HIV, lupus, infertility, arthritis, psoriasis, and other health problems. However, growth varies in the context of medical illness due to variation in symptom onset, etiology, threat to life, recovery trajectory, chronicity, permanence of change, and life context. Additional research is required to understand how benefit finding and growth function along these dimensions.

Cross-References

- ▶ Coping
- ▶ Hardiness
- ▶ Optimism
- ▶ Perceived Benefits
- ▶ Positive Psychology
- ▶ Posttraumatic Growth
- ▶ Resilience

References and Readings

Affleck, G., & Tennen, H. (1996). Construing benefits from adversity: Adaptational significance and dispositional underpinnings. *Journal of Personality, 64*(4), 899–922.

Bower, J. E., Low, C. A., Moskowitz, J. T., Sepah, S., & Epel, E. (2008). Benefit finding and physical health: Positive psychological changes and enhanced allostasis. *Social and Personality Psychology Compass, 2*, 223–244.

Helgeson, V. S., Reynolds, K. A., & Tomich, P. L. (2006). A meta-analytic review of benefit finding and growth. *Journal of Consulting and Clinical Psychology, 74*(5), 797–816.

Joseph, S., & Linley, P. A. (Eds.). (2008). *Trauma, recovery, and growth: Positive psychological perspectives on posttraumatic stress*. Hoboken, NJ: John Wiley & Sons.
Linley, P. A., & Joseph, S. (2004). Positive change following trauma and adversity: A review. *Journal of Traumatic Stress, 17*(1), 11–21.
Lopez, S. J., & Snyder, C. R. (Eds.). (2009). *Oxford handbook of positive psychology* (2nd ed.). New York: Oxford University Press.
Park, C. L., & Helgeson, V. S. (2006). Growth following highly stressful life events: Current status and future directions. *Journal of Consulting and Clinical Psychology, 74*(5), 791–796.
Park, C. L., Lechner, S. C., Antoni, M. H., & Stanton, A. L. (Eds.). (2009). *Medical illness and positive life change: Can crisis lead to personal transformation?* Washington, DC: American Psychological Association.
Tennen, H., & Affleck, G. (2002). Benefit-finding and benefit-reminding. In C.R. Snyder & S.J. Lopez (Eds.), *Handbook of positive psychology* (pp. 584–597). New York: Oxford University Press.
Tennen, H., & Affleck, G. (2009). Assessing positive life change: In search of meticulous methods. In C.L. Park, S.C. Lechner, M.H. Antoni, & A.L. Stanton (Eds.), *Medical illness and positive life change: Can crisis lead to personal transformation?* (pp. 31–49). Washington, DC: American Psychological Association.

Benefit-Risk Estimation

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Benefit-risk profile; Benefit-risk ratio; Risk-benefit assessment; Risk-benefit ratio

Definition

The benefit-risk estimate can be determined as follows:

$$\text{Benefit-risk estimate} = \frac{\text{Estimate (probability and degree) of benefit}}{\text{Estimate (probability and degree) of harm}}$$

In addition to the likelihood (probability) of benefit and of harm, the degree of both likely occurrences is important. For a given likelihood of a certain benefit, a 1 in 100 chance of a very mild adverse consequence (event) may be acceptable to a patient, but a 1 in 100 chance of a severe event might not.

The term "estimated" is used rather than calculated since, while a calculation (a division) is performed, the two values involved in the calculation are themselves estimates rather than precise quantitative statements (An analogous argument is used in the entry titled "▶ Sample Size Estimation.") The term "benefit-risk ratio" is commonly seen in the literature, but, following this argument, the term "ratio" can be seen to imply a degree of precision that is not (currently) possible in benefit-risk assessment.

The term "benefit-risk balance" is also meaningful. A favorable benefit-risk balance is one in which the estimate of benefit is sufficiently greater than the estimate of harm to make a decision to proceed with an intervention, and an unfavorable balance is one in which the estimate of benefit is not so. For a given intervention, the benefit-risk balance can differ for individual patients. While two patients may be considered likely to gain similar therapeutic benefit from the intervention, it is possible that concurrent illnesses, genetic susceptibility, and/or other factors present in one of them may increase the likelihood of harm to the point where the balance becomes unfavorable.

Benefit-risk assessments for an intervention are also potentially influenced by the availability of subsequent interventions. A new intervention may offer additional therapeutic benefit while maintaining a given level of safety. This would lessen the relative benefit-risk profile of the older intervention. The same would be true for a new intervention offering similar therapeutic benefit but doing so in such a way that the estimate of harm is decreased.

Cross-References

▶ Efficacy
▶ Sample Size Estimation

Benefit-Risk Profile

▶ Benefit-Risk Estimation

Benefit-Risk Ratio

▶ Benefit-Risk Estimation

Benefits of Exercise

Klaus Gebel[1,3] and Ding Ding[2]
[1]School of Education, University of Newcastle, Callaghan, NSW, Australia
[2]Graduate School of Public Health/Department of Family Preventive Medicine, San Diego State University/University of California San Diego, San Diego, CA, USA
[3]City Futures Research Centre, University of New South Wales, Sydney, Australia

Synonyms

Exercise; Motor behavior; Physical activity

Definition

Physical activity is defined as body movement produced by skeletal muscle that results in energy expenditure above resting level and can be accumulated at any time, for example, during work, household, transportation, or during leisure time. Exercise is a subset of leisure-time physical activity that is usually structured, planned, repetitive, and has the purpose of providing recreation, improving or maintaining physical fitness, or enhancing other components of health or well-being (US Department of Health and Human Services, 1996). Physical fitness includes cardiorespiratory fitness, muscle strength, body composition, and flexibility (Thompson et al., 2003). Metabolic fitness is also increasingly recognized as an important component of fitness which is closely related to physical activity levels, as well as cardiovascular and musculoskeletal fitness (Hassinen et al., 2010).

Description

Physical activity recommendations were traditionally only focused on vigorous exercise to achieve fitness and health benefits (American College of Sports Medicine, 1978). However, in the following years, emerging evidence from clinical and epidemiological studies showed that moderate-intensity physical activity, such as walking, is also associated with significant health benefits (Dunn et al., 1999). This has led to a shift in the physical activity paradigm from a sole focus on fitness and performance to a broader public health perspective (Haskell, 2009). The current physical activity guidelines from major governmental and professional organizations state that health benefits can be gained through moderate physical activity, vigorous exercise, or a combination of both (Haskell et al., 2007; Nelson et al., 2007; US Department of Health and Human Services, 2008; World Health Organization, 2010).

These recommendations and guidelines are based on numerous epidemiological and clinical studies that have established the health benefits of regular physical activity and exercise (Bassuk & Manson, 2009; Lee & Paffenbarger, 1998; Lee, Sesso, Oguma, & Paffenbarger, 2003; Sesso, Paffenbarger, & Lee, 2000). There is a broad base of evidence that an active life-style reduces the risk of coronary heart disease, stroke, type 2 diabetes, some cancers, and osteoporosis; improves mental health and lipid profiles; lowers blood pressure; facilitates weight loss and maintenance; and increases longevity (Courneya & Friedenreich, 2011; Dishman, Washburn, & Heath, 2004; Haskell et al., 2007; US Department of Health and Human Services, 1996; Bouchard et al., 2012).

There are specific recommendations for physical activity and exercise for different age groups. Children and adolescents (5–17) should

engage in moderate- to vigorous-intensity physical activity for 60 min or more per day. On at least 3 days per week, this should include vigorous exercise with muscle- and bone-strengthening activities (US Department of Health and Human Services, 2008). Of particular relevance to children and adolescents is that physical activity and exercise promote a healthy growth and positively affect the social (Malina, Bouchard, & Bar-Or, 2004) and cognitive development (Sibley & Etnier, 2003).

Adults aged 18–65 should accumulate at least 150 min of moderate physical activity per week, such as walking, on at least five, preferably all, days of the week, or alternatively at least 75 min of vigorous exercise per week. For more significant health benefits, adults should engage in 300 min of moderate physical activity or 150 min of vigorous exercise per week. Additionally, twice a week, adults should engage in exercise that maintains or increases muscular strength or endurance (Haskell et al., 2007; US Department of Health and Human Services, 2008).

The recommendations for older adults (65+ years) are similar to those for adults with additional emphasis on exercise that improves flexibility and balance (Nelson et al., 2007). Of particular relevance to the elderly is that exercise reduces the risk of falls and associated injuries and has therapeutic benefits for various chronic diseases, such as coronary heart disease, hypertension, osteoarthritis, claudication, and chronic pulmonary disease. Furthermore, physical activity and exercise help in the treatment of depression and anxiety disorders, delay cognitive impairment and disability, and improve functional ability, mobility, and overall quality of life in the elderly. Maintaining a sufficient level of mobility is critical to independent living for older adults (Nelson et al., 2007). Therefore, physical activity and exercise can not only add years to life, but life to years.

Cross-References

► Physical Activity and Health

References and Readings

American College of Sports Medicine. (1978). The recommended quantity and quality of exercise for developing and maintaining fitness in healthy adults. *Medicine & Science in Sports, 10*, VII–X.

Bassuk, S. S., & Manson, J. E. (2009). Physical activity, fitness, and the prevention of cardiovascular disease. In I.-M. Lee, S. N. Blair, J. Manson, & R. S. Paffenbarger Jr. (Eds.), *Epidemiologic methods in physical activity studies* (pp. 158–177). New York: Oxford University Press.

Bouchard, C., Blair, S. N., & Haskell, W. (Eds.) (2012). Physical activity and health (2nd ed.). Champaign: Human Kinetics.

Courneya, K., & Friedenreich, C. (Eds.) (2011). *Physical activity and cancer*. Heidelberg: Springer.

Dishman, R. K., Washburn, R. A., & Heath, G. W. (2004). *Physical activity epidemiology*. Champaign, IL: Human Kinetics.

Dunn, A. L., Marcus, B. H., Kampert, J. B., Garcia, M. E., Kohl, H. W., III, & Blair, S. N. (1999). Comparison of lifestyle and structured interventions to increase physical activity and cardiorespiratory fitness. *Journal of the American Medical Association, 281*, 327–334.

Haskell, W. (2009). Evolution of physical activity recommendations. In I.-M. Lee, S. N. Blair, J. E. Manson, & R. S. Paffenbarger (Eds.), *Epidemiologic methods in physical activity studies* (pp. 283–301). Oxford, UK: Oxford University Press.

Haskell, W. L., Lee, I.-M., Pate, R. R., Powell, K. E., Blair, S. N., Franklin, B. A., et al. (2007). Physical activity and public health: Updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Medicine & Science in Sports & Exercise, 39*, 1423–1434.

Hassinen, M., Lakka, T. A., Hakola, L., Savonen, K., Komulainen, P., Litmanen, H., et al. (2010). Cardiorespiratory fitness and metabolic syndrome in older men and women: The dose responses to Exercise Training (DR's EXTRA) study. *Diabetes Care, 33*, 1655–1657.

Lee, I.-M., & Paffenbarger, R. S., Jr. (1998). Physical activity and stroke incidence: The Harvard Alumni Health Study. *Stroke, 29*, 2049–2054.

Lee, I.-M., Sesso, H. D., Oguma, Y., & Paffenbarger, R. S., Jr. (2003). Relative intensity of physical activity and risk of coronary heart disease. *Circulation, 107*, 1110–1116.

Malina, R., Bouchard, C., & Bar-Or, O. (2004). *Growth, maturation, and physical activity* (2nd ed.). Champaign, IL: Human Kinetics.

Nelson, M. E., Rejeski, W. J., Blair, S. N., Duncan, P. W., Judge, J. O., King, A. C., et al. (2007). Physical activity and public health in older adults: Recommendation from the American College of Sports Medicine and the American Heart Association. *Medicine & Science in Sports & Exercise, 39*, 1435–1445.

Sesso, H. D., Paffenbarger, R. S., Jr., & Lee, I.-M. (2000). Physical activity and coronary heart disease in

men: The Harvard Alumni Health Study. *Circulation, 102*, 975–980.
Sibley, B. A., & Etnier, J. L. (2003). The relationship between physical activity and cognition in children: A meta-analysis. *Pediatric Exercise Science, 15*, 243–256.
Thompson, P. D., Buchner, D., Pina, I. L., Balady, G. J., Williams, M. A., Marcus, B. H., et al. (2003). Exercise and physical activity in the prevention and treatment of atherosclerotic cardiovascular disease: A statement from the Council on Clinical Cardiology (Subcommittee on Exercise, Rehabilitation, and Prevention) and the Council on Nutrition, Physical Activity, and Metabolism (Subcommittee on Physical Activity). *Circulation, 107*, 3109–3116.
US Department of Health and Human Services. (1996). *Physical activity and health – a report of the surgeon general*. Atlanta, GA: Centers for Disease Control and Prevention.
US Department of Health and Human Services. (2008). *2008 Physical activity guidelines for Americans*. Washington, DC: US Department of Health and Human Services.
World Health Organization. (2010). *Global recommendations on physical activity for health*. Geneva: World Health Organization.

Benson, Herbert

Stephanie Ann Hooker
Department of Psychology, University of Colorado Denver, Denver, CO, USA

Biographical Information

Dr. Herbert Benson

Herbert Benson was born in 1935 in Yonkers, New York. He graduated from Wesleyan University in 1957 with a B.A. in Biology and received his medical degree from the Harvard Medical School in 1961. He is currently the Director Emeritus of the Benson-Henry Institute (BHI) and the Mind/Body Medical Institute Associate Professor of Medicine at Harvard Medical School. In his career, spanning more than 40 years, Benson is considered to be the pioneer of mind/body medicine and to be one of the first Western physicians to integrate spirituality and healing into medicine (Massachusetts General Hospital, 2011a).

Major Accomplishments

Benson recognized that in contrast to the "fight-or-flight response" to stress, there must be an opposite physiological reaction to bring the body back to a state of homeostasis; this he defined as the relaxation response (Benson, 1975). This response is identified by marked decreases in respiration rate, metabolism, and heart rate and increases in alpha brain waves. Furthermore, Benson argued that this response could be mentally controlled and induced, similarly to biofeedback responses. Thus, he studied practitioners of Yoga, Zen, and Transcendental Meditation to fully understand this process. Interestingly, he found that individuals who were recently trained in the relaxation response had similar physiological responses during practice to highly trained experts in Yoga or Zen (Benson, 1975). Indeed, subsequent studies showed that these responses were not unique to Transcendental Meditation, but could be harnessed in a restful, hypometabolic state and, therefore, can be elicited through activities such as diaphragmatic breathing, knitting, chi gong, prayer, Yoga, jogging, Tai Chi, and progressive muscle relaxation. The key to eliciting the relaxation response is a repetitive thought, prayer, or movement and a casual return to that repetition if an intruding thought enters the mind (Massachusetts General Hospital, 2011b). Benson continues to teach and lecture about the beneficial effects of the relaxation response in counteracting stress. The revolutionary book, *The Relaxation Response*, was first published in 1975

and continues to be reprinted and translated into many different languages.

In addition to his work on the relaxation response, Benson advocates mind-body medicine through his work at the Benson-Henry Institute for Mind-Body Medicine at Massachusetts General Hospital. There, he supports a three-part system for treating patients: (1) pharmaceuticals, (2) surgery and procedures, and (3) "self-care" composed of mind/body interactions like nutrition, relaxation, exercise, and spirituality. He believes that this system is best because many patients are affected by stress-related conditions and need self-care to treat the person as a whole (Massachusetts General Hospital, 2011b).

Benson has authored more than 180 scientific publications and 12 books. More than five million copies of his books have been printed and translated into many languages. Many institutions have supported his research, including the National Institutes of Health, The John Templeton Foundation, and the Fetzer Institute. He has received numerous awards, including Fellow for the American College of Cardiology, a Presidential Citation from the American Psychological Association, and four honorary doctorates. Benson continues to lecture widely about mind/body medicine, striving to build awareness of the field and bridge the gap between Eastern and Western medicine.

References and Readings

Benson, H. (1975). *The relaxation response*. New York: Morrow.

Benson, H. (1979). *The mind/body effect*. New York: Simon & Schuster.

Benson, H. (1984). *Beyond the relaxation response*. New York: Times Books.

Benson, H. (1996). *Timeless healing: The power of biology and belief*. New York: Scribner.

Benson, H. (2000). *The relaxation response – Updated and expanded* (25th Anniversary ed.). New York: Avon.

Benson, H., & Proctor, W. (2003). *The breakout principle*. New York: Scribner.

Benson, H., & Proctor, W. (2010). *Relaxation revolution*. New York: Scribner.

Benson, H., Stuart, E., & The Staff of the Mind/Body Medical Institute. (1994). *The wellness book*. New York: Carol.

Casey, A., & Benson, H. (2004). *Mind your heart*. New York: Free Press.

Casey, A., & Benson, H. (2006). *The Harvard Medical School guide to lowering your blood pressure*. New York: McGraw-Hill.

Massachusetts General Hospital. (2011a). *Dr. Herbert Benson*. Retrieved July 15, 2011, from http://www.massgeneral.org/bhi/about/benson.aspx

Massachusetts General Hospital. (2011b). *Benson-Henry Institute for Mind-Body Medicine*. Retrieved July 15, 2011, from http://www.massgeneral.org/bhi/about/

Bereavement

Benjamin Hidalgo
Department of Psychiatry, Medical College of Wisconsin, Milwaukee, WI, USA

Synonyms

Grief; Mourning

Definition

Bereavement is the state of being deprived of something or someone, especially a loved one lost to death. Persons in a state of bereavement experience grief, as a normal emotional reaction to the major loss.

Cross-References

- ▶ Caregiver/Caregiving and Stress
- ▶ Death, Sudden
- ▶ End-of-Life Care
- ▶ Grief Counseling
- ▶ Grieving

References and Readings

Corless, I., Germino, B. B., & Pittman, M. A. (Eds.). (2003). *Dying, death, and bereavement: A challenge for living* (2nd ed.). New York: Springer.

Dunn, D. S., & Civitello, T. (2009). Grief is many things: Current perspectives on bereavement. *Journal of Social and Clinical Psychology, 28*(7), 937–939.

Hardy-Bougere, M. (2008). Cultural manifestations of grief and bereavement: A clinical perspective. *Journal of Cultural Diversity, 15*(2), 66–69.
Morgan, J. D., Laungani, P., & Palmer, S. (Eds.). (2009). *Death and bereavement around the world* (Reflective essays, Vol. 5). Amityville, NY: Baywood.
Qualls, S. H., & Kasl-Godley, J. E. (Eds.). (2011). *End-of-life issues, grief, and bereavement: What clinicians need to know*. Hoboken, NJ: John Wiley & Sons.
Stroebe, M. S., Hansson, R. O., Schut, H., Stroebe, W., & Van den Blink, E. (2008). *Handbook of bereavement research and practice: Advances in theory and intervention*. Washington, DC: American Psychological Association.

Bereavement Counseling

► Grief Counseling

Bereavement Therapy

► Grief Counseling

Beta Cells

Luigi Meneghini
Diabetes Research Institute, University of Miami, Miami, FL, USA

Synonyms

Insulin-producing cell

Definition

Beta cells are cells contained within the islets of Langerhans (islets), which specifically produce and secrete insulin into the circulation, in response to a variety of stimuli, most notably blood glucose. Beta cells are part of the endocrine system and are essential in glucose regulation and homeostasis. They achieve this through a balance with counter regulatory hormones, such as glucagon, which is produced by the beta cell's neighbor in the islet, the alpha cell. When beta cells have difficulty in secreting insulin properly (type 2 diabetes) or are completely nonfunctional (type 1 diabetes), diabetes develops. The beta cells co-secrete with insulin a peptide called amylin, which is thought to also be important in metabolic regulation, by slowing gastric emptying, suppressing glucagon secretion, and increasing satiety. Beta cells can also be stimulated to release insulin by a number of antidiabetic medications such as sulfonylureas, glinides, GLP (glucagon-like peptide)-1 receptor agonists, and DPP (dipeptidyl peptidase)-4 inhibitors. Beta cells can also be used to reverse hyperglycemia when islet cells are isolated from cadaveric donors and transplanted into the liver of patients with type 1 diabetes.

Cross-References

► Type 1 Diabetes
► Type 2 Diabetes

References and Readings

Joslin, E. P., & Kahn, C. R. (2005). *Joslin's diabetes mellitus* (14th ed.). Philadelphia: Lippincott Williams & Willkins.

Bias

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Participation bias; Selection bias; Systematic bias

Definition

Bias is the difference between the true value of a particular quantity and an estimate of the quantity

obtained from scientific investigation. Randomization is a process designed to reduce bias as much as possible.

Description

Various influences can introduce error into the assessment of treatment effects: As an example, here systematic bias is discussed. If all of the subjects in one treatment group share a characteristic that is not present in any of the subjects in the other treatment group(s), it is not possible to ascribe differences between the groups to the influence of central interest in the study, i.e., the different treatments received by the groups. Putting all relatively tall subjects in one group and all relatively short subjects in another group would be an example of systematic bias. Another example would be putting all relatively heavy subjects in one group and all relatively less heavy subjects in the other. If the behavioral intervention (treatment) of interest was a putative means of reducing blood pressure, there is a certain degree of biological plausibility that the difference in body weights in the treatment groups could influence the results.

Participation bias (Volunteer bias): Participation bias refers to the differential likelihood of certain individuals accepting invitations to participate in a research investigation (often in a survey) and others not responding or declining to participate.

Recruitment of subjects for participation in various types of experimental methodologies has to acknowledge certain biases. Consider the randomized controlled clinical trial. Subjects are recruited before randomization to treatment groups occurs, and it is the intent that subjects complete their participation in the trial regardless of which treatment they were randomized to. (In the classic double-blind trial, they would not know to which treatment they had been randomized.) In contrast, consider mailed (or electronically mailed) invitations to participate in a research survey. If the survey addresses a disease that you have, or that close friends or family members have, there is a higher likelihood that you will agree to take part in the research study by answering the questions asked by the survey than there is for healthy subjects who have little knowledge of (and possibly little interest in) the disease or condition of research interest.

Selection bias: Selection bias occurs when study subjects are allowed to select into which treatment group in the study they would like to be placed.

Cross-References

▶ Randomized Clinical Trial

Big Five, The

▶ Five-Factor Model of Personality

Binge

▶ Binge Drinking

Binge Drinking

Brian Borsari[1,2] and John Hustad[3]
[1]Department of Veterans Affairs Medical Center, Mental Health and Behavioral Sciences Service, Providence, RI, USA
[2]Department of Behavioral and Social Sciences, Center for Alcohol and Addiction Studies, Brown University, Providence, RI, USA
[3]Department of Medicine and Public Health Sciences, Penn State College of Medicine, Hershey, PA, USA

Synonyms

Alcohol; Bender; Binge; Blood alcohol concentration; College students; Dangerous drinking; Excessive drinking; Harmful drinking; Heavy

episodic drinking; High-risk drinking; Intoxication; Problem drinking; Risky drinking episode

Definition

The most widely used and accepted contemporary definition of the phrase "binge drinking" was published on February 5, 2004, by the NIAAA National Advisory Council. This definition for binge drinking put forth by a special task force assigned the responsibility of defining binge drinking and its differentiations from other patterns of alcohol use states that:

> A 'binge' is a pattern of drinking alcohol that brings blood alcohol concentration to 0.08 gram percent or above. For the typical adult, this pattern corresponds to consuming 5 or more drinks (male), or 4 or more drinks (female) in about 2 hours. Binge drinking is clearly dangerous for the drinker and for society (National Institute on Alcohol Abuse and Alcoholism, 2004).

This definition was released with the following caveats:

(a) A "drink" refers to half an ounce of alcohol (e.g., one 12 oz. beer, one 5 oz. glass of wine, or one 1.5 oz. shot of distilled spirits).
(b) Binge drinking is different from "risky drinking" (reaching a peak BAC of .05 g percent to .08 g percent) and a "bender" (2 or more days of sustained heavy drinking).
(c) For some individuals (e.g., older people or people taking other drugs or certain medications), the number of drinks needed to reach a binge-level BAC is lower than the "typical adult."
(d) People with risk factors for the development of alcoholism have increased risk with any level of consumptions.
(e) For pregnant women, any drinking presents risk to the fetus.
(f) Drinking by persons under the age of 21 is illegal.

Description

Multiple definitions of the term "binge drinking" have existed and been revised over time. Prior to 1991, a "binge" was often thought of to be an intense, multi-day or week long period of drinking that often done in a solitary fashion. The purpose of binge drinking was to become intoxicated, and a loss of control was a component of such a binge drinking episode. The binge was often accompanied by significant consequences such as blackouts, injuries and often ended in incarceration, alcohol poisoning, and/or inpatient treatment. Although a binge was occasionally referred to as a single event, the amounts of alcohol consumed were related to high levels of intoxication and related impairment over more than 1 day. Furthermore, binge drinking episodes, or binges, would often be separated by extended periods of sobriety.

In the United States, the meaning behind the phrase "binge drinking" underwent a drastic change after the term became widely used in the in research college student drinking in the early 1990s. Specifically, Dr. Henry Wechsler of the Harvard School of Public Health started to conduct large-scale surveys of college student alcohol use – the College Alcohol Survey (CAS). The CAS surveyed over 50,000 students at 140 colleges and universities in the United States. The first of these surveys was conducted in 1993, and subsequent surveys were conducted in 1997, 1999, and 2001 (Wechsler & Wuethrich, 2002). Originally defined as having 5 drinks per occasion, the definition was later amended to 5 drinks on one occasion for men and 4 drinks on one occasion for women. Furthermore, this definition lacked a time frame during which the drinks were consumed. Through the many research articles published from the CAS, as well as subsequent articles by other researchers that examined the phenomenon, "binge drinking" was conceptualized as the threshold for which alcohol-related harms became significantly more likely for the drinker. Furthermore, students who shared the environment with students engaging in binge drinking also experienced consequences such as having their sleep and studies disturbed, their property damaged, and even being assaulted. These "secondary effects" were also a source of concern for school administrators, and a commonly cited reason to increase prevention and intervention efforts on college campuses.

Dr. Wechsler's revised definition of the term binge was frequency used outside of research by the media to describe many high-profile alcohol-related fatalities and other related consequences. Thus, the revised definition of binge drinking was integrated into popular terminology.

During the 1990s and early 2000s, there was debate regarding the updated definition of "binge drinking" as Dr. Wechsler's definition of binge drinking was markedly different than the historical definition of a multi-day episode of heavy alcohol use. One concern about the Dr. Wechsler's definition is that it failed to differentiate between individuals who drank to the point of unconsciousness (e.g., BACs of .30 g/dL and higher) within individuals who met the binge drinking threshold but reached low levels of intoxication. In addition, the term binge drinking was often used in the media to describe sensational events related to high levels of intoxication. There was some concern that the use of binge drinking to primarily describe excessive levels of consumption would cause some individuals to falsely believe that the term binge drinking was synonymous to extreme intoxication. Furthermore, there was debate regarding whether the use of Dr. Wechsler's definition of binge drinking led to inaccurate beliefs about the actual occurrence of risky alcohol use at a time where a growing body of research indicated that overestimates about the frequency of alcohol use was positively related to increased personal use.

Synonyms of the term binge drinking were introduced to differentiate the historical and the current definition for binge drinking, and these synonyms included heavy episodic drinking, risky drinking episode, harmful drinking, dangerous drinking, excessive drinking, and problem drinking (Carey, 2001). In addition, NIAAA attempted to address limitations regarding Dr. Wechsler's definition of binge drinking by including level of intoxication as a criterion for whether a binge drinking episode occurred, as well as highlighting the importance of individual differences since some individuals may be at a heightened level of risk depending on their predisposition. Concurrent with the focus on binge drinking in the United States as a single, discrete drinking event researchers and policymakers around the world attempted to determine how many drinks needed to be consumed to constitute a binge drinking episode. Indeed, global cutoffs included a half bottle of spirits or two bottles of wine on one occasion (Sweden); double the daily recommended amount of alcohol – about 2–3 drinks (or 1 or 2 for women) (England); 6 bottles of beer (Finland) and 6 or more units of alcohol (women) and 8 or more units of alcohol for men (United Kingdom). However, to date, there is no worldwide consensus on the amount of alcohol that needs to be consumed to qualify as a binge drinking episode.

As with the term "binge drinking," there is a lack of consensus regarding how many binge drinking episodes one must experience in order to be classified as a "binge drinker." Researchers have used time frames used to capture binge drinking episodes have ranged from the past week to the past year. Overall, a 6-month time frame has been determined to be most informative in linking binge drinking to alcohol-related consequences. Dr. Wechsler and colleagues further differentiated between binge drinkers (one or two binge drinking episodes in the past 2 weeks) and frequent binge drinkers (two or more binge episodes in the past 2 weeks). However, this definition has not been universally accepted.

Because no stable definition of the term binge drinking or binge drinker exists, care must be taken to clearly define binge drinking when interpreting or disseminating research findings, especially when including different studies. As the definition of binge drinking has been clarified (defined in most studies as five or more drinks on one occasion for men and women) since 1991, research has implemented the construct into interviews and surveys with a variety of populations. That said, it is possible to identify some trends in the literature on binge drinking. Binge drinking typically peaks in adolescence and then decline as people get older, with the lowest rates found in individuals over 65 years old. Binge drinking is the most common style of drinking among adolescents, with over 90% of the alcohol consumed by high school students being imbibed while binge drinking. This is of concern as establishing this pattern of alcohol

use early in life may lead to continued binge drinking throughout the lifespan, as well as increase the risk for developing alcohol dependence. Research with adolescents and adults consistently indicates that binge drinking is related to injuries, violence, driving while intoxicated, unsafe sexual practices, and death. In addition, binge drinking in pregnant women can cause significant danger to the fetus such as fetal alcohol spectrum disorders. In the elderly, preliminary evidence suggests that binge drinking may be associated with the onset of dementia. The association of binge drinking with the concurrent use of other substances, such as tobacco, is also a public health concern.

Regarding gender and ethnic differences in binge drinking, men continue to binge drink more than women, accounting for as many as 81% of adult binge drinking episodes. Caucasians also report binge drink more than any other racial group, with African–Americans exhibiting the lowest rates of binge drinking.

Given the health implications, binge drinking has been the target of a variety of prevention and intervention efforts. In the college setting, the social norms marketing campaigns (SNM) became a widespread approach, with mixed results. In college students and other populations, individual brief motivational interventions (BMIs) have been administered in a wide variety of contexts, formats, and settings, and research indicates a consistent small to moderate effect on decreasing the frequency of binge drinking and related consequences. Interventions administered via the Internet have also demonstrated efficacy with college students and adults, and new approaches to screening and intervention through the web continue to be developed.

References and Readings

Alcohol: Problems and Solutions site, maintained by Dr. David Hanson. Retrieved from http://www2.potsdam.edu/hansondj/index.html

Carey, K. B. (2001). Understanding binge drinking: Introduction to the special issue. *Psychology of Addictive Behaviors, 15*, 283–286.

Courtney, K. E., & Polich, J. (2009). Binge drinking in young adults: Data, definitions, and determinants. *Psychological Bulletin, 135*, 142–156.

Dr. Wechsler's College Alcohol Survey website. Retrieved from http://www.hsph.harvard.edu/cas/About/index.html

Herring, R., Berridge, V., & Thom, B. (2008). Binge drinking: An exploration of a confused concept. *Journal of Epidemiology and Community Health, 62*, 476–479.

National Institute on Alcohol Abuse and Alcoholism. (2004). NIAAA council approves definition of binge drinking. *NIAAA Newsletter, 3*, 3.

The Nemours Center for Children's Health Media's website addressing binge drinking in adolescents: http://kidshealth.org/teen/drug_alcohol/alcohol/binge_drink.htmF

The National Institute of Alcohol Abuse and Alcoholism website. Retrieved from http://www.niaaa.nih.gov/Pages/default.aspx

Wechsler, H., & Wuethrich, B. (2002). *Dying to drink: Confronting binge drinking on college campuses*. Emmaus, PA: Rodale.

Binge Eating

Simon Sherry[1] and Skye Fitzpatrick[2]
[1]Department of Psychology, Dalhousie University, Halifax, Nova Scotia, Canada
[2]Department of Psychology, Ryerson University, Toronto, ON, Canada

Definition

Binge eating involves rapidly eating a very large amount of food in a relatively short period of time. Other key characteristics of binge eating include feeling out of control when eating, eating until uncomfortably full, eating apart from others, eating in the absence of hunger, and marked distress regarding overeating. Binge eating is distinguishable from other symptoms of disordered eating. Bulimia nervosa, for example, is a broader pattern of disordered eating including not only binge eating but also compensatory behaviors (e.g., dieting, purging, or exercising to avoid weight gain) and excessive concerns over body size, shape, and weight.

Description

Binge eating is usually conceptualized with reference to either a dimensional framework (with binge eating understood as lying along a continuum of severity ranging from mild to severe) or a categorical framework (with individuals suffering from severe binge eating understood as belonging to a qualitatively discrete diagnostic category). Binge Eating Disorder is a provisional diagnostic criteria set provided for further study in the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 2000). Binge Eating Disorder generally appears to represent a reliable and a valid diagnostic category. Subdiagnostic symptoms of binge eating are also important, as such symptoms negatively impact health and functioning and may herald the occurrence of more severe symptoms of disordered eating.

Epidemiological data suggest binge eating is a common and an impairing problem that most frequently occurs in wealthy, industrialized nations. The onset of binge eating is usually in late adolescence or in young adulthood. With a female-to-male ratio of 3-to-2, binge eating is the least gender-specific form of disordered eating. The prevalence of Binge Eating Disorder ranges from 1% to 4% in samples of community members and from 15% to 50% in samples from weight-control programs. An estimated 8% of individuals who are obese have Binge Eating Disorder. Binge eating is tied to health problems such as obesity, diabetes, and gastrointestinal dysfunction. Moreover, psychiatric difficulties and binge eating frequently co-occur, with mood, anxiety, substance use, and personality problems often accompanying binge eating. Binge eating is also associated with functional impairment in social, personal, familial, and occupational roles.

Several putative factors are involved in the onset and the maintenance of binge eating. Both personality traits (such as perfectionism) and Personality Disorders (such as Borderline Personality Disorder) are risk factors for binge eating. Negative affect is also implicated in binge eating, with binge eating conceptualized as a way of momentarily escaping negative affect. Evidence suggests unsatisfying interpersonal relationships (e.g., hostile interactions) and other interpersonal problems (e.g., evaluative fears) are related to binge eating. Cognitive biases such as strongly basing self-worth on control over eating are also tied to binge eating, and dietary restraint appears to play a key role in binge eating, with binge eating representing an attempt to compensate for caloric deprivation. Ultimately, no one single factor is responsible for binge eating and a confluence of the above factors appears to trigger and to maintain binge eating.

Binge eating is a treatable problem. Randomized controlled trials indicate cognitive behavioral therapy (Wilson & Fairburn, 2007) and interpersonal psychotherapy (Tanofsky-Kraff & Wilfley, 2010) are efficacious interventions for binge eating. Cognitive behavioral therapy focuses on establishing behavioral patterns that reduce binge eating (e.g., regular, moderate meals and snacks) and challenging dysfunctional cognitions that maintain binge eating (e.g., irrational cognitive distortions about dieting). Interpersonal psychotherapy focuses on identifying current interpersonal problem areas contributing to binge eating (e.g., marital disputes) and then improving those problem areas. Randomized trials also suggest antidepressants, especially selective serotonin reuptake inhibitors, are linked to short-term decreases in binge eating (Bodell & Devlin, 2010). Long-term effects of medications on binge eating are unknown. Combining psychotherapy and medication does not appear to result in greater reductions in binge eating.

Cross-References

- ▶ Bulimia
- ▶ Obesity
- ▶ Randomized Controlled Trial

References and Readings

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.

Bodell, L. P., & Devlin, M. J. (2010). Pharmacotherapy for binge-eating disorder. In C. M. Grilo & J. E. Mitchell (Eds.), *The treatment of eating disorders: A clinical handbook* (pp. 402–413). New York: Guilford Press.

Tanofsky-Kraff, M., & Wilfley, D. E. (2010). Interpersonal psychotherapy for the treatment of eating disorders. In W. S. Agras (Ed.), *The Oxford handbook of eating disorders* (pp. 348–372). New York: Oxford University Press.

Wilson, G. T., & Fairburn, C. G. (2007). Treatments for eating disorders. In P. E. Nathan & J. M. Gorman (Eds.), *A guide to treatments that work* (pp. 579–609). New York: Oxford University Press.

Biobehavioral Mechanisms

Catherine Benedict
Department of Psychology, University of Miami, Coral Gables, FL, USA

Synonyms

Biomarkers

Definition

Biobehavioral mechanisms within behavioral medicine refer to the interaction of biological, psychosocial, behavioral, and environmental factors that contribute to health-related outcomes and disease status. Biological processes are believed to mediate the influence of psychosocial, behavioral, and environmental factors on health and disease outcomes.

Description

Research concerned with biobehavioral mechanisms of health and disease has primarily focused on the impact of psychosocial, behavioral, and environmental factors on biological processes of the immune and endocrine systems. These factors can impact biological responses individually and/or synergistically and may include both acute and chronic effects. Psychological distress in response to stressors (i.e., negative life events, both acute and chronic) has been related to alterations in immune and endocrine functioning and much of this work has focused on the effects of stress on immunocompetence and inflammatory responses. Importantly, relationships among relevant biological, psychosocial, behavioral, and environmental factors are often bidirectional and synergistic in nature.

"Biobehavioral mechanism" is a term used to suggest biomarker research linked to psychological, behavioral, and sociocultural factors. Pioneering work first discovered the interrelationship between psychological stress and physiologic responses of the nervous, endocrine, and immune systems. Among the first researchers to evaluate these relationships were Walter Cannon and Hans Selye. It was Cannon (1939) who first identified the role of the autonomic nervous system (ANS) in the fight-or-flight stress response. Selye (1952, 1975) later demonstrated interactions of the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system (SNS) that lead to subsequent changes in immune functioning and the lymphoid organs in response to psychological stress. This led to a global immunosuppression model of stress and immunity. Over the years and with advancing technology, increasingly sophisticated methods for evaluating these relationships have been developed and our understanding of the mechanisms by which psychological and behavioral factors influence biological processes and health outcomes has advanced. Psychoneuroimmunology (PNI) has emerged as a field of study that is primarily concerned with interactions between the central nervous system, the endocrine system, and the immune system and the impact of these interactions on health and disease. The HPA axis and the sympathetic-adrenal medullary (SAM) axis are the primary pathways by which these systems interact. Much of this work has been concerned with the systemic effects of psychological stress on the regulatory processes of the HPA and SAM axes and the association between chronic inflammation and acute and/or chronic health conditions. Biomarkers are used to

objectively measure (ab)normal biological processes, pathogenic processes, disease progression, and response to treatment or intervention.

The HPA and SAM axes produce glucocorticoid hormones (e.g., cortisol) and catecholamines in response to stress. These neuroendocrine products bind to receptors on a number of immune cells, such as lymphocytes, monocytes, machrophages, and granulocytes, altering cellular activity and immune functioning (e.g., cell trafficking, proliferation, cytokine secretion, antibody production, and cytolytic activity; Padgett & Glaser, 2003; Rabin, 1999). Cytokines mediate and control inflammatory and immune responses. One of the effects of chronic activation of the stress response is downregulation of cortisol production and increases in the release of various pro-inflammatory cytokines such as C-reactive protein (CRP), interleukin-2 (IL-2), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-alpha). The nature and duration of stress (e.g., acute vs. chronic) has been identified as determining factors influencing the degree to which psychological stress leads to alterations in neuroendocrine functioning and immune dysregulation. Although both acute and chronic stress has been related to increased activation of inflammatory responses and alterations in immune function, longer-term or chronic stressors have been shown to have a more substantial impact.

"Biobehavioral mechanisms within behavioral medicine" is also a term that has been used to imply markers of disease activity. Both animal and human models have demonstrated that the interaction of the neuroendocrine and immune systems in response to stress influence a number of health-related outcomes. A lot of the emphasis looking at biobehavioral mechanisms has evaluated the interaction of stress and biomarker activity. Classic studies linking psychological and behavioral factors to biological mechanisms have demonstrated the negative effects of stress on immune functioning. Psychological stress has been shown to exacerbate viral and bacterial pathogenesis, increases susceptibility to viruses, slows wound healing, and alters autoimmune diseases (Black, 2003; Cohen et al., 1991; Kiecolt-Glaser et al., 1995; Padgett et al., 1998). Studies have demonstrated that subjects inoculated with a vaccine show poorer immunologic responses during times of stress (e.g., medical students during exams, caregiving for a spouse with dementia and Alzheimer's disease; Glaser et al., 1992, 2000, 2001; Jabaaij et al., 1996; Kiecolt-Glaser, Glaser, Gravenstein, Malarkey, & Sheridan, 1996; Vedhara et al., 1999) and this relationship has been shown to be dose dependent (Cohen et al., 1991). Furthermore, individuals who show delayed, weaker, or shorter-lived responses to vaccines are more likely to experience clinical illness and longer-lasting infections (Padgett & Glaser, 2003). Stress-induced alterations in neuroendocrine and immune responses have also been studied within a number of disease models including insulin resistance and type II diabetes (Black, 2003), cardiovascular health (e.g., atherosclerotic disease and hypertension; Black, 2003), cancer (Andersen, Kiecolt-Glaser, & Glaser, 1994; Andersen et al., 1998), human immunodeficiency virus (HIV; Antoni et al., 1990; Cruess et al., 2000), and multiple sclerosis (Ackerman, Martino, Heyman, Moyna, & Rabin, 1998).

Psychological stress may also impact biological processes indirectly through related effects on health behaviors. That is, efforts undertaken to manage the demands of stress or cope with an acute or ongoing stressor may lead to unhealthy behavior changes. For example, distressed individuals often have appetite and/or sleep disturbances and are more likely to self-medicate with alcohol and other drugs, including caffeine use and cigarette smoking. Likewise, health behaviors may have a direct effect on biological processes, independent of psychological stress. Immune functioning has been related to objective measures of sleep, nutrition, alcohol intake, and drug use. These factors may contribute independent effects and/or their interaction may lead to additive affects on neuroendocrine and immune functioning. For example, substance abuse has been directly related to immune dysfunction and indirectly through related effects on nutrition. Overall, poor health behaviors may interact with

psychological and biological processes in bidirectional ways, contributing to and exacerbating the effects of stress on health and disease.

Cross-References

- ▶ Behavioral Medicine
- ▶ Psychoneuroimmunology

References and Readings

Ackerman, K. D., Martino, M., Heyman, R., Moyna, N. M., & Rabin, B. S. (1996). Immunologic response to acute psychological stress in MS patients and controls. *Journal of Neuroimmunology, 68*, 85–94.

Ackerman, K. D., Martino, M., Heyman, R., Moyna, N. M., & Rabin, B. S. (1998). Stressor-induced alteration of cytokine production in multiple sclerosis patients and controls. *Psychosomatic Medicine, 60*, 484–491.

Ader, R., Cohen, N., & Felten, D. (1995). Psychoneuroimmunology: Interactions between the nervous system and the immune system. *Lancet, 345*, 99–103.

Ader, R., Felten, D. L., & Cohen, N. (2001). *Psychoneuroimmunology* (3rd ed.). San Diego, CA: Academic Press.

Andersen, B. L., Farrar, W. B., Golden-Kreutz, D., Kutz, L. A., MacCallum, R., Courtney, M. E., et al. (1998). Stress and immune responses after surgical treatment for regional breast cancer. *Journal of the National Cancer Institute, 90*, 30–36.

Andersen, B. L., Kiecolt-Glaser, J. K., & Glaser, R. (1994). A biobehavioral model of cancer stress and disease course. *American Psychologist, 49*(5), 389–404.

Antoni, M. H., August, S., LaPerriere, A., Baggett, H. L., Klimas, N., Ironson, G., et al. (1990). Psychological and neuroendocrine measures related to functional immune changes in anticipation of HIV-1 serostatus notification. *Psychosomatic Medicine, 52*, 496–510.

Aragona, M., Muscatello, M. R. A., Losi, E., Panetta, S., la Torre, F., Pastura, G., et al. (1996). Lymphocyte number and stress parameter modifications in untreated breast cancer patients with depressive mood and previous life stress. *Journal of Experimental Therapeutics and Oncology, 1*, 354–360.

Benjamini, E., Coico, R., & Sunshine, G. (2000). *Immunology: A short course* (4th ed.). New York: Wiley-Liss.

Biondi, M. (2001). Effects of stress on immune functions: An overview. In R. Ader, D. L. Felten, & N. Cohen (Eds.), *Psychoneuroimmunology* (3rd ed., pp. 189–226). San Diego, CA: Academic Press.

Black, P. H. (2003). The inflammatory response is an integral part of the stress response: Implications for atherosclerosis, insulin resistance, type II diabetes and metabolic syndrome X. *Brain, Behavior, and Immunity, 17*, 350–364.

Cannon, W. B. (1939). *The wisdom of the body*. New York: Norton.

Cohen, S., Tyrrell, D. A. J., & Smith, A. P. (1991). Psychological stress and susceptibility to the common cold. *New England Journal of Medicine*, *325*(9), 606–612.

Cruess, D. G., Antoni, M. H., McGregor, B. A., Kilbourn, K. M., Boyers, A. E., Alferi, S. M., et al. (2000). Cognitive-behavioral stress management reduces serum cortisol by enhancing benefit finding among women being treated for early stage breast cancer. *Psychosomatic Medicine*, *62*, 304–308.

Cruess, S., Antoni, M., Kilbourn, K., Ironson, G., Klimas, N., Fletcher, M. A., et al. (2007). Optimism, distress, and immunologic status in HIV-infected gay men following Hurricane Andrew. *International Journal of Behavioral Medicine, 7*, 160–182.

Glaser, R., Kiecold-Glaser, J. K., Bonneau, R. H., Malarkey, W., Kennedy, S., & Hughes, J. (1992). Stress-induced modulation of the immune response to recombinant hepatitis B vaccine. *Psychosomatic Medicine, 54*, 22–29.

Glaser, R., Sheridan, J., Malarkey, W. B., MacCallum, R. C., & Kiecolt-Glaser, J. K. (2000). Chronic stress modulates the immune response to a pneumococcal pneumonia vaccine. *Psychosomatic Medicine*, *62*, 804–807.

Glaser, R., MacCallum, R. C., Laskowski, B. F., Malarkey, W. B., Sheridan, J. F., & Kiecolt-Glaser, J. K. (2001). Evidence for a shift in the Th-1 to Th-2 cytokine response associated with chronic stress and aging. *Journal of Gerontology: Medicine Sciences*, *56*(8), M477–M482.

Jabaaij, L., van Hattum, J., Vingerhoets, J. J. M., Oostveen, F. G., Duivenvoorden, H. J., Ballieux, R. E. (1996). Modulation of immune response to rDNA hepatitis B vaccination by psychological stress. *Journal of Psychosomatic Research*, *41*(2), 129–137.

Kang, D., Rice, M., Park, N., Turner-Henson, A., & Downs, C. (2010). Stress and inflammation: A biobehavioral approach for nursing research. *Western Journal of Nursing Research, 32*(6), 730–760.

Kiecolt-Glaser, J. K., Marucha, P. T., Mercado, A. M., Glaser, R. (1995). Slowing of wound healing by psychological stress. *The Lancet*, *346*, 1194–1196.

Kiecolt-Glaser, J. K., Glaser, R., Gravenstein, S., Malarkey, W. B., & Sheridan, J. (1996). Chronic stress alters the immune response to influenza virus vaccine in older adults. *Proceedings of the National Academy of Sciences, United States of America, 93*, 3043–3047.

Padgett, D. A., Sheridan, J. F., Dorne, J., Bernston, G. G., Candelora, J., & Glaser, R. (1998). Social stress and the reactivation of latent herpes simplex virus type 1. *Proceedings of the National Academy of Sciences of the United States of America*, *95*, 7231–7235.

Padgett, D. A., & Glaser, R. (2003). How stress influences the immune response. *TRENDS in Immunology, 24*(8), 444–448.

Rabin, B. S. (1999). Stress, immune function, and health: The connection. New York, NY: Wiley Liss.
Segerstrom, S. C., & Miller, G. E. (2004). Psychological stress and the human immune system: A meta-analytic study of 30 years of inquiry. *Psychological Bulletin, 130*(4), 601–630.
Selye, H. (1952). *The story of the adaptation syndrome*. Montreal, Canada: Acta.
Vedhara, K., Cox, N. K., Wilcock, G. K., Perks, P., Hunt, M., Anderson, S., et al. (1999). Chronic stress in elderly carers of dementia patients and antibody response to influenza vaccination. *Lancet, 353*(9153), 627–631.

Biofeedback

Masahiro Hashizume
Department of Psychosomatic Medicine, Toho University, Ota-ku, Tokyo, Japan

Synonyms

Biofeedback control; Biofeedback therapy

Definition

Biofeedback denotes the use of electronic methods to amplify or convert imperceptible bodily information and feed this information back to the person concerned in a perceptible form such as light, sound, vibration, numbers, or graphical representation for the purposes of improving health and performance. This feedback can be used in training aimed at developing self-control over physiological functions and autonomic responses that are difficult to treat using conventional methods. This kind of therapy is known as biofeedback therapy or biofeedback control.

Description

Research on biofeedback began around 1960. Biofeedback involves two basic processes – the perception of physiological functions and the control of those functions. The physiological function to be brought under control must be continuously monitored with sufficient sensitivity to detect in real time change, and the change in the physiological measure must be reflected immediately to the person attempting to control the process.

In learning theory terms, biofeedback involves the operant conditioning of neuromuscular and autonomic nervous activity. Bodily reactions such as changes in muscle tone or heart rate are operant behavior that can be controlled through feedback. This control is based on the principle that if a certain reaction results in a reward, and if that response is rewarded every time it occurs, it will occur with increasing frequency. As a form of applied psychophysiology, clinical biofeedback assists persons to alter their own behaviors through systematic feedback of such physiological responses.

Biofeedback therapy can be applied to a wide range of vital reactions, including blood pressure, heart rate, heart rate variability (HRV), skin temperature, skin potential reflex, sweat gland action potential, electrocardiogram (ECG), electrogastrogram, electromyogram (EMG), respiration, and electroencephalogram (EEG). Especially, biofeed back displaying real-time electroencephalography is also called neurofeedback. Biofeedback therapy methods can be divided into those that seek to directly control bodily reactions associated with a specific condition and those that seek to indirectly control the condition through full body relaxation.

Biofeedback has been applied to many different fields. In medical care, it is used mainly to treat or prevent psychosomatic conditions such as bronchial asthma, hypertension, arrhythmia, tension-type headache, migraine, writer's cramp, and spasmodic torticollis. It is also said to be effective in the field of sports psychology to manage and relax the body and mind of athletes prior to competing, with electromyogram and skin temperature biofeedback being the most frequently used. Biofeedback is also deemed to be effective for post-stroke rehabilitation. The final goal in gaining control over a reaction is to be able to control the

reaction with just one's own powers, without using a monitor.

References and Readings

James, R. E. (1999). *Introduction to quantitative EEG and neurofeedback*. San Diego: Academic Press.
Pearce, S. (1999). *The practice of behavioural medicine*. Oxford: Oxford Science.
Schwartz, M. S. (2005). *Biofeedback: A practitioner's guide* (3rd ed.). New York: The Guilford Press.

Biofeedback Control

▶ Biofeedback

Biofeedback Therapy

▶ Biofeedback

Biological Indicators

▶ Biomarkers

Biological Markers

▶ Biomarkers

Biomarkers

Vivek Shetty
Oral & Maxillofacial Surgery, University of California, Los Angeles, CA, USA

Synonyms

Biological indicators; Biological markers

Definition

1. A biomarker is a characteristic that is objectively measured and evaluated as an indicator of normal biological processes, pathogenic processes, or pharmacologic responses to a therapeutic intervention.
2. A biomolecule whose presence, absence, or abnormal concentration in blood, urine, saliva, or other body fluids, and/or tissues indicates normal or diseased processes in the body.

Description

The body's adaptive response to psychological and physical stressors produces detectable changes in multiple biochemical and physiological processes, and potentially, any of these alterations may be used as biomarkers. As measurable characteristics that reflect the biological underpinnings of health and disease, biomarkers are increasingly used to augment self-report measures of stress and to illuminate the interactions between social, environmental, and behavioral factors with health outcomes. The attractiveness of biomarkers in behavioral medicine stems from their potential to reduce the reliability and validity issues inherent to subjective self-report assessments. Of particular interest are readily obtained, quantifiable and reliable biological measures that reveal existing psychological states and serve as harbingers of future health disorders.

Most biomarkers used in behavioral medicine are selected for their ability to reflect activity in one or more of the three main stress response systems: the sympathetic-adrenal-medullary (SAM) axis, the hypothalamic-pituitary-adrenal (HPA) axis, and the immune system. Activation of the SAM axis is the body's immediate physiological response to acute stressors and the released catecholamines, epinephrine, and norepinephrine are commonly used as biomarkers to reflect SAM axis activity. HPA axis activation is a longer-term hormonal response that starts manifesting 15–20 min following stressor onset. Primary indicators of the HPA axis activation include cortisol, dehydroepiandrosterone (DHEA), its sulfated form (DHEA-S), and adrenocorticotropin hormone (ACTH).

Crosstalk with the neuroendocrine system also produces alterations in the immune system that vary according to stressor duration. For instance, short-term stressors provoke increases in the levels of the quick-acting, all-purpose immune cells such as the natural killer (NK) cells. Prolonged or chronic stressors produce more robust changes in immune biomarkers, including suppressed activity and proliferation of NK cells, and increases in inflammatory markers such as proinflammatory cytokines (e.g., IL-6, TNF-α) and C-reactive protein (CRP). Alternate expressions of stress-related immunosuppression include slower wound healing and weaker seroconversion rates following vaccination.

Utilized individually or as part of a panel representing the multiple response pathways, biomarkers can be used to identify the risk of developing disease (antecedent biomarkers), screen for subclinical disease (screening biomarkers), recognize overt disease (diagnostic biomarkers), categorize disease severity (staging biomarkers), or predict disease course and response to therapy (prognostic biomarkers). Recognizing that simple, univariate tactics (i.e., use of one biomarker) do not fully address the complex process of adjustment and adaptation to multiple recurring stressors, biobehavioral researchers are progressively transitioning to more comprehensive, multivariate approaches (i.e., use of multiple biomarkers). An illustrative example is the integration of hormonal mediators of the stress response, cortisol and epinephrine, with blood pressure and waist-hip ratio to operationalize the construct of allostatic load, the cumulative physiological toll exacted by the body's efforts to adapt to life experiences. By using an allostatic load index representing neuroendocrine, immune, metabolic, and cardiovascular system functioning, a variety of studies have demonstrated greater prediction of age-related health and cognitive declines over and beyond traditional methods employed in biopsychosocial investigations.

The intrinsic value of any biomarker derives from its measurement properties and as such, an ideal biomarker should be accurate (i.e., match the actual value of the health construct being measured), have high sensitivity and specificity for the outcome of interest, and explain a reasonable proportion of the outcome, independent of other established predictors. However, the desirable properties of a biomarker can vary with its intended use. Features such as low costs and high sensitivity, specificity, and predictive values are important for a screening biomarker. In contrast, features such as costs, sensitivity, and specificity are less important in prognostic biomarkers because only individuals with disease are tested and they serve as their own controls (i.e., baseline values are compared with follow-up values). The transition from putative biomarker to a known valid biomarker status occurs through a multistep confirmatory process. Although the biomarker literature frequently uses the term "biomarker validation" to describe the authentication process, it is important to distinguish between validation (assay or method validation) and qualification (or clinical validation or evaluation). Method validation refers to the process of assessing the assay or measurement performance characteristics (e.g., accuracy, precision, selectivity, sensitivity, and reproducibility). Biomarker qualification is the more appropriate term for describing the graded, evidentiary process for linking the biomarker with biological processes and clinical endpoints relevant to the intended application.

On a practical level, a biomarker can have clinical utility only if it can be extracted easily and unobtrusively, is reproducibly obtained in a standardized fashion, and the measurement is readily accessible and easy to interpret by the end-user. These practical considerations drive the concomitant development of robust, low-cost, and portable biosensors that will allow biomarkers to be detected and measured reliably in places as diverse as remote field environments, community hospitals, or even at home. The truncated biosampling-reporting cycle afforded by these point-of-care biosensors will eventually allow a time-sampling protocol that is sensitive to common sources of biomarker variability (e.g., diurnal variation, timing of collection relative to stressor) and enable time-series psychophysiological measurements in naturalistic settings. As advances in behavioral research and biomarker development converge with innovations in biosensing technology and systems biology, one can expect that the

ready access to accurate and reliable biomarker information will enable more precise, predictive, and personalized health care.

Cross-References

- ▶ Biobehavioral Mechanisms
- ▶ Salivary Biomarkers

References and Readings

Atkinson, A. J., Colburn, W. A., DeGruttola, V. G., DeMets, D. L., Downing, G. J., Hoth, D. F., et al. (2001). Biomarkers and surrogate endpoints: Preferred definitions and conceptual framework. *Clinical Pharmacology and Therapeutics, 69*(3), 89–95.

Gruenewald, T. L., Seeman, T. E., Karlamangla, A. S., & Sarkisian, C. A. (2009). Allostatic load and frailty in older adults. *Journal of the American Geriatrics Society, 57*(9), 1525–1531.

Kiecolt-Glaser, J. K., McGuire, L., Robles, T. F., & Glaser, R. (2002). Psychoneuroimmunology: Psychological influences on immune function and health. *Journal of Consulting and Clinical Psychology, 70*(3), 537–547.

Piazza, J. R., Almeida, D. M., Dmitrieva, N. O., & Klein, L. C. (2010). Frontiers in the use of biomarkers of health in research on stress and aging. *The Journals of Gerontology, Series B: Psychological Sciences and Social Sciences, 65B*(5), 513–525.

Shetty, V., Zigler, C., Robles, T. F., Elashoff, D., & Yamaguchi, M. (2011). Developmental validation of a point-of-care, salivary α-amylase biosensor. *Psychoneuroendocrinology, 36*(2), 193–199.

Singh, I., & Rose, N. (2009). Biomarkers in psychiatry. *Nature, 460*(7252), 202–220.

Biopsychosocial Model

Eleanor Miles
Department of Psychology, The University of Sheffield, Western Bank, Sheffield, UK

Definition

The view that illness and health can be best understood as a result of the interaction between physiological, psychological, and sociocultural factors.

Description

The biopsychosocial model, originally advanced by George L. Engel (1977), views disease and health as the product of physiological, psychological, and sociocultural variables. This viewpoint stands in contrast to the biomedical model, in which disease is viewed in terms of deviation from normal biological functioning, and where the experience and etiology of illness are understood solely in terms of biological factors, such as genetic predispositions or physiological dysfunctions. Engel argued that this model was too narrowly focused, and that a greater emphasis needed to be placed on the role of psychosocial factors.

The idea that psychological and sociocultural factors could have an influence upon illness had already been recognized (in the field of psychosomatic medicine, for example). For example, factors such as negative beliefs about an illness (e.g., helplessness) and avoidance behaviors may act to worsen symptoms, whereas the presence of active coping strategies and social support may have a positive effect on the course of an illness. A key aspect of the biopsychosocial model is the importance it places on the interconnections between the three domains of biological, psychological, and social functioning. For example, psychological factors can both influence biological functioning (e.g., alterations in autonomic nervous system function and hormone production), and can also be influenced by biological functioning (e.g., disease may cause cognitive impairments or contribute to depression and anxiety). The model holds that an illness can be best understood by considering its psychological and sociocultural effects as well as its biological ones, and that the cause and progression of an illness can also be influenced by all three of these factors, not just biological ones. In other words, the biopsychosocial model suggests that both the etiology and the expression or prognosis of illness are best understood as the result of an interaction between biological, psychological, and sociocultural variables.

The biopsychosocial model can help to explain why patients with the same disease or physical pathology may experience their illness,

and respond to treatment, in very different ways. In terms of clinical practice, the model encourages clinicians to consider all relevant psychological and social factors when making a diagnosis, and implies that treatment should address all of these factors rather than just the biological component of illness. More generally, the model has also been influential in encouraging researchers and clinicians to integrate knowledge from different domains in order to better understand illnesses and how they can be treated (see Borrell-Carrio, Suchman, & Epstein, 2004, for further discussion). The field of behavioral medicine can be seen as an application of the principles of the biopsychosocial model to medical practice and research. The recent growth in application of psychological theories to understanding and influencing health behavior (such as the theory of planned behavior) is also a reflection of the biopsychosocial model's influence. Numerous studies have validated this model by showing that psychosocial factors predict onset and progression of cardiac diseases, the common cold, recovery from surgery and cancer prognosis, independent of biomedical and demographic variables, and that psychological interventions may also improve the health outcomes of some of these conditions.

Cross-References

- ▶ Behavioral Medicine
- ▶ Engel, George
- ▶ Sociocultural Differences

References and Readings

Borrell-Carrio, F., Suchman, A. L., & Epstein, R. M. (2004). The biopsychosocial model 25 years later: Principles, practice, and scientific inquiry. *Annals of Family Medicine, 2*(6), 576–582.

Engel, G. L. (1977). The need for a new medical model: A challenge for biomedicine. *Science, 196*(4286), 129–136.

Quill, T. E., Frankel, R. M., & McDaniel, S. H. (Eds.). (2004). *The biopsychosocial approach: Past, present and future*. Rochester, MN: University of Rochester Press.

White, P. (Ed.). (2005). *Biopsychosocial medicine: An integrated approach to understanding illness*. New York: Oxford University Press.

Bipolar Disorder, with Seasonal Pattern

- ▶ Seasonal Affective Disorder

Birth Control

- ▶ Family Planning

Birth Control, Family Planning

- ▶ Contraception

Birth Planning

- ▶ Family Planning

Birth Prevention

- ▶ Family Planning

Birth Weight

Linda C. Baumann and Alyssa Karel
School of Nursing, University of Wisconsin-Madison, Madison, WI, USA

Definition

The birth weight of a newborn is an indicator of chance for survival and long-term development and can also reflect the health status of the birth

mother. Normal birth weight is classified as babies born between the 10th and 90th percentiles of all babies of the same gestational age. Low birth weight is a major problem of newborns worldwide. There are three categories of low birth weight: low birth weight (LBW) is less than 2,500 g or about 5.5 lb; very low birth weight (VLBW) is less than 1,500 g; and extremely low birth weight (ELBW) is less than 1,000 g. Low-birth-weight infants are either born small for their gestational age (SGA) or born prematurely. A preterm birth is a gestation of less than 37 weeks and is a major cause of LBW. Small for gestational age births are more common in developing countries.

High birth weight is far less common worldwide but can still have major consequences on the health of both the mother and the infant. High birth weight or large for gestational age (LGA) babies weigh greater than the 90th percentile of all babies of the same gestational age, which for full-term infants is about 4,000–4,500 g. Maternal diabetes is the most common cause of LGA babies. Maternal risks associated with LGA babies include increased vaginal delivery time, increased chance of a cesarean delivery, lacerations of the birth canal, and maternal hemorrhage. The newborn is at risk of problems related to glucose regulation and respiratory distress.

Low-birth-weight infants are at increased risk of mortality and morbidity throughout their lives. As infants, risks include an inability to maintain body temperature, infection, difficulty gaining weight, respiratory distress syndrome, neurological problems, and sudden infant death syndrome. Later in life they are at risk of for poor muscle development, cognitive disabilities and are more likely to remain below average on height and weight charts.

Demographic, medical, behavioral and environmental maternal risk factors for having a LBW infant include age <18 or >35, low socioeconomic status, low education level, and ethnicity (African Americans have increased risk of having LBW infants when compared to Latina American and Caucasian mothers). Risk factors include a previous LBW infant and a woman who was herself a LBW infant, infection, placental problems, poor weight gain/poor nutrition, or first or second trimester bleeding. Behavioral risk factors include smoking and use of drugs or alcohol during pregnancy. Environmental risk factors include poverty, maternal stress, and delayed or a lack of prenatal care.

References and Readings

Bradley, P. F., & Zimmerman, J. (2006). *Pediatric critical care* (3rd ed.). Philadelphia: Mosby Elsevier.

Normal birthweight it critical to the future health and development. (2009). Retrieved August 17, 2010, from http://www.childinfo.org/low_birthweight_overview.html

Taeusch, H. W., Ballard, R. A., & Gleason, C. A. (2005). *Avery's diseases of the newborn* (8th ed.). Philadelphia: Mosby Elsevier.

The Children's Hospital of Philadelphia. (2010). *Large for gestational age*. Retrieved December 17, 2010, from http://www.chop.edu/healthinfo/large-for-gestational-age-lga.html

The Children's Hospital of Philadelphia. (2010). *Low birth weight*. Retrieved August 16, 2010, from http://www.chop.edu/healthinfo/low-birthweight.html

Wong, D., Hockenberry, M., Wilson, D., Perry, S., & Lowdermilk, D. (2006). *Maternal child nursing care* (3rd ed.). St Louis: Mosby Elsevier.

Bladder Carcinoma

▶ Cancer, Bladder

Blood Alcohol Concentration

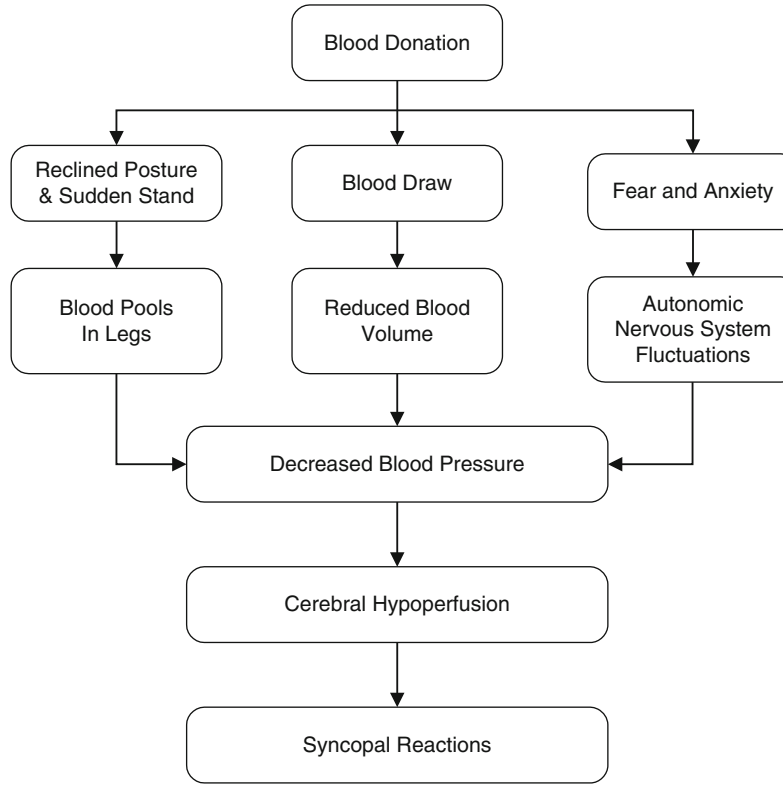
▶ Binge Drinking

Blood Donation

Christopher France and Janis L. France
Department of Psychology, Ohio University, Athens, OH, USA

Description

The blood donation process places a unique set of physiological and psychological demands on the



Blood Donation, Fig. 1 Potential contributors to syncopal reactions in response to blood donation

donor. As shown in Fig. 1, these include postural challenge upon standing after a prolonged period of reclined seating, loss of blood volume, and potential fear and anxiety. These factors can combine to produce reductions in cerebral perfusion that, in turn, can result in a range of reactions from mild presyncopal (i.e., pre-faint) symptoms such as dizziness or lightheadedness to periods of syncope (i.e., fainting or loss of consciousness) that can last for a few seconds in mild cases to minutes in more severe cases.

Risk of Syncopal Reactions to Blood Donation

Overall, the risk for presyncopal and syncopal symptoms is low, with a recent study of over six million whole blood donors reporting presyncopal reactions in 2.5% of all donations and loss of consciousness in only 0.1% (Eder et al., 2008). However, in a related report, it was noted that 16–17-year-old donors had a presyncopal rate of 8.9% and a loss of consciousness rate of 0.3% (Eder, Hillyer, Dy, Notari, & Benjamin, 2008). These findings are consistent with other reports that presyncopal symptoms occur 2.6–9 times more often among first-time blood donors as compared to repeat donors. And young, female, first-time donors are at particularly high risk with on-site syncopal reactions rates as high as 16.1%. Importantly, additional reactions can occur after a donor leaves the blood collection site, and analysis of reported cases indicate that 10–15% of all syncopal episodes occur off-site (Kamel et al., 2010; Newman, 2004). These off-site reactions are particularly problematic as they are more likely to be associated with a fall and an injury to the head. It has been estimated that 1 in 9,300 donations results in a health-care visit due to syncopal reactions and that one third of such visits relate to an injury sustained during a fall (Newman, 2004).

Impact of Syncopal Reactions on Blood Donor Retention

Although a number of factors shape individual decisions to donate, retrospective studies of

existing blood donors demonstrate that the experience of adverse reactions is a particularly important barrier to retention. For example, in a retrospective survey of over 30,000 blood donors (Thomson et al., 1998), donors' perception of physical well-being during or after donation was the single strongest predictor of intent to donate again. Donors who rated their physical well-being during or after donation as "fair to poor" reported an anticipated attrition rate that was six times higher than those who rated their well-being as "good to excellent." Similarly, a comparison of current versus "lapsed" donors (i.e., previous donors who had not donated within the past 2 years) revealed that a positive donation experience was one of the most important determinants of return behavior (Germain et al., 2007). Similar findings have also been observed in prospective analyses of donor behavior. For example, a study of nearly 90,000 whole blood donors revealed that those who did not experience a presyncopal or syncopal reaction returned to donate at a rate of 64% in the following year as compared to a rate of 40% for donors who experienced a reaction (France, Rader, & Carlson, 2005).

Reducing the Risk of Syncopal Reactions

A number of intervention strategies have been adapted to the blood donation context to address the specific physiological and psychological demands illustrated in Fig. 1. These include applied muscle tensing techniques to enhance venous return, predonation water consumption to acutely increase total peripheral resistance and resting blood pressure, and distraction techniques to reduce fear and anxiety.

Using Applied Muscle Tension to Attenuate Venous Pooling. Repeated, rhythmic muscle contraction procedures have been used for decades to treat fainting reactions in individuals with blood and injury/injection phobia. For example, individuals with blood and injury phobia can learn to make voluntary muscular contractions when faced with feared stimuli, and these actions can increase blood flow to the brain and prevent faintness (Foulds, Wiedmann, Patterson, & Brooks, 1990). Because this technique has obvious practical implications for preventing blood donation reactions, they have also been attempted with volunteer blood donors. In a series of studies, donors were randomly assigned to either donation-as-usual or an applied muscle tension group that watched a brief predonation video on the use of applied muscle tension during donation (Ditto & France, 2006; Ditto, France, Albert, & Byrne, 2007; Ditto, France, Lavoie, Roussos, & Adler, 2003; Ditto, Wilkins, France, Lavoie, & Adler, 2003). On the whole, these studies demonstrated that compared to controls who did not watch the video, donors who learned the muscle tensing technique (1) reported lower levels of presyncopal symptoms, (2) were less likely to have their donation chair reclined by the phlebotomist, and (3) expressed greater confidence that they would donate blood again in the future. Interestingly, while the beneficial effects of muscle tensing have been demonstrated in both males and females (Ditto & France, 2006; Ditto et al., 2007; Ditto, Wilkins et al., 2003), in some studies, these effects have been restricted to female donors (Ditto, France et al., 2003). In part, more consistent findings for female donors may be related to the fact that they are, on average, at greater risk for reactions.

Using Water to Compensate for Blood Volume Reductions. Drinking water elicits acute increases in sympathetic nervous system activity and total peripheral resistance that may help maintain blood pressure during donation, and this simple intervention has been shown to significantly delay syncopal reactions to head-up tilt testing (Lu et al., 2003). Although water consumption immediately prior to donation will do little to restore blood volume reductions at the time of donation (due to delays in absorption time), the acute cardiovascular effects of drinking water may help to offset the reductions in blood pressure that can occur with the loss of approximately 500 ml of blood. Consistent with this notion, healthy young blood donors who drank 500 ml of bottled water approximately 30 min prior to donation reported reduced presyncopal symptoms as compared to donors who did not

drink water (Hanson & France, 2004). Further, there was no relationship between total body water levels at baseline and reported reactions, suggesting that the benefit of predonation water loading arises from acute rather than chronic hydration. The beneficial effects of predonation water consumption were subsequently replicated in a sample of nearly 9,000 high school donors, and findings from this study suggested that this intervention was most effective when the donor consumed the water closer to the time of the actual blood draw (Newman, Tommolino et al., 2007).

Using Distraction to Reduce Donation Anxiety. For many years, patients have been encouraged to divert their attention from stressful medical procedures as a means of reducing pain and distress. Empirical evidence suggests that many diversions such as music, videos, and reading can have significant benefits by reducing patient anxiety. In the blood donation context, donors who engage in coping strategies that involve either thinking about being elsewhere or explicitly trying to divert attention away from the donation procedures experience less distress (Kaloupek, White, & Wong, 1984; Kaloupek & Stoupakis, 1985). Conversely, those who do not engage in distraction report a decreased likelihood of making future donations (Kaloupek et al., 1984). More recently, audiovisual distraction was assessed as a potential method of reducing presyncopal reactions in first-time blood donors (Bonk, France, & Taylor, 2001). Results indicated that donors who preferred avoidant coping (e.g., turning away from the sight of the needle) were less likely to experience negative reactions when they watched a 3-D movie while giving blood. Those who preferred vigilant coping (e.g., attending to the donation process) were neither helped nor hurt by watching the movie (Bonk et al., 2001). Combined with other studies that did not observe a similar benefit of distraction (Ferguson, Singh, & Cunninham-Snell, 1997), these findings suggest that individual differences in coping style preferences, opportunities for choice, and perceptions of control may play an important role in reducing anxiety and risk for syncopal reactions.

Conclusion

Although the overall rate of syncopal and presyncopal reactions is low, they remain a safety concern and a deterrent to both initial and repeat donation attempts. Even a small reduction in the percentage of first-time donors who experience a syncopal reaction would have a major impact on the blood supply, as a positive initial donation experience can be the difference between a single unit of blood donated and a lifetime contribution of several hundred units. Further, failing to address the experience of such reactions may have a reverberating negative impact on donor recruitment; donors share their stories and in so doing may discourage others in their circle of friends and family from future donations. As described above, a number of strategies may help to reduce the risk for syncopal reactions; however, this is a relatively new area of research and additional studies are needed to address such questions as: (1) Who is most likely to benefit from these interventions? (2) What is the optimum timing for the application of individual strategies relative to the blood draw? (3) What procedures are most practical and effective in the blood donation context? (4) What methods of instruction will maximize donor adherence?

References and Readings

Bonk, V. A., France, C. R., & Taylor, B. K. (2001). Distraction reduces self-reported physiological reactions to blood donation in novice donors with a blunting coping style. *Psychosomatic Medicine, 63*(3), 447–452.

Ditto, B., & France, C. R. (2006). The effects of applied tension on symptoms in French-speaking blood donors: A randomized trial. *Health Psychology, 25*(3), 433–437.

Ditto, B., France, C. R., Albert, M., & Byrne, N. (2007). Dismantling applied tension: Mechanisms of a treatment to reduce blood donation-related symptoms. *Transfusion, 47*(12), 2217–2222.

Ditto, B., France, C. R., Lavoie, P., Roussos, M., & Adler, P. S. (2003). Reducing reactions to blood donation with applied muscle tension: A randomized controlled trial. *Transfusion, 43*(9), 1269–1275.

Ditto, B., Wilkins, J. A., France, C. R., Lavoie, P., & Adler, P. S. (2003). On-site training in applied muscle tension to reduce vasovagal reactions to blood donation. *Journal of Behavioral Medicine, 26*(1), 53–65.

Eder, A. F., Dy, B. A., Kennedy, J. M., Notari, E. P., Iv, Strupp, A., Wissel, M. E., et al. (2008). The American Red Cross donor hemovigilance program: Complications of blood donation reported in 2006. *Transfusion, 48*(9), 1809–1819.

Eder, A. F., Hillyer, C. D., Dy, B. A., Notari, E. P., 4th, & Benjamin, R. J. (2008). Adverse reactions to allogeneic whole blood donation by 16- and 17-year-olds. *JAMA: The Journal of the American Medical Association, 299*(19), 2279–2286.

Ferguson, E., Singh, A. P., & Cunninham-Snell, N. (1997). Stress and blood donation: Effects of music and previous donation experience. *British Journal of Psychology, 88*(2), 277–294.

Foulds, J., Wiedmann, K., Patterson, J., & Brooks, N. (1990). The effects of muscle tension on cerebral circulation in blood-phobic and non-phobic subjects. *Behaviour Research and Therapy, 28*(6), 481–486.

France, C. R., Rader, A., & Carlson, B. (2005). Donors who react may not come back: Analysis of repeat donation as a function of phlebotomist ratings of vasovagal reactions. *Transfusion and Apheresis Science, 33*(2), 99–106.

Germain, M., Glynn, S. A., Schreiber, G. B., Gélinas, S., King, M., Jones, M., et al. (2007). Determinants of return behavior: A comparison of current and lapsed donors. *Transfusion, 47*(10), 1862–1870.

Hanson, S. A., & France, C. R. (2004). Predonation water ingestion attenuates negative reactions to blood donation. *Transfusion, 44*(6), 924–928.

Kaloupek, D. G., & Stoupakis, T. (1985). Coping with a stressful medical procedure: Further investigation with volunteer blood donors. *Journal of Behavioral Medicine, 8*(2), 131–148.

Kaloupek, D. G., White, H., & Wong, M. (1984). Multiple assessment of coping strategies used by volunteer blood donors: Implications for preparatory training. *Journal of Behavioral Medicine, 7*(1), 35–60.

Kamel, H., Tomasulo, P., Bravo, M., Wiltbank, T., Cusick, R., James, R. C., et al. (2010). Delayed adverse reactions to blood donation. *Transfusion, 50*(3), 556–565.

Lu, C. C., Diedrich, A., Tung, C. S., Paranjape, S. Y., Harris, P. A., Byrne, D. W., et al. (2003). Water ingestion as prophylaxis against syncope. *Circulation, 108*(21), 2660–2665.

Newman, B. H. (2004). Blood donor complications after whole-blood donation. *Current Opinion in Hematology, 11*(5), 339–345.

Newman, B., Tommolino, E., Andreozzi, C., Joychan, S., Pocedic, J., & Heringhausen, J. (2007). The effect of a 473-mL (16-oz) water drink on vasovagal donor reaction rates in high-school students. *Transfusion*, *47*(8), 1524–1533.

Thomson, R. A., Bethel, J., Lo, A. Y., Ownby, H. E., Nass, C. C., & Williams, A. E. (1998). Retention of 'safe' blood donors. The Retrovirus Epidemiology Donor Study. *Transfusion, 38*(4), 359–367.

Blood Glucose

Adriana Carrillo and Carley Gomez-Meade
Department of Pediatrics, Miller School of Medicine, University of Miami, Miami, FL, USA

Synonyms

Blood sugar

Definition

Blood glucose concentrations are maintained by tight regulation of glucose production and glucose utilization by insulin- and non-insulin-dependent tissues. Blood glucose levels are usually in the range of 70–99 mg/dL during fasting. Postprandial blood glucose levels might rise up to 140 mg/dl transiently. Blood glucose less than 70 mg/dL is considered hypoglycemia. Three main sources of glucose include gut absorption after ingestion of carbohydrates, endogenous glucose production from glycogenolysis (breakdown of glycogen), and gluconeogenesis (formation of glucose from amino acids, lactate, and glycerol). Only the liver and kidney provide the enzymes necessary for these two processes. The brain depends on continuous plasma glucose supply and cannot use free fatty acid as an energy source. Normoglycemia is essential to preserve cognitive functions, and long-term hypoglycemia or hyperglycemia can result in serious neurological sequela. Tissue-specific transport proteins are responsible for glucose transport from the extracellular to the intracellular space. GLUT-1 and GLUT-3 are glucose transporters that are non-insulin dependent but could be upregulated in prolonged hypoglycemia (Kronenber, Melmed, Polonsky, & Larsen, 2008; Lifshitz, 2007).

Hormones that Regulate Glucose

Insulin is the main glucose-lowering hormone and acts by suppressing glucose production and enhancing glucose use by insulin-dependent

tissues. Insulin secretion is tightly regulated by many factors including exogenous glucose, hormones, and the autonomic nervous system. Counter-regulatory hormones including glucagon, growth hormone, cortisol, and catecholamines increase glucose concentration by stimulating glycogenolysis and gluconeogenesis. Response to hypoglycemia involves a decrease in insulin levels, increase in glucagon levels stimulating glycogenolysis, and secretion of epinephrine that stimulates glucose production from liver and kidney and decreases glucose use. Growth hormone and cortisol stimulate glucose production and limit glucose use. Hormonal response to hypoglycemia occurs in a timely manner having acute lowering of insulin and increases in glucagon within minutes, and epinephrine being a critical hormone in a defect of glucagon. Growth hormone and cortisol are secreted in prolonged hypoglycemia, and their deficiency should be suspected in infants or children with persistent hypoglycemia (Lifshitz, 2007).

Blood Glucose Measurements

Blood glucose can be measured in plasma or in whole blood. Glucose concentration is lower in whole blood than in plasma. To convert whole blood glucose to plasma requires multiplication by 1.15. The majority of laboratories provide reports of plasma glucose. Blood glucose is reported in mg/dl in the United States, but other countries use international units. To convert to IU requires division of mg/dl by 18 and equals mmol/L (Kronenber, Melmed, Polonsky, & Larsen, 2008).

Cross-References

- ▶ Hyperglycemia
- ▶ Hypoglycemia

References and Readings

Kronenber, H., Melmed, S., Polonsky, K., & Larsen, P. R. (2008). *Williams textbook of endocrinology* (11th ed.). Philadelphia: Saunders Elsevier.

Lifshitz, F. (2007). *Pediatric endocrinology* (5th ed.). New York: Informa Healthcare.

Blood Pressure

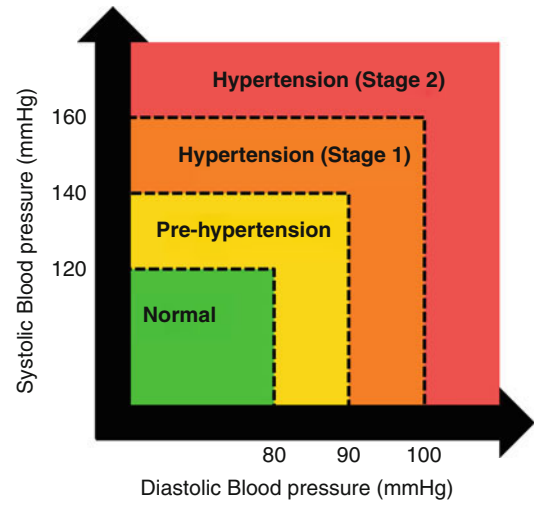
Annie T. Ginty
School of Sport and Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

Synonyms

Diastolic blood pressure (DBP); Systolic blood pressure (SBP)

Definition

Blood pressure is the hydrostatic pressure exerted by circulating blood on the walls of a blood vessel; it is highest in the aorta and large systemic areas. Blood pressure is recorded by two values: systolic blood pressure, which is measured after the heart contracts, and diastolic blood pressure, which is measured before the heart contracts. Systolic blood pressure <120 mmHg and diastolic blood pressure <80 mmHg are considered within normal range. A person is considered to have hypertension when their systolic blood pressure is greater than 140 mmHg or their diastolic blood pressure is greater than 90 mmHg.



Blood Pressure, Fig. 1 Categories of blood pressure

See chart below for blood pressure classifications. Blood pressure is commonly used by physicians as a way to gauge overall cardiovascular function and health of individuals (Fig. 1).

Cross-References

- ▶ Blood Pressure, Elevated
- ▶ Blood Pressure, Measurement of
- ▶ Systolic Blood Pressure (SBP)

References and Readings

Tortora, G. J., & Grabowski, S. R. (1996). *Principles of anatomy and physiology* (8th ed.). New York: Harper Collins College Publishers.

Blood Pressure Reactivity or Responses

Brian M. Hughes
School of Psychology, National University of Ireland, Galway, Galway, Ireland

Synonyms

Cardiovascular response/reactivity; Cardiovascular stress responses; Hemodynamic response/reactivity; Hemodynamic stress responses

Definition

A blood pressure *response* is a change in blood pressure that occurs following exposure to a stimulus. In behavioral medicine, the term is reserved for responses to psychological stress. Blood pressure responses can be quantified as the arithmetic difference between blood pressure measured during a true resting state and that measured during exposure to a stressor. Blood pressure *reactivity* refers to an individual's characteristic pattern of blood pressure responses across time.

Description

Basis of the Blood Pressure Response to Stress

As cognitive stress is disruptive to a person's mental resting state, it is associated with visceral neurological changes in brain function caused by the contemplation, initiation, and maintenance of those activities required by the mental stress response. As a consequence, psychological stress can be expected to result in an investment of mental resources that precipitates observable physiological responses.

Specifically, responding to stressors involves immediate psychological processes relating to emotion, working memory, and decision-making, which in turn invoke neurological activity in the frontal cortex, as well as in the amygdala and the bed nuclei of the stria terminalis within the limbic system. This neurological activity establishes outflow beyond the prefrontal cortex via the brainstem and the hypothalamus, which is intrinsic to the person's psychological and behavioral response (Lovallo & Gerin, 2003). The hypothalamus is also responsible for moderating a number of the body's metabolic regulatory functions; as such, hypothalamic mediation of psychological responses to stress results in virtually simultaneous effects on autonomic and endocrinal outputs, including shifts in cardiovascular function. Indeed, parameters of cardiovascular function are known to be highly sensitive to mental stress, such that even small changes at the cognitive level (e.g., the shifting of attention from one visual stimulus to another) will be followed by measurable changes in many cardiovascular functions, including blood pressure.

Reactivity as a Stable Pattern of Responses

To date, a large body of research has examined the nature, extent, and consequences of blood pressure responding to psychological stress. Such research has established that, all other things being equal, cardiovascular responding is temporally stable within persons (i.e., that a person will tend to exhibit uniform levels of reactivity across tasks, relative to other people) and across time (i.e., that a person's characteristic

level of reactivity in early life will reflect their level of reactivity later in life).

A key feature of the way blood pressure responds to mental stress is that response patterns, while differing across individuals, appear to be stable within individuals. A number of empirical studies have suggested that the magnitude of a person's cardiovascular responses to stress can maintain consistency across very long periods of time, including weeks, years, and even decades (e.g., Hassellund, Flaa, Sandvik, Kjeldsen, & Rostrup 2010). Further indication of the biologically inherent nature of cardiovascular response patterns can be gleaned from the fact that similar patterns of long-term consistency have been observed across species (De Jonge, Bokkers, Schouten, & Helmond, 1996). An important implication of this phenomenon is that people can be classified with some reliability in terms of their dispositional response tendencies. It is this consistent and dispositional pattern of blood pressure responses that is generally referred to as blood pressure "reactivity."

Accordingly, while some stressors are associated with greater cardiovascular responses than others, people's dispositional patterns of blood pressure reactivity appear to maintain further consistency across contexts. A number of different studies have suggested that people's characteristic levels of blood pressure responding to cognitive stress tasks (i.e., whether people are consistently low or high reactors when compared with peers) generalize across different task domains, including mental arithmetic, public speaking, and reaction time testing.

The majority of studies into blood pressure responses to stress seek to elicit stress responses experimentally using standardized presentations of contrived cognitive stressors, usually in laboratory settings. Typically, researchers seek to present cognitive challenges that are novel to participants, in order to avoid the confounding of stress response data with the effects of participants' prior experience of performing particular tasks. This raises the question as to whether such laboratory-based measures validly reflect blood pressure responses to stress that occur in daily life surroundings. Studies that specifically assess laboratory-to-field comparability have confirmed that there is some similarity between artificial research tasks and naturalistic environmental stressors, although little such research has examined the generalizability of cardiovascular variables other than systolic blood pressure, diastolic blood pressure, and heart rate.

Etiological Significance

The usual function of a blood pressure reaction is to prepare the organism to respond behaviorally to a stressor by providing nutritional supply to the major organs needed for physical action. However, as pointed out by Cannon (1929) in his description of the fight-or-flight response, such physical readiness is of less relevance when the threats in the environment require psychological responses rather than physical ones. As such, it can be said that psychological stress provokes a degree of cardiovascular responding beyond that which is metabolically necessary. It has been demonstrated that the decrement between such elevated cardiovascular responses and metabolic needs is such that even relatively mild mental stress can exert an impact on the cardiovascular system equivalent to that of rigorous physical exercise. Further, the extent of this exaggeration appears to be greater among persons who have higher resting blood pressure (Balanos et al., 2010). The fact that blood pressure responses to stress are disproportionate to physiological demands is believed to contribute to the onset and progression of cardiovascular disease in individuals in whom such responses are particularly elevated (Obrist, 1981). This implication of elevated blood pressure responding in the etiology of cardiovascular disease is commonly referred to as the "cardiovascular reactivity hypothesis" (e.g., Krantz & Manuck, 1984; Obrist, 1981).

The reactivity hypothesis offers a number of possible explanations for the centuries-old observation that psychological stress can damage physical health. Several underlying mechanisms have been proposed. Due to their metabolic disproportionality to physical demands, blood pressure responses to psychological stress may serve to permanently disrupt physiological

homeostasis in ways that lead to the gradual resetting of blood pressure (Obrist, 1981). In addition, the repeated eliciting of responses may contribute to cardiac and vascular hypertrophy (Lovallo & Gerin, 2003). Elevated levels of blood pressure reactivity may enhance disease risk by increasing serum levels of low-density lipoproteins while lowering levels of high-density lipoproteins (Raitakari et al., 1997). Further, elevated reactivity may be part of the exaggerated sympathetic responding that leads to increased blood insulin concentrations, which themselves are known to increase hypertension risk (Nazzaro et al., 2002). Finally, elevated reactivity may contribute to atherosclerosis through raising serum concentrations of proinflammatory cytokines (Georgiades, 2007).

A number of empirical studies have corroborated such interpretations by linking heightened cardiovascular reactivity with future hypertension development (e.g., Carroll et al., 2001), atherosclerosis (e.g., Jennings et al., 2004), increased left ventricular mass (e.g., Murdison et al., 1998), and coronary heart disease morbidity and mortality (e.g., Treiber et al., 2003).

Most research has studied the adverse implications of blood pressure responses that are elevated. However, two important caveats have emerged. Firstly, while consistent elevations in blood pressure can be expected to precipitate adverse outcomes over protracted periods of time, they may be beneficial for health when elicited over the short term in response to acute stress. Previous research has suggested that acute stress can stimulate immune effectiveness and, crucially, that cardiovascular reactivity is positively associated with indices of enhanced immune responding (Phillips, Carroll, Burns, & Drayson, 2009). The detrimental cardiovascular impact of sustained elevations in blood pressure tends to be lessened by the fact that responses to stressors reduce over time due to processes of adaptation (Kelsey, Soderlund, & Arthur, 2004), an effect that is itself enhanced among psychologically healthy persons (Hughes, Howard, James, & Higgins, 2011). Secondly, blood pressure responses that are unusually *low* may also be detrimental to health. A number of cross-sectional screening studies have found significant associations between low acute responding and markers of poor health, including elevated levels of depression and obesity (Phillips, 2011). In summary, elevated blood pressure responses to stress may reflect advantageous processes in the short term (as suggested by the association between short-term response and immune strength), so long as such responses are not sustained in the longer term (as implied by associations between elevated reactivity [i.e., sustained responding] and cardiovascular disease end points). Correspondingly, suppressed blood pressure responses may reflect a maladaptive physiological pattern, associated with generally poor health.

Measurement Issues

Blood pressure responses to stress are typically investigated using orthodox measures of systolic and diastolic blood pressure, which collectively represent the impact of circulating blood on the walls of blood vessels. For many decades, these variables, along with heart rate, have comprised the most technically feasible and thus most studied indices of cardiovascular function in behavioral medicine research. Due to the fact that direct arterial assessment of blood pressure would be particularly invasive (and thus stressful), most studies of blood pressure responses to stress have utilized automated sphygmomanometry. Laboratory and field studies usually employ tabletop and ambulatory monitors, respectively. However, while much epidemiological data exists on systolic and diastolic blood pressure and their associated psychosomatic pathways, these variables lack granularity when compared to the multifaceted biodynamics underlying cardiovascular function as a whole, which although more informative are also more difficult to measure. In addition, conventional measurement of blood pressure tends to be noncontinuous and so does not facilitate real-time tracking. Instead, measures are based on episodic readings returned no more frequently than around once every 2 min.

Alternative technological approaches have been developed to enable real-time tracking of cardiovascular function by returning continuous

beat-to-beat measurement of systolic and diastolic blood pressure, as well as ready monitoring of a range of hemodynamic variables underlying these parameters. Such underlying variables may include specific dimensions of cardiac function (such as cardiac output, stroke volume, heart rate variability) and vascular function (such as total peripheral resistance, total arterial compliance, and aortic impedance), as well as neurological variables (such as baroreflex sensitivity). Measurement of such variables is facilitated by photoelectric plethysmography, typically via a finger cuff, which is suitably noninvasive (and thus nonstressful) for most psychological research.

Such devices facilitate the close monitoring of the components of blood pressure, which in turn enables the testing of hypotheses relating to these components. For example, blood pressure is influenced by both vascular and cardiac arousal. Vascular and cardiac arousal appear to be affected differently by independent changes in alpha- and beta-adrenergic nervous system activity. In turn, alpha- and beta-adrenergic responses are believed to reflect different types of psychological coping states (namely, vigilance- and attack-response postures) (Sherwood & Turner, 1992). Accordingly, analyses of hemodynamic response patterns based on independent changes in vascular and cardiac parameters allow researchers to drill down into the qualitative aspects of stress responding and its impact on cardiovascular health (Obrist, 1981).

Influencing Factors

Blood pressure responding to stressors is sensitive to a range of contextual and environmental cues and contingencies. For example, average levels of cardiovascular response are proportionate to the level of cognitive challenge present and can be increased or reduced by proximal factors such as task complexity, emotional comfort, and social context.

Among the main categories of factors that have been found to be relevant are the following: task-specific factors (aspects of stressors that influence the degree of cardiovascular impact, such as computational complexity, task feedback, emotional content, personal or social significance); environment factors (contextual aspects that influence the degree of cardiovascular impact, such as immediate physical environment, time of day, work or home location); physiological factors (aspects of personal biological function that influence the degree of cardiovascular impact, such as sleep levels, habitual and acute caffeine intake, habitual tobacco use, oral contraception use, body mass index, age); social factors (social or interpersonal variables that influence the degree of cardiovascular impact, such as social assistance during stressors, audiences during stressors, availability of social support, aspects of personal social network); and person-level psychological factors (psychological or emotional variables that influence the degree of cardiovascular impact, such as depression, anxiety, or other psychiatric symptoms, and personality types, subtypes, and traits).

Accumulating evidence of the associations between such variables and blood pressure responses to stress is important in two respects. Firstly, it enables researchers to adequately control extraneous influences on blood pressure responses when conducting studies examining disease-relevant physiological mechanisms. Secondly, it provides insight into the ways in which these variables themselves contribute to cardiovascular disease. For example, associations between particular personality traits and blood pressure reactivity may help explain why these same traits emerge as correlates of disease outcomes in epidemiological research.

Cross-References

- ▶ Ambulatory Blood Pressure
- ▶ Ambulatory Monitoring
- ▶ Blood Pressure, Measurement of
- ▶ Cardiovascular Psychophysiology: Measures
- ▶ Cytokines
- ▶ Heart Rate
- ▶ Hypertension
- ▶ Hypothalamic-Pituitary-Adrenal Axis

- Immune Responses to Stress
- Perceived Stress
- Psychophysiological
- Psychophysiology: Theory and Methods
- Stress Test
- Systolic Blood Pressure (SBP)

References and Readings

Balanos, G. M., Phillips, A. C., Frenneaux, M. P., McIntyre, D., Lykidis, C., Griffin, H. S., et al. (2010). Metabolically exaggerated cardiac reactions to acute psychological stress: The effects of resting blood pressure status and possible underlying mechanisms. *Biological Psychology, 85*, 104–111.

Cannon, W. B. (1929). *Bodily changes in pain, hunger, fear, and rage*. New York: Appleton-Century-Crofts.

Carroll, D., Smith, G. D., Shipley, M. J., Steptoe, A., Brunner, E. J., & Marmot, M. G. (2001). Blood pressure reactions to acute psychological stress and future blood pressure status: A 10-year follow-up of men in the Whitehall II study. *Psychosomatic Medicine, 63*, 737–743.

De Jonge, F. H., Bokkers, E. A. M., Schouten, W. G. P., & Helmond, F. A. (1996). Rearing piglets in a poor environment: Developmental aspects of social stress in pigs. *Physiology and Behaviour, 60*, 389–396.

Georgiades, A. (2007). Hyperreactivity (cardiovascular). In G. Fink (Ed.), *Encyclopedia of stress* (2nd ed., pp. 372–376). Burlington, MA: Academic Press.

Hassellund, S. S., Flaa, A., Sandvik, L., Kjeldsen, S. E., & Rostrup, M. (2010). Long-term stability of cardiovascular and catecholamine responses to stress tests: An 18-year follow-up study. *Hypertension, 55*, 131–136.

Hughes, B. M., Howard, S., James, J. E., & Higgins, N. M. (2011). Individual differences in adaptation of cardiovascular responses to stress. *Biological Psychology, 86*, 129–136.

Jennings, J. R., Kamarck, T. W., Everson-Rose, S. A., Kaplan, G. A., Manuck, S. B., & Salonen, J. T. (2004). Exaggerated blood pressure responses during mental stress are prospectively related to enhanced carotid atherosclerosis in middle-aged Finnish men. *Circulation, 110*, 2198–2203.

Kelsey, R. M., Soderlund, K., & Arthur, C. M. (2004). Cardiovascular reactivity and adaptation to recurrent psychological stress: Replication and extension. *Psychophysiology, 41*, 924–934.

Krantz, D. S., & Manuck, S. B. (1984). Acute psychophysiologic reactivity and risk of cardiovascular disease: A review and methodologic critique. *Psychological Bulletin, 96*, 435–464.

Lovallo, W. R., & Gerin, W. (2003). Psychophysiological reactivity: Mechanisms and pathways to cardiovascular disease. *Psychosomatic Medicine, 65*, 36–45.

Murdison, K. A., Treiber, F. A., Mensah, G., Davis, H., Thompson, W., & Strong, W. B. (1998). Prediction of left ventricular mass in youth with family histories of essential hypertension. *American Journal of the Medical Sciences, 315*, 118–123.

Nazzaro, P., Ciancio, L., Vulpis, V., Triggiani, R., Schirosi, G., & Pirrelli, A. (2002). Stress-induced hemodynamic responses are associated with insulin resistance in mild hypertensives. *American Journal of Hypertension, 15*, 865–871.

Obrist, P. (1981). *Cardiovascular psychophysiology: A perspective*. New York: Plenum.

Phillips, A. C. (2011). Blunted cardiovascular reactivity relates to depression, obesity, and self-reported health. *Biological Psychology, 86*(2), 106–113.

Phillips, A. C., Carroll, D., Burns, V. E., & Drayson, M. (2009). Cardiovascular activity and the antibody response to vaccination. *Journal of Psychosomatic Research, 67*, 37–43.

Raitakari, O. T., Pitkänen, O.-P., Lehtimäki, T., Lahdenperä, S., Iida, H., Ylä-Herttuala, S., et al. (1997). In vivo low density lipoprotein oxidation relates to coronary reactivity in young men. *Journal of the American College of Cardiology, 30*, 97–102.

Sherwood, A., & Turner, J. R. (1992). A conceptual and methodological overview of cardiovascular reactivity research. In J. R. Turner, A. Sherwood, & K. C. Light (Eds.), *Individual differences in cardiovascular response to stress* (pp. 3–32). New York: Plenum.

Treiber, F. A., Kamarck, T., Schneiderman, N., Sheffield, D., Kapuku, G., & Taylor, T. (2003). Cardiovascular reactivity and development of preclinical and clinical disease states. *Psychosomatic Medicine, 65*, 46–62.

Blood Pressure, Elevated

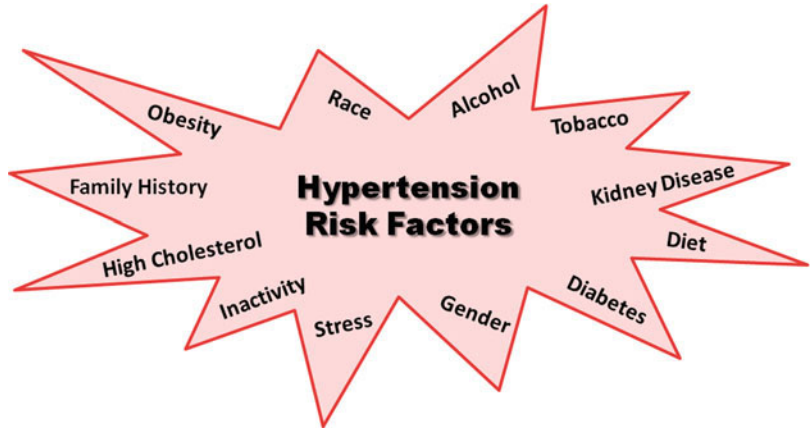
Annie T. Ginty
School of Sport and Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

Synonyms

High blood pressure

Definition

Hypertension (elevated blood pressure) refers to sustained high blood pressure in the arteries.



Blood Pressure, Elevated, Fig. 1 Risk factors for elevated blood pressure

A systolic blood pressure greater than 140 mmHg or a diastolic blood pressure higher than 90 mmHg (millimeters of mercury) is classified as hypertensive. The elevated arterial pressure requires the heart to use more energy to pump; this can eventually lead to myocardial infarction (heart attack) or heart failure. Individuals with hypertension are at an increased risk for cardiovascular disease and have a shorter life expectancy. Hypertension or high blood pressure cannot be attributed to any one identifiable cause. Some causes include diet, lack of exercise, stress, metabolic defects, and heredity factors. A more complete list is displayed in the figure below. Hypertension is more common in industrialized societies (Fig. 1).

Cross-References

- ▶ Blood Pressure, Measurement of
- ▶ Diastolic Blood Pressure (DBP)
- ▶ Systolic Blood Pressure (SBP)

References and Readings

Carretero, O. A., & Oparil, S. (2000). Essential hypertension, Part 1: Definition and etiology. *Circulation, 101*, 329–335.

Vanden, A. J., Sherman, J. H., & Luciano, D. S. (2001). *Human physiology: Mechanisms of body function*. New York: McGraw-Hill.

Blood Pressure, Measurement of

Annie T. Ginty
School of Sport and Exercise Sciences, The University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Elevated blood pressure; Hypertension

Definition

Blood pressure is often measured using a noninvasive technique called a sphygmomanometer. Sphygmomanometer is derived from the Greek word sphygmos, meaning "pulse." A cuff is wrapped around the upper arm of the individual and inflated to a level which is much higher than the expected systolic blood pressure measurement. Then a stethoscope is placed over the brachial artery and when no sounds are picked up it means the artery has been collapsed by the pressure against the walls preventing blood from flowing. Systolic blood pressure is measured by slowly releasing the pressure in the cuff until it eventually reaches a point where tapping sounds are heard, which is caused by blood spurting with each pulse. This occurs during ventricular contraction. Diastolic blood pressure is measured by continuing to

let the air out of the cuff, and when sounds are no longer heard, a reading is taken; no sounds mean blood is continuously flowing through the artery. This occurs during ventricular relaxation.

Measuring blood pressure is a quick and non-invasive way to obtain a general index of cardiovascular function of individuals. It is also one of the most common physiological measures used in behavioral medicine research (Andreassi, 2006).

Cross-References

- ▶ Diastolic Blood Pressure (DBP)
- ▶ Hypertension
- ▶ Systolic Blood Pressure (SBP)

References and Readings

Andreassi, J. L. (2006). *Psychophysiology: Human behavior and physiological response*. Hillsdale, NJ: Lawrence Erlbaum Associates.

Blood Sugar

- ▶ Blood Glucose

Blood Vessel Wall

- ▶ Endothelial Function

Body Composition

Sarah Messiah
Department of Pediatrics, University of Miami, Miami, FL, USA

Synonyms

Anthropometrics; Body mass index; Fat mass; Fat-free mass

Definition

Body composition is the proportion of fat, muscle, and bone of an individual's body. It is most often expressed as percentage of body fat and percentage of lean body mass (LBM) or as a ratio of lean mass to fatty mass. Lean mass includes muscle, bone, skin, internal organs, and body water. Fatty mass consists primarily of body fat (subcutaneous fat) and internal essential fat surrounding organs (visceral or intra-abdominal fat). Two people of the same height and same body weight can appear completely different from each other due to different body compositions. Body composition can provide important information about an individual's possible risk for cardiovascular disease or diabetes.

The American Dietetic Association recommends that a healthy adult male's body should have between 8% and 17% fat and a female should have 10–21% (ADA, 2009). Levels significantly above these amounts may indicate excess body fat. Athletes, leaner individuals, and more muscular individuals will have a body fat percentage lower than these levels.

Body Composition Measurement

Body composition (particularly body fat percentage) can be measured in several ways. Anthropometric measurements usually include height, weight, body mass index (BMI), waist circumference, waist-to-hip ratio, and percentage of body fat. These measures are then compared to reference standards to assess weight status and the risk for various diseases. Anthropometric measurements require precise measuring techniques to be valid but are the simplest and least expensive way to measure body composition.

Body mass index (BMI) is defined as an individual's body weight divided by the square of his or her height (kg/m^2). It is used to estimate an individual's adiposity based on his/her height, assuming an average body composition. BMI is not a direct measure of percentage body fat, but because of the simplicity of measurement and calculation, it is the most widely used diagnostic tool to identify those who are underweight, normal, overweight, obese, or morbidly obese.

Waist Circumference. A high waist circumference (WC) is associated with an increased risk for type 2 diabetes, dyslipidemia, hypertension, and cardiovascular disease when BMI is between 25 and 34.9. (In adults, a BMI greater than 25 is considered overweight and a BMI greater than 30 is considered obese.) Changes in WC over time can indicate an increase or decrease in abdominal fat. Increased abdominal fat is associated with an increased risk of heart disease. WC is measured by locating the upper hip bone and placing a measuring tape around the abdomen (ensuring that the tape measure is horizontal).

Waist-to-Hip Ratio. The waist-to-hip ratio (WHR) has been used as an indicator of potential risk of developing serious health conditions. Research shows that people with "apple-shaped" bodies (with more weight around the waist) face more health risks than those with "pear-shaped" bodies who carry more weight around the hips. While the subject is standing, hip circumference is measured at the point yielding the maximum circumference over the buttocks using a tape measure to measure to the nearest 1 cm. The waist-hip ratio equals the waist circumference divided by the hip circumference.

Percent Body Fat. The most common method of measuring body fat is to assess skinfold thickness using a set of measurement calipers to measure the depth of subcutaneous fat in multiple places on the body. These measurements are then used to estimate total body fat with a margin of error of approximately 4% points. The measurement can use three to nine different standard anatomical sites around the body but typically include the abdominal area, the subscapular region, arms, buttocks, and thighs. The right side is usually only measured for consistency. The tester pinches the skin at the appropriate site to raise a double layer of skin and the underlying adipose tissue, but not the muscle. The calipers are then applied 1 cm below and at right angles to the pinch, and a reading in millimeters (mm) is taken 2 s later. The mean of two measurements should be taken. If the two measurements differ greatly, a third should then be done, then the median value taken.

Another common method of measuring body composition is bioelectrical impedance analysis (BIA), which uses the resistance of electrical flow through the body to estimate body fat. Partly because of a demand for faster and easier methods of evaluating body composition, BIA has become a widely used method of estimating percent body fat. The use of BIA is based on the principle that the conductivity of an electrical impulse is greater through fat-free tissue than it is through fatty tissue. Current-injector electrodes are placed just below the phalangeal-metacarpal joint in the middle of the dorsal side of the right hand and below the metatarsal arch on the superior side of the right foot. Detector electrodes are placed on the posterior side of the right wrist, midline to the pisiform bone on the medial (fifth phalangeal) side with the foot semiflexed.

Total body or estimated total body scans using dual energy x-ray absorptiometry (DEXA) give accurate and precise measurements of body composition, including bone mineral content (BMC), bone mineral density (BMD), lean tissue mass, fat tissue mass, and percent body fat results. The person lays on the whole body scanner, with the x-ray sources mounted beneath a table and a detector overhead. The person is scanned with photons that are generated by two low-dose x-rays at different energy levels. The body's absorption of the photons at the two levels is measured. The ratios can be then used to predict total body fat, fat-free mass, and total body bone mineral content. The procedure can take about 10–20 min. DEXA can also distinguish regional as well as whole body parameters of body composition. As such, it is considered a reference standard.

Body composition is also estimated using cross-sectional imaging methods like magnetic resonance imaging (MRI) and computed tomography (CT). Since MRI and CT give the most precise body composition measures to date, many pharmaceutical companies are very interested in these new procedures to estimate body composition measures before and after drug therapy, especially in drugs that might change body composition.

Cross-References

▶ Body Mass Index

References and Readings

Position of the American Dietetic Association. (2009). Weight management. *Journal of the American Dietetic Association, 109*, 330–346.

Body Fat

M. Di Katie Sebastiano
Kinesiology, University of Waterloo, Waterloo, ON, Canada

Synonyms

Adipose tissue

Definition

Body fat generally refers to adipose tissue, a complex connective tissue with specific roles in metabolism and endocrine function. While the terms "adipose tissue" and "body fat" can be used synonymously, body fat is most often used in the context of body composition, while adipose tissue is more often used when describing the physiological properties of fat. Fat consists of a variety of different cells including adipocytes (fat storage cells), connective tissue matrix (nonliving material to nourish the cells), nerve tissue, stromal vascular cells, and immune cells. Traditionally, adipose tissue was thought of as a passive storage depot of excess energy; however, recently, the specific roles of adipose tissue in endocrine function and metabolism have been identified.

Description

Body composition describes the proportion of lean and fat mass a person carries. Often, percent (%) body fat is used as a descriptor of body composition. Lohman, Houtkooper, and Going (1997) created recommended % body fat levels for children and physically active adults. They suggest average % body fat for healthy adults aged to be 18–35 is 13% for males and 28% for females or 18% for males and 32% for females aged 36–55. Severe excess of body fat, when compared to lean body mass, is known as obesity. An obese individual has a percentage of body fat greater than 22% in males and greater than 35% in females aged 18–35. For an older adult population (aged 36–55), obesity is defined as a body fat of greater than 25% for males and 38% for females (Lohman et al., 1997). The World Health Organization provides general classifications of overweight and obese individuals derived from population. These classifications are based on body mass index (BMI), one of the most common classifications of excessive body fat. BMI provides a ratio of a person's weight (in kg) to their height (in meters squared). Overweight individuals have BMI greater than 25.0 kg/m^2 and less than 29.9 kg/m^2 while individuals classified as obese have a BMI greater than 30 kg/m^2. Morbid obesity occurs in anyone with a BMI greater than 35 kg/m^2 (WHO, 2006). While BMI provides a quick and easy measure of body composition, it does not give any indication of % body fat. There are other methods available to provide a more complete description of body composition. These techniques, unlike weight and BMI, allow for the more precise measurement of body fat and fat-free mass (FFM) to better identify individuals who have excessive body fat.

Bioelectrical impedance analysis (BIA) crudely measures FFM and body fat. It measures body density based on the conduction of an electrical current applied to the organism. The intra- and extracellular fluids serve as electrical conductors for the current, while the cellular membranes act as electrical condensers. Lean body tissue contains a greater percentage of water (70–75%) while fat mass contains only 10–15% water (Kyle et al., 2004a). As such lean tissue conducts the current better than fat mass. BIA is based on the relationship between lean tissue's water concentration and conductance. BIA generates a resistance and reactance value that can then be used to calculated total body water, FFM,

and body fat. There are many different equations that have been developed to determine FFM and body fat from BIA measures and they range from generalized equations to population specific equations; it is up to the evaluator to choose the most appropriate equation. There are two excellent reviews that discuss the various equations for BIA analysis (see Kyle et al., 2004a and Kyle et al., 2004b). BIA is still a rather crude measure of body composition as it is based on many assumptions and the status of the participant, such as hydration level, fasting state, and caffeine ingestion, can significantly influence the results of BIA analysis. It, however, is easily accessible and does not require a trained technician to use.

Another method of body composition analysis that is often used in the field to measure body fat is the anthropometric measure of skinfold thickness. Skinfold thickness is often employed in combination with other anthropometric measures such as height, weight, and body circumference to create a general description of a participant's body composition. Also, skinfold measures are inexpensive and require little equipment, i.e., they can be easily used in a variety of settings. Skinfold measures are based on the premise that 30–70% of body fat is located subcutaneously, or just below the skin, and is proportional to visceral fat, or fat surrounding the internal organs and tissue. Calipers are then used to measure the thickness of subcutaneous fat (Heyward, 2006). From the skinfold measures, many equations have been developed to calculate the body density, percentage of body fat. Again these equations can be generalized to healthy or specific clinical populations. However, skinfold measures are still based on a variety of assumptions and requires skilled technicians for accurate measures. It is also a relatively insensitive measure of body fat and cannot detect differences over a short term.

Hydrostatic weight was at one time considered to be a gold standard of body composition analysis. It works by measuring the body volume of an individual submerged underwater. From body volume, body density is then calculated. One can then estimate body fat and fat-free mass from density values described in the literature. While hydrostatic weighing provides a reliable and valid measure of body density and body fat, it has a high subject burden (underwater submersion), it cannot differentiate between the different components of fat-free mass (muscle, organs, etc.) and relies on the assumed densities of FFM and body fat (Heyward, 2006).

Air displacement plethysmography (ADP) works on the same principle as hydrostatic weighing. However, body volume is determined via air displacement instead of water displacement. It has much less subject burden than hydrostatic weighing and is fast, noninvasive, and accessible to a wider range of body compositions. However, it also has the same limitations as hydrostatic weighing in that it cannot differentiate between the different components of fat mass and relies on the assumed densities of FFM and body fat (Heyward, 2006).

Dual-energy X-ray absorptiometry (DXA) uses very low dose radiation to differentiate between soft tissue and bone. Fat tissue is then estimated from the specific attenuation characteristics of soft tissue. DXA is able to distinguish between body fat, lean tissue, and bone, unlike any of the previously mentioned methods of body composition analysis. It is highly precise, and allows for the separation between different regions of the body. DXA, however, cannot differentiate between different compartments (i.e., visceral, subcutaneous, etc.) of fat and lean tissue (Heymsfield et al., 1997).

Computerized tomography (CT) uses X-ray attenuation to detect the different tissues and reconstruct an image of specific fat tissues (i.e., subcutaneous versus visceral), lean tissues (i.e., skeletal muscle, kidneys, liver), and bones. A trained technician can then use software to precisely quantify the amount of muscle and adipose tissue from just a single CT image. CT image analysis also allows differentiation between adipose tissue depots and individual muscle. CT imaging, however, exposes the participant to large doses of radiation, is very expensive, and requires highly skilled technicians to not only take the scan, but also to precisely quantify the amount of muscle and fat. It is still one of the most accurate methods to determine body composition at the tissue-organ level (Heymsfield, 2008).

Magnetic resonance imaging (MRI) is also one of the most accurate methods to determine

body composition. MRI involves the generation of a magnetic field where atomic protons behave like magnets and become aligned in the magnetic field. The protons are then activated by radio waves and absorb energy. A signal is then generated and is used to develop regional and cross-sectional images of the whole body. Fat, muscle, visceral organs, and bone are then precisely quantified (Heymsfield, 2008). MRI has the benefit of trivial radiation exposure, which lowers the risk for the participant. It is also the best method of body fat analysis as it can use multiple images and whole body or serial measures to get a precise measure of body fat and lean tissue. Again, it is very expensive, requires high technical skill, and the images can be affected by respiration.

Each of these methods can generate the amount of body fat and the % of body fat an individual possesses with varying degrees or accuracy and ease of use and cost. It is within the discretion of the individual to determine the most appropriate method.

Cross-References

- ▶ Adipose Tissue
- ▶ Body Composition
- ▶ Body Mass Index
- ▶ Obesity

References and Readings

Heymsfield, S. B. (2008). Development of imaging methods to assess adiposity and metabolism. *Internal Journal of Obesity, 32*(Suppl 7), S76–S82.

Heymsfield, S. B., Wang, Z., Baumgartner, R. N., & Ross, R. (1997). Human body composition: Advances in models and methods. *Annual Review of Nutrition, 17*, 527–558.

Heyward, V. H. (2006). Assessing body composition. In *Advanced fitness assessment and exercise prescription* (5th ed., pp. 171–211). Windsor, ON: Human Kinetics.

Kyle, U. G., Bosaues, I., DeLorenzo, A. D., Deurenberg, P., Elis, M., Gomez, J. M., & Pichard, C. (2004a). Bioelectrical impedance analysis – part I: Review of principles and methods. *Clinical Nutrition, 23*(6), 1226–1243.

Kyle, U. G., Bosaues, I., DeLorenzo, A. D., Deurenberg, P., Elis, M., Gomez, J. M., & Pichard, C. (2004b). Bioelectrical impedance analysis - part II: Utilization in clinical practice. *Clinical Nutrition, 23*(6), 1430–1453.

Lohman, T. G., Houtkooper, L., & Going, S. (1997). Body fat measurement goes high-tech: Not all are created equal. *ACSM's Health and Fitness Journal, 16*, 92–96.

World Health Organization (WHO). (2006). *Global database on body mass index*. http://apps.who.int/bmi/index.jsp?introPage=intro.html. Accessed Jan 2011.

Body Image

Rachel Millstein
SDSU/UCSD Joint Doctoral Program in Clinical Psychology, University of California, San Diego/San Diego State University, San Diego, CA, USA

Synonyms

Appearance evaluation; Body perception; Body size satisfaction

Definition

Body image refers to how a person experiences or feels about his or her body size, weight, shape, or functionality. Body image is a broadly defined and measured construct. Some of the more commonly studied aspects are: body size satisfaction, body size estimation, weight appropriateness, self-perceived overweight, figure rating scale: current-ideal discrepancy, and appearance evaluation. Body image can influence weight-loss behaviors, emotional well-being, and health behaviors.

Description

Body image is a central component of emotional well-being and self-perception, involving the subjective experience and evaluations of the appearance of one's body. Body size satisfaction is the attitudinal component of body image, reflecting the feelings about one's own body, versus perceptual body image which describes one's estimated size. Media, peer, environmental, and personality characteristics can influence

a person's body image and satisfaction. Satisfaction with body image is a major factor which determines self-esteem and health-related behaviors, such as smoking, healthful nutrition patterns, and engaging in physical activity.

Approximately two-thirds of the US adult population is overweight (BMI $>=$ 25–29.9 kg/m^2) or obese (BMI $>=$ 30 kg/m^2) and excess weight is a known risk factor for diseases such as cardiovascular disease, type 2 diabetes, cancer, and mental illness. Concomitant with the increasing body size among the US population have been the counter-influences of national public health initiatives to reduce obesity and cultural preferences toward leaner figures. Obesity and overweight are also associated with decreased quality of life and poor body image. Poor or distorted body image can be a key factor in the etiology of eating disorders (anorexia and bulimia nervosa) and depression. Body Dysmorphic Disorder (BDD) is a newly recognized mental health condition characterized by an excessive concern with one's body image. While most of the body size satisfaction literature addresses weight concerns, it could also encompass feelings about one's height or specific body areas. Understanding the interrelationships between actual body size, perceived body size, and body image can be useful in determining motivation for weight loss, weight-control practices, and improving self-concept.

In studies of body image, women have consistently been found to view themselves as heavier than they actually are and desire a thinner figure. Research shows that African-American, Hispanic, and American Indian/Alaska native women all tend to display higher levels of satisfaction than white women. Many studies have also found the pattern of white women expressing greater body size dissatisfaction and at lower BMIs than their African-American or Hispanic peers. Women also show body size dissatisfaction at a lower BMI than men. National surveys have shown men to be more satisfied with their body size, even if they are overweight. Overall, men tend to show less awareness of being overweight and the necessity of losing weight if overweight or obese. Men appear to ascribe less importance to their body size than do women, which may account for these discrepancies in image and weight-control behaviors. Men who are dissatisfied with their size or weight tend to be split between wanting to gain muscle weight and wanting to lose excess fat, generally striving toward the muscular ideal male body type.

The associations between body size satisfaction and weight-loss practices are complex and depend on a variety of factors, such as actual or perceived body size, psychological factors, and health status, and they may differ by race and sex. In general, more people who report poor body image are likely to indicate that they are trying to lose weight, compared to those with low or no body image dissatisfaction. Traditionally, it has been reported that women who are dissatisfied with their body size or image tend to choose diet as a weight-loss strategy, while men dissatisfied with their bodies or body image focus more on exercise and diet in order to build muscle and lose weight. Dissatisfaction with body size and poor body image may lead women to avoid physical activity. Attempts to promote healthy weight loss, weight maintenance strategies, and positive body image may be best suited to encouraging appropriate physical activity, nutrition behaviors, and realistic body image and beauty expectations.

Cross-References

- ▶ Anorexia Nervosa
- ▶ Binge Eating
- ▶ Body Composition
- ▶ Body Fat
- ▶ Body Mass Index
- ▶ Bulimia
- ▶ Obesity
- ▶ Overweight
- ▶ Self-Concept
- ▶ Self-Esteem
- ▶ Self-Image

References and Readings

Anderson, L. A., Eyler, A. A., Galuska, D. A., Brown, D. R., & Brownson, R. C. (2002). Relationship of satisfaction with body size and trying to lose weight in a national

survey of overweight and obese women aged 40 and older, United States. *Preventive Medicine, 35*, 390–396.
Cash, T. F., & Pruzinsky, T. (Eds.). (1990). *Body images: Development, deviance, and change*. New York: Guilford Press.
Cash, T. F., & Pruzinsky, T. (Eds.). (2004). *Body image: A handbook of theory, research, and clinical practice*. New York: Guilford Press.
Chang, V. W., & Christakis, N. A. (2003). Self-perception of weight appropriateness in the United States. *American Journal of Preventive Medicine, 24*, 332–339.
Flynn, K. J., & Fitzgibbon, M. (1998). Body images and obesity risk among black females: A review of the literature. *Annals of Behavioral Medicine, 20*, 13–24.
Friedman, K. E., Reichmann, S. K., Costanzo, P. R., & Musante, G. J. (2002). Body image partially mediates the relationship between obesity and psychological distress. *Obesity Research, 10*, 33–41.
Grogan, S. (2006). Body image and health: Contemporary perspectives. *Journal of Health Psychology, 11*, 523–530.
McCabe, M. P., & Ricciardelli, L. A. (2004). Body image dissatisfaction among males across the lifespan: A review of past literature. *Journal of Psychosomatic Research, 56*, 675–685.
Must, A., Spadano, J., Coakley, E. H., Field, A. E., Colditz, G., & Dietz, W. H. (1999). The disease burden associated with overweight and obesity. *Journal of the American Medical Association, 282*, 1523–1529.
Ogden, C. L., Carroll, M. D., Curtin, L. R., McDowell, M. A., Tabak, C. J., & Flegal, K. M. (2006). Prevalence of overweight and obesity in the United States, 1999–2004. *Journal of the American Medical Association, 295*, 1549–1555.

Body Language

▶ Communication, Nonverbal
▶ Nonverbal Communication

Body Mass Index

Sarah Messiah
Department of Pediatrics, University of Miami, Miami, FL, USA

Synonyms

Anthropometric; Body measurement; Height; Weight

Definition

Body mass index (BMI) is defined as an individual's body weight divided by the square of his or her height (standard unit of measure is kg/m^2) (Table 1). It is used to estimate an individual's adiposity based on his/her height, assuming an average body composition. BMI is not a direct measure of percentage body fat, but because of the simplicity of its measurement and calculation, it is the most widely used diagnostic tool to identify those who are underweight, normal weight, overweight, obese, or morbidly obese. The most significant limitation of BMI is that the formula does not take into account phenotypical characteristics such as muscle mass (e.g., athletes who may be classified as "overweight" or "obese" according to their BMI, yet have a very low overall percent of body fat), bone mass, and frame size as well as varying proportions of fat, cartilage, and water weight. However, research has shown that BMI correlates well with direct measures of body fat, such as underwater weighing and dual energy x-ray absorptiometry (DEXA) (Mei et al., 2002; Garrow & Webster, 1985).

Since the early 1980s, the World Health Organization (WHO) has used BMI as the standard for recording obesity statistics worldwide (World Health Organization, 1995, 2000, 2004) (Table 2). In the United States, The Centers for Disease Control and Prevention (CDC) monitor BMI in both the pediatric and adult populations to generate prevalence estimates of underweight, normal weight, and overweight in the population (Ogden, Carroll, Curtin, Lamb, & Flegal, 2010). For adults 20 years old and older, BMI is interpreted using standard weight status categories that are the same for all ages and for both men and women.

For children and teens in the range of ages 2–20 years, the interpretation of BMI is both age and sex specific. While the BMI number is calculated the same way for children and adults, the criteria used to interpret the meaning of the BMI number for children and teens are different from those used for adults. For children and teens, BMI age- and sex-specific percentiles are used for two reasons: (1) the amount of body fat changes with age and (2) the amount of body fat

Body Mass Index, Table 1 How to calculate body mass index (BMI)

SI units	BMI = mass (kg)/(height (m))^2
	Note: Since height is commonly measured in centimeters, divide height in centimeters by 100 to obtain height in meters
Imperial/US customary units	BMI = mass (lb) × 703/(height (in))^2
	BMI = mass (lb) × 4.88/(height (ft))^2

Body Mass Index, Table 2 The international classification of adult underweight, overweight, and obesity according to body mass index

Classification	BMI(kg/m^2)	
	Principal cutoff points	Additional cutoff points
Underweight	<18.50	<18.50
Severe thinness	<16.00	<16.00
Moderate thinness	16.00–16.99	16.00–16.99
Mild thinness	17.00–18.49	17.00–18.49
Normal range	18.50–24.99	18.50–22.99
		23.00–24.99
Overweight	≥25.00	≥25.00
Pre-obese	25.00–29.99	25.00–27.49
		27.50–29.99
Obese	≥30.00	≥30.00
Obese class I	30.00–34.99	30.00–32.49
		32.50–34.99
Obese class II	35.00–39.99	35.00–37.49
		37.50–39.99
Obese class III	≥40.00	≥40.00

Source: Adapted from WHO (1995, 2000, 2004)

differs by gender. Because of these factors, the interpretation of BMI is both age and sex specific for children and teens. Therefore, the CDC BMI-for-age growth charts take into account these differences and allow translation of a BMI number into a percentile for a child's sex and age. The percentiles fall into specific categories to define underweight, normal weight, overweight, and obese (Table 3).

For infants and children ages 0–24 months, the CDC recommends that health-care providers use the WHO growth standards to monitor growth via weight-for-length measurements that are sex specific to identify how children should grow when provided optimal conditions.

Body Mass Index, Table 3 Weight status categories for the calculated BMI-for-age percentile, United States pediatric population

Weight status category	Percentile range
Underweight	Less than the 5th percentile
Healthy weight	5th percentile to less than the 85th percentile
Overweight	85th to less than the 95th percentile
Obese	Equal to or greater than the 95th percentile
Morbidly obese	Equal to or greater than the 97th percentile

BMI is used as a screening tool to identify possible weight problems in both. However, BMI is not a diagnostic tool. For example, a person may have a high BMI, but to determine if excess weight is a health risk, a health-care provider would need to perform further assessments. These assessments might include skinfold thickness measurements; evaluations of diet, physical activity, and family history; and other appropriate health screenings and laboratory tests.

Cross-References

- Body Composition
- Obesity
- Overweight

References and Readings

Centers for Disease Control and Prevention, Division of Nutrition, Physical Activity and Obesity, National Center for Chronic Disease Prevention and Health Promotion. (2011). Body Mass Index. Accessed January 4, 2011, from http://www.cdc.gov/healthyweight/assessing/bmi/index.html

Garrow, J. S., & Webster, J. (1985). Quetelet's index (W/H2) as a measure of fatness. *International Journal of Obesity, 9*, 147–153.

Mei, Z., Grummer-Strawn, L. M., Pietrobelli, A., Goulding, A., Goran, M. I., & Dietz, W. H. (2002). Validity of body mass index compared with other body-composition screening indexes for the assessment of body fatness in children and adolescents. *American Journal of Clinical Nutrition*, *75*(6):978–985.

Ogden, C. L., Carroll, M. D., Curtin, L. R., Lamb, M. M., & Flegal, K. M. (2010). Prevalence of high body mass

index in US children and adolescents, 2007–2008. *Journal of the American Medical Association, 303*, 242–249.
World Health Organization. (1995). *Physical status: the use and interpretation of anthropometry*. Report of a WHO Expert Committee. WHO Technical Report Series 854. Geneva, Switzerland: Author.
World Health Organization. (2000). *Obesity: preventing and managing the global epidemic*. Report of a WHO Consultation. WHO Technical Report Series 894. Geneva, Switzerland: Author.
World Health Organization Expert Consultation. (2004). Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *The Lancet, 363*, 157–163.

Body Measurement

▶ Body Mass Index

Body Perception

▶ Body Image

Body Size Satisfaction

▶ Body Image

Bogalusa Heart Study

Sarah Messiah
Department of Pediatrics, University of Miami, Miami, FL, USA

Synonyms

Childhood origins of cardiovascular disease; Cohort study; Longitudinal study

Definition

The Bogalusa Heart Study, originating in Bogalusa, Louisiana, has been focused on examining the early natural history of cardiovascular disease (CVD), coronary artery disease, and essential hypertension among a semirural community–based cohort of black and white children and young adults for over 30 years.

Description

The Bogalusa Heart Study, originating in Bogalusa, Louisiana, has been focused on examining the early natural history of cardiovascular disease (CVD), coronary artery disease, and essential hypertension among a semirural community–based cohort of black and white-children and young adults (Freedman et al., 2010; Voors, Foster, Frerichs, Webber, & Berenson, 1976). For over 30 years, study subjects identified in early childhood have been followed, and their anthropometrics, blood pressure, heart rate, blood cholesterol levels, as well as several other clinical end points that characterize CVD have been periodically measured (Voors, Webber, Frerichs, & Berenson, 1977). The investigators have described the incidence and prevalence of biologic and behavioral CVD risk factors in these children. Their population has enabled them to document differences not only between males and females but also between blacks and whites. The results from the Bogalusa Heart Study have clearly documented that atherosclerosis has its basis in childhood and that prevention can and must begin at the early ages and have resulted in hundreds of publications in the scientific literature (National Heart, Lung, and Blood Institute, http://clinicaltrials.gov/ct2/show/NCT00005129).

Design

The initial survey of over 3,500 children was initiated in 1973–1974 and was restricted to children from ages 2 ½ to 14 (Webber, Frank, Smoak, Freedman, & Berenson, 1987). A physical examination that included collecting anthropometric data, hemoglobin, blood pressure, serum lipids, and a health history was conducted. In 1976–1977, the second cross-sectional survey of over 4,000 children expanded the eligibility criteria to include children ages 5–17 years old. This

survey included information on salt intake, smoking, health beliefs, and attitudes, and for girls ages 8–17, menstrual history and oral contraceptive use. The third survey of over 3,500 children in 1978–1979 also collected skinfold thickness and two measurements of heart rate. The fourth survey of over 3,300 children in 1981–1982 added data on alcohol use, type A behavior, peer networks, and dieting habits.

The Bogalusa Heart Study continued to use a cross-sectional and longitudinal design with the general cross-sectional survey of approximately 3,700 Bogalusa children ages 5–17 in 1988–1989 in the sixth screen and additional longitudinal studies to recall children in defined subgroups for more intensive evaluation. Half of the 12,000 children screened since 1973 had been studied three or more times. There were several other cohort groups and studies. The Newborn-Infant Cohort Study was designed to describe distributions, interrelationships, and trends through time for blood pressure, serum lipid and lipoprotein concentrations, dietary intake patterns, and anthropometric measurements. Four hundred and forty infants born between January 1, 1974, and June 30, 1975, were examined at birth, at 6 months, and yearly at ages 1–4 and at 7, 10, and 13 years for cardiovascular risk factor variables. The Post-High School Study examined young adults ages 21–30 who previously were examined as children ages 5–14 in the first Bogalusa Heart Study screening in 1973–1974. The population included approximately 4,603 young adults originally screened and any other children or adolescents examined for the first time in any subsequent surveys.

The fifth screening began in 1988 and extended through December 1991. The Pediatric Pathology Risk Factor Program, which began in 1978, documented the relationship of cardiovascular disease risk factors to anatomic and pathologic changes. A local information system was established to obtain family or coroner's consent to autopsy any deceased resident between the ages of 3 and 26 in the Bogalusa area. Autopsy specimens were collected from over 100 deceased children and young adults, of whom approximately 40% had been previously examined in the Bogalusa Heart Study. Major activity during 1988–1991 involved 24-h dietary recall collections on all the 1963, 1966, and 1968 birth cohorts attending the Post-High School Study. A food frequency questionnaire was also self-administered to all the Post-High School Study participants. The use of these two dietary methodologies, 24-h dietary recall and food frequency questionnaire, provided data to assess the nutrient composition of diets of young adults, assess the weekly consumption of individual foods, compare nutrient composition data with food frequency data, and compare dietary intakes at the post-high school age with those of school age.

Several substudies were conducted using the Bogalusa Heart Study population. Among them were the impact of childhood obesity on risk factors, the relationship of apolipoproteins A-I and B in children to parental myocardial infarction, and the relationship between left ventricular size, as demonstrated by echocardiography and blood pressure distribution (Freedman et al., 2008). The study was renewed in Fiscal Year 1992 in order to follow up the previously examined young adults for development of abnormal levels of cardiovascular risk factors and clinical disease.

In 1997, the study was renewed and extended through June 2002 in order to study the impact of genetic factors on the evolution from childhood cardiovascular risk factors to subclinical and clinical morbidity in an adult population, ages 20–40, who had been followed over a long period of time. The study also seeks to study the association of risk factor phenotypes to anatomic changes in the cardiovascular system, as seen by necropsy. The population for genotype-phenotype studies includes approximately 1,400 siblings derived from 178 longitudinal birth cohorts. The cardiovascular phenotypes include obesity, blood pressure, lipids, lipoproteins, apoproteins, homocysteine, glucose-insulin, fibrinogen, plasminogen activator inhibitor-1, and von Willebrand factor. Environmental risk factors consist of sociodemographic characteristics, tobacco and alcohol use, oral contraception, physical activity, and diet. Subclinical morbidity includes echo-Doppler measurements of cardiac-carotid structure and function. Using robust sibling pair linkage

methods, a genome-wide search involving 391 markers with spacing of 10 cM is conducted for genes which influence quantitative traits. This is supplemented with 41 highly polymorphic markers located in or near candidate genes likely to be related to obesity, lipoprotein metabolism, blood pressure, insulin resistance, diabetes, atherogenesis, and thrombosis. The study is shifting from a population-based epidemiologic study to a family-based genetic epidemiologic study.

Over the past three decades, the Bogalusa Heart study has resulted in the following key scientific findings:

- Observations clearly show that the major etiologies of adult heart disease, atherosclerosis, coronary heart disease, and essential hypertension begin in childhood. Documented anatomic changes occur by 5–8 years of age.
- CVD risk factors can be identified in early life. Normative values from a large biracial (black-white) population (approximately 10,000 individuals) are available for comparison.
- The levels of risk factors in childhood are different than those in the adult years. Levels change with growth phases, i.e., in the first year of life, during puberty and adolescence, in the transition to young adulthood, and in adulthood.
- Autopsy studies show atherosclerotic lesions in the aorta and coronary vessels, and changes in the kidney vasculature relate strongly to clinical CVD risk factors, clearly indicating atherosclerosis and hypertension begin in early life.
- Gender and race contrasts are a major contribution to the research findings. It is well known that blacks have more hypertension and diabetes, white males have more early coronary artery disease, and women show a lag in the development of heart disease.
- Environmental factors are significant and influence dyslipidemia, hypertension, and obesity. Those that are controllable include diet, exercise, and cigarette smoking.
- Lifestyles and behaviors that influence CVD risk are learned and begin early in life. Healthy lifestyles should be adopted in childhood because they are critical to modulation of risk factors later in life. Primary care physicians, pediatricians, and cardiologists can play a major leadership role in the prevention of adult heart diseases beginning in childhood. Physicians are encouraged to obtain risk factor profiles on children, along with a family history of heart disease.

Cross-References

- ▶ Coronary Heart Disease
- ▶ Health Disparities
- ▶ Hypertension
- ▶ Longitudinal Research

References and Readings

Freedman, D. S., Fulton, J. E., Dietz, W. H., Pan, L., Nihiser, A. J., Srinivasan, S. R., et al. (2010). The identification of children with adverse risk factor levels by body mass index cutoffs from 2 classification systems: The Bogalusa Heart Study. *American Journal of Clinical Nutrition, 92*(6), 1298–1305.

Freedman, D. S., Patel, D. A., Srinivasan, S. R., Chen, W., Tang, R., Bond, M. G., et al. (2008). The contribution of childhood obesity to adult carotid intima-media thickness: The Bogalusa Heart Study. *International Journal of Obesity, 32*(5), 749–756.

Voors, A. W., Foster, T. A., Frerichs, R. R., Webber, L. S., & Berenson, G. S. (1976). Studies of blood pressure in children, ages 5–14 years, in a total biracial community: The Bogalusa Heart Study. *Circulation, 54*(2), 319–327.

Voors, A. W., Webber, L. S., Frerichs, R. R., & Berenson, G. S. (1977). Body height and body mass as determinants of basal blood pressure in children–The Bogalusa Heart Study. *American Journal of Epidemiology, 106*(2), 101–108.

Webber, L. S., Frank, G. C., Smoak, C. G., Freedman, D. S., & Berenson, G. S. (1987). Cardiovascular risk factors from birth to 7 years of age: the Bogalusa Heart Study. Design and participation. *Pediatrics, 80*, 767–778.

Brain

- ▶ Brain, Cortex
- ▶ Brain, Imaging
- ▶ Brain, Tissue
- ▶ Hypothalamus

Brain and Spinal Cord

▶ Central Nervous System

Brain Damage

Mary Spiers
Department of Psychology, Drexel University, Philadelphia, PA, USA

Synonyms

Brain injury; Brain trauma

Definition

Brain damage causes physical damage to the brain that may range from microscopic neuronal damage to damage that extends over a large area of the brain. Although, technically, brain damage may result from a number of conditions including disease (e.g., dementias) and developmental conditions (e.g., autism, learning disabilities), the term "brain damage" is usually associated with brain injury. Brain injuries fall into two general categories related to mechanism of injury: traumatic and nontraumatic. Traumatic brain injuries (TBI) result from external forces and are often the result of falls, motor vehicle accidents, or being struck or assaulted. In wartime, blast injuries may also cause TBI. Nontraumatic brain injuries may result from a variety of events or infectious processes including, for example, cerebral vascular accidents (i.e., stroke), anoxia, tumor, seizures/epilepsy, or encephalitis. Brain damage typically results in impairment in brain functioning compared to previous or age-appropriate levels of functioning. The degree and pattern of behavioral change following brain damage is associated with the severity and location of injury.

Cross-References

▶ Brain, Injury
▶ Traumatic Brain Injury

References and Readings

Zillmer, E. A., Spiers, M. V., & Culbertson, W. C. (2008). *Principles of neuropsychology* (2nd ed.). Belmont, CA: Wadsworth/Thompson Learning.

Brain Imaging

▶ Brain, Imaging
▶ Neuroimaging

Brain Injury

▶ Brain Damage
▶ Traumatic Brain Injury

Brain Lesion

▶ Brain, Injury

Brain Pathology

▶ Brain, Injury

Brain Trauma

▶ Brain Damage
▶ Traumatic Brain Injury

Brain Tumor

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

Brain tumors (BT) are tumors which reside in the brain or spinal cord. They include abnormal cell proliferation of neurones or glia cells, in the cranial nervous, in the skull, in the limbic system, in the brain stem, or in the spinal cord. Metastasis may develop in the cortex. Severity of BT varies greatly, since this depends on their size, location, type, and stage of development upon detection. Those all are referred to as primary BT. Since often BT are hidden from the eye due to the skull, BT may be only detected via direct brain scans. Another type of BT are secondary BT, where peripheral tumors metastasize to the brain, mainly including lung, breast, colon cancers, and melanoma. Primary BT are among the ten most fatal cancers, with over 51,000 new cases diagnosed every year in the USA alone (Starkweather et al., 2011).

The most common type of BT is astrocytoma, where 75% of patients die within 5 years of diagnosis. Glioblastomas are one type of BT, with a median survival time of 14.6 months (Schneider, Mawrin, Scherlach, Skalej, & Firsching, 2010). Among the proven risk factors of BT are high-dose ionizing radiation, inherited genetic syndromes, and brain lymphomas associated with AIDS (Davis, 2007). The role of cellular phones in the etiology of BT is still under intense investigation. Often, psychological aspects are uniquely affected in BT, given the location of the tumor and its possible impact on mood, behavior, and cognition (Weitzner, 1999). Psychological factors such as depressive symptoms have been found to independently predict poor prognosis in BT (Starkweather et al., 2011). Furthermore, the latter authors reviewed studies and suggest that the neuroimmune interactions which are under control of astrocytes can in fact contribute to the depressive symptoms and the changes in tumor microenvironment seen in astrocytoma. Given the poor prognosis, observed psychological changes, and understood fear of having a BT, patients and close ones often require special psychological help for this tumor. Thus, BT are a clear example where behavior medicine can play a pivotal role in both research and treatment.

Cross-References

▶ Cancer Survivorship

References and Readings

Davis, F. S. (2007). Epidemiology of brain tumors. *Expert Review of Anticancer Therapy, 7*, S3–S6.
Schneider, T., Mawrin, C., Scherlach, C., Skalej, M., & Firsching, R. (2010). Gliomas in adults. *Deutsches Ärzteblatt International, 107*, 799–807.
Starkweather, A. R., Sherwood, P., Lyon, D. E., McCain, N. L., Bovbjerg, D. H., & Broaddus, W. C. (2011). A biobehavioral perspective on depressive symptoms in patients with cerebral astrocytoma. *Journal of Neuroscience Nursing, 43*, 17–28.
Weitzner, M. A. (1999). Psychosocial and neuropsychiatric aspects of patients with primary brain tumors. *Cancer Investigation, 17*, 285–291.

Brain Wave

Alyssa Haney[1] and Michele L. Okun[2]
[1]Department of Psychiatry, School of Medicine, University of Pittsburgh, Pittsburgh, PA, USA
[2]Sleep Medicine Institute and Department of Psychiatry, School of Medicine, University of Pittsburgh, Pittsburgh, PA, USA

Synonyms

Cortical activity; EEG

Definition

Brain waves are the physical representation of the brain's electrical charge and activity.

Electroencephalography (EEG) is the technique used to record electrical activity. EEG measures voltage fluctuations resulting from ionic current flows within the neurons of the brain. Brain wave activity is recorded from different standard sites on the scalp according to the international electrode placement system. Recording electrical activity requires measurement of voltage between two electrode sites. The electrical activity between electrode pairs is evaluated in terms of amplitude and frequency. Amplitude ranges from 5 to 200 μV. Frequency of EEG activity generally ranges from 0 to 100 Hz (American EEG Society, 1994; Niedermeyer and da Silva, 2004; Towle et al., 1993).

Brain wave activity can be recorded during wakefulness as well as sleep. With regards to sleep, stage N1 sleep or drowsiness is characterized by a fading of the alpha waves and increases in beta activity and a small increase in theta activity. As sleep deepens, high-voltage, single or complex theta or delta waves, which are called vertex sharp waves, appear. Stage N2 sleep is characterized by increased numbers of vertex sharp waves (K-complexes) and centrally predominant spindle waves, sinusoidal 12–14 Hz activity. Stage N3 sleep or slow wave sleep (SWS) is characterized by progressively higher amplitude and low-frequency delta waves (American EEG Society, 1994).

The frequencies are indicated by Greek letters:

- Delta – 0 to 4 Hz
- Theta – 4 to 8 Hz
- Alpha – 8 to 12 Hz
- Beta – 12 to 32 Hz
- Gamma – 3 to 100 Hz

Cross-References

▶ Brain
▶ Sleep

References and Readings

American EEG society. (1994). Guideline fifteen: Guidelines for polygraphic assessment of sleep-related disorders (polysomnography). *Journal of Clinical Neurophysiology, 11*(1), 116–124.

Niedermeyer, E., & da Silva, F. L. (2004). *Electroencephalography: Basic principles, clinical applications, and related fields*. Philadelphia: Lippincot Williams & Wilkins.

Towle, V. L., Bolaños, J., Suarez, D., Tan, K., Grzeszczuk, R., Levin, D. N., et al. (1993). The spatial location of EEG electrodes: Locating the best-fitting sphere relative to cortical anatomy. *Electroencephalography and Clinical Neurophysiology, 86*(1), 1–6.

Brain, Cortex

Elliott A. Beaton
Department of Psychiatry and Behavioral Sciences and the M.I.N.D. Institute, University of California-Davis, Sacramento, CA, USA

Synonyms

Brain; Cerebrum; Fissure; Frontal; Gyrus/Gyri (pl); Lobes; Occipital; Parietal; Sulcus; Telencephalon; Temporal

Definition

The cerebral cortex is the outermost gray matter layer of the telencephalon or cerebrum of the brain.

Description

This entry describes the cerebral cortex in humans. Although there is a high degree of conservation across vertebrate species and especially in mammals, readers should refer elsewhere for phenotypic and functional details regarding other animals.

The cerebral cortex (Latin for "Brain" and "Bark" respectively) is the outermost layer of the cerebrum (also known as the telencephalon). The wrinkled or undulating appearance of the cortex is a result of folding that allows for greater surface area within the confines of the skull. The furrows are referred to as "sulci" (plural for sulcus and

Latin for "furrow") or "fissures" which are simply larger sulci that serve as important navigational landmarks. The rounded ridges between the sulci are the gyri (plural for "gyrus" and Latin from *gyre* meaning whirling or circular). There are individual differences in the patterning of the gyri and sulci, but the general pattern is highly correlated across individuals within species. The cerebral cortex consists of two hemispheres (right and left) that are connected by large bundles axons called commissures: the anterior, the posterior, and the corpus callosum. The largest of these white matter bundles is the corpus callosum and its fibers connect to corresponding regions of each hemisphere.

The surface of the cortex can be described in terms of the superolateral, medial, and inferior surfaces. The inferior surface can be further divided into the orbital and tentorial surfaces but it is commonly described according to the four of six visible lobes from anterior (front) to posterior (back) in a transverse or sagittal view: frontal cortex, temporal cortex, parietal cortex, and occipital cortex (there are also the insular and limbic subcortical lobes). These are named for the cranial bones they sit below and are demarcated by certain sulci and fissures and by hemisphere (i.e., right and left). The frontal and parietal lobes are divided by the central sulcus also known as the sulcus of Rolando. The temporal lobe is divided from the frontal and parietal lobes by the lateral sulcus also known as the Sylvian fissure. The occipital lobe is roughly delimited from the parietal and temporal lobes by the parieto-occipital sulcus converging with a line drawn upward from the preoccipital notch where the cerebellum meets the cerebral cortex.

The phylogenic history and evolution of the human brain is represented in the layers of the cortex. The outermost is the neocortex (Latin for "new bark") also known as the neopallium (Latin = "new mantel") or isocortex (Greek = "equal rind"). After the neocortex is the phylogenically older allocortex (Greek = "other cortex" also known as the archipallium). Next is the older still paleocortex or palepallium (Greek = "old cortex") and the oldest of all, the archaeocortex or archipallium.

In humans, the cerebral cortical surface area is on average between 2,200 and 2,850 cm^2. Typically, cortex is further organized by cell type in six layers or *laminae* that vary in total thickness from 5 mm at the precentral gyrus to 1.5 mm at the frontal and temporal poles with an average thickness of 3 mm. From the surface to the interior, the six layers of the cortex include: (1) molecular layer with mostly dendrites and long axons; (2) external granular layer with mostly small pyramidal cells; (3) pyramidal cell layer; (4) internal granular layer with small pyramidal and stellate cells; (5) inner pyramidal layer of large pyramidal cells; and, (6) the multiform or spindle-cell layer. The neurons of these layers number between 2.6 and 20 billion in the cortex with 0.6×10^9 synapses per mm^3. Regions of the cortex vary in terms of *laminae* thickness, in addition to cellular morphology, which involves the appearance of cells and their axons and dendrites.

There are a variety of other systems for mapping out the cortex including three-dimensional stereotactic coordinates or regions divided based on underlying cytoarchitectonic organization of the cortical tissue referred to as Brodmann's areas (BA). The cortex can also be classified by the general function associated with tissue within the region boundary but it should be noted that brain function requires coordination across and among a variety of brain regions.

For example, the primary visual cortex (also known as striate cortex; BA17) is part of the occipital lobe and is involved in processing visual information. Primary auditory cortex (BA41) is found on the lower surface of the lateral fissure that separates the temporal lobe from the frontal and parietal lobes. Somatosensory information processing occurs in the postcentral gyrus (also known as the primary somatosensory area; BA1,2,3) of the parietal lobe. Integration of visual and somatosensory information also occurs in the parietal lobe. Anterior to the somatosensory cortex are the primary (BA4) and secondary (BA6) motor cortices. Groups of cells within these regions can be quite specialized for a given perceptual or motor function.

Broadly, interpretation, planning, action, learning, and memory occur in the rest of the cerebral cortex in the association areas. The central sulcus serves to divide the adjacent anterior

and posterior cortical regions into the motor association cortex (also called the pre-motor cortex; BA6) and somatosensory association cortex respectively. The visual association cortex abuts the primary visual cortex in the most posterior part of the occipital lobes and the somatosensory association cortex in the parietal lobes, encompassing the lower half of the occipital and extending along the lateroventral temporal lobes.

The auditory association cortex encompasses roughly the upper temporal lobe. The language cortex is lateralized with the dominant hemisphere (commonly the left hemisphere) engaged in reception and production of language, and analogous areas in the non-dominant hemisphere (commonly the right hemisphere) that are involved in producing and understanding voice inflection and tone that provide information about the emotional content of speech. Within this region is a receptive language region known as Wernicke's area (BA22) that extends from the angular (BA39) and supramarginal (BA40) gyri. The expressive language area known as Broca's area (BA44 and 45) is located in the inferior fontal gyrus.

Particularly developed in humans is the most anterior part of the frontal cortex (often called the prefrontal cortex), which is rostral to the motor association cortex. The prefrontal cortex (PFC) is the slowest to mature and continues to develop well into young adulthood. The PFC is further divided into regions such as the orbitomedial (BA11 and 12) and dorsolateral (BA 9 and 10) PFC. These areas are associated with particular aspects of executive function such as impulse control, emotion regulation and reactivity, planning, judgment, working memory, and abstract reasoning.

References and Readings

Fuster, J. (2008). *The prefrontal cortex* (4th ed.). London: Academic/Elsevier.

Gazzaniga, M. S. (Ed.). (2004). *The cognitive neurosciences* (4th ed.). Cambridge, MA: MIT Press.

Kandel, E. R., Schwartz, J. H., & Jessell, T. M. (Eds.). (2000). *Principles of neuroscience* (4th ed.). New York: McGraw-Hill.

Nolte, J. (2009). *The human brain: An introduction to its functional anatomy* (6th ed.). Philadelphia: Mosby/Elsevier.

Brain, Imaging

Elliott A. Beaton
Department of Psychiatry and Behavioral Sciences and the M.I.N.D. Institute, University of California-Davis, Sacramento, CA, USA

Synonyms

Brain imaging; Computerized tomography (CT); Diffuse optical imaging (DOI); Event-related optical imaging (EROI); Functional magnetic resonance imaging (fMRI); Imaging; Magnetic resonance imaging (MRI); Positron emission tomography (PET)

Definition

Neuroimaging broadly refers to the relatively noninvasive technologies and techniques for localizing, measuring, and visualizing central nervous system function and structure. Common neuroimaging methodologies include magnetic resonance imaging (MRI), positron emission tomography (PET), and computerized tomography (CAT/CT).

Description

Neuroimaging refers to a collection of techniques used to noninvasively view structure and function of living brain tissue. The methods used to visualize brain tissue have evolved significantly over the last several decades from using x-ray technologies to the more recent and increasingly ubiquitous (nuclear) magnetic resonance imaging.

Contrast X-rays and Computerized Axial Tomography (CAT/CT)

X-ray photography creates images by passing x-rays through the body and onto a photographic plate by taking advantage of variation in x-ray radiation absorption of different tissues. Certain molecules and denser materials absorb more radiation and thus less reaches the photographic plate.

This method is excellent for seeing skeletal bones that appear on the photographic plate with a high degree of contrast compared to surrounding tissues. It is less useful for tissues that do not strongly differ in x-ray radiation absorption such as parts of the brain. One method to get around this is to introduce a radiopaque agent to increase contrast by differentially absorbing x-rays. This allows for the visualization of the cerebral ventricular and circulatory systems. Pneumoencephalography involves injecting air into the cerebral ventricular system to briefly displace cerebral spinal fluid (CSF), and cerebral angiography involves injecting a radiopaque dye into a cerebral artery. These methods are limited in the information they produce but can be used to examine general brain atrophy, damage, or displacement of blood vessels.

The next step in the evolution of x-ray imaging of the living brain was the introduction of computed tomography (CT) which is sometimes referred to as computerized axial tomography (CAT). However, CT is more appropriate, because "axial" merely refers to the plane of image acquisition, and images can just as easily be acquired in the coronal or sagittal planes. CT utilizes an x-ray detector rather than a photographic plate. The x-ray source and detector are mounted opposite one another on a rotating ring inside a tube that encircles the person being scanned. The CT scanner captures numerous images of the brain from several angles as the x-ray source and detector rotate around the head. These images are then reconstructed by a computer to make three-dimensional multi-slice images of the living brain. The brighter and darker areas of the images are described as "hyperdense" and "hypodense," respectively, with grayish components of the images as "isodense." Water and CSF appears almost black, white matter darker than gray matter, and skull as nearly white. Variation in image intensity is more carefully delineated in Hounsfield units (HU) with water having an HU of 0, CSF an HU between 8 and 18, gray matter and white matter HU = 37–41 and 30–34, respectively, and bone HU = 600–2,000. CT scans have the benefit of being relatively inexpensive and having very fast acquisition times making them particularly valuable tools for detecting recent brain trauma and intracranial lesions in emergency situations. Limitations of CT scanning include poorer contrast between brain tissue types and the number of CT scan any one person can have at a given time is limited because of safety requirements to limit exposure to x-ray radiation. Furthermore, while CT can be used to visualize brain structure, it is not useful for measuring brain function while engaging in a process or activity. Other methods allowing for accurate localization of brain function include positron emission tomography (PET) and functional magnetic resonance imaging (fMRI). Imaging equipment that combine CT and PET technologies in one package are now commonly available and increase information yield and utility with the practical benefit of taking up less space than dedicated CT and PET scanners.

Single Photon/Positron Emission Tomography (SPECT/PET)

Positron emission tomography (PET) and single photon emission computerized tomography (SPECT) are used to image brain activity. This method also uses radiation and radiation detectors but rather than shooting an x-ray through the material to be imaged, PET utilizes radiolabeled tracers in the form of chemicals that have specific actions within the brain. For example, fluorine-18-labeled 2-fluoro-2-deoxy-D-glucose (18F-FDG) is a commonly used radiotracer. When 18F-FDG is injected into the carotid artery, it is rapidly taken up by metabolically active neurons during an experimental task as it very similar to glucose. However, it cannot be metabolized like glucose and thus accumulates in active brain regions where it slowly breaks down. The radioactive label (or ligand) gives off photons (i.e., S*P*ECT) as a result of a nuclear process where a proton in the nucleus is converted into a neutron, neutrino, and a positron (i.e., *P*ET). Both the neutrino and the positron are then ejected from the nucleus. The kinetic energy of the ejected positron both varies and declines at a rate that depends on the nature of the surrounding material. When an ejected positron meets an

electron, it creates an annihilation reaction where the electron and the positron turn into two photons that travel in opposite directions (180°) of each other. These photons are measured as a line by two of a series of scintillation detectors mounted in opposition from one another. The images created by the PET scanner are not images of the brain itself; rather, they are images created from the relative distributions of detected amounts of radioactivity in brain regions of interest.

PET is powerful methodology that can be used to study hemodynamics, drug action localization, receptor function, metabolism, and even molecular processes including DNA synthesis. PET is particularly valuable in detecting disease processes that may be evident as metabolic variation but are not yet manifested as anatomical abnormality that could be detected using CT or MRI. However, PET images can be effectively combined with CT or MRI images providing accurate localization of accumulated radioactivity. PET is also advantageous in that radiation exposure is relatively limited. The primary limitation of PET is the necessity for local access to a cyclotron to produce radiotracers. The radiotracers have a very short half-life and thus must be made in close physical proximity to the PET scanner and utilized quickly. The limitations of PET and CT have led to a significant increase in application of methods that do not utilize hard radiation like x-rays or radiolabels that are expensive to produce. Structural and functional magnetic resonance imaging (sMRI and fMRI, respectively) and the recent emergence of near infrared spectroscopic imaging (NIRSI) allow for detailed analyses of both brain structure and function in the living brain.

Magnetic Resonance Imaging (MRI)/ Functional Magnetic Resonance Imaging (fMRI)

Magnetic resonance imaging (MRI) methods produce images of the brain and other bodily regions that are high in both contrast and resolution. Although some MRI methods utilize contrast agents, MRI does not expose patients or study participants to ionizing radiation. Rather, this technique utilizes a very powerful homogeneous and stable electromagnetic field.

This brief description of how MRI works is limited to "classical"/Newtonian physics, but quantum mechanical descriptions are available elsewhere. Protons are found in all of the nuclei of the atoms that make up the body, but conventional MRI utilizes hydrogen protons. Hydrogen protons spin randomly with their magnetic moments "pointing" in random directions until they are in the influence of the strong magnetic field of the MRI scanner where they all align in parallel with the direction (z-axis) of the external field generated by the electromagnet. Application of a radiofrequency (RF) pulse is applied to the z-axis aligned hydrogen protons with an excitation/receiver coil. As a result of absorbed energy from the RF pulse, the hydrogen protons move or "flip" into a higher energy state that is antiparallel to the z-axis toward the x-y plane. With the removal of the RF pulse, the hydrogen protons "relax" or move back into alignment with the external electromagnetic field along the z-axis and release the absorbed energy form the RF pulse as electromagnetic waves that are detected by the excitation/receiver coil and other magnetic gradient coils in three dimensions.

Static contrast methodologies are used to generate anatomical images of the brain. Depending on the type of RF pulse applied, the images highlight different types of tissue or fluids. Static contrast between tissue types is achieved by three properties of protons in tissues: (1) the proton density (i.e., how many hydrogen protons are in the region), (2) proton relaxation times along the z-axis (i.e., the longitudinal relaxation time or T1), and (3) proton relaxation times along the x-y plane (i.e., the transverse relaxation time or T2). Motion contrasts detect dynamic properties of protons in tissues and fluids to generate images of blood flow, capillary irrigation, perfusion, and diffusion of water.

Functional MRI (fMRI) refers to MRI methodologies that estimate brain activity. Brain slices are repeatedly imaged over time allowing for statistical contrast of experimental manipulations. The most common is blood oxygen level–dependant (BOLD) fMRI. BOLD fMRI methods exploit changes in levels of oxygen in the blood that result from the metabolic demands of brain tissue during

neural activity. Active brain tissue utilizes oxygen and the change from an oxygenated state to a deoxygenated state can be detected because deoxygenated blood is paramagnetic. Other methods include perfusion or dynamic-contrast MRI, which measures changes in blood volume using an injected paramagnetic contrast agent such as gadolinium, or magnetic resonance spectroscopy (MRS) which measures localized levels of brain metabolites. There is also diffusion MRI that measures diffusion coefficients of water in brain tissue. Diffusion tensor imaging (DTI) examines the water diffusion coefficients in neighboring voxels to estimate the shapes and directions of white matter tracts.

MRI possesses advantages over CT and PET including very high-resolution images that can be acquired without ionizing radiation. In most MRI procedures, no contrast agent is needed and the procedures are completely noninvasive. MRI still requires significant safety procedures though. The magnet is always active, and any objects that are susceptible to magnetism can become dangerous projectiles within the boundaries of the field. Furthermore, patients and study participants must be screened for metallic objects or medical devices such as pacemakers in and on their bodies.

Diffuse Optical Imaging or Tomography (DOI/DOT) and Near Infrared Spectroscopy (NIRS)

Diffuse optical imaging (DOI) and near infrared spectroscopy (NIRS) are relatively new applications for measuring relative changes in blood volume and oxygenation via hemoglobin levels as a proxy for cellular metabolism. These methods exploit changes in the properties of near IR light projected through tissue in the absorptive spectra and light scattering properties of water, oxygenated hemoglobin, and deoxygenated hemoglobin. Like BOLD fMRI, DOI measures the hemodynamic response as blood flows to the active tissue supplying oxygen to satisfy the metabolic needs of neurons in the active region. Changes in the way that light moves through brain tissue from the IR source to the IR detector can be computationally modeled and blood flow to particular brain regions can be examined based on the placement of the IR source and detectors.

Modeling how light moves through the various tissues of the head is a complex process that contributes to DOI and NIRS limitations. One method of simplifying the model is to assume the brain region being scanned is essentially "flat" and that the tissues do not differ in their optical properties. However, anatomical MRI can be combined with DOI/NIRS to provide a better model of absorption and scattering of light with bone and other head tissues. Advantages of this technology include a high degree of portability, rapid data acquisition, relative low cost, and complete noninvasiveness. The primary disadvantages result from the lack of robust spatial resolution and that imaging is limited to surface and near-surface brain tissue.

Cross-References

- ▶ Magnetic Resonance Imaging (MRI)
- ▶ Neuroimaging

References and Readings

Azar, F., & Intes, X. (Eds.). (2008). *Translational multimodal optical imaging*. Norwood, MA: Artech House.

Christian, P. E., & Waterstram-Richm, K. M. (Eds.). (2012). *Nuclear medicine and PET/CT technology and techniques* (7th ed.). St. Louis, MO: Mosby.

Hanson, S. J., & Bunzl, M. (Eds.). (2010). *Foundational issues in human brain mapping*. Cambridge, MA: MIT Press.

Huttel, S. A., Song, A. W., & McCarthy, G. (2008). *Functional magnetic resonance imaging* (2nd ed.). Sunderland, MA: Sinauer.

Jezzard, P., Mathews, P. M., & Smith, S. S. (2001). *Functional MRI: An introduction to methods*. New York: Oxford University Press.

Jiang, H. (2010). *Diffuse optical tomography*. Boca Raton, FL: CRC Press/Taylor & Francis.

Mettler, F. A., & Guiberteau, M. J. (2006). *Essentials of nuclear medicine imaging* (5th ed.). Philadelphia: Saunders/Elsevier.

Romans, L. (2011). *Computed tomography for technologists: A comprehensive text*. Baltimore: Wolters Kluwer Health/Lippincott Williams & Wilkins.

Wahl, R. L., & Beanlands, R. S. B. (Eds.). (2009). *Principles and practice of PET and PET/CT* (2nd ed.). Philadelphia: Lippincott Williams & Wilkins/Wolters Kluwer.

Brain, Injury

Eric Roy
Department of Kinesiology, University of Waterloo, Waterloo, ON, Canada

Synonyms

Brain damage; Brain lesion; Brain pathology

Definition

A brain injury refers to any damage that occurs to the brain. Brain injuries can be classified in various ways: specific types of injury, primary and secondary, focal and diffuse.

Description

Types of Injury

Brain injuries can be developmental in origin while others are acquired through trauma, stroke, or neurodegenerative processes such as Alzheimer disease. The focus here will be on brain injury acquired through trauma. Regardless of etiology, brain injury can have primary and secondary as well as focal and diffuse effects (Jallo, & Loftus, 2009; Weber, & Maas, 2007).

Primary Versus Secondary Injury

A primary injury arises during the initial blow to the brain. At the moment of trauma, the physical structures of the brain are displaced. This displacement results in a contusion or bruise to the brain, damage to blood vessels, and stretching or tearing of axons. The blood–brain barrier and the tissues covering the brain called the meninges may also be damaged. As well the cells in the brain may be damaged and die. Different tissues in the brain have varying thresholds of deformation or response to mechanical loading and so vary in their potential for injury. Depending on the location and intensity of the forces during the initial traumatic event, some tissues in the brain may experience greater forces and so may be more affected than others.

There are two principal mechanisms of primary injury. One involves actual trauma to the brain arising from the brain coming in contact with the inside of the skull. The point of initial contact results in what is called the coup injury. Depending on the forces involved in the trauma, the brain may in a sense bounce off the coup location inside the skull and move in the opposite direction. The point at which the brain contacts the skull results in the contrecoup injury. Depending on the force of the trauma, there can be a bleeding or hemorrhaging in the brain. This bleeding can be epidural or extradural meaning outside the dura mater, the outer most of three membranes or meninges covering the brain. There may also be subdural bleeding. This bleeding can lead to an epidural or subdural contusion or hematoma.

The other mechanism of primary injury arises from the whiplash effect with the head rotating on the neck due to the acceleration and deceleration forces present in some traumatic accidents such as in motor vehicle accidents. These acceleration-deceleration forces result in shearing strains which may cause tearing of blood vessels deep in the brain resulting in petechial hemorrhages. These strains also causing tearing of the axons or diffuse axonal injury.

The primary injuries lead to secondary injuries which result from processes precipitated by the trauma. Secondary injury begins within hours of the primary injury and plays an important role in the eventual outcome. While most people who suffer a traumatic brain injury recover to varying degrees, about 40% deteriorate due this secondary damage. Secondary injury results from complications associated with the primary injury which include cerebral hypoxia (low oxygen levels in the brain), hypotension (low blood pressure), cerebral edema (brain swelling), and increased intracranial pressure (pressure within the skull). Large increases in intracranial pressure can lead to pushing the brain (herniation) through the hole in the base of the skull called the foramen magnum.

Other secondary damage includes meningitis, acidosis (high acid levels in the blood), and

hypercapnia (high levels of carbon dioxide in the blood). Secondary injury can also be caused by release and imbalances in brain chemicals called neurotransmitters. One effect is called excitotoxicity which can cause neurodegeneration through the action of free radicals. Cerebral autoregulation is another type of secondary injury which affects the regulation of blood flow to the brain. Breakdown in the blood–brain barrier as well as cerebral ischemia are also secondary changes in brain function following injury.

Focal and Diffuse Injury

Focal injury refers to injury which occurs in a specific location in the brain. Diffuse injuries involve damage over a more widespread region. Focal injuries arise from a blow to the head which affects the underlying brain area. Diffuse injuries most often arise from acceleration-deceleration forces rather than a blow to the head. It is common for these types of injury to occur at the same time. Focal and diffuse injuries can occur in the context of trauma but also from other forms of brain injury such as stroke.

In the context of trauma, a focal injury arises from direct forces such as when the head strikes the inside of the windshield in a motor vehicle accident or the ice surface in a hockey game. These types of focal injury involve the skull remaining intact. Other focal injuries involve penetration of the skull such as in gunshot wounds to the head. Focal injuries are associated with symptoms arising from damage to the affected brain area such as the loss of hand function on one side of the body due to damage in the motor area on the opposite side of the brain.

Diffuse injury involves damage to the brain over a more widespread area. Diffuse may be a misnomer for this type of injury since the damage often involves multiple focal injuries spread over wide areas in the brain. In the context of trauma, such injuries arise from shearing forces associated with acceleration-deceleration forces resulting in diffuse axonal injuries and tearing of blood vessels. In the context of stroke, diffuse injury arises from multiple strokes occurring around the same time in different brain areas.

Diffuse axonal injury is most often seen in traumatic brain injury and refers to damage to the white matter tracts arising from rotational shearing forces associated most often with deceleration forces in assaults or motor vehicle accidents. The major source of injury is to the axons, the part of the neuron that affords communication between the neurons. The axon appears white due to the myelination and so is called white matter and collectively these axons form white matter tracts. When the brain is decelerated, parts which vary in their densities and distances from the point of rotation slide over one another and so create shearing forces which serve to tear these tracts. The most common locations for diffuse axonal injuries include white matter tracts of the cerebral cortex, basal ganglia, thalamus, and the deep hemispheric nuclei. Diffuse axonal injury involves axonal separation at the point of the stretch with the part of the axon distal to this tear degrading. It was thought that the major reason for the axonal injury was due to the mechanical forces present at the moment of trauma (the primary injury). Now, it is believed that there are a series of biochemical changes which occur in response to the primary injury hours to days after the primary injury due to shearing forces.

Cross-References

- ▶ Anger Management
- ▶ Anxiety
- ▶ Brain Damage
- ▶ Brain Imaging
- ▶ CAT Scan
- ▶ Cognitive Impairment
- ▶ Dementia
- ▶ Depression: Symptoms
- ▶ Neuroimaging
- ▶ Neuropsychology
- ▶ Therapy, Occupational
- ▶ Therapy, Physical
- ▶ Therapy, Speech
- ▶ Trail-Making Test
- ▶ Traumatic Brain Injury

References and Readings

Coles, J. (2007). Imaging after brain injury. *British Journal of Anesthesia, 99*, 49–60.

Jallo, J., & Loftus, C. (2009). *Neurotrauma and critical care of the brain*. New York: Thieme Medical Publishers.

Weber, J., & Maas, A. (2007). *Neurotrauma: New insights into and treatment*. New York: Elsevier.

Brain, Regions

► Central Nervous System

Brain, System

► Central Nervous System

Brain, Tissue

Victoria Harms and Lorin Elias
Department of Psychology, University of Saskatchewan, Saskatoon, SK, Canada

Definition

Neural or brain tissue is specialized for communication through the transmission of electrical signals. The majority (approximately 98%) of neural tissue is found within the brain and the spinal cord. It is composed of two basic classes of cells: nerve cells (or neurons), which transmit communication signals, and glial cells, which act to support both the structure and function of neurons (Carlson, 2004).

Neurons

The basic functional unit of the brain is the neuron. Its functional role is to send and receive the electrical impulses that communicate messages about sensory, motor, and cognitive events throughout the brain. The average brain contains roughly 100 billion neurons. Although there are upwards of 1,000 different types of neurons, they all have the same basic structure and function.

Each neuron has a soma, or cell body, that performs all the basic metabolic functions required to keep the cell alive and functioning. At one end of the cell body are the dendrites; these are fine processes or branches that receive incoming information from other neurons. Together the cell bodies and dendrites of neurons compose the gray matter of the brain, so named for its pinkish-gray coloration. At the other end of the cell body is the axon, a long cylindrical projection that conducts signals from the cell body for transmission to other neurons. Most axons are surrounded by a fatty layer of tissue, called the myelin sheath, which helps speed the conduction of electrical signals along the axon. White matter is composed mostly of axons and is so named for the whitish appearance created by the myelin sheath (Nolte, 2009).

Information is transferred from one cell to another at communication sites called synapses. Individual neurons are not physically connected to one another; rather between two communicating cells is a tiny gap called the synaptic cleft. The electrical signals transmitted along the axon of the sending (presynaptic) cell trigger the release of specific chemicals (neurotransmitters) which travel across the synaptic cleft and bind to receptor sites on the receiving (postsynaptic) cell (Conners, 2005).

Glial Cells

Greek for "nerve glue," neuroglia, or glial cells, are the support cells of the brain. Outnumbering neurons at a ratio of approximately 10:1, glial cells make up over half of the volume of the brain (Pinel, 2006). Glia were traditionally thought to physically hold neurons together. However, it is now known that glial cells provide structure and support for neurons by surrounding the cell bodies and processes of neurons (Kandel, 1991). Although not directly responsible for information processing and transmission, glial cells do play a variety of essential roles in maintaining and supporting the function of neurons:

Glial cells act to support neurons and, in doing so, provide the overall physical structure for the brain.

Specialized glial cells (oligodendrocytes in the central nervous system, Schwann cells in the peripheral nervous system) produce and maintain the myelin sheath that insulates axons.

Glial cells act as a supply system providing oxygen and nutrients to neurons.

Some glia (e.g., astrocytes and microglia) perform important cleaning and protective roles, removing excess neurotransmitters, pathogens, and cellular debris left following cell death.

During the process of development, specialized glia (radial glial cells) act to guide the migration of neurons to their specific locations in the brain and to direct the path of axon growth.

Cell type	Function
Astrocyte	Structural support, regulation of ion concentrations in the extracellular fluid, provide nutrients to neurons, and clean up debris following neuronal death (phagocytosis)
Oligodendrocytes	Produce the myelin sheath around axons
Radial glia	Specialized astrocyte, directs the path of migrating neurons and guides axon growth during development
Ependymal glia	Create the wall of ventricles and secrete cerebrospinal fluid (CSF)
Microglia	Phagocytosis and immune function protecting the brain from microorganisms; also produce the inflammatory response following brain injury

Cross-References

▶ Brain
▶ Brain, Cortex

References and Readings

Carlson, N. R. (2004). *Physiology of behavior* (8th ed.). Toronto, ON: Pearson.

Conners, B. W. (2005). Synaptic transmission in the nervous system. In W. F. Boron & E. L. Boulpaep (Eds.), *Medical physiology* (pp. 295–324). Amsterdam: Elsevier/Saunders.

Kandel, E. R. (1991). Nerve cells and behavior. In E. R. Kandel, J. H. Schwartz, & T. M. Jessell (Eds.), *Principles of neuroscience* (4th ed., pp. 19–35). Toronto, ON: McGraw-Hill.

Nolte, J. (2009). *The human brain: An introduction to its functional neuroanatomy* (6th ed.). Amsterdam: Elsevier/Mosby.

Pinel, J. P. (2006). *Biopsychology* (6th ed.). Toronto, ON: Pearson.

Brain-Behavior Relationships

▶ Neuropsychology

BRCA1 and BRCA2

Heidi Hamann
Department of Psychiatry, UT Southwestern Medical Center, Dallas, TX, USA

Synonyms

Breast cancer genes; Breast/ovarian genetic testing

Definition

BRCA1 and BRCA2 are breast and ovarian cancer susceptibility genes first identified in the mid-1990s (Miki et al., 1994; Wooster et al., 1995). Mutations in BRCA1 and BRCA2 (BRCA1/2) are associated with significantly increased risks of breast and ovarian cancer among women and smaller increases in breast and prostate cancer among men. The development of predictive genetic testing for BRCA1/2 mutations was accompanied by concerns about psychological and behavioral consequences of learning one's test results (Lerman, Croyle, Tercyak, & Hamann, 2002). However, a meta-analysis by Hamilton, Lobel, and Moyer (2009) concluded that although general and cancer-specific psychological distress may rise immediately after testing for mutation carriers, it tends to return to baseline

levels over time. Mutation noncarriers tend to report short-term decreases in anxiety and long-term decreases in cancer-specific distress. Despite these overall findings, there is a need to more fully understand the social context associated with BRCA1/2 testing; for example, Hamann and colleagues (2008) found that siblings who had different genetic test results experienced more negative interpersonal responses than siblings who shared the same test result.

In addition to investigations of psychological reactions to BRCA1/2 testing, increased attention has been paid to the behavioral consequences of testing. Among mutation carriers, prophylactic breast and ovarian surgeries dramatically reduce associated cancer risks (Domchek et al., 2010). Although the evidence for risk reduction is less clear, BRCA carriers may also receive recommendations for intensive breast (e.g., mammogram, breast MRI) and ovarian (e.g., CA125, transvaginal ultrasound) surveillance. Of note is a recent study of long-term behavioral outcomes which reported that over 80% of female mutation carriers had obtained risk-reducing breast or ovarian surgeries, compared to much lower levels among women with uninformative or negative results (Schwartz et al., 2011). Rates of mammography were generally high for all tested women (66–92%); ovarian screening was less well utilized, but still higher among mutation carriers. In general, results indicate that BRCA testing has favorable effects on behaviors associated with cancer risk reduction.

Despite the accumulated data on BRCA1/2 testing, there is a continued need for outcome assessment among understudied groups, including racial and ethnic minorities and underinsured populations. A more complete picture of the psychosocial and behavioral correlates of BRCA testing will emerge with studies that include more diverse populations.

Cross-References

- ▶ Breast Cancer
- ▶ Cancer Risk Perceptions
- ▶ Cancer, Ovarian
- ▶ Genomics

References and Readings

Domchek, S. M., Friebel, T. M., Singer, C. F., et al. (2010). Association of risk-reducing surgery in BRCA1 or BRCA2 mutation carriers with cancer risk and mortality. *Journal of the American Medical Association, 304*, 967–975.

Hamann, H. A., Smith, T. W., Smith, K. R., Croyle, R. T., Ruiz, J. M., Kircher, J. C., et al. (2008). Interpersonal responses among sibling dyads tested for BRCA1/BRCA2 gene mutations. *Health Psychology, 27*, 100–109.

Hamilton, J. G., Lobel, M., & Moyer, A. (2009). Emotional distress following genetic testing for hereditary breast and ovarian cancer: A meta-analytic review. *Health Psychology, 28*, 510–518.

Lerman, C., Croyle, R. T., Tercyak, K. P., & Hamann, H. (2002). Genetic testing: Psychological aspects and implications. *Journal of Consulting and Clinical Psychology, 70*(3), 784–797.

Miki, Y., Swenson, J., Shattuck-Evans, D., Futreal, P. A., Harshman, K., Tavtigian, S., et al. (1994). A strong candidate for the breast and ovarian cancer susceptibility gene BRCA1. *Science, 266*, 66–71.

Schwartz, M. D., Isaacs, C., Graves, K. D., Poggi, E., Peshkin, B. N., Gell, C., et al. (2011). Long-term outcomes of BRCA1/BRCA2 testing: Risk reduction and surveillance. *Cancer*. doi:10.1002/cncr.26294.

Wooster, R., Bignell, G., Lancaster, J., Swift, S., Seal, S., Mangion, J., et al. (1995). Identification of the breast cancer susceptibility gene BRCA2. *Nature, 378*, 789–792.

Breast Cancer

Annette L. Stanton and Betina R. Yanez
Department of Psychology, University of California, Los Angeles, CA, USA

Synonyms

Breast carcinoma; Breast neoplasm

Definition

Breast cancer is a disease that involves uncontrolled division of abnormal cells in breast tissues, typically the ducts (tubes that carry milk to the nipple) and lobules (glands that make milk). Several different forms of breast cancer

exist, which have implications for distinct medical treatments. Breast cancer cells can spread through the blood and lymph systems to other parts of the body.

Description

In the United States, breast cancer is the most common form of cancer in women and their second leading cause of cancer death (after lung cancer). It is estimated that more than 230,000 women and 2,000 men will be diagnosed with breast cancer in 2011, and nearly 40,000 adults will die of the disease (Siegel, Ward, Brawley, & Jemal, 2011). Approximately one in eight women will be diagnosed with breast cancer in her lifetime. Incidence and mortality rates for breast cancer have decreased in the past decade. Health disparities exist, however; for example, African American women are less likely to be diagnosed with breast cancer than are white women, but more likely to die from the disease. In addition, women with no greater than a high school education are more likely to die from breast cancer than are more educated women. Worldwide, breast cancer is the most common cancer in women, with increasing incidence and much lower survival rates in developing and low-income countries.

Researchers and clinicians in behavioral medicine, health psychology, and related fields are contributing to understanding and improving the lives of women with breast cancer in at least four ways. First, researchers have focused on understanding and promoting early detection of breast cancer, which contributes to enhanced survival. Barriers to obtaining mammograms to detect breast cancer are well characterized, including contextual factors such as having no regular physician and no physician's recommendation for mammography, language barriers, lack of health insurance and access to screening, low education, and low social support for screening. Individual factors including lack of knowledge regarding breast cancer screening, low perceived risk of and worry about breast cancer, embarrassment about screening, and fatalistic beliefs that cancer is incurable also serve as barriers to early detection. Provision of reminders (e.g., letter, phone) for screening, video and print materials, and one-on-one education to overcome barriers are effective methods for increasing mammogram use (Baron et al., 2008). Although mammography rates have risen dramatically in the past three decades, a minority of eligible women have never had a mammogram, and many more do not receive mammograms on the recommended schedule. Effective interventions targeted to these groups are needed.

Researchers in behavioral medicine and health psychology also are investigating biobehavioral factors that might contribute to breast cancer initiation and progression. Behavioral factors that play a role in poorer breast cancer prognosis include lack of physical activity, weight gain/obesity, alcohol use, and nonadherence to breast cancer treatments (e.g., McTiernan, Irwin, & Vongruenigen, 2010). It is crucial to note that no psychological factor has been demonstrated to promote the initiation of breast cancer. Plausible models through which psychosocial and environmental factors might promote disease progression exist, however, and experimental research with nonhuman animals suggests that chronically stressful environments might contribute to the spread of breast cancer once it has developed (Antoni et al., 2006). Application of these findings to humans must proceed only through very carefully conducted research.

Characterizing processes of psychological and physical adjustment to breast cancer and its treatment and delineating factors that help and hinder women as they confront the disease represent another important area of research in behavioral medicine and health psychology. Clearly, diagnosis of and treatments for breast cancer often constitute profound stressors for women (e.g., Stanton, 2006). Best demonstrated in prospective research (i.e., studies in which psychological and physical health indicators are assessed prior to and after breast cancer diagnosis), women who are diagnosed with breast cancer evidence significant declines in physical, emotional, and social functioning/roles relative to women who are not diagnosed with breast cancer. Women are most

prone to distress and life disruption during particular phases of the cancer trajectory (e.g., diagnosis and treatment, medical treatment completion, cancer recurrence). For most women, psychological and physical adjustment improves such that overall quality of life is positive and indistinguishable from that of the general population by approximately 2 years after diagnosis. Specific problems can persist for some women, however, such as fatigue/sleep problems, cognitive disruption during and after chemotherapy (e.g., memory problems), depression, fear of cancer recurrence, and sexual problems (e.g., Bower, 2008). Intimate partners and other loved ones also face challenges as they go through the breast cancer experience (Andersen, 2009; Manne & Badr, 2008).

Longitudinal studies reveal contextual and individual factors that predict psychological adjustment in women who confront breast cancer diagnosis and treatment. For example, low education, social isolation or lack of interpersonal support, lack of satisfaction with the medical team, and holding negative expectancies about general and cancer-specific outcomes can contribute to an increase in distress in women with breast cancer. Coping through attempting to avoid thoughts and feelings related to breast cancer also predicts decrements in adjustment, whereas engagement in approach-oriented coping strategies (e.g., problem-focused coping, seeking social support, emotional expression, positive reappraisal) often is associated with more favorable psychological status across time. Treatment-related alterations in biological systems also may contribute to negative side effects of breast cancer, such as cancer-related fatigue. Theories of adjustment and associated research demonstrate that multiple aspects of the environment and the individual influence women's psychological and physical health during and after the breast cancer experience.

Finally, researchers and clinicians have developed and tested the efficacy of psychosocial and behavioral interventions to promote well-being and health in women with breast cancer. Although findings are not completely consistent, reviews of this literature suggest that such approaches as cognitive-behavioral interventions, relaxation techniques, and psychoeducational strategies are effective in improving psychological status (e.g., distress, depressive symptoms, anxiety), fatigue, and pain (e.g., Duijts, Faber, Oldenburg, van Beurden, & Aaronson, 2011; Tatrow & Montgomery, 2006; Zimmerman, Heinrich, & Baucom, 2007). Interventions to promote physical activity also appear effective in improving fatigue, body image, depressive symptoms, quality of life, and physical functioning in women with breast cancer (e.g., Duijts et al., 2011; McNeely et al., 2006). Psychosocial interventions also can affect physiological parameters, although the question of whether they can affect important health outcomes in women with breast cancer is far more controversial (McGregor & Antoni, 2009).

In sum, theory and research in behavioral medicine, health psychology, and associated fields have promoted early detection of breast cancer, contributed to delineation of biobehavioral factors relevant to disease progression, advanced the understanding of women's experience of breast cancer, and offered effective interventions to enhance quality of life and health in breast cancer survivors. Ongoing and future research promises to extend these findings to underserved groups (e.g., women with advanced breast cancer, low educational resources, diverse ethnicities) and to create maximally effective and efficient interventions for women and loved ones who confront the disease.

Cross-References

- ▶ American Cancer Society
- ▶ Cancer Risk Perceptions
- ▶ Cancer Screening/Detection/Surveillance
- ▶ Cancer Survivorship
- ▶ Cancer Treatment and Management
- ▶ Coping
- ▶ Coping Strategies
- ▶ Fatigue
- ▶ National Cancer Institute
- ▶ Psychosocial Impact

References and Readings

Andersen, B. L. (2009). In sickness and in health: Maintaining intimacy after breast cancer recurrence. *Cancer Journal, 15*, 70–73.

Antoni, M. H., Lutgendorf, S. K., Cole, S. W., Dhanhar, F. S., Sephton, S. E., McDonald, P. G., et al. (2006). The influence of bio-behavioural factors on tumour biology: Pathways and mechanisms. *Nature Reviews Cancer, 6*, 240–248.

Baron, R. C., Rimer, B. K., Breslow, R. A., Coates, R. J., Kerner, J., Melillo, S., et al. (2008). Client-directed interventions to increase community demand for breast, cervical, and colorectal cancer screening: A systematic review. *American Journal of Preventive Medicine, 35*(Suppl. 1), S34–S55.

Bower, J. E. (2008). Behavioral symptoms in patients with breast cancer and survivors. *Journal of Clinical Oncology, 26*, 768–777.

Duijts, S. F. A., Faber, M. M., Oldenburg, H. S. A., van Beurden, M., & Aaronson, N. K. (2011). Effectiveness of behavioral techniques and physical exercise on psychosocial functioning and health-related quality of life in breast cancer patients and survivors-a meta-analysis. *Psycho-Oncology, 20*, 115–126.

Manne, S., & Badr, H. (2008). Intimacy and relationship processes in couples' psychosocial adaptation to cancer. *Cancer, 112*(Suppl. 11), 2541–2555.

McGregor, B. A., & Antoni, M. H. (2009). Psychological intervention and health outcomes among women treated for breast cancer: A review of stress pathways and biological mediators. *Brain, Behavior, and Immunity, 23*, 159–166.

McNeely, M. L., Campbell, K. L., Rowe, B. H., Klassen, T. P., Mackey, J. R., & Courneya, K. S. (2006). Effects of exercise on breast cancer patients and survivors: A systematic review and meta-analysis. *Canadian Medical Association Journal, 175*, 34–41.

McTiernan, A., Irwin, M., & Vongruenigen, V. (2010). Weight, physical activity, diet, and prognosis in breast and gynecologic cancers. *Journal of Clinical Oncology, 28*, 4074–4080.

Siegel, R., Ward, E., Brawley, O., & Jemal, A. (2011). Cancer statistics, 2011: The impact of eliminating socioeconomic and racial disparities on premature cancer deaths. *CA: Cancer Journal for Clinicians, 61*(4), 212–236.

Stanton, A. L. (2006). Psychosocial concerns and interventions for cancer survivors. *Journal of Clinical Oncology, 24*, 5132–5137.

Tatrow, K., & Montgomery, G. H. (2006). Cognitive behavioral therapy techniques for distress and pain in breast cancer patients: A meta-analysis. *Journal of Behavioral Medicine, 29*, 17–27.

Zimmerman, T., Heinrich, N., & Baucom, D. H. (2007). "Does one size fit all?" Moderators in psychosocial interventions for breast cancer patients: A meta-analysis. *Annals of Behavioral Medicine, 34*, 225–239.

Breast Cancer Genes

▶ BRCA1 and BRCA2

Breast Carcinoma

▶ Breast Cancer

Breast Neoplasm

▶ Breast Cancer

Breast/Ovarian Genetic Testing

▶ BRCA1 and BRCA2

Brief Multidimensional Measure of Religiousness/Spirituality (BMMRS)

Kevin S. Masters
Department of Psychology, University of Colorado, Denver, CO, USA

Synonyms

Multidimensional measure of religiousness/spirituality

Definition

The Brief Multidimensional Measure of Religiousness/Spirituality (BMMRS) is a paper-pencil, self-report measure of different dimensions or facets of religiousness and spirituality (R/S) that was developed in 1999 a US national working group of experts supported by the Fetzer Institute in collaboration with the US

National Institute on Aging (NIA), part of the National Institutes of Health. The group's primary mission was to develop items for assessing health-relevant domains of religiousness and spirituality. Thus, the measure was specifically designed for use in health outcomes and other health research. It contains 38 items across 11 dimensions with two additional meaning dimension items in an appendix.

Description

Research into the relations between religiousness and spirituality (R/S) and health outcomes has increased in recent years. Scholars from many different disciplines including psychology, medicine, epidemiology, public health, sociology, nursing, anthropology, and other fields have launched empirical investigations not only to determine if there is a relation between R/S and health but also to ascertain the direction and strength of that relation and, more importantly for present purposes, its precise nature. That is, scholars recognize that both R/S and health are complex multidimensional constructs. Thus, questions at the global level (e.g., "Does R/S predict health?") are necessarily limited. More precise questions of the type "what aspects of R/S predict what dimensions or measures of health?" are likely to produce stronger and more consistent empirical findings.

It is in this context that the BMMRS was conceptualized and developed. Idler and colleagues (2003) describe specific procedures regarding delineation of domains as well as development and selection of items. An excellent summary may be found in Piedmont, Mapa, and Williams (2007). Briefly, the BMMRS was designed to provide a single-source measure of what are considered important domains of R/S that are likely to be significant for health outcomes research. Further, the instrument was to include both religiousness and spirituality. It was understood that though these terms are related, many investigators view them as distinguishable from one another. Thus, the authors of the BMMRS concluded that spirituality is generally concerned with the transcendent, addressing ultimate questions about life's meaning with the guiding belief that there is more to life than only what is seen or understood. Religiousness, on the other hand, is often the path to development of spirituality, but it also includes specific behavioral, social, doctrinal, and denominational characteristics that may or may not promote spiritual development. According to this formulation, it is possible to be religious and not spiritual, spiritual and not religious, or both spiritual and religious. Measures of both spirituality and religiousness were included in the BMMRS.

The specific dimensions assessed by the BMMRS include daily spiritual experiences (six items), values/beliefs (two items), forgiveness (three items), private religious practices (five items), religious and spiritual coping (seven items), religious support (four items), religious/spiritual history (three items), commitment (three items), organizational religiousness (two items), religious preference (one item), and overall self-ranking of religiousness and spirituality (two items). An appendix consists of two items assessing meaning. As intended, the BMMRS (or subscales) has begun to receive attention and use by investigators analyzing R/S and health outcomes. Recently, four studies (Masters et al., 2009; Neff, 2006; Piedmont et al., 2007; Stewart & Koeske, 2006) investigated the psychometric properties of versions of the scale (including both the longer scale and the BMMRS). Generally, these studies provide empirical support for the multidimensional construction of the scale, though it is also clear that these dimensions are considerably correlated with one another. This led Piedmont et al. (2007) to suggest that perhaps the scale should be considered multifaceted rather than multidimensional, a characterization that Masters and colleagues (2009) supported.

Cross-References

- ▶ Religion/Spirituality
- ▶ Religiousness/Religiosity
- ▶ Spirituality
- ▶ Spirituality and Health
- ▶ Spirituality, Measurement of

References and Readings

Fetzer Institute. (1999). *Multidimensional measurement of religiousness/spirituality for use in health research: A report of the Fetzer Institute/National Institute of Aging Working Group*. Kalamazoo, MI: Author.

Idler, E. L., Musick, M. A., Ellison, C. G., George, L. K., Krause, N., & Williams, D. R. (2003). Measuring multiple dimensions of religion and spirituality for health research. *Research on Aging, 25*, 327–365. doi:10.1177/0164027503252749.

Masters, K. S., Carey, K. B., Maisto, S. A., Caldwell, P. E., Wolfe, T. V., Hackney, H. L., et al. (2009). Psychometric examination of the brief multidimensional measure of religiousness/spirituality among college students. *The International Journal for the Psychology of Religion, 19*, 106–120. doi:10.1080/10508610802711194.

Neff, J. A. (2006). Exploring the dimensionality of "religiosity" and "spirituality" in the Fetzer multidimensional measure. *Journal for the Scientific Study of Religion, 45*, 449–459.

Piedmont, R. L., Mapa, A. T., & Williams, J. E. G. (2007). A factor analysis of the Fetzer/NIA Brief Multidimensional Measure of Religiousness/Spirituality (MMRS). *Research in the Social Scientific Study of Religion, 17*, 177–196.

Stewart, C., & Koeske, G. F. (2006). A preliminary construct validation of the multidimensional measurement of religiousness/spirituality instrument: A study of Southern USA samples. *The International Journal for the Psychology of Religion, 16*, 181–196.

Brief Symptom Inventory

Suzana Drobnjak
Department of Psychology, University of Zurich, Binzmuehlestrasse, Switzerland

Definition

The Brief Symptom Inventory (BSI) is a 53-item-self-report instrument. It is a short alternative to the complete Symptom Checklist-90-Revised (SCL-90-R). The BSI was designed to assess psychological symptoms during the last 7 days in medical patients, non-patients, and subjects for experimental studies. It can be used in both cross-sectional and longitudinal studies, and it can measure chronological sequences as well as pre- and post-ratings.

The BSI is composed of nine primary symptom dimensions (somatization, obsessive-compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, and psychoticism). It includes three global indices of distress (Global Severity Index, Positive Symptom Distress Index, and Positive Symptom Total), which measure the overall psychological distress level, the intensity of symptoms, and the number of self-reported symptoms. Each item of individual psychological stress can be answered on a 5-point scale, ranging from 0 = "not at all" to 4 = "extremely." It takes 8–10 min to answer all items.

The interpretation of the BSI is carried out in three steps. First, the three global indices of overall distress are used. Second, each primary symptom dimension is considered in order to determine the specific areas of psychopathology. Third, individual items are focused on discrete symptoms. The optimal interpretation is dependent on the integration of information from all three source levels. The test scores are reported in terms of standardized area T-scores.

For the evaluation of the BSI, different normative data for different samples were consulted. Among these were adult non-patients, adult psychiatric outpatients, adult psychiatric inpatients, and adolescent non-patients.

Based on a sample of 719 psychiatric outpatients, reliability coefficients were established. Internal consistency (Cronbach's alpha: 0.71 for psychoticism to 0.85 for depression) and test-retest reliabilities (Cronbach's alpha: 0.68 for somatization to 0.91 for phobic anxiety) show a high reliability. The Global Severity Index also revealed an excellent stability coefficient of 0.90. All primary symptom dimensions of the BSI also correlate highly with the comparable dimensions of the SCL-90-R, which makes the BSI a particularly apt shorter inventory. Moreover, the BSI has a high convergent, discriminant, and construct validity.

The BSI is used by professionals like psychologists, psychiatrists, physicians, nurses, and other healthcare professionals. It helps them to assess the psychological problems of their patients, to find an adequate support for care management decisions, and to measure and monitor patient

progress during and after treatment. Furthermore, by providing aggregated patient information, the BSI is an effective tool to measure the outcome of treatment programs. The BSI is used worldwide and therefore has been translated into two dozen languages, such as Spanish, French, and Italian.

More recently, the BSI-18 has been developed. The BSI-18 is a brief 18-item-srcreening inventory designed to screen for psychiatric disorder in medical and community populations. The items are selected from the anxiety, depression, and somatization dimensions of the SCL-90-R and BSI.

References and Readings

Derogatis, L. R. (1993). *BSI brief symptom inventory: Administration, scoring, and procedures manual* (4th ed.). Minneapolis, MN: National Computer Systems.

Derogatis, L. R., & Melisaratos, N. (1983). The brief symptom inventory: An introductory report. *Psychological Medicine, 13*(3), 595–605.

Zabora, J., Brintzenhofeszoc, K., Jacobsen, P., Curbow, B., Piantodosi, S., Hooker, C., et al. (2001). A new psychosocial screening instrument for use with cancer patients. *Psychosomatics, 42*, 241–246.

Bronchial Asthma

▶ Asthma
▶ Asthma and Stress
▶ Asthma: Behavioral Treatment

Bronchitis

Linda C. Baumann and Alyssa Karel
School of Nursing, University of Wisconsin-Madison, Madison, WI, USA

Definition

Bronchitis is a respiratory condition in which there is inflammation of the bronchi and excess mucus secretion in the airway. This excess secretion of mucus often causes a partial obstruction of the bronchi. Bronchitis can either be short lived and self-limiting (acute) or present with a recurrent productive cough (chronic).

Description

Most cases of acute bronchitis are preceded by an acute viral infection of the upper respiratory tract, but bacterial causes are also not uncommon. The most common symptom is a persistent cough accompanied by excessive production of mucus. Other symptoms include headache, chest discomfort, and shortness of breath, which is often made worse with exertion. Because acute bronchitis is generally self-limiting and will subside within 7–10 days, treatment is often supportive and antibiotic treatment is not recommended. Supportive treatment consists of fluids, rest, anti-inflammatory medications such as aspirin or acetaminophen, the use of a humidifier, and occasionally bronchodilators and cough suppressants.

Chronic bronchitis is characterized by persistent cough with excessive sputum production for at least 3 months for two consecutive years. Along with emphysema, a disease characterized by enlargement of alveoli distal to the terminal bronchiole, chronic bronchitis is one of the two major forms of chronic obstructive pulmonary disease (COPD). The conditions most often coexist and COPD is usually discussed as one disease state.

Chronic bronchitis is characterized by chronic inflammation of the airway, lung parenchyma, and vasculature. The inflamed airways result in hypertrophy of mucous glands causing excessive mucus in the airway and occlusion of small bronchi. Cycles of injury and repair of small airways cause narrowing and fibrosis. Changes in lung parenchyma results in emphysema and destruction of the lungs over time. Typically patients have problems with hypoxemia, airflow limitation, and hyperinflation of the lungs, gas exchange abnormalities, and pulmonary hypertension.

COPD is a major cause of death in the USA and worldwide. Smoking tobacco is the major behavioral risk factor for developing COPD. Although there has been a decrease in cigarette

smoking in the USA, there has been a marked increase in developing countries. Other risk factors in the development of COPD are exposure to occupational chemicals and dust, air pollution, infections and deficiency of the a_1-antitrypsin is a genetic risk factor.

COPD has both physiological and psychological consequences. Persons with COPD, when compared to the general population, experience higher rates of depression and anxiety, which can further complicate respiratory symptoms. Anxiety and depression negatively impact the quality of life of persons with COPD.

Non-pharmacological treatment modalities for COPD include cognitive behavioral therapy and pulmonary rehabilitation. Through progressive exercise, training of respiratory function, breathing exercises, education about medications, smoking cessation and physical activity, persons with COPD are often able to achieve an increased tolerance for exercise, decreased shortness of breath, and an improved quality of life.

Pharmacological interventions include tricyclic antidepressants and selective serotonin reuptake inhibitors (SSRIs). Benzodiazepines can also be used but the possibility of respiratory depression can be a serious side affect and therefore they should not be a first-line medication. Although presently no drug can reduce decline in lung function associated with COPD, bronchodilators can be used to improve ventilation. Commonly used bronchodilators include b_2-adrenergic agonists, anticholinergic agents, and methylxanthines. Oxygen therapy is also often used in treatment by raising partial pressure of O_2 in inspired air. Long-term O_2 therapy can improve survival, exercise capacity, and cognitive performance.

References and Readings

Baum, A., Newman, S., Weinman, J., West, R., & McManus, C. (1997). *Cambridge handbook of psychology, health and medicine*. Cambridge, UK: Cambridge University Press.

Brenes, G. A. (2003). Anxiety and chronic obstructive pulmonary disease: Prevalence, impact, and treatment. *Psychosomatic Medicine, 65*, 963–970.

Dains, J. E., Baumann, L. C., & Scheibel, P. (2007). *Advanced health assessment and clinical diagnosis in primary care* (3rd ed.). St. Louis, MO: Mosby.

Jarvis, C. (2008). *Physical examination and health assessment* (5th ed.). St. Louis, MO: Mosby Elsevier.

Lewis, S. L., Heitkemper, M. M., Dirksen, S. R., O'Brien, P. G., & Bucher, L. (2007). *Medical surgical nursing: Assessment and management of clinical problems* (7th ed.). St. Louis, MO: Mosby Elsevier.

Mikkelsen, R., Middelboe, T., Pisinger, C., & Stage, K. (2004). Anxiety and depression in patients with chronic obstructive pulmonary disease (COPD). A review. *Nordic Journal of Psychiatry, 58*(1), 65–70.

West, J. B. (2008). *Pulmonary pathophysiology: The essentials* (7th ed.). Baltimore: Lippincott Williams &Wilkins.

Brownell, Kelly D. (1951–)

Lara LaCaille
Department of Psychology, University of Minnesota Duluth, Duluth, MN, USA

Biographical Information

Kelly D. Brownell was born in 1951. He completed his undergraduate education at Purdue University and earned his Ph.D. in clinical psychology at Rutgers University in 1977. He then joined the faculty in the Department of Psychiatry at the University of Pennsylvania School of Medicine where he worked for 13 years. Since 1991, Brownell has been on the faculty at Yale University where he has served in a number of leadership roles including Master of Silliman College and Chair of the Department of Psychology.

Brownell is a professor, scientist, and internationally known expert on obesity. He is the co-founder and director of the Rudd Center for Food Policy and Obesity, the director of the Yale Center for Eating and Weight Disorders, and professor of psychology, epidemiology, and public health; institute for social and policy studies; and forestry and environmental studies at Yale University.

Major Accomplishments

Brownell's early research focused predominantly on the treatment of obesity. His "Learn Program for Weight Management" (Brownell, 2004), a treatment manual for patients, was first published in 1987 and has become the gold standard for behavioral weight loss interventions. Over the past decade, Brownell's emphasis has shifted to examining the deleterious effects of the "toxic food environment," the importance of obesity prevention, and the role of public policy in fighting the obesity epidemic. In particular, he has advocated for prohibiting fast foods and soft drinks in schools, restructuring school lunch programs, regulating food advertising aimed at children, taxing foods with poor nutritional value, and subsidizing the sale of healthy foods. His leadership in these efforts has been highly regarded. Time magazine identified Brownell as one of the "World's 100 Most Influential People" and referred to him as an "Obesity Warrior" due to his extensive efforts to translate science into public policy.

Brownell has published 14 books and over 300 scientific articles and chapters. His paper on "Understanding and Preventing Relapse" (Brownell, Marlatt, Lichtenstein, & Wilson, 1986), published in the American Psychologist, was listed as one of the most frequently cited papers in psychology. He has been the recipient of numerous prestigious awards including the James McKeen Cattell Award from the New York Academy of Sciences, the award for Outstanding Contribution to Health Psychology from the American Psychological Association, the Lifetime Achievement Award from Rutgers University, and the Distinguished Alumni Award from Purdue University. In addition, Brownell has served as president of many national and international organizations, including the Society for Behavioral Medicine, the Society for Advancement of Behavior Therapy, and the Division of Health Psychology of the American Psychological Association. He was elected to membership in the Institute of Medicine in 2006.

Cross-References

▶ Obesity: Prevention and Treatment

References and Readings

Brownell, K. D. (2004). *The LEARN program for weight management*. Dallas, TX: American Health Publishing Company.

Brownell, K. D., & Frieden, T. R. (2009). Ounces of prevention: The public policy case for taxes on sugared beverages. *The New England Journal of Medicine, 360*, 1805–1808.

Brownell, K. D., & Horgen, K. B. (2004). *Food fight: The inside story of the food industry, America's obesity crisis, and what we can do about it*. New York: McGraw-Hill/Contemporary Books.

Brownell, K. D., Marlatt, G. A., Lichtenstein, E., & Wilson, G. T. (1986). Understanding and preventing relapse. *The American Psychologist, 41*, 765–782.

Brownell, K. D., & Rodin, J. (1994). The dieting maelstrom: Is it possible and advisable to lose weight? *The American Psychologist, 49*, 781–791.

Fairburn, C. G., & Brownell, K. D. (eds) (2002). *Eating disorders and obesity: A comprehensive handbook* (2nd ed.). New York: Guilford Press.

Built Environment

Ding Ding
Graduate School of Public Health/Department of Family Preventive Medicine, San Diego State University/University of California San Diego, San Diego, CA, USA

Synonyms

Physical environment

Definition

The widely accepted definition for the built environment was provided by the Transportation Research Board and Institute of Medicine (2005). Broadly defined, the built environment refers to "land use patterns, the transportation system, and design features that together provide opportunities for travel and physical activity" (Transportation Research Board and Institute of Medicine, 2005). Specifically, land use patterns refer to "spatial distribution of human activities," such as land use for residential, business, and recreational purposes. The transportation system refers to the "physical infrastructure and services that provide the spatial links or connectivity among activities." Examples of the transportation system include roads, public transit, sidewalks, and bike lanes. Design refers to the "aesthetic, physical and functional qualities of the built environment." Design features are related to both land use and the transportation system, such as greeneries, architectural design, and streetscape. The field of built environment is drawn upon interdisciplinary collaboration from urban planning, architecture, design, engineering, policy, economics, and many other areas.

Description

There has been increasing recognition in recent years about the link between the built environment and public health. Research in this area has been guided by ecological models (Berrigan & McKinnon, 2008), which emphasize multi-level environmental influences on behaviors (Hovell, Wahlgren, & Adams, 2009; Sallis, Owen, & Fisher, 2008). Built environment attributes of neighborhoods, such as land use, zoning, layout, design, recreation facilities, and transportation infrastructures, are associated with a series of health behaviors and outcomes (Renalds, Smith, & Hale, 2010). The most frequently examined health outcome is obesity, as behaviors related to both energy consumption (e.g., dietary behaviors) and energy expenditure (e.g., physical activity) are influenced by the built environment (Papas et al., 2007; Sallis & Glanz, 2009).

Dietary Behaviors

Food can be accessed from stores, markets, and restaurants. The number, type, location, and accessibility of food outlets are directly associated with individuals' food choices (Glanz, Sallis, Saelens, & Frank, 2005). Studies consistently found that individuals living in neighborhoods with better access to healthy food outlets (e.g., grocery stores) have healthier dietary behaviors and weight outcomes (Morland, Diez Roux, & Wing, 2006; Morland, Wing, & Roux, 2002). Conversely, individuals living in neighborhoods with little or no access to healthy food options (i.e., "food deserts"(Smith & Morton, 2009)) or with concentrated fast-food restaurants have poorer diets and worse weight outcomes (Li, Harmer, Cardinal, Bosworth, & Johnson-Shelton, 2009; Moore, Diez Roux, Nettleton, Jacobs, & Franco, 2009).

Physical Activity

The association between the built environment and physical activity is complex due to the multidimensionality of both built environment and physical activity (Sallis, Adams, & Ding, 2011). Built environment attributes related to physical activity are usually categorized as neighborhood design, transportation infrastructures, and recreation facilities.

Walkable neighborhoods (sometimes synonymous with "traditional neighborhoods" or "smart growth" neighborhoods), characterized by high residential density, good street connectivity, and mixed land use, support physical activity, especially active transport (e.g., walking, biking for transport purpose). These types of neighborhoods provide destinations within close proximity and direct routes for walking and biking (Owen, Humpel, Leslie, Bauman, & Sallis, 2004; Saelens & Handy, 2008; Saelens, Sallis, & Frank, 2003). Access to and quality of transportation infrastructures such as sidewalks, bike lanes, and public transit systems provide opportunities for transport physical activity (Saelens et al., 2003; Sallis et al., 2009). Recreational facilities such as public parks and neighborhood streets provide locations for leisure-time physical activity (Sallis et al., 2011). On a "micro" scale, features,

conditions, and amenities within parks and streetscapes such as aesthetics, cleanliness, and vegetation are related to individuals' physical activity in these locations (Ellaway, Macintyre, & Bonnefoy, 2005; Kaczynski, Potwarka, & Saelens, 2008).

Environmental Quality

Unsustainable planning and development bring hazards and threats to the natural environment, which, in turn, affect public health (Dearry, 2004; Frumkin, Frank, & Jackson, 2004). Current patterns of land use and development have led to environmental consequences such as natural habitat loss, fragmentation, and water shortage (Environmental Protection Agency, 2001). Urban sprawl and increasing automobile travels contribute to air and water pollutions (Dearry, 2004; Frumkin et al., 2004). Auto-dependent neighborhoods encourage driving, which accounts for a large proportion of greenhouse gasses emission, a major cause of global climate change (Intergovernmental Panel on Climate Change, 2007).

In addition to the evidence from the above areas, other studies have found associations between the built environment and other aspects of public health, such as mental health, social capital, and alcohol abuse (Renalds et al., 2010). With emerging evidence supporting the link between the built environment and public health, better policies and planning are needed to engineer health-promoting and environmentally sustainable neighborhoods.

References and Readings

Berrigan, D., & McKinnon, R. A. (2008). Built environment and health. *Preventive Medicine, 47*(3), 239–240.

Dearry, A. (2004). Impacts of our built environment on public health. *Environ Health Perspect, 112*(11), A600–601.

Ellaway, A., Macintyre, S., & Bonnefoy, X. (2005). Graffiti, greenery, and obesity in adults: Secondary analysis of European cross sectional survey. *British Medical Journal, 331*(7517), 611–612.

Environmental Protection Agency, U. S. (2001). *Our built and natural environments: A technical review of the interactions between land use, transportation, and environmental quality*. Washington, DC: U.S. Environmental Protection Agency.

Frumkin, H., Frank, L., & Jackson, R. (2004). *Urban sprawl and public health: Designing, planning, and building for healthy communities*. Washington, DC: Island Press.

Glanz, K., Sallis, J. F., Saelens, B. E., & Frank, L. D. (2005). Healthy nutrition environments: Concepts and measures. *American Journal of Health Promotion, 19*(5), 330–333.

Hovell, M. F., Wahlgren, D. R., & Adams, M. (2009). The logical and empirical basis for the behavioral ecological model. In R. J. DiClemente, R. Crosby, & M. Kegler (Eds.), *Emerging theories and models in health promotion research and practice: Strategies for enhancing public health* (2nd ed.). San Francisco, CA: Jossey-Bass.

Intergovernmental Panel on Climate Change. (2007). *Climate change 2007: Climate change impacts, adaptation and vulnerability. Summary for policymakers. Contribution of working group II to the fourth assessment report of the intergovernmental panel on climate change*. Geneva: World Meteorological Organization.

Kaczynski, A. T., Potwarka, L. R., & Saelens, B. E. (2008). Association of park size, distance, and features with physical activity in neighborhood parks. *American Journal of Public Health, 98*(8), 1451–1456.

Li, F., Harmer, P., Cardinal, B. J., Bosworth, M., & Johnson-Shelton, D. (2009). Obesity and the built environment: Does the density of neighborhood fast-food outlets matter? *American Journal of Health Promotion, 23*(3), 203–209.

Moore, L. V., Diez Roux, A. V., Nettleton, J. A., Jacobs, D. R., & Franco, M. (2009). Fast-food consumption, diet quality, and neighborhood exposure to fast food: the multi-ethnic study of atherosclerosis. *American Journal of Epidemiology, 170*(1), 29–36.

Morland, K., Diez Roux, A. V., & Wing, S. (2006). Supermarkets, other food stores, and obesity: the atherosclerosis risk in communities study. *American Journal of Preventive Medicine, 30*(4), 333–339.

Morland, K., Wing, S., & Roux, A. D. (2002). The contextual effect of the local food environment on residents' diets: The atherosclerosis risk in communities study. *American Journal of Public Health, 92*(11), 1761–1767.

Owen, N., Humpel, N., Leslie, E., Bauman, A., & Sallis, J. F. (2004). Understanding environmental influences on walking: Review and research agenda. *American Journal of Preventive Medicine, 27*, 67–76.

Papas, M. A., Alberg, A. J., Ewing, R., Helzlsouer, K. J., Gary, T. L., & Klassen, A. C. (2007). The built environment and obesity. *Epidemiologic Reviews, 29*, 129–143.

Renalds, A., Smith, T. H., & Hale, P. J. (2010). A systematic review of built environment and health. *Fam Community Health, 33*(1), 68–78.

Saelens, B. E., & Handy, S. L. (2008). Built environment correlates of walking: A review. *Medicine and Science in Sports and Exercise, 40*(7 Suppl), S550–566.

Saelens, B. E., Sallis, J. F., & Frank, L. D. (2003). Environmental correlates of walking and cycling: Findings from

the transportation, urban design, and planning literatures. *Annuals of Behavioural Medicine, 25*(2), 80–91.
Sallis, J. F., Adams, M. A., & Ding, D. (2011). Physical activity and the built environment. In J. Cawley (Ed.), *The Oxford handbook of the social science of obesity*. New York: Oxford University Press.
Sallis, J. F., Bowles, H. R., Bauman, A., et al. (2009). Neighborhood environments and physical activity among adults in 11 countries. *American Journal of Preventive Medicine, 36*(6), 484–490.
Sallis, J. F., & Glanz, K. (2009). Physical activity and food environments: Solutions to the obesity epidemic. *The Milbank Quarterly, 87*(1), 123–154.
Sallis, J. F., Owen, N., & Fisher, E. B. (2008). Ecological models of health behavior. In K. Glanz, B. K. Rimer, & K. Viswanath (Eds.), *Health behavior and health education: Theory, research, and practice* (4th ed.). San Francisco: Jossey-Bass.
Smith, C., & Morton, L. W. (2009). Rural food deserts: Low/income perspectives on food access in Minnesota and Iowa. *Journal of Nutrition Education and Behavior, 41*(3), 176–187.
Transportation Research Board and Institute of Medicine. (2005). *Does the built environment influence physical activity? Examine the evidence 2005*. Washington, DC: Transportation Research Board.

Bulimia

Anna Maria Patino-Fernandez
Department of Pediatrics, University of Miami, Miami, FL, USA

Definition

The Diagnostic and Statistical Manual of Mental Disorders, fourth edition, Text Revision (APA, 2000), includes as the essential features of bulimia nervosa: (1) recurrent episodes of binge eating, characterized by eating in a discrete period of time an amount of food that would be considered excessive and a sense of lack of control over eating during the episode; (2) at least two binge/purge cycles a week, on average, for at least 3 months; (3) recurrent compensatory behaviors in order to prevent weight gain, such as vomiting, fasting, excessive exercise, and/or misuse of laxatives, diuretics, enemas, or other medications; and (4) self-evaluation is unduly influenced by body shape and weight. There are more women than men affected by this disorder which is often accompanied by depression and substance abuse.

Warning signs for bulimia nervosa (NIMH, 2011) include the following:
(a) Preoccupation with food
(b) Binge eating, usually in secret
(c) Vomiting after bingeing
(d) Abuse of laxatives, diuretics, and diet pills
(e) Denial of hunger or drugs to induce vomiting
(f) Compulsive exercise
(g) Swollen parotid glands
(h) Broken blood vessels in the eyes

Treatment of Bulimia Nervosa

Unless malnutrition is severe, the goal of treatment is to reduce or eliminate the person's binge eating and purging behavior. Psychotherapy has proven effective in helping to prevent the eating disorder from recurring and in addressing issues that led to the disorder (le Grange, Crosby, Rathouz, & Leventhal, 2007). As with anorexia, family therapy is recommended. Cognitive behavioral therapy (CBT) has also proven effective. Research has found that family-based treatment for adolescent bulimia nervosa is more effective and shows faster treatment effects than individual-based supportive psychotherapy (le Grange et al., 2007). CBT is an effective intervention for the purging and eating behaviors and associated symptoms of depression that often accompany bulimia nervosa (Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000). CBT for this disorder includes psychoeducation, self-monitoring (e.g., keeping a food diary as a way of becoming more aware of the types of situations that trigger bingeing), eliminating rigid dieting, strategies to reduce bingeing and purging, and application of behavioral strategies to establish more regular eating habits (e.g., self-reward for three meals plus two snacks at regular times of the day). CBT may also involve addressing cognitive distortions (e.g., certain foods are good or bad) and using exposure techniques for avoided food or anxiety-evoking situations. CBT is often combined with nutritional counseling and/or antidepressant medications. Reviews of antidepressant trials in adults have found short-term improvements in bulimic

symptoms and a small improvement in depressive symptoms (Romano, Halmi, Sarkar, Koke, & Lee, 2002). No studies with antidepressants have been conducted in children with bulimia.

Cross-References

▶ Eating Disorders: Anorexia and Bulimia Nervosa

References and Readings

Agras, W. S., Walsh, B. T., Fairburn, C. G., Wilson, G. T., & Kraemer, H. C. (2000). A multicenter comparison of cognitive-behavioral therapy and interpersonal psychotherapy for bulimia nervosa. *Archives of General Psychiatry, 57*, 459–466.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author. Text Revision.

le Grange, D., Crosby, R. D., Rathouz, P. J., & Leventhal, B. L. (2007). A randomized controlled comparison of family-based treatment and supportive psychotherapy for adolescent bulimia nervosa. *Archives of General Psychiatry, 64*, 1049–1056.

National Institute of Mental Health (NIMH). (2011). *Eating disorders*. Retrieved January 12, 2011 from http://www.nimh.nih.gov/health/publications/eating-disorders/complete-index.shtml

Romano, S. J., Halmi, K. J., Sarkar, N. P., Koke, S. C., & Lee, J. S. (2002). A placebo-controlled study of fluoxetine in continued treatment of bulimia nervosa after successful acute fluoxetine treatment. *The American Journal of Psychiatry, 151*, 96–102.

Bupropion (Wellbutrin, Zyban)

Michael Kotlyar[1] and John P. Vuchetich[2]
[1]Department of Experimental and Clinical Pharmacology, College of Pharmacy, University of Minnesota, Minneapolis, MN, USA
[2]Department of Psychiatry, University of Minnesota School of Medicine, Minneapolis, MN, USA

Synonyms

Wellbutrin®; Zyban®

Definition

Bupropion is a medication that is currently approved in the United States for the treatment of major depressive disorder, seasonal affective disorder, and to assist in smoking cessation (Product Information, 2010a, 2010b). Although its mechanism of action is not clear, it (or its metabolite) appears to inhibit the reuptake of norepinephrine and dopamine with little effect on serotonin reuptake (Baldessarini, 2006; Product Information, 2010a). Bupropion is available in three formulations: immediate release (usually taken three times daily), sustained release (usually taken twice daily), and extended release (taken once daily) (Teter, Kando, Wells, & Hayes, 2008). When used for smoking cessation, it is recommended that bupropion be initiated 1 to 2 weeks prior to the day that the patient is planning to quit smoking and treatment should be continued for 7 to 12 weeks, with longer therapy considered to prevent relapse for those who successfully quit during initial therapy (Fiore et al., 2008; Product Information, 2010b). When used to treat depression, the full therapeutic effects of bupropion may not occur for up to 8 weeks after treatment initiation, although some symptoms may start to improve sooner (American Psychiatric Association, 2010; Finley, 2009). The length of treatment depends on patient characteristics such as severity of the episode of depression and number of previous episodes. Clinicians should consult the latest clinical practice guidelines for the treatment of depression for specific recommendations regarding the length of time patients should be maintained on antidepressant treatment, but depending on patient characteristics the recommendations currently range from 4 months of therapy (from the time of symptom improvement) to indefinite maintenance of therapy (American Psychiatric Association, 2010; Finley, 2009). Due to bupropion's activity as both an antidepressant and a drug that increases smoking cessation rates, this medication may be a good choice for patients being treated for depression who would also like to quit smoking (American Psychiatric Association, 2010).

Side effects commonly associated with the use of bupropion include dry mouth, insomnia, agitation, jitteriness, skin reactions, and gastrointestinal complaints (such as nausea and vomiting) (American Psychiatric Association, 2010; Teter et al., 2008). Bupropion can increase the risk of seizures and is contraindicated in patients with seizure disorders or who may be at increased seizure risk (e.g., someone with a history of eating disorder or head injury, someone who may be withdrawing from alcohol or sedative hypnotic drugs) (Product Information, 2010a). Risk of seizures when using bupropion can be minimized by avoiding high doses, avoiding rapid titration, and being sure to split the total daily dose as recommended in the product labeling (e.g., three times daily and twice daily dosing for the immediate and sustained release formulations, respectively) (American Psychiatric Association, 2010; Product Information, 2010a). As with all antidepressants, bupropion carries a warning regarding increased suicidality, particularly in children and young adults during the initial stages of therapy (Product Information, 2010a). Bupropion can also be involved in drug-drug interactions since it is a potent inhibitor of the cytochrome P450 (CYP450) 2D6 isoenzyme which is responsible for metabolizing a large number of medications (Finley, 2009). Therefore, the potential for drug interactions should be considered prior to initiating or discontinuing therapy and managed as appropriate.

Cross-References

- ▶ Cessation Intervention (Smoking or Tobacco)
- ▶ Depression: Symptoms

References and Readings

American Psychiatric Association. (2010). Practice guideline for the treatment of patients with major depressive disorder. *The American Journal of Psychiatry, 167*(10 Suppl.), 1–124. Third Edition.

Baldessarini, R. J. (2006). Drug therapy of depression and anxiety disorders. In L. S. Goodman, A. Gilman, L. L. Brunton, J. S. Lazo, & K. L. Parker (Eds.), *Goodman & Gilman's the pharmacological basis of therapeutics* (11th ed.). New York: McGraw-Hill, Medical Publishing Division.

Finley, P. R. (2009). Mood disorders: Major depressive disorders. In M. A. Koda-Kimble, L. Y. Young, B. K. Alldredge, R. L. Corelli, B. J. Gugielmo, W. A. Kradjan, & B. R. Williams (Eds.), *Applied therapeutics: The clinical use of drugs* (9th ed.). Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins.

Fiore, M. C., Jaén, C. R., Baker, T. B., Bailey, W. C., Benowitz, N. L., Curry, S. J., et al. (2008). *Treating tobacco use and dependence: 2008 update. Clinical practice guidelines*. Rockville, MD: U.S. Department of Health and Human Services. Public Health Service.

Product Information (2010a). Wellbutrin XL (bupropion hydrochloride) Extended-Release tablet *GlaxoSmithKline*. Research Triangle Park.

Product Information (2010b). Zyban (bupropion hydrochloride) Sustained-Release tablet. *GlaxoSmithKline*. Research Triangle Park.

Teter, C. J., Kando, J. C., Wells, B. G., & Hayes, P. E. (2008). Depressive disorders. In J. T. DiPiro, R. L. Talbert, G. C. Yee, G. R. Matzke, B. G. Wells, & L. M. Posey (Eds.), *Pharmacotherapy: A pathophysiologic approach* (7th ed.). New York: McGraw-Hill Medical.

Bypass Surgery

Patricia Woltz
School of Nursing, University of Maryland, Baltimore, MD, USA

Synonyms

Coronary artery bypass graft (CABG); Heart bypass surgery

Definition

Coronary artery bypass grafting (CABG) is a revascularization procedure used to treat the advanced manifestations of coronary artery disease (CAD). In this type of heart surgery, healthy arteries or veins from elsewhere in the body are grafted to coronary arteries to increase blood flow and oxygen supply to the myocardium (heart muscle). Grafts are placed to bypass

coronary arteries significantly narrowed (stenosed) by arteriosclerosis.

Description

Coronary artery bypass surgery is one of the most common operations performed in the world. The goals of CABG are to prolong survival or to relieve symptoms of myocardial ischemia (e.g., angina). Other symptoms that may be improved following CABG include dyspnea, exercise tolerance, and quality of life. First introduced in the 1950s, today's conventional CABG procedure uses cardiopulmonary bypass (a heart-lung machine) and cardioplegia (intentional paralysis of the heart). Newer alternatives in CABG include off-pump (i.e., without cardiopulmonary bypass support), beating heart, and minimally invasive endoscopic approaches. A combination of approaches may be used during a single CABG procedure depending on the assessment of patient risks and benefits and provider preference and experience. Rapid evolution of new technologies in CABG as well as in the alternative therapies available to treat CAD has resulted in conflicting evidence from disparate clinical trials and registries. Consequently, consensus is lacking about the superiority of one approach over another (Eagle et al., 2004; Morrison, 2008).

Indications

The risks of CABG should be balanced against the expected symptom relief and survival benefit. CABG does not treat CAD pathophysiology. Vessel restenosis and graft blockage is common within 10–12 years following CABG (Eagle et al., 2005). Angina relieved by CABG typically recurs within 5–10 years (Bravata et al., 2007).

Ideally, a patient under consideration for CABG has developed medically refractory myocardial ischemia in the setting of optimal drug therapy. Indications for CABG outlined by the American College of Cardiology/American Heart Association guidelines for patients with stable angina, unstable angina, or myocardial infarction (MI) are based on the extent of CAD and left ventricular function (Eagle et al., 2004). Criteria include >70% narrowing of three, and in some cases two major coronary arteries, >50% narrowing of the left main coronary artery, or left ventricular ejection fraction < 0.55. The risk factors associated with mortality following CABG are the priority of the operation (emergent, urgent, or elective), advanced age, prior CABG, female gender, left ventricular ejection fraction < 0.40, number of major coronary arteries with significant stenoses, and percent stenosis of the left main coronary artery. While CABG continues to be the standard of care for patients with left main and three-vessel disease (Lee, Hillis, & Nabel, 2009), the evidence supporting this is being challenged by newer, alternative therapies (below).

Surgery

Prior to surgery, a coronary angiogram is conducted to estimate the extent of vessel blockage. Patients may have additional functional stress testing and perfusion imaging studies to evaluate the degree of inducible ischemia and myocardial viability.

Surgery begins with graft preparation. Internal mammary artery (IMA) grafts are preferred over other grafts because they demonstrate superior long-term patency (Umakanthan, Solenkova, Leacche, Byrne, & Ahmad, 2010). When feasible to preserve its native blood flow, an IMA graft is left in situ (connected) to the subclavian artery and its peripheral end anastomosed to the coronary artery beyond the area of stenosis. Saphenous veins harvested endoscopically with small incisions at the groin and knee may be used for multiple grafts.

The number of bypasses in a single operation may range from one to six. In conventional CABG, the chest is opened via a median sternotomy. Venous blood, which must be anticoagulated and kept warm, is rerouted to a cardiopulmonary bypass machine where it is oxygenated and then redirected back to the systemic arterial circulation. To stabilize the myocardium during graft placement, the heart is temporarily stopped using a potassium solution. Risks associated with use of cardiopulmonary bypass (e.g., thrombosis, emboli, bleeding, fluid

shifts, and temporary organ dysfunction) limit the length of CABG with cardiopulmonary bypass to less than 4 h, after which time the heart is restarted with controlled electrical shocks and the sternum wired closed (Gravlee, Davis, Stammers, & Ungerleider, 2008). The usual length of hospitalization following a conventional procedure is 4–6 days. For adequate wound healing, patients must avoid activities that put strain on the sternal incision for 12 weeks. Benefits of endoscopic CABG are smaller incisions, faster wound healing, shorter recovery times, and cost savings (Bravata et al., 2007). However, use of robotic devices, beating heart grafting, and limited visualization with endoscopic CABG procedures demand a high level of surgical skill and are more likely to result in incomplete revascularization. Recommended resources describing CABG approaches are Gomez and Gibson webcast (2007), Kuss, von Salviati, and Börgermann (2010), Sellke et al. (2005), and Umakanthan et al. (2010).

Complications

People undergoing CABG are at risk for the same complications as any surgery, plus some adverse events more common with or unique to CABG. The major complications associated with CABG are related to procedural success and durability. These include death, MI, stroke, neurocognitive impairment, renal dysfunction, mediastinitis, non-closure of the sternum, and need for revascularization. Myocardial infarction and stoke can occur due to emboli. Neurological abnormalities range from deterioration of intellectual function or memory to stupor or coma and may arise from hypoxia, emboli, hemorrhage, and metabolic abnormalities experienced during CABG (Knipp et al., 2004; Pepper, 2005). Mediastinitis and non-closure of the wound may arise with use of IMA grafts, which alter blood flow in the chest. Restenosis and graft blockage requiring revascularization can occur within months to years. Adverse events related to CABG surgery may be classified as perioperative or short-term (within 30 days of surgery), early (within the first year), and late effects (after the first year).

Alternative Therapy

Alternative therapies for CAD are risk factor modification, medical management, and percutaneous coronary intervention (PCI). Behavioral risk factor modification is a powerful tool that is often underemphasized in the treatment of CAD. Smoking cessation, diet modification, exercise, weight loss, and tight glycemic control in diabetics limit the progression of arteriosclerosis and prolong survival. Medical therapy for CAD includes aspirin, lipid-lowering agents (especially statins), beta-blockers, and angiotensin-converting enzyme inhibitors and/or angiotensin receptor blockers. Each of these drugs increases survival and has relatively few adverse outcomes (Morrison, 2006). Percutaneous coronary intervention is a group of catheter-based approaches that includes balloon angioplasty, bare metal stents, and drug-eluding stents. Percutaneous methods are revascularization alternatives to CABG surgery that are less costly, less invasive, performed more rapidly, and have shorter recovery times.

Whether to manage patients medically and whether to revascularize using CABG or PCI are current controversies. Revascularization is recommended for unstable angina and evolving MI; however, it is less clear how to best manage patients with stable angina. A recent study found that medical therapy plus CABG improved long-term outcomes over medical therapy alone in the management of asymptomatic patients with severe left ventricular dysfunction (Velazquez et al., 2011). In general, the incidence of restenosis is greater in PCI compared to CABG, while CABG has greater risks of mortality, stroke, and neurological complications. Percutaneous intervention is generally indicated over CABG in the setting of an acute MI or with comorbidities other than diabetes (Morrison, 2008). The newest PCI technology is drug-eluding stents which are designed to minimize restenosis in stented vessels, a common complication of bare metal stents. Drug eluding stents challenge the superiority of CABG to PCI and comparative study is currently underway. Overall, the decision to use CABG or PCI appears to be evolving to one that focuses on the complexity of coronary anatomy, patient

preferences, and potential risk and benefits depending on patients' medical states and their comorbidities (Lee et al., 2009).

References and Readings

Bravata, D. M., Gienger, A. L., McDonald, K. M., Sundaram, V., Perez, M. V., Varghese, R., et al. (2007). Systematic review: The comparative effectiveness of percutaneous coronary interventions and coronary artery bypass graft surgery. *Annals of Internal Medicine, 147*(10), 703–716.

Eagle, K. A., Guyton, R. A., Davidoff, R., Edwards, F. H., Ewy, G. A., Gardner, T. J., et al. (2004). ACC/AHA 2004 guideline update for coronary artery bypass graft surgery: Summary article: A report of the American college of cardiology/American heart association task force on practice guidelines (committee to update the 1999 guidelines for coronary artery bypass graft surgery). *Circulation, 110*(9), 1168–1176. doi:10.1161/01.CIR.0000138790.14877.7D.

Eagle, K., Guyton, R., Davidoff, R., Edwards, F., Ewy, G., Gardner, T., et al. (2005). ACC/AHA pocket guideline. *Coronary artery bypass surgery*. Retrieved from www.americanheart.org/downloadable/heart/1112977349318CABG%202005pocket.pdf

Gomez, M., & Gibson, D. (2007). Off pump coronary artery bypass – A beating heart procedure. *Live webcast*. Houston, TX: Memorial Hermann Heart & Vascular Institute. Retrieved from http://video.google.com/videoplay?docid=9014695099760440284#

Gravlee, G., Davis, R., Stammers, A., & Ungerleider, R. (2008). *Cardiopulmonary bypass: Principals and practice* (3rd ed.). Philadelphia: Lippincott Williams & Wilkins.

Knipp, S. C., Matatko, N., Wilhelm, H., Schlamann, M., Massoudy, P., Forsting, M., et al. (2004). Evaluation of brain injury after coronary artery bypass grafting. A prospective study using neuropsychological assessment and diffusion-weighted magnetic resonance imaging. *European Journal of Cardio-Thoracic Surgery: Official Journal of the European Association for Cardio-Thoracic Surgery, 25*(5), 791–800. doi:10.1016/j.ejcts.2004.02.012.

Kuss, O., von Salviati, B., & Börgermann, J. (2010). Off-Pump versus on-pump coronary artery bypass grafting: A systematic review and meta-analysis of propensity score analyses. *The Journal of Thoracic and Cardiovascular Surgery, 140*(4), 829–835. doi:10.1016/j.jtcvs.2009.12.022. 835. e1–13.

Lee, T. H., Hillis, L. D., & Nabel, E. G. (2009). CABG vs. Stenting – Clinical implications of the SYNTAX trial. *The New England Journal of Medicine, 360*, e10.

Morrison, D. (2006). PCI versus CABG versus medical therapy in 2006. *Minerva Cardioangiologica, 54*(4), 643–672.

Morrison, D. A. (2008). Extent of atherosclerotic disease and left ventricular function. In *Textbook of interventional cardiology* (5th ed., pp. 72–84). Philadelphia: Saunders Elsevier.

Pepper, J. (2005). Controversies in off-pump coronary artery surgery. *Clinical Medicine & Research, 3*(1), 27.

Sellke, F. W., DiMaio, J. M., Caplan, L. R., Ferguson, T. B., Gardner, T. J., Hiratzka, L. F., et al. (2005). Comparing on-pump and off-pump coronary artery bypass grafting: Numerous studies but few conclusions: A scientific statement from the American heart association council on cardiovascular surgery and anesthesia in collaboration with the interdisciplinary working group on quality of care and outcomes research. *Circulation, 111*(21), 2858–2864. doi:10.1161/CIRCULATIONAHA.105.165030.

Umakanthan, R., Solenkova, N. V., Leacche, M., Byrne, J. G., & Ahmad, R. M. (2010). Coronary artery bypass surgery. In P. Toth & C. Cannon (Eds.), *Comprehensive Cardiovascular Medicine in the Primary Care Setting*, (pp. 263–279). New York: Springer, doi:10.1007/978-1-60327-963-5_13.

Velazquez, E. J., Lee, K. L., Deja, M. A., Jain, A., Jain, A., Marchenko, A., et al. (2011). Coronary-artery bypass surgery in patients with left ventricular dysfunction. *The New England Journal of Medicine, 364*(17), 1607–1616. doi:10.1056/NEJMoa1100358.

C

CABG

► Coronary Artery Bypass Graft (CABG)

Cachectin

► Tumor Necrosis Factor-Alpha (TNF-Alpha)

Cachexia (Wasting Syndrome)

Travis Lovejoy
Mental Health & Clinical Neurosciences Division, Portland Veterans Affairs Medical Center, Portland, OR, USA

Synonyms

AIDS wasting; Cancer cachexia; Cardiac cachexia; HIV wasting; Slim disease

Definition

Cachexia is a syndrome characterized by the loss of lean body tissue, often including involuntary weight loss, accompanied by increased metabolic and proinflammatory cytokine activity. It is distinct from mere weight loss due to anorexia and from sarcopenia, which is characterized by the loss of lean body tissue replaced by fat mass with little or no resulting weight loss. Cachexia occurs in patients with chronic illnesses such as cancer, HIV/AIDS, chronic kidney disease, chronic heart failure, and chronic obstructive pulmonary disease.

Description

Etiology

The etiology of cachexia is multifactorial. Increased inflammatory processes in the form of cytokine production lead to metabolic dysregulation, such as increased resting energy expenditure, and may contribute to heightened protein degradation accompanied by decreased protein synthesis. Many patients with cachexia will also experience anorexia (i.e., a loss of appetite) and decreased nutrient absorption in the gastrointestinal tract, which accounts for concomitant weight loss. However, the overall loss of lean body tissue observed in patients with cachexia occurs independent of nutrient uptake.

Diagnosis

The multifactorial etiology and absence of a consensus definition for cachexia presents challenges to diagnostic uniformity. Most current diagnostic systems for cachexia assess at least some of the following: (1) percentage of unintentional body weight lost in a specific time frame (e.g., the past 12 months); (2) proportion of lean body mass to fat mass; (3) body mass index; (4) the presence of clinical symptoms such as

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

decreased muscle strength, fatigue, and decreased appetite; and (5) abnormal biochemistry such as increased inflammatory markers.

Treatment

Treatments for cachexia aim to restore lean body mass and improve quality of life. Pharmacological treatments have focused on (1) increasing appetite and caloric intake through the use of appetite stimulants; (2) maintaining and/or restoring lean body mass with testosterone, anabolic steroids, or human growth hormone; and (3) downregulating cytokine activity through the use of systemic anti-inflammatory medications. Non-pharmacological treatments include resistance training for muscle retention, nutritional counseling and supplementation to ensure adequate macro- and micronutrient intake, and targeted amelioration of conditions that may exacerbate cachexia such as opportunistic infections in those with compromised immune systems.

Psychosocial Impact of Wasting

Although cachexia has a gradual onset, its clinical manifestation occurs somewhat rapidly and often during advanced disease stages. Considerable reductions in physical activity, coupled with decreased appetite and metabolic changes, have a significant impact on patients' quality of life. Many patients with cachexia feel shame or embarrassment about their bodily changes and distance themselves from loved ones. Decreased libido may have deleterious effects on individuals' romantic partnerships.

The Role of Behavioral Medicine

Behavioral medicine plays a key role in the treatment of patients with cachexia. Behavioral medicine professionals can provide patient education regarding cachexia treatment options, deliver interventions to improve medication adherence, and offer counseling and instruction for tailored nutrition and exercise programs. The provision of psychotherapy that addresses acute psychiatric conditions, adjustment to chronic illness, and couples issues pertaining to sexuality can help to improve overall quality of life for persons diagnosed with cachexia.

Cross-References

- ▶ Body Composition
- ▶ Cytokines
- ▶ Sarcopenia
- ▶ Tumor Necrosis Factor-Alpha (TNF-Alpha)

References and Readings

Mantovani, G., Anker, S. D., Inui, A., Morley, J. E., Fanelli, F. R., Scevola, D., et al. (2006). *Cachexia and wasting: A modern approach*. New York: Springer.

Springer, J., von Haehling, S., & Anker, S. D. (2006). The need for a standardized definition for cachexia in chronic illness. *Nature Clinical Practice Endocrinology & Metabolism, 2*, 416–417.

Wanke, C., Kohler, D., & HIV Wasting Collaborative Consensus Committee. (2004). Collaborative recommendations: The approach to diagnosis and treatment of HIV wasting. *Journal of Acquired Immune Deficiency Syndromes, 37*, S284–S288.

Caffeine

▶ Coffee Drinking, Effects of Caffeine

Caloric Intake

Megan Roehrig[1], Jennifer Duncan[2] and Alyson Sularz[1]

[1]Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

[2]Department of Preventive Medicine, Feinberg School of Medicine Northwestern University, Chicago, IL, USA

Synonyms

Energy In; Energy Intake

Definition

Caloric intake is defined as the amount of energy consumed via food and beverage. A calorie is

a unit of energy that is defined as the amount of heat energy required to raise 1 g of water by 1°C. Calories are units that measure the energy in food as well as the energy produced, stored, and utilized by living organisms.

Daily caloric intake needs are determined by a variety of factors such as age, gender, height, weight, activity level, and genetics. Three well-documented formulas are used to calculate daily caloric needs: the Harris-Benedict equation (1919), the Mifflin-St Jeor equation (1990), and the Institute of Medicine's Dietary Reference Intake equation (2002). These equations determine the resting metabolic rate (RMR), which represents the minimum energy needed to maintain vital body functions. While the terms RMR and basal metabolic rate (BMR) are often used interchangeably, the BMR requires more stringent testing conditions and factor in calories needed based on the individual's activity level. The HHS/USDA 2005 recommendations for daily caloric intake requirements for healthy weight maintenance and prevention of obesity according to age, gender, and activity level are available at http://www.nhlbi.nih.gov/health/public/heart/obesity/wecan/downloads/calreqtips.pdf.

Caloric intake can be measured using objective and subjective methods. Common objective methods are calorimetry and the doubly labeled water technique, while common subjective methods are 24-h dietary recall interviews and food diaries. Objective measurements are highly accurate but costly to implement, while subjective measurements are less expensive but subject to greater error. In fact, subjective estimates can be off by as many as 800 kcal (Beasly, Riley, & Jean-Mary, 2004).

One pound of body weight is equal to approximately 3,500 cal. When caloric intake is equal to caloric expenditure, an energy balance is achieved and body weight is maintained. Weight loss occurs when caloric expenditure is greater than caloric intake. Conversely, weight gain is the result of greater caloric intake than caloric expenditure. Caloric imbalances in either extreme have multiple health risk implications, including obesity and eating disorders and their associated medical comorbidities.

Cross-References

▶ Fat, Dietary Intake

References and Readings

Harris, J. A., & Benedict, F. G. (1919). *A biometric study of basal metabolism in man*. Washington, DC: Carnegie Institution of Washington.

Mifflin, M., St Jeor, S., Hill, L., Scott, B., & Daugherty, S. (1990). A new predictive equation for resting energy expenditure in healthy individuals. *The American Journal of Clinical Nutrition, 51*(2), 241–247.

Rolls, B., & Barnett, R. (2000). *The volumetrics weight-control plan*. New York: Harper Collins.

Trumbo, P., Schlicker, S., Yates, A. A., Poos, M., & Food and Nutrition Board of the Institute of Medicine, The National Academies. (2002). Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein and amino acids. *Journal of the American Dietetic Association, 102*(11), 1621–1630.

U.S. Department of Health and Human Services and U.S. Department of Agriculture. (2005). *Dietary guidelines for Americans*. Washington, DC: U.S. Government Printing Office.

Cancer and Cigarette Smoking

▶ Cancer and Smoking

Cancer and Diet

Akihiro Tokoro
Department of Psychosomatic Medicine, National Hospital Organization, Kinki-Chuo Chest Medical Center, Sakai Osaka, Japan

Synonyms

Diet and cancer

Definition

A field in which the relationship between cancer and diet is examined from the interdisciplinary

perspectives of basic medicine, clinical epidemiology, preventive medicine, and behavioral medicine.

Description

The relationship between diet and cancer has recently been recognized as an area of scientific interest. Dietary factors are thought to be involved in 30% of cases of cancer in developed countries and in 20% in developing countries (Marian, 2010).

In 2004, the American Society of Clinical Oncology (ASCO) announced a goal of achieving prophylactic intervention for cancer prevention, with a focus placed on reduction of tobacco use, control of obesity, cancer-causing infections, and environmental carcinogens (Lippman & Bernard, 2004).

A WHO report (http://www.who.int/gho/en/) showed that 35% of adults aged ≥20 years old worldwide were overweight (body mass index [BMI]: ≥25 kg/m^2) and 12% were obese (BMI: ≥30 kg/m^2) in 2008. The rate of obesity has more than doubled since 1980.

Previous studies have suggested that unhealthy eating and lack of physical activity can affect the development and prognosis of some cancers, including breast cancer, colon cancer, and prostate cancer.

Research into the details of the association of diet with development of cancer is limited. However, a report by the International Agency for Research on Cancer (IARC) in 2002 showed that being overweight or obese is associated with an increased risk of cancer in both men and women (International Agency for Research on Cancer (IARC), 2002).

Based on these data, the American Cancer Society (ACS) guidelines (American Cancer Society guidelines on nutrition and physical activity for cancer prevention http://caonline.amcancersoc.org/content/vol56/issue5/) recommend:

1. Maintenance of a healthy weight throughout life
2. Adoption of a physically active lifestyle
3. Consumption of a healthy diet, with an emphasis on plant sources
4. Limited consumption of alcoholic beverages

Further research is required to examine the relationship between single dietary factors and development or progression of cancer and between health behaviors, including dietary lifestyle, and cancer.

Cross-References

▶ Cancer Prevention

References and Readings

American Cancer Society guidelines on nutrition and physical activity for cancer prevention. http://caonline.amcancersoc.org/content/vol56/issue5/

http://www.who.int/gho/en/

International Agency for Research on Cancer (IARC). (2002). Weight control and physical activity. In H. Vanio & F. Biaciani (Eds.), *IARC handbooks of cancer preventive effects*. Lyons: IARC Press.

Lippman, S. M., & Bernard, L. (2004). Cancer prevention and the American Society of Clinical Oncology. *Journal of Clinical Oncology, 22*(19), 3848–3851.

Marian, L. (2010). Diet and cancer. In *Psycho-Oncology* (2nd ed., pp. 22–27). New York: Oxford University Press.

Cancer and Physical Activity

Akihiro Tokoro
Department of Psychosomatic Medicine, National Hospital Organization, Kinki-Chuo Chest Medical Center, Sakai Osaka, Japan

Synonyms

Exercise and cancer; Physical activity and cancer

Definition

A field in which the relationship between cancer and physical activity is examined from the

interdisciplinary perspectives of basic medicine, clinical epidemiology, preventive medicine, rehabilitation, and behavioral medicine.

Description

The relationship between physical activity and cancer has recently been recognized as an area of scientific interest. The role of physical activity in preventing cancer has been examined in several epidemiological studies and several reviews of publications. An appropriate physical activity may reduce cancer risk and improve the quality of life of cancer patients (Marian, 2010).

Epidemiological evidence suggests that physical activity is associated with a reduced risk of colon and breast cancers. Some studies have also reported the link between physical activity and a reduced risk of endometrial (uterus), lung, and prostate cancers. More good news – physically active lifestyle helps you reduce your risk of heart disease, diabetes, and osteoporosis (American Cancer Society guidelines on nutrition and physical activity for cancer prevention http://www.cancer.org/acs/groups/cid/documents/webcontent/002577).

Based on several publications such as the American Cancer Society (ACS) guidelines (American Cancer Society guidelines on nutrition and physical activity for cancer prevention http://www.cancer.org/acs/groups/cid/documents/webcontent/002577), the Centers for Disease Control and Prevention (CDC) (State indicator report on physical activity, 2010) and the American Institute for Cancer Research (AICR) (http://www.aicr.org/reduce-your-cancer-risk/recommendations-for-cancer-prevention/recommendations_02_activity.html) recommend at least 30 min of moderate to vigorous physical activity, above usual activities, 5 or more days a week, and they say 45–60 min of intentional physical activity is more beneficial.

Further research is required to examine the role of physical activity in cancer survivorship and its correlation with quality of life and reduced cancer risk. The National Cancer Institute (NCI)-funded studies are exploring the ways in which physical activity may improve the prognosis and quality of life of cancer patients and survivors (National Cancer Institute fact, sheet, physical activity and cancer).

Cross-References

- Cancer and Diet
- Cancer Prevention
- Exercise
- Physical Fitness

References and Readings

American Cancer Society guidelines on nutrition and physical activity for cancer prevention. http://www.cancer.org/acs/groups/cid/documents/webcontent/002577

http://www.aicr.org/reduce-your-cancer-risk/recommendations-for-cancer-prevention/recommendations_02_activity.html

Marian, L. (2010). Exercise and cancer. In *Psycho-oncology* (2nd ed., pp. 28–32). New York: Oxford University Press.

National Cancer Institute fact, sheet, physical activity and cancer. http://www.cancer.gov/cancertopics/factsheet/prevention/physicalactivity

State indicator report on physical activity. (2010). http://www.cdc.gov/physicalactivity/downloads/PA_State_Indicator_Report_2010

Cancer and Smoking

Monica Webb Hooper
Department of Psychology, University of Miami, Coral Gables, FL, USA

Synonyms

Cancer and cigarette smoking; Cancer and tobacco smoking; Lung cancer and smoking

Definition

A cancer diagnosis represents a heterogeneous class of diseases characterized by uncontrolled

growth of malignant cells in the body. These cells form a tumor that starts in the epithelium, invades organs of the body and nearby tissue, has the capacity to metastasize to other sites through the bloodstream or lymph nodes, and may recur after surgical removal. The development of cancer may be influenced by hereditary and/or environmental factors.

Tobacco smoking is defined as the practice of burning and inhaling tobacco. The combustion from the burning allows the nicotine, tar, and other chemicals and toxins to be absorbed through the lungs. Cigarette smoking is the most prevalent form of consuming tobacco. Most national surveys define a current smoker as having smoked at least 100 (five packs) cigarettes in their lifetime and currently smokes on at least some days.

Description

Over 46 years of scientific research, including 29 reports from the US Surgeon General, has led to the unequivocal conclusion that cigarette smoking causes cancer. But, Dr. John Hill, first deduced that snuff (smokeless tobacco) might be cancerous in "Cautions Against the Immoderate Use of Snuff," written in 1761 (U.S. Department of Health and Human Services [USDHHS], 1982). The earliest scientific investigations on the positive association between smoking and cancer were published in the 1920s and 1930s (USDHHS, 1982). In 1950, four retrospective studies examining the smoking histories of lung cancer patients compared to controls were published, all indicating a positive link between smoking and cancer. The first Surgeon General's report with sufficient evidence to declare that smoking causes lung cancer was published in 1964 (U.S. Department of Health, Education, and Welfare [USDEW], 1964). At that time, smoking was causally linked to lung cancer among men, but there was insufficient evidence among women. Early on, the most prevalent lung cancers, squamous cell and epidermoid, were specifically associated with smoking. It was also found that the frequency of oat-cell and adenocarcinoma was greater among smokers compared to nonsmokers. In 1968, the Surgeon General's report concluded that smoking also caused lung cancer in women (USDHEW, 1968). Lung cancer remains the most common form of cancer among men and women.

Cigarette smoking is responsible for the majority of deaths due to cancer. Between 1995 and 1999, over 70% of cancer deaths among US males were attributable to smoking (USDHHS, 2004). During the same years, over 50% of cancer deaths among women were due to smoking. This corresponds to almost 1.5 million years of potential life lost among men, and almost 1 million years among women (USDHHS).

Some have questioned how a causal relationship could be determined between cigarette smoking and cancer. This is largely because random assignment and a control group are necessary preconditions to conclude that a cause-and-effect connection exists. However, the accumulation of robust associations over a long period of time can also be used to establish causality. The criteria used by the Surgeon General's report included the following: (1) the consistency of association; (2) the robustness of association; (3) the specificity of association; (4) the temporal nature of association; (5) the rationality of association; and (6) experimental and clinical autopsy-based evidence (USDHEW, 1967). Using these criteria, there is no doubt that cancer is caused by smoking.

Since the finding that smoking definitively causes cancer, the prevalence of cigarette smoking has declined. In 1965, the overall smoking prevalence was 42%, which decreased to 33% by 1971 (USDHEW, 1971). The rates of smoking sharply declined in the USA, although there was no change in the absolute number of smokers (53 million) over the 20-year period between 1951 and 1971. Since 2004, smoking rates have leveled off at about 20%. In 2010, 19.3% of adults (45 million) were current smokers (Centers for Disease Control and Prevention, 2011). The past decade witnessed an overall decline in the prevalence of cancer in the USA, which is directly related to declines in smoking.

With each Surgeon General's report, the evidence explicating the types of cancers caused by

Cancer and Smoking, Table 1 List of cancers caused by smoking

Lung cancer	Acute myeloid leukemia
Esophageal	Larynx
Stomach	Oral cavity
Pancreatic	Pharynx
Bronchial	Trachea
Kidney	Renal pelvic
Uterine cervical	Nasal cavity
Urinary bladder	

Cancer and Smoking, Table 2 Examples of known carcinogens in cigarette smoke (humans)

Category	Name
Aldehydes	Formaldehyde
Aromatic amines	2-naphthylamine
	4-aminobiphenyl
Metals and inorganic compounds	Arsenic
	Beryllium
	Nickel
	Chromium (hexavalent)
	Cadmium
Organic compounds	Vinyl chloride
Volatile hydrocarbons	Benzene

National Toxicology Program (2011)

smoking have increased. It is now well established that smoking damages almost every organ in the human body and causes at least 15 types of cancer (Table 1). There is a dose–response relationship between cancer mortality and the number of cigarettes smoked per day (USDHHS, 1982). Smoking a greater number of daily cigarettes leads to increased exposure to the 7,000 chemicals and toxins contained in each cigarette (USDHHS, 2010). Although addictive, the nicotine in cigarettes is not the source of cancer development. Rather, it likely results from the effects of the 69 carcinogens contained in cigarettes (USDHHS, 2010). There are several key chemicals in cigarettes that are known to be cancer causing in humans (Table 2). Among these dangerous chemicals are formaldehyde and arsenic.

The mechanisms that explain the causal relationship between smoking and cancer are complex. Genetic predisposition and polymorphisms are related to cancer risk among smokers and nonsmokers (USDHHS, 2010). Inhalation of the chemicals and toxins in cigarette smoke initiates genetic and cellular processes that lead to malignant tumor development. To date, the unique contribution of the carcinogens found in cigarettes to cancer is not fully known. But the evidence suggests that cigarette smoking leads to DNA damage. Repeated exposure to cancer-causing agents alters major cellular pathways through genetic mutation and the growth of DNA adducts. DNA adducts (i.e., DNA pieces that are chemically bonded to a carcinogen) are formed by cytochrome P-450 enzymes, which metabolize the carcinogens in cigarette smoke. Smokers with polymorphisms in the GSTM1 and CYP1A1 genes appear to have greater frequencies of DNA adducts compared to those without these polymorphisms. These processes facilitate unconstrained cell increases and inhibit the immune system's ability to reduce their progression and range.

Cigarette smoking is the single most important avoidable cancer risk behavior. Smoking cessation is the only method for stopping the pathogenic processes that ultimately lead to cancer. Thus, quitting smoking reduces the likelihood of a cancer diagnosis. A former smoker's chance of developing cancer declines gradually over time and depends on the extent of exposure to cigarette smoke. With the increasing duration of cessation, the overall rate of cancer mortality approaches that of nonsmokers (USDHHS, 1982). Ex-smokers of 15 years or more have lung cancer rates only two times greater than never-smokers.

The prevalence of smoking among people diagnosed with cancer approximates the national average. Many people erroneously believe that once a person has been diagnosed with cancer, the damage is already done; thus, there is no benefit of smoking cessation (USDHHS, 1990). However, the evidence indicates that continued smoking among cancer patients negatively affects their prognosis. Specifically, smoking is associated with increased risks of recurrence,

a second cancer, and decreased efficacy of cancer treatment. Thus, smoking cessation is also important for cancer patients and survivors.

In summary, cigarette smoking causes cancer. Indeed, smoking is the leading preventable cause of multiple cancers, including lung cancer. There is a dose–response relationship between daily smoking intensity and cancer mortality, but there is no safe level of smoking. The prevalence of smoking has declined since the first Surgeon General's report directly linking smoking to cancer, but about 20% of the US population continues to smoke. All of the biological mechanisms by which smoking leads to cancer are not yet elucidated; but it is known that smoking leads to DNA damage and reduces the immune system's ability to rid the body of cell overgrowth. Smoking cessation is the best way to reduce the risk of cancer and is beneficial even after a cancer diagnosis.

Cross-References

► Smoking Cessation

References and Readings

Centers for Disease Control and Prevention. (2011). Vital signs: Current cigarette smoking among adults aged ≥18 years – United States, 2005–2010. *Morbidity and Mortality Weekly Report, 60*, 1207–1212.

National Toxicology Program. (2011). *Report on carcinogens* (12th ed., 499 pp.). Research Triangle Park, NC: U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program.

U.S. Department of Health and Human Services. (1982). *The health consequences of smoking: Cancer. A report of the surgeon general*. Rockville, MD: U.S. Department of Health and Human Services, Public Health Service, Office on Smoking and Health, DHHS Publication No. (PHS) 82-50179.

U.S. Department of Health and Human Services. (1990). *The health benefits of smoking cessation. A Report of the Surgeon General*. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. DHHS Publication No. (CDC) 90-8416.

U.S. Department of Health and Human Services. (2004). *The health consequences of smoking: A report of the surgeon general*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health.

U.S. Department of Health and Human Services. (2010). *How tobacco smoke causes disease: The biology and behavioral basis for smoking-attributable disease: A report of the surgeon general*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health.

U.S. Department of Health, Education, and Welfare. (1964). *Smoking and health: Report of the advisory committee to the surgeon general of the public health service*. Washington, DC: U.S. Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control. PHS Publication No. 1103.

U.S. Department of Health, Education, and Welfare. (1967). *The health consequences of smoking. A public health service review: 1967*. Washington, DC: U.S. Department of Health, Education, and Welfare, Public Health Service, Health Services and Mental Health Administration. PHS Publication No. 1696.

U.S. Department of Health, Education, and Welfare. (1968). *The health consequences of smoking. 1968 supplement to the 1967 public health service review*. Washington, DC: U.S. Department of Health, Education, and Welfare, Public Health Service, 1. DHEW Publication No. 1696 (Suppl.).

U.S. Department of Health, Education, and Welfare. (1971). *The health consequences of smoking. A report of the surgeon general: 1971*. Washington, DC: U.S. Department of Health, Education, and Welfare, Public Health Service, Health Services and Mental Health Administration. DHEW Publication No. (HSM) 71-7513.

Cancer and Tobacco Smoking

► Cancer and Smoking

Cancer Cachexia

► Cachexia (Wasting Syndrome)

Cancer of the Uterine Cervix

► Cancer, Cervical

Cancer Prevention

Toru Okuyama
Division of Psycho-oncology and Palliative Care, Nagoya City University Hospital, Nagoya, Aichi, Japan

Synonyms

Screening

Definition

Despite the development of modern medicine, cancer is a leading cause of death and disability. Since the development of cancer is associated with many genetic and environmental factors, efforts to prevent cancer by decreasing environmental factors have been made.

There are three levels in the cancer prevention strategy. The primary prevention is to reduce exposures to risk factors contributing to develop cancer. It includes smoking cessation, treatment of viral infections including papillomavirus, hepatitis B and C virus, diet, physical activity, reducing exposures to sunshine, ionizing radiation, or some harmful material such as aniline dyes, benzenes, and asbestos. Currently, at least one-third of all cancer cases are thought to be preventable via avoiding these risk factors.

The secondary prevention intends to promote early detection and early treatment and, therefore, reduces significant morbidity or mortality by cancer. Screening cancers is the main attempt but has been proven effective for relatively few types of cancer, with a few exceptions. The U.S. Preventive Services Task Force currently recommends cervical cytology testing for cervical cancer screening, mammography for screening breast cancer, and fecal occult blood testing, sigmoidoscopy, or colonoscopy for screening colorectal cancer.

Tertiary prevention involves activities to minimize the negative impact or outcome of cancer and maximizing the quality of life after developing cancer. Some risk factors for the development of cancer may accelerate the advance of cancer and result in early recurrence or shorter survival. Examples include negative effects of continued smoking after development of lung cancer on survival and high fat consumption in promoting breast cancer recurrence. This level also includes rehabilitation programs and patient support programs.

Cross-References

▶ Cancer Screening/Detection/Surveillance
▶ Prevention: Primary, Secondary, Tertiary

References and Readings

American Cancer Society Cancer Prevention & Early Detection Facts & Figures. (2008). Retrieved March 29, 2012, from http://www.cancer.org/Research/CancerFactsFigures/CancerPreventionEarlyDetectionFactsFigures/cancer-prevention-early-detection-facts-figures-2008

Smith, R. A., Cokkinides, V., Brooks, D., Saslow, D., Shah, M., & Brawley, O. W. (2011). Cancer screening in the United States, 2011: A review of current American Cancer Society guidelines and issues in cancer screening. *CA: A Cancer Journal for Clinicians, 61*(1), 8–30.

U.S. Preventive Services Task Force. Retrieved March 29, 2012, from http://www.ahrq.gov/clinic/uspstfix.htm

Cancer Risk Perceptions

Michael E. Stefanek
Research and Collaborative Research, Indiana University, Bloomington, IN, USA

Synonyms

Health risk; Likelihood judgments; Risk perception

Definition

Risk is the likelihood that something will happen.

Risk is a combined function of the probability of loss and the consequences of loss

(e.g., severity of loss in the physical, psychological, social, and economic realms).

Risk is a population-based measure, the chance of something happening, as determined by its occurrence among a large group of people over time. An individual's risk varies considerably within a given numerical boundary of a population's risk, due to variations in personal, genetic, environmental, and behavioral factors.

Risk communication is the communication with individuals (not necessarily face to face) which addresses knowledge, perceptions, attitudes, and behavior related to risk.

Cancer risk perception is the judgment, based on cognitive and affective factors, of the chances that a given individual will develop cancer over a certain period of time. It can be significantly influenced by the way in which an individual's risk is communicated to him or her. Both "thinking" and "feeling" are critical components of risk perception in general and cancer risk perception in particular.

Description

The issue of risk perception and communication in the cancer arena has received increasing attention over the past decade (Klein & Stefanek, 2007; Peters, McCaul, Stefanek, & Nelson, 2006; Rothman and Kivniemi, 1999). This is due in large part to the increasing awareness that the judgment that people make about their likelihood of developing cancer has important implications. At the level of the individual, risk perceptions guide protective action, such as not smoking, exercise and dieting behavior, and undergoing screening tests for early detection of cancer. If the perception of risk is underestimated, such protective action may not occur. If the perception exceeds the objective risk, such perception may cause anxiety, depression, stress, or may even result in excessive screening behaviors or indulgence in "alternative" health practices that have no evidence base. At the community level, risk perceptions may guide responses by communities concerned about "cancer clusters" in their immediate environment, what might be causing such excess cancers, and what is needed to fix the problem. At the policy level, risk perceptions may influence funding for cancer research and the development of guidelines for screening to detect cancer early or genetic tests to identify individuals who may inherit a higher risk of developing cancer.

Risk Perceptions and Health Behavior

Arguably the most critical issue determining the importance of risk perceptions is determining if such perceptions promote healthy behavior. Outside of the cancer realm, a recent meta-analysis of vaccination behaviors did indeed find a consistent relationship between risk perceptions and behavior, supporting the role of risk perceptions as a core concept in health behavior theories (Brewer et al., 2007). Reviewing the link between risk perception and behavior in cancer, the relationship is present, but appears modest. A solid summary of this data is provided by McCaul, Magnan and Dillard (2009) and a systematic review by Edwards et al. (2006) focusing upon personalized risk communication for informed decision making related to screening tests. These summaries note a generally positive relationship in areas such as mammography screening and smoking cessation, but also report that such relationships may have any number of mediators or moderators involved in this risk perception – health behavior link, including worry, barriers to change, or the presence of a family history of cancer. Given the modest relationship between risk perceptions and health behaviors linked to cancer prevention or early detection, it is not surprising that direct evidence that changing risk perceptions will cause subsequent changes in behavior is less available. However, there is indirect evidence that such changes may occur. McClure (2002) reviewed a series of studies of interventions that have provided biomarker data (carbon monoxide feedback to smokers) and supported the role of changes in risk perception in smoking cessation. There is also some evidence that using "teachable moments" such as the diagnosis of cancer to support smoking cessation may be productive,

linked perhaps to a new appreciation of one's risk of death (McBride & Ostroff, 2003).

In sum, risk perception is but one of a number of variables impacting health behaviors most critical to cancer control such as healthy eating, physical activity, tobacco use, excessive alcohol intake, excessive sun exposure, and appropriate utilization of cancer screening tests.

Given the data to date that supports the role of risk perception in cancer control, it is important to have an understanding of the processes involved in how people develop their perception of risk, how it is measured, and future research needed to develop our understanding of cancer risk perceptions.

Risk Perception: The Role of Affect and Cognition

How do people think about risk? It is now recognized that our perceptions of risk are influenced by a host of cognitive and affective variables. In addition, "how" risk is communicated impacts how our perception of the risk of cancer may be formed. These processes often lead to biases and misperceptions that influence both laypeople and health-care providers. There are a host of such processes to consider, many reviewed by Klein and Stefanek (2007), Peters et al. (2006), and McCaul et al. (2009) in the context of cancer control and well explained by Slovic (2010) in a more general overview of risk and risk communication. A number of such cognitive processes involve mental "shortcuts" or "heuristics." Very briefly, these heuristics can be thought of as "rules of thumb" that are used often automatically to influence the perception of risk. These include the availability heuristic, representativeness heuristic, the anchoring heuristic, and the affect heuristic. As one example, the availability heuristic refers to the common practice of making judgments about the frequency of an event based upon the information that is most readily available. If such information is unrepresentative or incomplete, the subsequent judgment will be inaccurate. For instance, if someone is in the process of scheduling a flight and is exposed to several stories of airline crashes, this may make the person feel relatively more at risk than driving to his or her destination. Likewise, when someone hears a story of celebrities developing cancer, perhaps by repeated media exposure, he or she may overestimate the risk of developing similar cancers. While such "heuristics" may indeed be helpful and accurate, they hold the potential for very inaccurate estimates of risk.

In addition to cognitive influences, there is a growing appreciation of the role of "affect" or emotion in risk perception (Slovic, 2010). It has become clearer that people process information through two distinct modes: deliberative and experiential (Slovic, 2010), following what has become known as the "dual process" theory of thinking, with the "deliberative" system being logical, analytical, slower, and the "experiential" system being more affective, intuitive, and fast. While it is assumed that these systems interact in forming risk perceptions, much work is needed to determine how this process plays out in forming risk perceptions. The role of the "experiential" system may be even more contributory in the area of cancer risk perception, given the fear and anxiety that accompanies the image of cancer development and treatment.

Finally, how risk is presented may significantly influence the perception of risk. Risk estimates can be provided in ways that differ only in format. For instance, relative risk (RR) is most commonly used (e.g., the risk of cancer is 25% higher in group A than in group B) in medical journals and the media. Another approach is to provide the number needed to treat (NNT) (e.g., 300 people need to take medicine A in order to save 1 life). Finally, the information can be presented as an absolute frequency (1,000 people took medicine A and 3 developed cancer; 1,000 people did not take medicine A and 4 developed cancer). Formats for conveying risk are critical since individuals are not mathematically fluent, nor do they have stable opinions about the magnitude of any given risk (Lipkus, 2007). This issue of presentation format becomes key, given the recognition over the past several years of the influence of numeracy in risk communication.

Conveying Risk Estimates

Whether perceptions of risk impact decisions and behavior relies on how messages of risk

magnitudes are conveyed. It is important to emphasize that communicating risk is not equivalent to communicating numbers. In fact, there is some controversy in the field of risk perception about the degree to which presenting risk in numerical format is critical to an individual's understanding of risk over time and links to behavior change. That is, what may be key is not specific numerical reasoning, but simply whether an individual has an understanding of the risk in a general fashion, whether she or he understands the "gist" of the risk (Reyna, 2004). Presenting risk verbally (e.g., "you have a somewhat higher than average risk") provides an overall sense of risk, but may fail to communicate the exact magnitude of risk, and is not helpful in making direct comparisons of risk across individuals. Numbers may be more precise than verbal representations of risk and provide a bit of scientific credibility to the risk communication, and most people express a preference for numbers. However, they do not address "gut" reactions or intuition well and do not provide clear information to individuals who may struggle with numerical competency. This latter issue of "innumeracy" is a critical one since increasing evidence indicates that a large proportion of individuals, even highly educated ones, struggle with numbers (Lipkus, Samsa, & Rimer, 2001; Schwartz, Woloshin, Black, & Welch, 1997). In fact, Schwartz et al. (1997) found that numeracy was strongly linked to being able to accurately use information about the benefit of mammography and called for more effective formats to present risks and benefits of mammography. Overall, there are few "best practices" cleanly established, although the call to present information both numerically and visually (graphs, tables) has been proposed (Lipkus, 2007).

Summary and Future Directions

It is clear that cancer risk perceptions are but one of many influences on health behavior related to cancer control. However, there is growing evidence that risk perception generally and cancer risk perception specifically can impact health behavior. In addition to cognitive influences on risk perception, it is essential to acknowledge the role of affect in the perception of risk. This includes not only incident affect (emotional state when risk information is communicated) but also "integral" affect (i.e., affect specifically related to the risk in question). It is not in the too distant past that cancer was viewed as a death sentence, with disfiguring and toxic treatments. Thus, the image of or beliefs surrounding cancer can clearly "link" to negative affect, which may then influence the perception of risk. Another key issue is how best to convey risk information. In addition, numeracy impacts the perception of risk. Many people have problems dealing with frequencies, percentages, or fractions, which impacts accurate risk representations, and the use of verbal labeling to transmit risk information risks being less than specific and perhaps quite different in meaning than the communicator(s) of such risk meant to transmit.

Continuing research is needed to determine how best to present risk while also utilizing what we do know about presenting such information in as "transparent" a manner as possible (Kutz-Micke, Gigerenzer, & Martignon, 2008). We must also continue to explore cultural differences in risk perception and how such influences may impact both perceptions and risk communication in order to intervene most effectively to enhance cancer control behaviors.

References and Readings

Brewer, N. T., Chapman, G. B., Gibbons, F. X., Gerrard, M., McCaul, K. D., & Weinstein, N. D. (2007). Meta-analysis of the relationship between risk perception and health behavior: The example of vaccination. *Health Psychology, 26*(2), 136–145.

Edwards, A. G. K., Evans, R., Dundon, J., Haigh, S., Hood, K., & Elwyn, G. J. (2006). Personalised risk communication for informed decision making about taking screening tests. *Cochrane Database of Systematic Reviews, 4*, 1–66. doi:10.1002/14651858.CD001865.pub2.

Klein, W. M., & Stefanek, M. (2007). Cancer risk elicitation and communication: Lessons from the psychology of risk perception. *CA: A Cancer Journal for Clinicians, 57*, 147–167.

Kutz-Micke, E., Gigerenzer, G., & Martignon, L. (2008). Transparency in risk communication: Graphical and analog tools. *Annals of the New York Academy of Sciences, 1128*, 18–28.

Lipkus, I. M. (2007). Numeric, verbal, and visual formats of conveying health risks: Suggested best practices and future recommendations. *Medical Decision Making, 27*, 696–713.
Lipkus, I. M., Samsa, G., & Rimer, B. (2001). General performance on a numeracy scale among highly educated samples. *Medical Decision Making, 21*, 7–44.
McBride, C. M., & Ostroff, J. S. (2003). Teachable moments for promoting smoking cessation: The context of cancer care and survivorship. *Cancer Control, 10*, 325–333.
McCaul, K. D., Magnan, R. E., & Dillard, A. (2009). Understanding and communicating about cancer risk. In S. M. Miller, D. J. Bowen, R. T. Croyle, & J. H. Rowland (Eds.), *Handbook of cancer control and behavioral science* (pp. 133–150). Washington, DC: American Psychological Association Press.
McClure, J. B. (2002). Are biomarkers useful treatment aids for promoting health behavior change? *American Journal of Preventive Medicine, 22*, 200–207.
Peters, E., McCaul, K., Stefanek, M., & Nelson, W. (2006). A heuristics approach to understanding cancer risk perception: Contributions from judgment and decision-making research. *Annals of Behavioral Medicine, 31*(1), 45–52.
Reyna, V. F. (2004). How people make decisions that involve risk. *Current Directions in Psychological Science, 13*(2), 60–66.
Rothman, A. J., & Kiviniemi, M. T. (1999). Treating people with information: An analysis and review of communicating health risk information. *Journal of the National Cancer Institute Monographs, 25*, 44–51.
Schwartz, L. M., Woloshin, S., Black, W. C., & Welch, G. H. (1997). The role of numeracy in understanding the benefit of screening mammography. *Annals of Internal Medicine, 127*, 966–972.
Slovic, P. (2010). *The feeling of risk*. Washington, DC: Earthscan.

Cancer Screening/Detection/Surveillance

Tainya C. Clarke and David J. Lee
Department of Epidemiology and Public Health, Miller School of Medicine, University of Miami, Miami, FL, USA

Synonyms

Cancer prevention

Definition

Cancer screening is the use of diagnostic tests and procedures to detect the presence of cancerous tissue before it is symptomatic. There are recommended routine screening tests for some of the more prevalent cancers. The parameters (such as age, time intervals) set for screening recommendations increase the likelihood that tests may detect the disease rather than the disease presenting itself symptomatically.

Description

According to the President's Cancer Panel, 41% of Americans will develop cancer in their lifetime (Reuben, 2010); however, data from the National Health Interview Survey indicates that only 75% of the US population adheres to recommended routine colorectal, breast, cervical, and prostate cancer screenings (National Health Interview Survey [NHIS], 1997–2010). Screening is important because it increases the probability of finding a cancerous growth in its early stage, despite the lack of any noticeable symptoms. Finding a cancerous growth in its earliest stage (i.e., during its period of *sojourn*), or in some cases before it becomes *palpable*, increases the likelihood of successfully treating the disease before it spreads. Additionally, there must be sufficient evidence that treatment initiated earlier as a result of screening will lead to an improved outcome (National Cancer Institute [NCI], 2011).

Cancer screening may reveal no tumor or the presence of a cancerous growth, which is then classified by stage. The concept of *staging* as a general classification of localized, regional, and distant disease was developed in the 1940s (NCI, 2011). Staging describes the severity of a person's cancer based on the extent of the primary tumor. One of the more detailed and more widely used staging systems is the *Tumor, Node, Metastasis (TNM) system*. In the TNM system, the tumor size, the status of the lymph nodes, as well as the status of distant metastases (spreading to other parts of the body) are also categorized (NCI, 2011). The statuses of these

core elements are aggregated into stages 0 through 4 and are associated with the likelihood of disease survival. Adherence to recommended routine screenings usually leads to discovery of tumors in their earliest stage. This includes *in situ*, where any abnormal cells present are only in the layer of cells in which they developed, or *localized*, wherein the cancer is limited to the organ in which it originated, and has not spread.

There are several recommended routine cancer screenings, most of which are age specific, some of which are gender specific (Table 1). Recommendations to patients are usually made by primary care physicians, but most screening tests are performed by physicians or technicians specializing in that particular field. Adherence to recommended routine cancer screening has led to the discovery of early stage tumors and has prevented the development of advance stage cancers. This has in turn resulted in an increase in quality adjusted life years and saves thousands of dollars in medical expenditure. There are several cancer registries in the USA which maintain records of reported tumors and that work closely with hospitals, cancer research centers, and agencies responsible for cancer surveillance. The continued surveillance of screening behavior within the population, the chronicling of cancer staging in addition to monitoring associated morbidity and mortality rates provide valuable information treatment and survival.

Cancer surveillance and screening are carried out by several agencies and responsible programs such as the Surveillance, Epidemiology, and End Results (SEER) Program of the National Cancer Institute (NCI), and the Center for Disease Control's National Program of Cancer Registries-Cancer Surveillance System (NPCR-CSS). Cancer surveillance involves the measurement and monitoring of cancer incidence, survival, morbidity, and mortality for persons with cancer. Surveillance also assesses of genetic predisposition of a population, environmental risks in addition to population cancer health and risk behavior (NCI, 2010a).

Cancer screening is not without controversy, and there are ongoing debates regarding whether the harms associated with some tests outweigh their benefits. Screening tests may present unnecessary physical and psychological risks for persons being tested. Some screening procedures have been known to cause bleeding, while others have resulted in perforation of the lining of sensitive organs (Morbidity and Mortality Weekly Report [MMWR], 2010) (see Table 1). The risks of screening tests may be further increased as the test results may not always be valid (i.e., a test may fail to detect a cancerous growth, and this kind of *false-negative* result can lead to a delay in treatment and/or removal of the cancer). Contrarily, sometimes a test may detect a cancer when there is none present. This *false-positive* test result causes undue stress and anxiety and usually leads to the patient being submitted to further tests, which may also have risks (Levin et al., 2008).

The ability of a screening test to detect cancer in a person who truly has the disease (*sensitivity*) or failure to find cancer in a person who is truly negative for the disease (*specificity*) is of outmost importance in determining the gold standard for screening tests. A reliable screening test should have both high sensitivity and high specificity.

History of Screening

While cancers were being surgically removed as early as the 1700s, screening for the disease did not begin until the late nineteenth century. This was as a result of an insightful discovery by a mid-nineteenth century, German pathologist named Rudolf Virchow. Virchow discovered that cancerous tumors were the result of abnormal growth of normal cells (McNeely, 2002), which laid the foundation for early detection.

Cancer screening has evolved since its institution in the early 1900s, and advances in detection techniques have resulted in the early discovery of cancerous cell growth. This is attributed to the highly sophisticated screening tools used for various tests and procedures. The most common types of screening tests are imaging and laboratory tests. Imagining tests include x-ray mammograms for breast cancer screening and computed tomography (CT) scans used to detect or confirm the presence of brain, lung, and bone cancers, among others. Papanicolaou (Pap) tests for

Cancer Screening/Detection/Surveillance, Table 1 Advantages and disadvantages of some common cancer screening tests

Screening exam		Current recommendations	Benefits/advantages	Risks/disadvantages
Breast cancer screening	Mammography (a digital or film x-ray picture of the breast)	Women ≥40 years should have mammograms every 1–2 years	Only proven reliable method of detection of small abnormal tissue growths confined to the milk ducts (ductal carcinoma in situ)	False positive- which may lead to unnecessary additional testing
		Women who are at higher than average risk of breast cancer should talk with their health care providers regarding frequency of screening and age at which to start	Detects all types of breast cancers, including invasive ductal and lobular cancers	Over diagnosis may lead to the treatment of clinically insignificant cancers. This may result in breast deformity, thromboembolic events, lymphedema, development of new cancers, or toxicities due to chemotherapy
	Clinical breast exam	Every 3 years for women in their 20s and 30s and every year for women ≥40 years	Lead to a decrease in breast cancer cause specific mortality among women 50 –69 years	False-negatives lead to a false sense of security and a delay in cancer diagnosis
Cervical cancer screening	Pap test	Recommended for women at least 3 years after having first vaginal intercourse, but no later than 21 years old	Reduces mortality from cervical cancer by finding cancers when they are most treatable	Regular Pap tests lead to additional diagnostic procedures (e.g., colposcopy) and treatment for low-grade squamous intraepithelial lesions (LSIL), with long-term consequences for fertility and pregnancy
		Regular Pap test every 1 year or newer liquid-based Pap test every 2 years		
		Women ≥30 who have had 3 consecutive normal Pap test results may get screened every 2–3 years. Women ≥30 years may also get screened every 3 years with either the conventional or liquid-based Pap test, in addition to the human papilloma virus (HPV) test		
		Women ≥70 years with 3 or more consecutive normal Pap tests in and no abnormal Pap test results in the last 10 years may choose to stop having Pap tests		
		Women who have had a total hysterectomy for non-cancer related reasons may stop having Pap-tests		

(*continued*)

Cancer Screening/Detection/Surveillance, Table 1 (continued)

Screening exam		Current recommendations	Benefits/advantages	Risks/disadvantages
Colorectal screening	Flexible sigmoidoscopy	Men and women ≥50 years	Allows the doctor to view the rectum and the entire colon	Examines only the rectum and the lower part of the colon. Any polyps in the upper part of the colon will be missed
		Every 5 years[a], or	Doctor can perform a biopsy and remove polyps or other abnormal tissue during the test, as needed	
	Colonoscopy	Every 10 years, or		Requires thorough cleansing of the colon before the test. Some form of sedation is used in most cases
	CT colonography (virtual colonoscopy)	Every 5 years[a]		Risks tearing or perforation of the lining of the colon
	Double-contrast barium enema	Every 5 years[a], or		cannot perform a biopsy or remove polyps during the test
	Fecal occult blood test (gFOBT)	Annually[b], or	Not an invasive procedure, hence complications are rare	Additional procedures are necessary if the test indicates an abnormality
	Fecal immunochemical test (iFOBT/FIT)	Annually[b], or	No sedation is necessary	Fecal tests fail to detect most polyps and some cancers
			No cleansing of the colon is necessary	Dietary changes recommended a few days prior to gFOBT but not iFOBT
			FOBT does not cause bleeding, tearing or perforation of the lining of the colon	Colonoscopy required if the test indicates an abnormality
Digital rectal exam (DRE)	Annually	Interval uncertain (possibly 3–5 years)[b]	No cleansing of the colon is necessary	Only detects abnormalities in the lower part of the rectum
Stool DNA test (sDNA)	Interval uncertain (possibly every 3–5 years)			
Prostate cancer screening	Prostate specific antigen (PSA) blood test	Discuss with physician the pros and cons of receiving a baseline PSA and if conducted, when another test would be necessary	Detects the disease in its early stage among high risk men	Detects small non life threatening cancers that leads to over diagnosis and complications from unnecessary treatment
	PSA velocity test (How PSA measures rise over time)			Causes unnecessary anxiety
	PSA density test (Ratio of PSA level to size of prostate gland)	Men at higher than normal risk (Blacks, men whose father, brother or son have been diagnosed with prostate cancer)		Elevated PSA levels may be due to other noncancerous conditions such as benign prostatic hyperplasia and prostatitis
	Percent-free PSA (Ratio of unattached PSA in blood to total PSA)	Discuss screening with physician at 45 years		

(*continued*)

Cancer Screening/Detection/Surveillance, Table 1 (continued)

Screening exam		Current recommendations	Benefits/advantages	Risks/disadvantages
	Age-specific PSA range	Men ≥50 years discuss the harms and benefits of PSA screening with physician		
	Digital rectal exam	Men with a previous PSA of ≥4 ng/ml in the blood, should be retested if discussion with physician dictates a necessity		

[a]If the test is positive, a colonoscopy should be done
[b]The multiple stool take-home tests should be used. One test done by the doctor in the office is not adequate for testing. A colonoscopy should be done if the test is positive

cervical cancer screening and prostate-specific antigen (PSA) tests for prostate cancer screening are typically confirmed by laboratory tests. Other screening tools include ultrasound, magnetic resonance imaging (MRI), and fine-needle biopsy. Additionally, proteomics have been used to diagnose and identify the best treatment for specific individuals, and genetic testing has been used to confirm whether women tested may have an increased probability of developing a certain type of cancer (NCI, 2011).

The use of vaginal smears (Pap test) for cervical cancer screening was established in the late 1930s by George Papanicolaou (Papanicolaou and Traut, 1941). Colorectal screening began in the 1940s and was conducted with a rigid proctoscope until the introduction of the flexible sigmoidoscope in the 1980s (Grossman, 1998). Breast cancer screening was implemented in the 1960s (Fletcher, 2011), while PSA serum test was approved for prostate cancer screening by the Food and Drug Administration in the early 1990s (NCI, 2010b).

Recommending Authorities

Several authorities on cancer issues periodically update screening guidelines. Some of the more prominent agencies which make screening recommendations include the following: the American Cancer Society (ACS), the American College of Radiology (ACR), the American College of Obstetricians and Gynecologists (ACOG), and the National Cancer Institute (NCI). The ACS, the ACR, and the NCI issue guidelines for all cancer types considered amenable to screening, while the ACOG makes recommendations for female gender–related cancers such as breast and cervical cancer screening (American Cancer Society [ACS], 2011b; American College of Obstetrics and Gynecologists [ACOG], 2009, 2011; NCI, 2011). The United States Preventive Services Task Force (USPSTF) is another organization that makes screening recommendations. They are a small independent panel of nongovernment medical experts who have a strong foundation in preventive and evidence-based medicine (United States Preventive Services Task Force [USPSTF], 2011). The panel usually comprises of general doctors (such as family physicians, internists, physician specialists, pediatricians, nurses, and health behavior specialists). There is some amount of disagreement regarding some screening guidelines among these different groups, especially with reference to the recent controversial changes in breast cancer screening.

The USPSTF assigns one of five letter grades to each of its recommendations; a level of certainty regarding net benefits accompanies each letter grade (USPSTF, 2011). The USPSTF also uses an I-statement/grade when there is insufficient evidence to assess the balance of benefits and harms of the recommended service. These recommendation processes and methods are outlined in a procedure manual and are based on evidence-based medicine (USPSTF).

Controversial Recommendations

In 2009, the USPSTF recommended changing the breast cancer screening guidelines for women from annual mammograms beginning at age 40 to every other year beginning at age 50 after determining that the benefits of annual mammograms beginning at age 40 did not outweigh the potential risks (Pickert, 2011). They argued that increased mammography screenings would lead to a greater likelihood of false positives, psychological stress, depression, overexposure to radiation, and unnecessary surgery.

The scientific panel supporting the USPSTF's decision strongly believes that much of the abnormal cell growth detected in women in their 40s could be detected in their 50s with no adverse effects from the delay. However, there have been numerous studies, including that by Anders et al. (2008), which show aggressive fast-growing cancers in younger women, which would in fact contradict the USPSTF. The ACS, ACR, ACOG, and the NCI still recommend annual screenings, beginning at age 40 (ACOG, 2011; ACR, 2008; ACS, 2011a; NCI, 2011).

Prostate cancer screening is equally contentious and is notorious for detecting false positives and false negatives. PSA testing does not distinguish tumors that would cause no harm from clinically significant tumors, which results in overdiagnosis and overtreatment (Pickert, 2011). In October 2011, the USPSTF issued a 'D' grade for PSA screening, thus recalling previous recommendations of annual screenings for men who do not have an increased risk of getting the disease. The USPSTF has determined that there is a moderate or high certainty that PSA screening offers no net benefit and that the harms from associated tests and exams outweigh the benefits of the screening.

As agencies try to improve on screening recommendations and clinical practice, careful consideration must be made with regard to the public health message being communicated to the general population. New scientific discoveries, frequent changes in recommendations, and disagreements between recommending authorities cast doubts among the general public and dissuade persons from adhering to recommended screenings. In an effort to increase the number of early detections and reduce the incidence of avoidable cancers within the population, it is important to resolve existing controversies and reduce the frequency of changes in recommendations. These inconsistencies may result in confusion, mistrust, and a negative attitude and behavior toward recommended cancer screenings.

Surveillance

Public health officials often consider the proportion of the population that must participate in a screening program for one death to be prevented within a defined time interval. This proportion is dependent on the disease characteristics as well as other population parameters. Epidemiologists and population scientists often investigate the measures of risks within a particular population; this translates to the implementation of public health policy and screening guidelines as well as helps dictate the actions taken by medical practitioners.

Ongoing surveillance conducted by the aforementioned recommending authorities has identified disproportionately lower screening behavior among certain subsets of the US population. African American and Hispanics are less likely to seek recommended cancer screening compared to their non-Hispanic White counterparts (Vidal et al., 2009). Uninsured Americans and those living below the poverty income level are less likely to report having a routine place of care and thus less likely to get recommended screening advice from a medical professional. Blue-collar workers and workers in the service industry are less likely to adhere to recommended screenings when compared to persons employed in the white-collar job sector (Vidal et al.). In the United States, this information (on the noninstitutionalized civilian population) is collected and stored in several population health databases. As such, epidemiologists and behavioral scientists are able to assess the adherence to screening in conjunction with some of the more common social determinants of health and demographic information. With this information, we are also able to correctly identify groups of persons that are at a higher risk and therefore require more frequent

screening. These analyses lead to reports which further drive policies and influence research and investigations into current recommendations and screening practices. It is of utmost importance to not only recommend cancer screening but also provide the public with information on the associated harms and benefits to early detection and encourage persons to take a more active role in managing cancer-related and other preventive health behavior.

The information in the table below has been adopted from the American Cancer Society and the National Cancer Institute. It provides an overview of general screening recommendations and their associated advantages and disadvantages.

Cross-References

- ▶ American Cancer Society
- ▶ National Cancer Institute

References and Readings

American Cancer Society. (2011a). *Guidelines for early the detection of cancer*. Retrieved August 26, 2011, from www.cancer.org

American Cancer Society. (2011b). *Cancer facts and figures 2011*. Atlanta, GA: American Cancer Society. Retrieved August 25, 2011, from www.cancer.org

American College of Obstetricians and Gynecologists. (2011). Breast cancer screening. *Obstetrics and Gynecology, 118*, 372. ACOG Practice Bulletin No. 122.

American College of Obstetrics and Gynecologists. (2009). *ACOG announces new pap smear and cancer screening guidelines*. Retrieved August 26, 2011, from www.acog.org

American College of Radiology. (2008). *ACR practice guideline for the performance of screening and diagnostic mammography*. Retrieved August 26, 2011, from www.acr.org

Anders, C. K., Hsu, D. S., Broadwater, G., Acharya C.R., Foekens J.A., Zhang Y. et al. (2008). Young age at diagnosis correlates with worse prognosis and defines a subset of breast cancers with shared patterns of gene expression. *Journal of Clinical Oncology*, *26*, 3324–3330.

Fletcher, S. W. (2011). Breast cancer screening: A 35-year perspective. *Epidemiologic Reviews, 133*, 165–175.

Grossman, S. (1998). A new era in colorectal screening and surveillance. *The Permanente Journal, 2*, 1.

Levin, B., Lieberman, D. A., McFarland, B., Smith R.A., Brooks D. Andrews K.S. et al. (2008). Screening and surveillance for the early detection of colorectal cancer and adenomatous polyps: A joint guideline from the American Cancer Society, the US Multi-Society Task Force on Colorectal Cancer, and the American College of Radiology. CA: *A Cancer Journal for Clinicians*, *58*, 130.

McNeely, I. F. (2002). *Medicine on a grand scale: Rudolf Virchow, liberalism, and the public health*. London: Welcome Trust Centre for the History of Medicine at University College.

Morbidity and Mortality Weekly Report (MMWR). (2010). Surveillance of screening detected cancers (colon and rectum, breast, and cervix)-United States, 2004–2006. *Morbidity and Mortality Weekly Report. Surveillance Summaries, 59*, 1.

National Cancer Institute (2010a). Cancer Trends Progress Report – 2009/2010. Retrieved August 23, 2011 from http://progressreport.cancer.gov.

National Cancer Institute (2010b). Cancer Advances in Focus: Prostate Cancer. Retrieved August 23, from http://www.cancer.gov/cancertopics/factsheet/cancer-advances-in-focus/prostate.

National Cancer Institute. (2011). *Cancer screening overview*. Retrieved August 29, 2011, from http://www.cancer.gov

National Health Interview Survey (NHIS). 1997–2010. Retrieved September 3, 2011, from http://www.cdc.gov/nchs/nhis/quest_data_related_1997_forward.htm

Papanicolaou, G. N., & Traut, H. F. (1941). The diagnostic value of vaginal smears in carcinoma of the uterus. *American Journal of Obstetrics and Gynecology, 42*, 193–206.

Pickert, K. (2011, June 2). The Screening Dilemma. *Time Magazine* on line. Retrieved August 26, 2011, from http://www.time.com/time/specials/packages/article/0,28804,2075133_2075127_2075108-2,00.html

Reuben, S. H. (2010). *Reducing environmental cancer risk: What we can do now*. President's Cancer Panel. U.S. Department of Health and Human Services 2008–2009 Annual Report.

United States Preventive Task Force. (2011). *Recommendations*. Retrieved August 26, 2011, from http://www.uspreventiveservicestaskforce.org

Vidal L, LeBlanc WG, McCollister KE, Arheart KL, Chung-Bridges K, Christ S, Caban-Martinez AJ, Lewis JE, Lee DJ, Clark J 3rd, Davila EP, Fleming LE. Cancer screening in US workers. (2009). Cancer screening in US workers. *American Journal of Public Health*, *99*(1), 59–65.

Cancer Survivor

- ▶ Cancer Survivorship

Cancer Survivorship

Steven C. Palmer
Abramson Cancer Center, University of Pennsylvania, Philadelphia, PA, USA

Synonyms

Cancer survivor

Definition

Cancer survivorship, as a construct, is a recognition of the large number of individuals living with cancer and its aftermath. The term represents an expanded emphasis placing quality of life on a par with efforts to prolong and lengthen survival. Although there is general agreement that "cancer survivorship" represents a distinct concept within the cancer experience trajectory, the definition of *who* is a survivor and *when* one transitions from patient to survivor status is less clear and depends more on sociological and political considerations than empirical data. The most common definition and that preferred by the Office of Cancer Survivorship at the National Cancer Institute is that cancer survivorship starts at "the time of diagnosis" and continues "through the balance of (the survivor's) life. Family members, friends, and caregivers are also impacted by the survivorship experience and are therefore included in this definition."

Description

Origin

Cancer survivorship is a relatively new area of study. Only since the 1970s could more than half of those adults diagnosed with cancer expect to live at least 5 years. "Cancer survivorship," as a construct, was introduced in the mid-1980s by the National Coalition for Cancer Survivorship. The definition introduced by the founder of that organization defines an individual as a cancer survivor from diagnosis throughout the remainder of his or her life and includes family, friends, and informal caregivers. Thus, survivorship was broadly defined and inclusive of those with whom the patient interacted intimately and from whom the patient received support. The impetus for this definition appears to be a desire to shift the focus away from the concept of the "cancer victim" to one in which individuals were seen as actively coping with the range of physical, psychological, and social sequelae that occur throughout the cancer experience from diagnosis to end of life.

Although this is the most common view of when cancer survivorship begins, other views exist, as well. Historically, the medical field has endorsed a more circumscribed definition of a cancer survivor as an individual who has completed active cancer treatment and experienced a period of at least 5 years of disease-free status. Thus, survivorship is more or less equated with "cure" of primary disease, and the focus is clearly on the aftereffects of cancer and its treatment. Others have defined a cancer survivor as "someone who has completed initial treatment and has no apparent evidence of active disease, or is living with progressive disease and may be receiving treatment but is not in the terminal phase of illness, or someone who has had cancer in the past." Again, this focuses away from the immediate effects of cancer and treatment and toward issues of posttreatment well-being.

Characteristics of Cancer Survivors

As of 2008, there were approximately 11,900,000 cancer survivors in the United States, representing about 4% of the population. As can be seen in Table 1, cancer is disproportionately a disease of older adults, and almost 60% of cancer survivors are aged 65 and older, although these individuals represent only about 12% of the total population, while less than 1% of cancer survivors are aged 19 or younger. Thus, issues of cancer survivorship occur most commonly in the context of physical comorbidities that are frequent among older adults.

The number of cancer survivors is growing in the USA, due to earlier detection of breast,

Cancer Survivorship, Table 1 Cancer survivorship in USA by age group (2008)

Age group	Proportion of survivors
Less than 19 years	<1%
20–39 years	4%
40–64 years	36%
+65 years	59%

prostate, and colorectal cancer and, to a lesser extent, improved treatments. Currently, more than 66% of adults diagnosed with cancer can expect to live at least 5 years and 75% of children can expect to live at least 10 years following a cancer diagnosis. The total number of survivors is expected to increase at even a faster pace as the number of individuals aged 60 and older increases due to population growth and the aging of the "baby boomer" population. Simultaneously, the number of individuals who have survived cancer for a long period of time is expected to increase. Currently, more than 15% of cancer survivors have lived 20 or more years from initial diagnosis.

In terms of specific cancer sites, female breast cancer survivors represent the plurality of survivors (22%), followed by prostate cancer survivors (20%), colorectal cancer survivors (9%), gynecological cancer survivors (8%), hematological cancer survivors (8%), urinary tract cancer survivors (7%), and melanoma survivors (7%).

Racial and ethnic minorities are somewhat underrepresented among cancer survivors, particularly African Americans who represent approximately 13% of the total population but only 8% of cancer survivors. Considering all cancers, African Americans are more likely than other racial groups to die following a cancer diagnosis and less likely to survive for extended periods (e.g., $\geq$10 years). The precise reasons for this disparity are presently unknown, but likely include a complex interplay of social, cultural, and economic factors, with access to adequate medical care and poverty playing key roles. What is clear is that following diagnosis, racial and ethnic minorities experience relatively worse outcomes including greater chance of cancer recurrence, increased mortality, and decreased overall survival times.

Seasons of Cancer Survival

In 1985, Mullen described the "seasons" of survival, each of which is centered around a different stage of disease and treatment, and each of which focuses on a specific set of concerns. These seasons, acute, extended, and permanent survival, are described in Table 2.

Long-Term and Late Effects of Cancer and Its Treatment

As noted, long-term survival is now the norm for most cancer patients. This increased survival time has come with a cost; cancer and its treatment can and often do lead to decreased quality of life. Some of these effects are caused directly by tumor burden itself, while others are related to treatment exposures. That is, they are the unintended consequences of exposure to surgery, toxic chemotherapy, and ionizing radiation, among other treatments. The side effects of cancer and its treatment can occur in physical, psychological, or social domains and are often conceptualized in terms of long-term or late effects.

Long-term effects are those side effects that arise during cancer treatment and persist following the treatment period. These effects can last for months or years following cancer treatment, but may resolve over time. For example, many people treated with certain chemotherapies develop peripheral nerve damage during treatment that can affect hearing, balance, or touch for years afterward. *Late effects* on the other hand, are those symptoms or toxicities that are either undetected or absent during active treatment but arise only afterward. In many cases, late effects may not be recognized for years following cancer treatment. Research into the long-term and late effects of cancer and its treatment is ongoing, better understood in cases of pediatric cancer than adult-onset cancer, and not well-developed in terms of prevalence estimates or an understanding of when they might arise or how long they might last before resolving. Although many long-term and late effects of treatment are

Cancer Survivorship, Table 2 Seasons of survival (Mullen 1985)

Season	Disease and treatment stage	Physical concerns	Psychosocial concerns
Acute survival	Diagnosis, primary treatment	Pain control, side effects	Mortality issues, fear, distress, coping with treatment
Extended survival	Remission, completion of primary treatment	Physical limitations, treatment effects, loss of strength, fatigue	Fear of recurrence, body image
Permanent survival	Cure of disease	Long-term and late effects of treatment, second tumors, reproductive health	Employment, insurance, coping with diminished health

specific to particular cancer sites or treatment regimens, there are a number of common symptoms and experiences.

Physical Effects of Cancer and Its Treatment

The most common physical symptom reported by cancer patients regardless of cancer site or treatment is fatigue, with more than 50% of cancer patients reporting fatigue at some point in the survivorship trajectory. Pain is also a common long-term effect of cancer treatment, with more than 40% of cancer patients reporting pain at some point and more than one-fifth reporting pain as long as 2 years after diagnosis, most commonly resulting from surgical intervention. Other physical effects that can arise from cancer, surgery, radiation, chemotherapy, or hormonal exposures include second cancers, bone difficulties such as osteoporosis, cardiovascular and coronary dysfunction, fertility difficulties, hormonal deficiencies, sexual dysfunction, hematological problems, immunosuppression, lymphedema, pulmonary difficulties, and problems with renal function.

Psychosocial Effects of Cancer and Its Treatment

The term "distress" has been used to describe the emotional experience of cancer survivors in a nonpathologizing and nonstigmatizing manner. The most commonly used conceptualization of distress, from the National Comprehensive Cancer Network, defines it as "a multifactorial unpleasant emotional experience of a psychological (cognitive, behavioral, emotional), social, and/or spiritual nature that may interfere with the ability to cope effectively with cancer, its physical symptoms and its treatment. Distress extends along a continuum, ranging from common normal feelings of vulnerability, sadness, and fears to problems that can become disabling, such as depression, anxiety, panic, social isolation, and existential and spiritual crisis." Thus, distress encompasses a broad range of emotional experiences that range from normative feelings of sadness and fear about the future to more chronic and interfering experiences such as depression and anxiety.

Psychosocial difficulties such as distress appear to be fairly common among early cancer survivors, although many of these difficulties are mild in severity and tend to decrease over time from diagnosis. One exception to this appears to be an increase in negative emotional experience among individuals nearing end of life or entering into palliative care.

Essentials of Survivorship Care

To improve the outcomes achieved by the ever increasing number of cancer survivors, the Institute of Medicine has outlined four components essential to quality survivorship care. First, survivorship care should focus on *prevention* of new and recurrent cancers, as well as other late effects of treatment. Second, there should be an emphasis on *surveillance* for new, recurrent, or spread of cancer as well as medical and psychosocial late effects. Third, there is a need for *intervention* to assist survivors in dealing with the consequences of cancer and its treatment. Fourth, given the complex medical environments in which cancer survivors find themselves, *coordination of care*, particularly between specialty and primary providers, is essential to ensuring that survivors' needs are met.

References and Readings

Ganz, P. A. (2009). Survivorship: Adult cancer survivors. *Primary Care: Clinics in Office Practice, 36*, 721–741.
Harrington, C. B., Hansen, J. A., Moskowitz, M., Todd, B. L., & Feuerstein, M. (2010). It's not over when it's over: Long-term symptoms in cancer survivors – A systematic review. *International Journal of Psychiatry in Medicine, 40*(2), 163–181.
Hewitt, M., Greenfield, S., & Stovall, E. (Eds.). (2006). *From cancer patient to cancer survivor: Lost in transition*. Washington, DC: The National Academies Press.
Mullen, F. (1985). Seasons of survival: Reflections of a physician with cancer. *New England Journal of Medicine, 313*, 270–273.

Cancer Treatment and Management

Deidre Pereira and Megan R. Lipe
Department of Clinical Health and Psychology, University of Florida, College of Public Health and Health Professions, Gainesville, FL, USA

Synonyms

Chemotherapy; Radiation therapy; Surgery; Surgical resection

Definition

Medical intervention in cancer commonly involves multiple modalities including surgery, systemic chemotherapy, and radiation therapy. For patients newly diagnosed with cancer, surgery is typically the first of these methods as it is commonly used to confirm a diagnosis, determine the severity of the disease (i.e., stage and grade), and in some cases, for "tumor debulking." Radiation therapy is used in more than half of all patients with cancer, either as a definitive treatment, or in combination with surgical interventions and/or chemotherapy. Finally, chemotherapy involves the use of drugs to target and destroy rapidly dividing cancer cells. As a treatment, its effectiveness is dependent upon the type and severity of disease, but it can be used to shrink tumors, control the spread of disease, or cure cancer (i.e., remission).

Description

Cancer patients are at an increased risk for psychological and physiological distress throughout the treatment and management of their disease. The most common types of treatment, as previously discussed, impact the body physically, mentally, and emotionally. Behavioral and psychosocial strategies can not only be used to supplement these medical treatments but also reduce the physical and psychological side effects of cancer treatment and management. The primary symptom of concern and of focus in the psychosocial treatment of individuals with cancer is *distress*. The National Comprehensive Cancer Network (NCCN), an organization that has composed widely used guidelines for distress management, posits that the term "distress" is more acceptable in its use because it carries a less stigmatizing connotation. NCCN defines distress in cancer as "A multifactorial unpleasant emotional experience of a psychological (cognitive, behavioral, emotional), social, and/or spiritual nature that may interfere with the ability to cope effectively with cancer, its physical symptoms and its treatment."

Patients are at an increased risk for distress if they have a history of a psychiatric disorder or substance abuse, are cognitively impaired, have language, literacy, or physical barriers to communication, have severe comorbid illness, experience uncontrolled physical symptoms, have spiritual/religious concerns, have inadequate social support, or have additional stressors including family conflict, financial stressors, dependent children, limited access to healthcare, or have a history of abuse. Providers participating in the care of cancer patients should assess for these risks and also be aware of times associated with increased vulnerability for distress. Periods of increased vulnerability are often marked by times of change or novelty in a patient's cancer experience or feelings of uncertainty. For example, awaiting diagnosis and/or treatment, altering treatment modality, transitioning into survivorship, recurrence, and end of life are often circumstances under which distress may be more likely to manifest.

In addition, distress is associated with physical side effects of disease and/or cancer treatment including pain, fatigue, nausea, and insomnia. These symptoms are very common in cancer patients and often result from psychological distress and physiological effects of cancer treatment. As such, there is a bidirectional relationship among these symptoms and distress such that unpleasant physical side effects may prompt distress while the experience of emotional distress may also exacerbate and maintain these physical symptoms.

Psychosocial cancer management and treatment must first begin with a brief distress screening. It is optimal that these be completed earlier in a patient's cancer experience (i.e., upon diagnosis). NCCN provides a brief, validated distress screening tool that measures recent distress and the presence of factors (i.e., practical, family, emotion, spiritual, or physical problems) that may contribute to distress. Patients who endorse clinically significant levels of distress should then be referred to the appropriate service(s), depending on their individual needs. Cancer patients experiencing distress may benefit from assistance from a mental health professional, social worker, and/or chaplain.

Patients referred to a mental health professional should undergo a more comprehensive evaluation in order to further understand the difficulties they are having and to inform treatment decisions. These evaluations will commonly assess for suicidality, mood or anxiety disorders, adjustment disorders, substance-abuse disorders, personality disorders, and cognitive impairments secondary to disease and/or cancer treatment. Sleep disorders are assessed through a thorough sleep and medical history and objective evaluations such as polysomnography. Results from these evaluations are disseminated to all other providers involved in the patient's care in order to ensure that their comprehensive treatment plan is tailored to their individual needs.

Treatment for distress and its associated symptoms secondary to cancer is multifaceted in nature. While psychotherapy is indicated in patients with mild to severe distress, antidepressants and anxiolytics can be used to supplement psychotherapy in individuals with moderate to severe distress. Psychological interventions including cognitive-behavioral therapy (CBT), supportive psychotherapy, and family or couples therapy have been shown to help cancer patients manage distress and improve quality of life. Cognitive-behavioral treatments in cancer often focus on increasing problem solving skills and addressing maladaptive thought patterns that promote feelings of depression, anxiety, and/or guilt. Behavioral management strategies are also used with cancer patients to decrease the psychosomatic manifestation of distress. Fatigue in cancer can be managed through the practice of relaxation, distraction, exercising to increase energy, improving sleep, and emotional support. Individuals with insomnia benefit from behavioral treatments that place focus on creating a comfortable sleep environment that promotes sleep (i.e., stimulus control), avoiding behaviors that contribute to poor sleep such as drinking caffeine and napping, and addressing any emotional concerns that may contribute to poor sleep. Behavioral techniques can also be used in addition to anti-nausea/vomiting medications and analgesics to help patients relax and feel more in control of nausea and/or pain following cancer treatment. Guided imagery allows the patient to mentally transition to a more pleasant, safe place, and to distract oneself from the nausea and/or pain. Likewise, cancer patients can utilize hypnosis or learn self-hypnosis in order to block physical discomfort and pain during and after treatment procedures. Lastly, progressive muscle relaxation and biofeedback can also be used to help patients increase awareness of tension, anxiety, and other bodily changes in order to achieve relaxation and prevent nausea, pain, or insomnia.

The initial screening, comprehensive evaluation, and treatment are essential to ensuring that cancer patients navigate their experiences effectively; however, it is also important to conduct assessments of distress at further points along the cancer experience. As previously mentioned, there are periods of time in which patients may have an increased vulnerability to distress and it is during these transitional periods in which their distress should be reassessed and treated as necessary.

Given its complex nature, physical and psychological distress throughout the process of cancer treatment should be assessed and managed through comprehensive involvement by all providers in the patient's healthcare team. This requires that the patient's healthcare team function in an environment in which interdisciplinary work is promoted and in which there is regular, open communication among all providers involved in the patient's care and management of their disease.

Cross-References

- ▶ Cancer Prevention
- ▶ Cancer Survivorship
- ▶ Cancer, Types of
- ▶ Cancer: Psychosocial Treatment

References and Readings

Abeloff, M. D., Armitage, J. O., Niederhuber, J. E., Kastan, M. B., & Gillies McKenna, W. (2008). *Clinical oncology* (4th ed.). Philadelphia: Churchill Livingstone.

American Cancer Society. (2010). *Fatigue in people with cancer*. Retrieved May 26, 2011, from http://www.cancer.org

American Cancer Society. (2010). *Nausea and vomiting*. Retrieved May 26, 2011, from http://www.cancer.org

American Cancer Society. (2010). *Pain control: A guide for those with cancer and their loved ones*. Retrieved May 26, 2011, from http://www.cancer.org

National Cancer Institute. (2010). *Nausea and vomiting PDQ*. Retrieved May 26, 2011, from http://www.cancer.gov

National Cancer Institute. (2010). *Pain PDQ*. Retrieved May 26, 2011, from http://www.cancer.gov

National Cancer Institute. (2010). *Sleep disorders PDQ*. Retrieved May 26, 2011, from http://www.cancer.gov

National Comprehensive Cancer Network. (2011). *The NCCN clinical practice guidelines in oncology: Distress management* [Version 1.011]. Retrieved May 26, 2011, from http://www.nccn.org

Cancer Types

- ▶ Cancer, Types of

Cancer, Bladder

Heather Honoré Goltz[1,2], Marc A. Kowalkouski[1], Stacey L. Hart[3] and David Latini[4]
[1]HSR&D Center of Excellence, Michael E. DeBakey VA Medical Center (MEDVAMC 152) Houston, TX, USA
[2]Department of Social Sciences, University of Houston-Downtown, Houston, TX, USA
[3]Department of Psychology, Ryerson University, Toronto, ON, Canada
[4]Scott Department of Urology, Baylor College of Medicine, Houston, TX, USA

Synonyms

Bladder carcinoma; Urothelial carcinoma of the bladder

Definition

Bladder cancer research has almost exclusively employed epidemiological or clinical research approaches, leaving it understudied from a behavioral medicine perspective. Yet, research provides many opportunities for clinicians and researchers to develop targeted bladder cancer prevention and survivorship interventions for mental health, diet and exercise, fatigue, smoking cessation, and other areas.

Description

Bladder Anatomy and Histopathology

Bladder cancer originates in the urinary bladder, a hollow, muscular organ that collects urine from the kidneys via the ureters and excretes it via the urethra (Konety & Carroll, 2007; Pashos, Botteman, Laskin, & Redaelli, 2002). Before excretion, urine is stored in the lumen, which is surrounded by several cell layers comprising the bladder wall. The innermost layer, or urothelium (epithelium), directly contacts urine in the lumen.

The second layer, or lamina propria, consists of subepithelial connective tissue. The third layer, the muscularis propria, contains smooth muscle. The final layer contains perivesical fat tissue.

Approximately 70–80% of newly diagnosed US bladder cancers are confined to the urothelium or lamina propria (i.e., Ta, Tis, T1; also called superficial or nonmuscle invasive [NMIBC]; Sexton et al., 2010). Remaining diagnoses are classified as muscle invasive (MIBC), where the tumor has invaded the muscularis propria (i.e., T2, T3). Once a bladder tumor begins invading surrounding organs, it becomes T4 (Jacobs, Lee, & Montie, 2010; Konety & Carroll, 2007; Pashos et al., 2002). Most US bladder cancer patients (90%) have transitional cell carcinoma; the remaining have squamous cell carcinomas (5%), adenocarcinomas (1–2%), primary small cell carcinoma, or other tumor histologies (Sexton et al., 2010).

Bladder Cancer Epidemiology and Risk Factors

Bladder cancer incidence, morbidity, and mortality vary by country (Botteman, Pashos, Redaelli, Laskin, & Hauser, 2003). It is the fifth most common cancer in the United States and the second most commonly diagnosed urologic cancer (Altekruse et al., 2010). From 1988 to 2008, the number of US diagnoses increased by more than 50% (Shariat et al., 2009). The United States had an estimated 70,530 new bladder cancer diagnoses and 14,680 deaths in 2010 (Jacobs et al., 2010).

While many cases contain no explicit ties to carcinogenic exposure, bladder cancer has several well-established biological, sociodemographic, and environmental risk factors (Pashos et al., 2002). Men are three to four times more likely to receive a diagnosis than women. While men have higher lifetime risk for developing bladder cancer, women tend to present with later-stage disease and worse prognosis for 5-year survival, even controlling for tumor stage and grade (Jacobs et al., 2010; Pashos et al., 2002; Shariat et al., 2009). Bladder cancer diagnoses among adolescents and young adults remain relatively rare (Sexton et al., 2010). Over three quarters of cases occur in individuals 60 years and over. Race/ethnicity is also important. Caucasian Americans are twice as likely to develop bladder cancer as African Americans. Despite lower incidence, African Americans are diagnosed at advanced-stage disease and have higher mortality rates, even after controlling for tumor characteristics (Konety & Carroll, 2007; Pashos et al., 2002; Sexton et al., 2010).

Environmental risk factors for developing bladder cancer include behavioral risk factors and occupational or chemical exposures (Pashos et al., 2002; Sexton et al., 2010). Less than 10% of individuals diagnosed with bladder cancer report a positive family health history. Smoking is the primary environmental risk factor (Jacobs et al., 2010; Pashos et al., 2002; Sexton et al., 2010). Additional behavioral risk factors include diet/nutrition, specific herbal supplements, chronic urinary tract infection or inflammation, parasitic infection, arsenic-contaminated water, and pelvic radiation. Chemicals linked to increased bladder cancer risk include aniline dyes, aromatic amines, cyclophosphamide, and specific analgesics. At-risk occupations include autoworkers; metalworkers; hairdressers; painters; and paper, leather, dye, and rubber plant workers (Jacobs et al., 2010; Sexton et al., 2010).

Bladder Cancer Symptoms and Detection

Approximately 80–90% of patients diagnosed with bladder cancer present with gross or microscopic amounts of blood in the urine (hematuria; Pashos et al., 2002). As there is a small latent period between bladder cancer development and symptom onset, hematuria is considered the most important symptom (Pashos et al.; Sexton et al., 2010). Twenty percent of patients report other symptoms, including flank pain, painful urination (dysuria), increased urgency or frequency of urination, and inability to urinate (Pashos et al., 2002). Many bladder cancer symptoms, particularly hematuria, are also symptomatic of urinary tract infections, benign prostatic hyperplasia, and other benign conditions. Women may inadvertently be misdiagnosed with gynecological conditions or chronic urinary tract infections in lieu of bladder cancer, contributing to delayed diagnosis (Jacobs et al., 2010).

Physicians suspecting bladder cancer as a potential explanation for these symptoms perform a physical exam and health-history assessment, including smoking history/status and chemical/occupational exposures (Pashos et al., 2002). Clinicians may use intravenous or retrograde pyelography, ultrasound, computed tomography, positron emission tomography, or magnetic resonance imaging to check for urinary tract tumors (Sexton et al., 2010). More commonly, physicians rely on cystoscopy, involving insertion of a camera attached to flexible tubing into the bladder via the urethra while the patient is under local anesthetic (Pashos et al., 2002; Sexton et al., 2010). This procedure is considered the "gold standard" for detecting bladder cancer and allows direct visualization of the urethra and urothelium for tumors (Sexton et al.). Urine cytology, or a bladder wash, is often performed adjunctive to cystoscopy to check for hematuria and bladder cancer cells pretreatment and during posttreatment surveillance (Pashos et al., 2002; Sexton et al., 2010; see below). Early detection of cancer recurrence is linked to reduced morbidity and mortality, although only 40% of bladder cancer survivors are adherent with surveillance (Schrag et al., 2003). Behavioral medicine interventions are warranted in this particular area of cancer control.

Nonmuscle-Invasive Bladder Cancer (NMIBC) Treatment

Transurethral resection of the bladder tumor (TURBT) is a first-line treatment for NMIBC. TURBT may be performed under anesthesia and serves diagnostic, prognostic, and therapeutic functions. Individuals with low risk for progression (i.e., those with low-grade Ta tumors) may be treated using TURBT alone. A repeat TURBT may be performed to restage individuals with high risk for progression (e.g., high-grade T1) within the first month of initial diagnosis (Jacobs et al., 2010; Konety & Carroll, 2007; Sexton et al., 2010). Intravesical chemotherapies such as mitomycin C and immunotherapies such as bacillus Calmette-Guérin (BCG) may be used immediately post-TURBT or as maintenance therapy to treat persistent microscopic tumors, prevent reimplantation or tumor formation, and reduce the chance of stage/grade progression (Jacobs et al., 2010; Konety & Carroll, 2007; Sexton et al., 2010). Common side effects of TURBT include bleeding and infection, whereas intravesical therapies are associated with dysuria, fever, chills, and increased frequency of urination (Pashos et al., 2002; Shariat et al., 2009). BCG intravesical therapy is linked to erectile difficulties; there may also be treatment-related female sexual issues. Patients who have recurrent, high-grade NMIBC unresponsive to intravesical therapy may eventually undergo partial or radical cystectomy (Pashos et al., 2002; Sexton et al., 2010; see below).

Muscle-Invasive Bladder Cancer (MIBC) Treatment

Individuals with MIBC may require more intensive treatment. A "curative" treatment involves radical cystectomy, in which the entire bladder is removed and some adjacent lymph nodes and organs. Male patients may have the prostate and seminal vesicles removed, while women may have their uterus, fallopian tubes, ovaries, and anterior vagina wall removed (Konety & Carroll, 2007). Patients then receive some form of urinary diversion so that they can continue to collect and excrete urine. Options include ileal conduit (i.e., urine is stored in a small portion of intestine and drained through a stoma in the abdomen into an ostomy bag), neobladder (i.e., urine is collected in a section of small intestine connected to the urethra, allowing "normal" urination), and continent cutaneous pouch (i.e., urine is stored in a small portion of the intestine and drained through a stoma via catheter; Jacobs et al., 2010; Konety & Carroll, 2007; Pashos et al., 2002).

Postoperative complication rates and side effects vary by diversion type. Daytime and nighttime incontinence, urinary retention, internal bleeding, infection, wasting syndrome, diarrhea, renal failure, and vitamin deficiencies are some short- and long-term effects (Pashos et al., 2002). Additional side effects include sexual dysfunction and infertility. While cystectomy is considered the gold standard for MIBC treatment, there are bladder-preservation alternatives

for poor surgical candidates due to age, health status, or other factors, or whose beliefs and values preclude surgery. Alternatives include TURBT alone or in combination with external-beam radiation therapy and/or systemic chemotherapy; however, survival rates are generally lower than those from radical cystectomy (Konety & Carroll, 2007).

Bladder Cancer Surveillance

The risk for bladder cancer recurrence is higher than for any other cancer but varies by tumor grade. For example, the 3-year recurrence rates for Ta- and T1-stage tumors are 40–70% and 50–80%, respectively (Schrag et al., 2003). Therefore, surveillance is an important disease-management strategy.

Bladder cancer is also the most expensive cancer in terms of cost per patient per year and lifetime costs per patient. Current estimates place total patient costs at almost $3 billion US dollars per year, of which an estimated 60% goes to monitoring and treatment of recurrence. NMIBC treatment and monitoring represents a substantial portion of these costs (Botteman, Pashos, Redaelli, et al., 2003).

Physicians vary in terms of their surveillance protocols. American Urological Association guidelines recommend intensive follow-up consisting of cystoscopy and cytology every 3 months in years 1 and 2, semiannual cystoscopy and cytology in years 3 and 4, and annual cystoscopy and cytology in years 5–10 or for life (American Urological Association, 2007). Given that cystoscopic examinations are time-consuming and invasive, current adherence rates to bladder surveillance are estimated at about 40% (Schrag et al., 2003). Individuals who are older, non-Caucasian, less-educated, and living in urban geographic locales or low-income areas are significantly more likely to be nonadherent with surveillance (Schrag et al.).

Issues in Bladder Cancer Survivorship

More than 500,000 bladder cancer survivors currently live in the United States (Altekruse et al., 2010), yet little is known about their survivorship needs, particularly those stemming from gender and race/ethnic disparities or psychosocial factors (e.g., fear of recurrence, social constraint and support, psychological distress, and anxiety) (Botteman, Pashos, Hauser, Laskin, & Redaelli, 2003). Given the chronic nature of this disease and related symptoms, bladder cancer survivors may benefit from targeted, culture- and literacy-appropriate patient health education interventions that impact lifestyle/behavior change, symptom management, health-related quality of life, and treatment/surveillance adherence. Limited patient education materials are available from The American Cancer Society (www.cancer.org), Bladder Cancer Advocacy Network (www.bcan.org), and National Cancer Institute (www.cancer.gov). There are few research-tested bladder cancer interventions; however, interventions designed for prostate cancer may be helpful to survivors.

Cross-References

- ▶ American Cancer Society
- ▶ Cancer and Smoking
- ▶ Health Disparities
- ▶ National Cancer Institute
- ▶ Occupational Health
- ▶ Smoking and Health

References and Readings

Altekruse, S. F., Kosary, C. L., Krapcho, M., Neyman, N., Aminou, R., Waldron, W., et al. (Eds.). (2010). *SEER cancer statistics review, 1975–2007*. Retrieved January 17, 2011, from http://seer.cancer.gov/csr/1975_2007

American Urological Association. (2007). *American Urological Association: Guideline for the management of nonmuscle invasive bladder cancer: (Stages Ta, T1, and Tis): 2007 update*. Baltimore: Author.

Botteman, M. F., Pashos, C. L., Hauser, R. S., Laskin, B. L., & Redaelli, A. (2003). Quality of life aspects of bladder cancer: A review of the literature. *Quality of Life Research, 12*, 675–688.

Botteman, M. F., Pashos, C. L., Redaelli, A., Laskin, B. L., & Hauser, R. S. (2003). The health economics of bladder cancer: A comprehensive review of the published literature. *Pharmacoeconomics, 21*, 1315–1330.

Jacobs, B. L., Lee, C. T., & Montie, J. E. (2010). Bladder cancer in 2010: How far have we come? *CA: Cancer Journal for Clinicians, 60*, 244–272.
Konety, B. R., & Carroll, P. R. (2007). Urothelial carcinoma: Cancers of the bladder, ureter, & renal pelvis. In E. A. Tanagho & J. W. McAninch (Eds.), *Smith's general urology* (17th ed.). New York: McGraw-Hill Professional.
Pashos, C. L., Botteman, M. F., Laskin, B. L., & Redaelli, A. (2002). Bladder cancer epidemiology, diagnosis, and management. *Cancer Practice, 10*(6), 311–322.
Schrag, D., Hsieh, L. J., Rabbani, F., Bach, P. B., Herr, H., & Begg, C. (2003). Adherence to surveillance among patients with superficial bladder cancer. *Journal of the National Cancer Institute, 95*(8), 588–597.
Sexton, W. J., Wiegand, L. R., Correa, J. J., Politis, C., Dickinson, S. I., & Kang, L. C. (2010). Bladder cancer: A review of non-muscle invasive disease. *Cancer Control, 17*(4), 256–268.
Shariat, S. F., Sfakianos, J. P., Droller, M. J., Karakiewicz, P. I., Meryn, S., & Bochner, B. H. (2009). The effect of age and gender on bladder cancer: A critical review of the literature. *British Journal of Urology International, 105*, 300–308.

Cancer, Cervical

Deidre Pereira[1] and Stephanie L. Garey[2]
[1]Department of Clinical and Health Psychology, College of Clinical Health and Health Professions, University of Florida, Gainesville, FL, USA
[2]Department of Clinical and Health Psychology, College of Public Health and Health Professions, University of Florida, Gainesville, FL, USA

Synonyms

Cancer of the uterine cervix; Cervical adenocarcinoma; Invasive cervical cancer; Squamous cell carcinoma of the cervix (SCCC)

Definition

Cervical cancer is a slow-growing cancer that develops in the lower portion of the uterus, known as the cervix. Approximately 1 in every 145 women will develop invasive cervical cancer in her lifetime. In 2010, an estimated 12,200 women were diagnosed with cervical cancer (ACS, 2010). Significant racial/ethnic disparities exist in cervical cancer incidence rates, with African American and Hispanic/Latino women having elevated rates compared to White, Asian American/Pacific Islander, and American Indian/Alaska Native women (NCI, 2011).

Cervical cancer typically begins as a precancerous condition, known as cervical intraepithelial neoplasia (CIN; (ACS, 2011)). Patients who do not undergo regular pelvic exams and Pap tests are likely to develop one of the two main types of cervical cancer: squamous cell cervical carcinoma or cervical adenocarcinoma. The majority of cases, approximately 80–90%, are squamous cell carcinomas of the cervix (ACS). Persistent infection with human papillomaviruses (HPVs) has been identified as the cause of cervical cancer in the majority of cases. There are over 150 types of HPV, including 40 types that are transmitted sexually (NCI). HPV types 16 and 18 are considered to be carcinogenic (cancer-causing) to humans and have been classified as Group 1 carcinogens by the International Agency for Research on Cancer/World Health Organization. Accordingly, cervical infection with HPV types 16 and 18 confers high risk for the transformation of CIN to cervical cancer and causes over 70% of all cervical cancers (ACS). In contrast, HPV types 6 and 11 have been classified as possibly carcinogenic to humans (Class 2B carcinogens) and are mostly implicated in the development of anogenital condylomata (genital warts; (ACS)). Women who are sexually active at a young age or have many sexually partners are at a greater risk for HPV infection. As of 2011, the Federal Drug Administration (FDA) has approved the use of two vaccines for the prevention of the most common types of HPV infection (ACS; NCI). One vaccine prevents four HPV types (6, 11, 16, and 18) and is indicated for use in females and males 9–26 years of age. A second vaccine protects against two HPV types (16 and 18) and is indicated for use only in females 10–25 years of age (NCI). Additional risk factors for cervical cancer include smoking cigarettes and exposure to secondhand smoke, a high number of full-term pregnancies, long-term use of oral contraceptives, and immunosuppression (ACS).

Screening for cervical cancer includes regular pelvic exams and a Papanicolaou (Pap) test to screen for precancerous or malignant changes in cervical cells (ACS). If the Pap test detects abnormal cells, an HPV DNA test is conducted to determine whether HPV infection is present (NCI). Furthermore, a colposcopic examination can be performed to identify abnormal areas on the cervix visually and allow for a biopsy of cervical tissue (ACS). If invasive cancer is identified, additional imaging studies may be conducted to determine if the cancer has metastasized (spread) to distant organs and facilitate staging. Women who have early-stage cervical cancer often do not experience any symptoms; however, once the cancer has progressed and spread to proximal tissues, women may experience abnormal vaginal bleeding, unusual discharge, and pain during intercourse (ACS).

Treatment of cervical cancer can include surgery, chemotherapy, and radiation therapy. As with all cancers, the appropriate treatment choice depends largely on the stage of disease. Surgical methods for treating cervical cancer include cryosurgery, laser surgery, conization, hysterectomy, and trachelectomy (NCI). Pelvic exenteration or lymph node dissection may be performed when there has been spread or recurrence of cervical cancer (ACS; NCI). The most common types of chemotherapy that target cervical cancer include cisplatin, paclitaxel, topotecan, ifosfamide, and 5-fluorouracil (5-FU; ACS)). Six to seven weeks of radiation treatment may also be used to treat cervical cancer (ACS). Certain stages of cervical cancer may require a combination of chemotherapy and radiation, known as chemoradiation.

The 5-year survival rate of cervical cancer is 71% (ACS). While the number of deaths due to cervical cancer has decreased across the last several decades, mortality rates have remained steady since approximately 2003. African American women have the highest cervical cancer mortality rates compared to all other racial/ethnic groups, which may be partly due to the fact that African American women are less likely to be diagnosed with early-stage cervical cancer than White women (ACS).

Cross-References

- ▶ Cancer Prevention
- ▶ Cancer Survivorship
- ▶ Cancer, Types of
- ▶ Cancer: Psychosocial Treatment
- ▶ Reproductive Health
- ▶ Women's Health

References and Readings

American Cancer Society. (2010). *Cancer facts & figures 2010*. Atlanta, GA: Author.

American Cancer Society. (2011). Cervical cancer. In *Learn about cancer*. Retrieved February 27, 2011, from http://www.cancer.org/cancer/cervicalcancer/index

National Cancer Institute. (2011). Cervical cancer. In *Cancer topics*. Retrieved February 27, 2011, from http://www.cancer.gov/cancertopics/types/cervical

Cancer, Colorectal

Hiromichi Matsuoka
Department of Psychocomatic Medicine, Kinki University Faculty of Medicine, Osakasayama, Osaka, Japan

Synonyms

Colorectal cancer

Definition

Colorectal cancer (CRC) is the third most frequently diagnosed cancer in men and women in the United States. Patients with localized colon cancer have a 90% five-year survival rate (Jemal et al., 2009).

Colorectal cancer mortality can be reduced by early diagnosis and by cancer prevention through polypectomy. Therefore, the goal of CRC screening (CRCS) is to detect cancer at an early, curable stage as well as to detect and remove clinically significant adenomas (Levin et al., 2008).

Screening tests that can detect both early cancer and adenomatous polyps are encouraged. Current technology falls into two broad categories: structural tests and stool/fecal-based tests. Regular screening with the fecal occult blood test (FOBT) or sigmoidoscopy facilitates earlier detection of CRC and lowers mortality. Screening colonoscopy may decrease CRC incidence through early detection and removal of precancerous polyps. Reported interventions to promote the FOBT have included patient reminders through use of personal media, approaches that reduce structural barriers such as mailing of FOBT kits, and use of provider assessment and feedback (Baron et al., 2008; Hardcastle et al., 1996; Kronborg, Fenger, Olsen, Jorgensen, & Sondergaard, 1996; Mandel et al., 1993; Selby, Friedman, Quesenberry, & Weiss, 1992; Shapiro et al., 2008; Winawer et al., 1993).

Other preventive health behaviors are positively associated with CRCS, including a recent mammogram or Pap test for women, a recent prostate-specific antigen (PSA) test for men, a cholesterol test, dental visit, seat belt use, fruit and vegetable consumption, and physical activity (See ff et al., 2004).

CRC is predominantly a disease of Westernized countries, indicating that components of the Western lifestyle may contribute to the risk. A large body of evidence has implicated modifiable lifestyle factors as causes of colorectal cancer, including smoking, lack of physical activity, body composition, alcohol intake, and diet (Shapiro, See ff, & Nadel, 2001).

Aspirin taken for several years at doses of at least 75 mg daily reduced long-term incidence and mortality due to colorectal cancer. Benefit was greatest for cancers of the proximal colon, which are not otherwise prevented effectively by screening with sigmoidoscopy or colonoscopy (Rothwell et al., 2010).

Type C has emerged as a behavioral pattern, coping style, or personality type that predisposes people to or is a risk factor for the onset and progression of cancer. Type C has been described as a personality that is overcooperative, stoical, unassertive, patient, avoiding conflict, compliant with external authorities, unexpressive through suppression or denial of negative emotions, self-sacrificing, and predisposed to experiencing hopelessness and depression. There is evidence of connections among personality, stress and cancer, as well as among personality, stress, and the autonomic, endocrinological, and immune systems. These psychological characteristics can be considered as cancer risk factors. Nevertheless, a type C or cancer-prone personality should be understood in terms of its synergic interactions with genetic, biological, and environmental factors (Eysenck, 1994).

Significant barriers to advanced cancer patients receiving mental health treatment for distress have been reported in the literature. Monthly monitoring of distress in older patients using telephone monitoring and educational materials, along with referral for appropriate help, has been found to be efficient means of reducing anxiety and depression, compared with patients who received only educational materials (Kornblith et al., 2006).

Acupuncture, transcutaneous electrical nerve stimulation, supportive group therapy, self-hypnosis, and massage therapy may provide cancer pain relief in dying patients (Pan, Morrison, Ness, Fugh-Berman, & Leipzig, 2000).

Cross-References

- ▶ Aspirin
- ▶ Colorectal Cancer
- ▶ Lifestyle

References and Readings

Baron, R. C., Rimer, B. K., Breslow, R. A., et al. (2008). Client-directed interventions to increase community demand for breast, cervical, and colorectal cancer screening. *American Journal of Preventive Medicine, 35*(1S), S34–S55.

Eysenck, H. J. (1994). Personality, stress and cancer prediction and prophylaxis. *Advances in Behavior Research and Therapy, 16*, 167–215.

Hardcastle, J. D., Chamberlain, J. O., Robinson, M. H. E., et al. (1996). Randomized controlled trial of fecal-occult-blood screening for colorectal cancer. *Lancet, 348*(9040), 1472–1477.

Jemal, A., Siegel, R., Ward, E., Hao, Y., Xu, J., & Thun, M. J. (2009). Cancer statistics, 2009. *CA: A Cancer Journal for Clinicians, 59*, 225–249.

Kornblith, A. B., Dowell, J. M., Herndon, J. E., 2nd, Engelman, B. J., Bauer-Wu, S., Small, E. J., et al. (2006). Telephone monitoring of distress in patients aged 65 years or older with advanced stage cancer: A cancer and leukemia group B study. *Cancer, 107*(11), 2706–2714.

Kronborg, O., Fenger, C., Olsen, J., Jorgensen, O. D., & Sondergaard, O. (1996). Randomized study of screening for colorectal cancer with fecal-occult-blood test. *Lancet, 348*, 1467–1471.

Levin, B., Lieberman, D. A., McFarland, B., et al. (2008). Screening and surveillance for the early detection of colorectal cancer and adenomatous polyps, 2008: A joint guideline from the American Cancer Society, the US Multi-Society Task Force on Colorectal Cancer, and the American College of Radiology. *CA: A Cancer Journal for Clinicians, 58*, 130–160.

Mandel, J. S., Bond, J. H., Church, T. R., et al. (1993). Reducing mortality from colorectal cancer by screening for fecal occult blood. Minnesota Colon Cancer Control Study. *The New England Journal of Medicine, 328*(19), 1365–1371.

Pan, C. X., Morrison, R. S., Ness, J., Fugh-Berman, A., & Leipzig, R. M. (2000). Complementary and alternative medicine in the management of pain, dyspnea, and nausea and vomiting near the end of life. A systematic review. *Journal of Pain and Symptom Management, 20*(5), 374–387.

Rothwell, P. M., Wilson, M., Elwin, C. E., Norrving, B., Algra, A., Warlow, C. P., et al. (2010). Long-term effect of aspirin on colorectal cancer incidence and mortality: 20-year follow-up of five randomised trials. *Lancet, 376*(9754), 1741–1750. Epub 2010 Oct 21.

See ff, L. C., Nadel, M. R., Klabunde, C. N., et al. (2004). Patterns and predictors of colorectal cancer test use in the adult US population: Results from the 2000 National Health Interview Survey. *Cancer, 100*(10), 2093–2103.

Selby, J. V., Friedman, G. D., Quesenberry, C. P., Jr., & Weiss, N. S. (1992). A case–control study of screening sigmoidoscopy and mortality from colorectal cancer. *The New England Journal of Medicine, 326*(10), 653–657.

Shapiro, J. A., See ff, L. C., & Nadel, M. R. (2001). Colorectal cancer-screening tests and associated health behaviors. *American Journal of Preventive Medicine, 21*(2), 132–137.

Shapiro, J. A., See ff, L. C., Thompson, T. D., Nadel, M. R., Klabunde, C. N., & Vernon, S. W. (2008). Colorectal cancer test use from the 2005 National Health Interview Survey. *Cancer Epidemiology, Biomarkers & Prevention, 17*(7), 1623–1630.

Winawer, S. J., Zauber, A. G., Ho, M. N., The National Polyp Study Workgroup, et al. (1993). Prevention of colorectal cancer by colonoscopic polypectomy. *The New England Journal of Medicine, 329*, 1977–1981.

Cancer, Lymphatic

Hiroe Kikuchi
Department of Psychosomatic Research, National Institute of Mental Health, National Center of Neurology and Psychiatry, Tokyo, Japan

Synonyms

Lymphoma

Definition

Lymphatic cancer is a cancer of the lymphatic system, which is a part of the immune system. It is also called lymphoma. Although lymphoma commonly affects lymph node, it also affects other organs such as spleen.

The new World Health Organization classification (Swerdlow et al., 2008) is usually used to classify lymphoma, and lymphoma is divided into Hodgkin lymphoma and non-Hodgkin lymphoma (B cell and T/NK cell). Staging is basically based on distribution of the lesions. Non-Hodgkin lymphoma is clinically classified into indolent, aggressive, and highly aggressive.

Symptoms and signs of lymphoma are lymphadenopathy which is without tenderness and mobile, fever, fatigue, nocturnal sweating, weight loss, bloating sensation, etc.

In Hodgkin lymphoma, radiotherapy with or without chemotherapy is used for a limited stage and chemotherapy is the first choice for an advanced stage. High-dose chemotherapy with autologous hematopoietic stem cell transplantation is considered for recurrent or refractory cases.

In non-Hodgkin lymphoma, treatment is selected depending on the pathological classification and the grade of malignancy. In B cell indolent lymphoma, radiotherapy or surgical therapy is used for a limited stage and chemotherapy or careful follow-up without any therapy is selected for an advanced stage. Monoclonal

antibody called rituximab is also used. In aggressive B cell lymphoma, chemotherapy combined with rituximab is generally used and radiotherapy is combined for a limited stage. High-dose chemotherapy with autologous peripheral blood stem cell transplantation is conducted at first remission in a high-risk group. In highly aggressive B cell lymphoma, treatment which is used for acute lymphoblastic leukemia is applied. Although no standard therapy is established for T cell lymphoma, chemotherapy is often applied. Eradication of *Helicobacter pylori* is used for gastric mucosa-associated lymphoid tissue (MALT) lymphoma.

Cross-References

► Chemotherapy

References and Readings

Swerdlow, S. H., Campo, E., Harris, N. L., Jaffe, E. S., Pileri, S. A., Stein, J., et al. (Eds.). (2008). *WHO classification of tumours of haematopoietic and lymphoid tissues*. Lyon: IARC.

Cancer, Ovarian

Ashley Nelson[1] and Erin Costanzo[2]
[1]Department of Psychiatry, School of Medicine and Public Health, University of Wisconsin-Madison, Madison, WI, USA
[2]Department of Psychiatry, Carbone Cancer Center, University of Wisconsin-Madison, Madison, WI, USA

Synonyms

Ovarian carcinoma; Ovarian neoplasm

Definition

Ovarian cancer is a malignancy of the tissue of the ovary and most commonly consists of either an epithelial cell tumor, arising from cells on the surface of the ovary, or a germ cell tumor, in which the cancer forms in the egg cells. Approximately 22,000 new cases of ovarian cancer were reported in the United States in 2010, and ovarian cancer ranks as the fifth most common cause of death among women with cancer (American Cancer Society [ACS], 2010). Because ovarian cancer is difficult to detect during the early stages, women are often diagnosed with advanced disease that has spread to the lymph nodes or metastasized to other organs. The overall 5-year survival rate for women diagnosed with ovarian cancer is 46% (ACS, 2010). The course of treatment varies by tumor type and stage but may include chemotherapy, radiation therapy, and/or surgery to remove the tumor and/or one or both ovaries (oophorectomy).

Description

A diagnosis of ovarian cancer and its corresponding treatment has been associated with significant decrements in emotional, physical, and functional quality of life (Arriba, Fader, Frasure, & von Gruenigen, 2010). Women with ovarian cancer often report pervasive fatigue, disrupted sleep, and limitations in their ability to be active. Moreover, younger women typically experience loss of fertility, and many survivors report sexual concerns that persist well beyond the period of physical recovery. Not surprisingly, emotional distress is common, including symptoms of depression and anxiety. Distress can persist well after treatment ends, with survivors commonly reporting fear of a cancer recurrence and significant anxiety around follow-up clinic visits and diagnostic tests (Arriba et al., 2010).

Research is also beginning to delineate biobehavioral mechanisms by which behavioral factors may alter physiological pathways associated with ovarian tumor growth and development, including effects on immunosuppression, inflammation, and angiogenesis (growth of new blood vessels to nourish the tumor) (Costanzo, Sood, & Lutgendorf, 2011). For example, ovarian cancer patients who report greater distress and have

more limited social support show poorer NK cell activity in peripheral blood and tumor infiltrating lymphocytes as compared to women with lower distress and better support (Lutgendorf et al., 2005). Greater depressive symptoms and less social support have also been associated with elevated levels of pro-angiogenic cytokines including interleukin-6 and vascular endothelial growth factor (VEGF) (Costanzo et al., 2011). Moreover, ovarian cancer patients who report more depressive symptoms show higher and less variable levels of nocturnal cortisol (Lutgendorf et al., 2008). Stress hormones, including norepinephrine and epinephrine, have been shown to increase VEGF production and the in vitro invasiveness of ovarian tumor cells (Lutgendorf et al., 2003; Sood et al., 2006). These findings suggest that behavioral factors may play an important role not only in quality of life, but also in ovarian cancer outcomes.

Cross-References

- ▶ Cortisol
- ▶ Epinephrine
- ▶ Natural Killer Cell Activity
- ▶ Norepinephrine/Noradrenaline
- ▶ Vascular Endothelial Growth Factor (VEGF)

References and Readings

American Cancer Society. (2010). *Cancer facts & figures 2010*. Atlanta, GA: Author.

Arriba, L. N., Fader, A. N., Frasure, H. E., & von Gruenigen, V. E. (2010). A review of issues surrounding quality of life among women with ovarian cancer. *Gynecologic Oncology, 119*, 390–396.

Costanzo, E. S., Sood, A. K., & Lutgendorf, S. K. (2011). Biobehavioral influences on cancer progression. *Immunology and Allergy Clinics of North America, 31*, 109–132.

Lutgendorf, S. K., Cole, S., Costanzo, E., Bradley, S., Coffin, J., Jabbari, S., et al. (2003). Stress-related mediators stimulate vascular endothelial growth factor secretion by two ovarian cancer cell lines. *Clinical Cancer Research, 9*, 4514–4521.

Lutgendorf, S. K., Sood, A. K., Anderson, B., McGinn, S., Maiseri, H., Dao, M., et al. (2005). Social support, psychological distress, and natural killer cell activity in ovarian cancer. *Journal of Clinical Oncology, 23*, 7105–7113.

Lutgendorf, S. K., Weinrib, A. Z., Penedo, F., Russell, D., DeGeest, K., Costanzo, E. S., et al. (2008). Interleukin-6, cortisol, and depressive symptoms in ovarian cancer patients. *Journal of Clinical Oncology, 26*, 4820–4827.

Sood, A. K., Bhatty, R., Kamat, A. A., Landen, C. N., Han, L., Thaker, P. H., et al. (2006). Stress hormone-mediated invasion of ovarian cancer cells. *Clinical Cancer Research, 12*, 369–375.

Cancer, Prostate

Marc A. Kowalkouski[1], Heather Honoré Goltz[1,2], Stacey L. Hart[3] and David Latini[4]
[1]HSR&D Center of Excellence, Michael E. DeBakey VA Medical Center (MEDVAMC 152), Houston, TX, USA
[2]Department of Social Sciences, University of Houston-Downtown, Houston, TX, USA
[3]Department of Psychology, Ryerson University, Toronto, ON, Canada
[4]Scott Department of Urology, Baylor College of Medicine, Houston, TX, USA

Synonyms

Carcinoma of the prostate; Neoplasm of the prostate; Prostatic adenocarcinoma

Definition

Prostate cancer originates in the prostate, a walnut-shaped gland in the male reproductive system. Fluid secreted by the prostate nourishes and transports sperm. Over 95% of cancers of the prostate are adenocarcinomas, originating in glandular tissue. The prostate is made up of three distinct zones of glandular tissue. Approximately 70% of cancers develop in the peripheral zone, 10–20% in the transition zone, and 5–10% in the central zone. Most prostate cancers are slow growing, and survival rates are high, particularly for men with localized disease.

Description

Prostate Cancer Epidemiology

Aside from skin cancer, prostate cancer is the most common malignancy and the second most common cause of cancer death among men in the United States. Roughly 1 in 6 men will be diagnosed with prostate cancer in their lifetime. In 2010, an estimated 220,000 incident cases of prostate cancer were diagnosed in America, mostly among men over age 70. Additionally, more than 30,000 prostate cancer deaths were projected, second only to lung cancer for cancer deaths among American men.

The etiology of prostate cancer is not well understood. However, the male sex hormone testosterone, particularly at high levels, can accelerate the reproduction and growth of existing cancer cells in the prostate. Increasing age is the most important risk factor for prostate cancer. A positive family history is also associated with increased risk. Additionally, African American men have higher incidence and mortality rates than Whites. Since 1975, incidence rates in the United States have fluctuated, slightly decreasing since 2000. Substantial changes can be traced to the introduction of the prostate-specific antigen (PSA) screening test in the 1980s.

Prostate Cancer Screening

Screening for prostate cancer includes serum PSA testing and digital rectal examination. Due to the widespread implementation of PSA testing, over 90% of prostate cancers are detected at early stages, when the disease is localized to the prostate and easiest to treat. However, PSA testing is not without controversy, and guidelines for screening differ. Please see *Prostate-Specific Antigen* for additional information.

Prostate Cancer Diagnosis

Prostate cancer may be suspected if the PSA level is greater than 4 ng per mL. When prostate cancer is suspected, a core needle biopsy is performed for tissue analysis. If the tissue biopsy confirms the presence of cancer, further testing (e.g., computed tomography or magnetic resonance imaging) may be completed to determine whether the cancer has spread to other parts of the body. A Gleason score (range: 2–10) is also calculated to evaluate the growth rate of the cancer, dependent on the appearance of tumor cells under microscope. A high Gleason score indicates advanced disease. Together, tumor stage and Gleason score are used to determine prognosis and to direct treatment decisions. Today, nearly all prostate cancers are detected when tumors are confined to the prostate (i.e., Gleason score 6 or 7).

Prostate Cancer Treatment

Many treatment options are available to men diagnosed with localized prostate cancer (e.g., active surveillance, radiotherapy, and surgery). However, there is currently no consensus regarding optimal treatment. Each treatment impacts quality of life differently – ranging from urinary, sexual, and bowel dysfunction to more systemic concerns, such as weight gain, bone loss, hot flashes, and depression. Therefore, individuals must make decisions based upon their own personal preferences, clinical characteristics, and a variety of external factors, including provider recommendations. Most men experience decreased sexual potency, regardless of treatment. However, men undergoing radical prostatectomy suffer most from urinary problems, while radiotherapy is associated with poor bowel function. The physical side effects associated with prostate cancer treatment can severely affect a man's quality of life. Additionally, the emotional and psychological distress associated with symptoms, as well as complications in spouse or partner relationships, can further diminish quality of life. Given the slow-growing nature of most prostate cancers, some individuals may consider deferring treatment to maintain better quality of life. Active surveillance involves routine monitoring of patients diagnosed with early-stage, low-grade prostate cancer, in lieu of definitive treatment. However, this option carries its own burden, primarily the uncertainty and anxiety associated with having an "untreated" cancer. For men with advanced stages of disease, additional treatment options are available (e.g., hormonal therapy, chemotherapy).

Current Medical Research and Interventions in Prostate Cancer

A major concentration in current prostate cancer research focuses on the evaluation of potential factors affecting the observed racial disparity. Several projects are attempting to identify genetic and other variants that may increase incidence and mortality in African American men. Furthermore, there is an emphasis on identifying additional biomarkers to improve detection and prognostic accuracy. Studies are also being conducted to compare the effectiveness of active surveillance for disease management with immediate treatment. Finally, for men with advanced hormone-refractory disease, improved chemotherapy regimens are being evaluated (e.g., docetaxel and cabazitaxel).

Current Behavioral Medicine Research and Interventions in Prostate Cancer

Given the array of treatment options available to men and the lack of consensus concerning best practices, decision-making tools are essential to assist in determining which treatment option is most in congruence with their values and lifestyle preferences. In a review of treatment decision-aid studies, aids were found to decrease distress, increase knowledge, and support shared decision making.

Additionally, only a limited number of evidence-based behavioral medicine interventions have been developed to address quality-of-life concerns in this population. The first of these interventions was developed from work done in breast cancer and shown to be effective in improving quality of life. Unfortunately, the results have been more mixed in interventions focusing on improving psychosocial distress. Reductions in distress have generally been modest and of short duration. However, other research suggests that men adjust to changes in functional status and symptom distress improves over time.

Developing these materials and programs should be an immediate priority for behavioral medicine researchers. Promising results have been shown in adapting cognitive behavioral stress-management programs, peer-support, nurse-led, and telephone-based interventions. Less work has been done with subgroups among prostate cancer patients and survivors. Only one intervention has been developed to provide psychosocial support for men on active surveillance. Little data exist on gay and bisexual men with prostate cancer. No interventions have focused on the particular needs of single men, for whom treating erectile dysfunction related to prostate cancer may be particularly challenging.

Cross-References

► Prostate-Specific Antigen (PSA)

References and Readings

American Cancer Society. (2010). *Cancer facts and figures 2010*. Atlanta, GA: Author.

Bailey, D. E., Jr., Wallace, M., & Mishel, M. H. (2007). Watching, waiting and uncertainty in prostate cancer. *Journal of Clinical Nursing, 16*, 734–41.

Eton, D. T., & Lepore, S. J. (2002). Prostate cancer and HRQOL: A review of the literature. *Psychooncology, 11*, 307–26.

Gore, J. L., Gollapudi, K., Bergman, J., Kwan, L., Krupski, T. L., & Litwin, M. S. (2010). Correlates of bother following treatment for clinically localized prostate cancer. *Journal of Urology, 184*, 1309–15.

Green, G. L., Sands, L. P., Latini, D. M., Kaniu, P., Barker, J. C., Chren, M. M., et al. (2009). Values insight and balance scales (VIBES-PC): Psychometric characteristics in the prostate cancer clinical setting. *Annals of Behavioral Medicine, 37*, S37.

Knight, S. J., & Latini, D. M. (2009). Sexual side effects and prostate cancer treatment decisions: Patient information needs and preferences. *Cancer Journal, 15*, 41–4.

Latini, D. M., Elkin, E., Cooperberg, M. R., Sadetsky, N., DuChane, J., Carroll, P. R., et al. (2006). Differences in clinical characteristics and disease-free survival for Latino, African-American, and non-Latino white men with localized prostate cancer: Data from CaPSURE™. *Cancer, 106*, 789–795.

Latini, D. M., Hart, S. L., Coon, D. W., & Knight, S. J. (2010). Sexual rehabilitation after prostate cancer: Current interventions and future directions. In V. T. DeVita, T. S. Lawrence, & S. A. Rosenberg (Eds.), *Cancer: Principles & practice of oncology – Advances in oncology* (Vol. 1, pp. 22–28). Philadelphia: Lippincott Williams & Wilkins.

Lin, G. A., Aaronson, D. S., Knight, S. J., Carroll, P. R., & Dudley, R. A. (2009). Patient decision aids for prostate cancer treatment: A systematic review of the literature. *CA: A Cancer Journal for Clinicians, 59*, 379–90.

Litwin, M. S., Hays, R. D., Fink, A., Ganz, P. A., Leake, B., Leach, G. E., et al. (1995). QoL outcomes in men

treated for localized prostate cancer. *Journal of the American Medical Association, 273*, 129–135.
Rottman, N., Dalton, S. O., Bidstrup, P. E., Würtzen, H., Hoybye, M. T., Ross, L., et al. (2011). No improvement in distress and quality of life following psychosocial cancer rehabilitation. A randomised trial. *Psychooncology*, doi: 10.1002/pon.192. Accessed 8 Feb 2011 [Epub ahead of print]
Tanagho, E. A., & McAninch, J. W. (Eds.). (2008). *Smith's general urology* (17th ed.). New York: McGraw-Hill.

Cancer, Testicular

Catherine Benedict
Department of Psychology, University of Miami, Coral Gables, FL, USA

Synonyms

Nonseminoma; Seminoma; Testicular neoplasms

Definition

Testicular cancer is a type of cancer that forms in the tissue of one or both testicles, the male reproductive glands located in the scrotum. There are several different types of testicular cancer but most cases originate in germ cells (cells that make sperm) and are called testicular germ cell tumors. Testicular germ cell tumors may be further categorized into seminomas and nonseminomas. Seminoma tumors are a slower growing and less aggressive form of testicular cancer. They are usually isolated to the testicle or testes and are particularly sensitive to radiation treatment. Nonseminoma tumors are faster growing and more aggressive. This form of testicular cancer tends to occur in younger men.

Description

Testicular cancer is not common and accounts for only 1% of all cancers in men. There are an estimated 8,290 new diagnoses and about 350 deaths due to testicular cancer each year. It is most common in young and middle-aged men such that about 9 out of 10 testicular cancers occur in men between the ages of 20 and 54. Treatment is very successful and the risk of dying from testicular cancer is low.

Factors that may increase the risk for developing testicular cancer include abnormal testicle development, such as Klinefelter's syndrome, undescended testicle (cryptorchidism), personal or family history of testicular cancer, age, and ethnicity. Non-Hispanic white men are more likely than African-American and Asian-American men to develop testicular cancer. The risk of Hispanic/Latino men developing this type of cancer is between that of Asians and non-Hispanic whites.

Signs and symptoms of testicular cancer include a lump or enlargement in either testicle; a feeling of heaviness in the scrotum; a sudden collection of fluid in the scrotum; pain or discomfort in a testicle, scrotum, abdomen, or lower back; and enlargement or tenderness of the breasts. However, many men do not experience symptoms, even when the cancer has spread to other organs.

Diagnosis and Treatment

Initial diagnosis generally involves an ultrasound or biopsy. To determine whether the cancer has spread outside of the testicle, a computerized tomography (CT) scan to look for signs of cancer in the abdominal lymph nodes or blood tests to look for elevated tumor markers may be used. The staging of the cancer will depend on the results of these tests. There are three stages of testicular cancer: Stage I cancer is limited to the testicle (localized); Stage II cancer has spread to the lymph nodes in the abdomen (regional); and Stage III cancer has spread to other parts of the body and most commonly includes the lungs, liver, bones, and/or brain (distant).

Cross-References

► American Cancer Society

References and Readings

Chung, P., Mayhew, L. A., Warde, P., Winquist, E., & Lukka, H. (2010). Management of stage I seminomatous testicular cancer: A systematic review. *Clinical Oncology, 22*(1), 6–16.

Feldman, D. R., Bosl, G. J., Sheinfeld, J., & Motzer, R. J. (2008). Medical treatment of advanced testicular cancer. *Journal of the American Medical Association, 299*(6), 672–684.

Glendenning, J. L., Barbachano, Y., Norman, A. R., Dearnaley, D. P., Horwich, A., & Huddart, R. A. (2010). Long-term nerologic and peripheral vascular toxicity after chemotherapy treatment for testicular cancer. *Cancer, 116*(10), 2322–2331.

Howlader, N., Noone, A. M., Krapcho, M., Neyman, N., Aminou, R., Waldron, W., et al. (Eds.). (2011). *SEER cancer statistics review, 1975–2008*. Bethesda, MD: National Cancer Institute. Retrieved from http://seer.cancer.gov/csr/1975_2008/, based on November 2010 SEER data submission, posted to the SEER web site, 2011.

Huyghe, E. (2008). Testicular cancer. In: Editor-in-Chief: K. Heggenhougen, Editor(s)-in-Chief, *International encyclopedia of public health* (pp. 309–318). Oxford: Academic Press.

van den Belt-Dusebout, A. W., de Wit, R., Gietema, J. A., Horenblas, S., Louwman, M. W., Ribot, J. G., Hoekstra, H. J., Ouwens, G. M., Aleman, B. M., & van Leeuwen, F. E. (2007). Treatment-specific risks of second malignancies and cardiovascular disease in 5-year survivors of testicular cancer. *Journal of Clinical Oncology, 25*(28), 4370–4378.

Cancer, Types of

Yoshinobu Matsuda
National Hospital Organization, Kinki-Chuo Chest Medical Center, Sakai shi, Osaka, Japan

Synonyms

Cancer types

Definition

Cancer is a term used for a disease in which abnormal cells divide uncontrollably and invade other tissues.

Cancer includes many forms of disease, and there are more than 100 different types of cancer. Most cancers are named for the organ or type of cell from which they originate. Cancer types can also be grouped into broader categories.

The main categories of cancer include:

Carcinoma – cancer that begins in the skin or in tissue that lines or covers internal organs

Sarcoma – cancer that arises from bone, cartilage, fat, muscle, blood vessels, or other connective or supportive tissue

Leukemia – cancer that starts in blood-forming tissue such as the bone marrow

Lymphoma and myeloma – cancers that begin in the cells of the immune system

Central nervous system cancers – cancers that begin in the tissue of the brain or spinal cord

The cancers that are diagnosed with the greatest frequency in the United States are bladder cancer, breast cancer, colorectal cancer, endometrial cancer, kidney cancer, leukemia, lung cancer, melanoma, non-Hodgkin lymphoma, pancreatic cancer, prostate cancer, and thyroid cancer.

Some behavioral factors have been reported to have an association with the incidences of cancer, cancer screening, cancer recurrence, and cancer mortality. For example, cigarette smoking contributes significantly to mortality rates for lung cancer, oral cancer, and cancers of the esophagus, larynx, bladder, stomach, pancreas, kidney, and cervix.

Each type of cancer has characteristic-associated behavioral factors (see the section on a particular cancer for more information).

Cross-References

▶ Breast Cancer
▶ Cancer, Bladder
▶ Cancer, Cervical
▶ Cancer, Colorectal
▶ Cancer, Lymphatic
▶ Cancer, Ovarian
▶ Cancer, Prostate
▶ Cancer, Testicular
▶ Carcinoma
▶ Kaposi's Sarcoma

References and Readings

Holland, J. C. (2009). *Psycho-oncology* (2nd ed.). New York: Oxford University Press.
National Cancer Institute. www.cancer.gov

Cancer: Psychosocial Treatment

Frank J. Penedo[1], Catherine Benedict[2] and Bonnie McGregor[3]
[1]Department of Medical Social Sciences & Lurie Comprehensive Cancer Center, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA
[2]Department of Psychology, University of Miami, Coral Gables, FL, USA
[3]Fred Hutchinson Cancer Research Center, Seattle, WA, USA

Synonyms

Behavioral oncology; Psycho-oncology; Psychosocial intervention; Psychosocial oncology

Definition

Psychosocial treatment in oncology covers a broad range of effective therapies that have yet to become the standard of care for most cancer patients. Psychosocial therapies help cancer patients and their families emotionally adjust to diagnosis and treatment, cope with treatment-related side effects (e.g., fatigue, pain, nausea), improve adherence to chemotherapy regimens, and improve health behaviors. Therapies can include cognitive behavioral therapy, hypnosis and guided imagery, mindfulness-based therapies, cognitive behavioral stress management, couple- and family-based therapy, play therapy for children, and motivational interviewing for behavior change. Psychosocial therapy is typically administered by clinical psychologists, psychiatrists, social workers, nurses, and more recently, via web-based interventions and telephone counseling. This therapy can be administered in both individual and group settings at multiple points along the cancer continuum, from before diagnosis among people at elevated risk for cancer, to many years after active treatment has completed. Psychosocial therapies have been shown in numerous studies to improve not only psychological (e.g., reduce distress) and quality of life outcomes, but also physical outcomes (e.g., improve immune function and physical functioning) among cancer survivors in need of therapy.

Description

Natural History

A cancer diagnosis can be considered an existential crisis in the lives of many of those affected and can result in increased distress, changes in emotional roles, social roles, physical functioning, and quality of life for most people who are diagnosed. Cancer patients have higher rates of clinically significant psychological disorders than their non-diagnosed age-matched peers. Several factors tend to influence the extent of psychological distress in response to a cancer diagnosis. These include younger age at diagnosis, history of mental illness and premorbid psychological functioning, stage at diagnosis and prognosis, and social support or other resources available (e.g., health insurance). As they do with other major life events, individuals with a cancer diagnosis rely on a variety of strategies to cope with the changes their diagnosis and treatment bring. Notably, most patients do not experience clinically significant symptoms of distress or dysfunction and, over time, typically 1–2 years, most patients will weather the crisis and return to baseline levels of functioning. However, for a significant number of individuals, full emotional and physical recovery can take much longer. Psychosocial intervention can facilitate emotional and physical adjustment to and recovery from cancer diagnosis and treatment.

Indications and Assessment

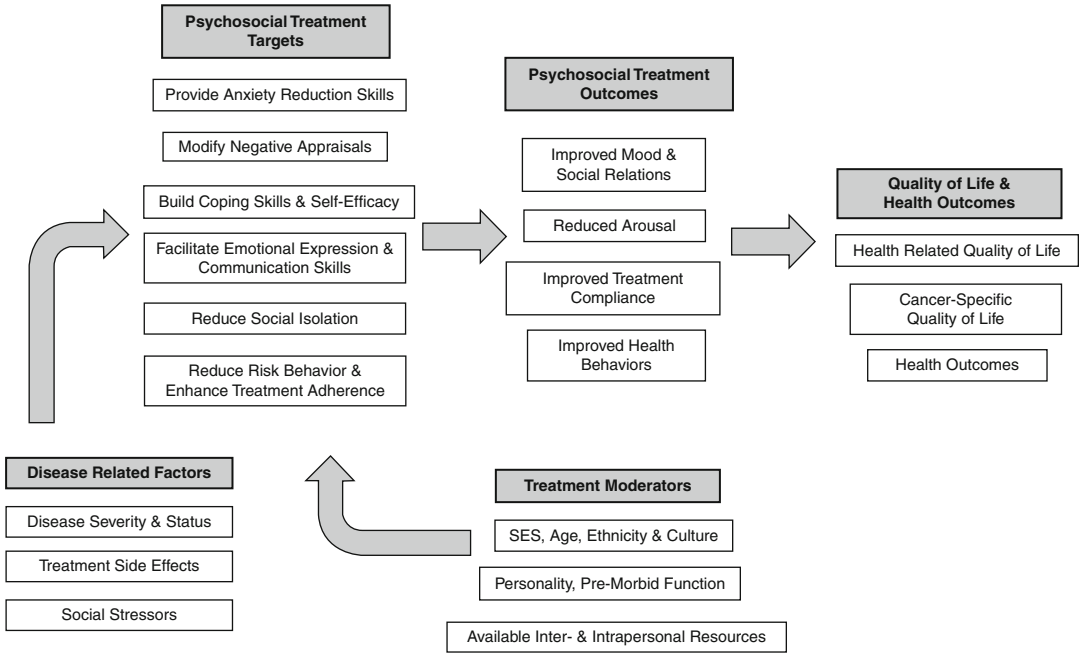
Psychosocial intervention is indicated not only when the patient is reporting elevated levels of distress, depression, or anxiety (which should be

assessed regularly during cancer treatment and posttreatment follow-up visits, see below), but also when a patient reports difficulty with pain management, fatigue, cognitive complaints, or problems with sexual functioning. Assessment and patient education regarding available psychosocial interventions is warranted at routine intervals during cancer treatment and care, as many patients who might benefit from intervention may not be reporting symptoms at the time and early intervention can be effective prophylaxis against later symptom development. Commonly used instruments to assess psychosocial and physical well-being include a distress thermometer or validated measures of mood and affect, such as the Hospital Anxiety and Depression Scale (HADS). To address concerns regarding changes in cognitive functioning, neuropsychological testing may be warranted. For those who experience severe levels of distress and/or meet clinical criteria for a mental health disorder, evaluation for pharmacologic treatment may be warranted.

Psychosocial Treatment Modalities

Several psychosocial intervention models in cancer have shown success in reducing distress, improving quality of life, and facilitating the overall posttreatment adjustment period. Psychosocial treatment approaches have ranged from open support groups and psychoeducational programs that are based on information provision, to supportive group therapy approaches and individual treatments that are structured to provide a nurturing environment to express concerns over the multiple challenges associated with cancer survivorship. Both individual and group-based interventions based on cognitive behavioral intervention models that blend a variety of therapeutic techniques (e.g., cognitive restructuring, relaxation training) have shown success in improving health-related quality of life across multiple cancer populations. Other intervention approaches include mindfulness-based stress reduction, emotional expression, symptom management, health behavior change, and motivational interviewing. A significant amount of research has shown that effective therapy components in multimodal intervention efforts include techniques such as relaxation training (e.g., guided imagery) to lower arousal, disease information and management, an emotionally supportive environment where participants can address fears and anxieties, behavioral and cognitive coping strategies, and social support skills training. Therapeutic processes by which participants benefit from intervention include giving and receiving information, sharing experiences, reducing social isolation, and providing patients with coping skills that facilitate self-efficacy and sense of control over the cancer experience. Some evidence suggests that cancer patients may benefit more from structured interventions than purely supportive ones; this may be due to learning skills with which they can more effectively cope with cancer-related changes after the intervention has ended (e.g., stress management). Interventions may also be couple or family based, depending on the goals of therapy and targeted outcomes, and may be administered at all phases of the cancer continuum, from post-diagnosis and treatment decision making to end of life or long-term survivorship time periods. Such interventions can be delivered via several modalities including face-to-face and technology-based individual and group-based formats.

The model in Fig. 1 proposes that cancer patients and survivors may benefit from psychosocial interventions that target multiple components. For example, teaching anxiety reduction skills can provide a way to reduce anxiety, tension, and other forms of stress responses and, thus, help the survivor achieve a sense of mastery over disease-related and general stressors. The use of cognitive restructuring techniques can help patients identify links between thoughts, emotions, and bodily changes, and increase their ability to identify commonly used distorted thoughts and understanding of how these thoughts can interfere with emotional well-being, effective management of the disease, and multiple domains of quality of life. Participants in these interventions can also benefit from techniques that challenge cognitive, behavioral, and interpersonal coping strategies by increasing awareness of the use of maladaptive coping strategies to deal with



Cancer: Psychosocial Treatment, Fig. 1 Conceptual model of psychosocial treatment interventions

stress and disease-related challenges. Therefore, attention is given to replacing inefficient and indirect ways of dealing with stressors and promotes both emotion and problem-focused strategies while increasing patients' ability to adaptively express both positive and negative emotions. These intervention models also promote identifying and utilizing beneficial sources of social support, as well as providing self-management skills to engage in positive lifestyle changes and behaviors. Communication skills are also targeted, particularly those specific to interacting with health care professionals and communicating concerns about functional limitations and treatment-related side effects with the spouse/partner, family, and friends.

Within the intervention model, disease-related factors provide several considerations for psychosocial treatment approaches. Disease severity (localized vs. advanced disease) and status (disease free survival vs. recurrent disease) significantly influences the experience of the cancer patient and survivor. For example, advanced and recurrent diseases are characterized by greater psychosocial compromises such as greater levels of anxiety, depression, and interpersonal disruption, as well as existential concerns regarding the end of life. Similarly, treatment type and timing within the cancer survivorship continuum will pose varying psychosocial and physical responses that need to be considered. Some treatments are characterized by immediate functional limitations with a slow recovery that invariably does not reach baseline functioning over 1–2 years posttreatment. In contrast, other treatments have a more insidious side effect trajectory with the greatest consequences surfacing up to 1 year posttreatment. Therefore, an awareness and knowledge of the trajectories of treatment-related side effects must be considered as these symptoms will vary by treatment type. It is also critical to understand ongoing stressors not specifically related to cancer such as financial burdens or other major life events that may be impacting quality of life as these will also influence the efficacy of psychosocial treatments. Furthermore, a series of possible treatment moderators need to be considered. Older patients will be more likely to have multiple comorbidities and functional limitations

that will impact health-related quality of life outcomes. Socioeconomic status can play a significant role in treatment adjustment as it has been consistently associated with health-related quality of life outcomes via its influence on treatment compliance and follow-up. It is also critical to have a good understanding of pretreatment psychological functioning. Cancer patients with prior histories of psychological dysfunction such as depression, anxiety, or interpersonal difficulties seem to have greater difficulties in adjusting to the multiple challenges faced posttreatment. Similarly, low levels of education and a lack of interpersonal resources have also been shown to significantly impact adjustment. Therefore, any intervention approach needs to consider multiple disease-related characteristics and possible treatment moderators as these will likely interact with intervention efficacy and influence psychosocial treatment outcomes.

Psychosocial Effects of Intervention

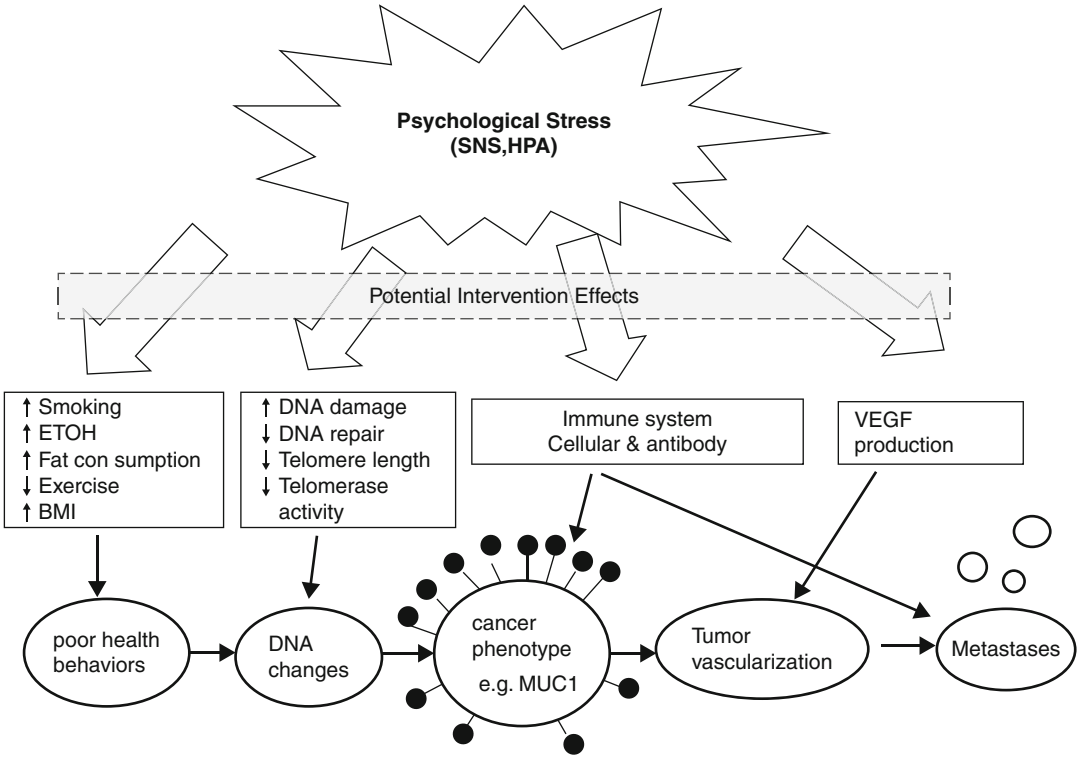
There is a large literature documenting the effectiveness of psychosocial intervention with cancer patients. Interventions have demonstrated positive effects across a range of psychosocial and physical outcomes, including symptoms of depression and anxiety, and cancer-related fear, social functioning, and disease- and treatment-related symptoms (e.g., fatigue, nausea, pain). Although findings have been mixed with reports of nonsignificant effects as well, several reviews of the literature have concluded that the majority of psychotherapeutic interventions among cancer patients demonstrate some improvement in psychosocial adjustment. Notably, sociodemographic factors (e.g., age, education, and socioeconomic status), premorbid psychological and physical functioning, social support, coping styles, and certain personality traits (e.g., neuroticism, interpersonal sensitivity, and social inhibition) have been associated with increased risk of adjustment difficulties following cancer diagnosis and treatment, suggesting that there may also be considerable variability in baseline functioning and response to intervention efforts.

Biological Effects of Intervention

Psychological distress can influence tumor progression via many different pathways (e.g., genetic changes, immune surveillance, pro-angiogenic processes). For example, there are data to suggest that psychological intervention can influence important neuroendocrine (e.g., cortisol) and immune function pathways, especially lymphocyte proliferation and TH1 cytokine production. One landmark study showed that women with metastatic breast cancer who participated in an expressive supportive group therapy intervention lived about twice as long as women in the comparison condition. This effect has been partially replicated in a subset of women with estrogen-receptor-negative tumors. While some groups have attempted to replicate survival findings, and with only limited success, other teams conducted studies focusing on neuroendocrine and immune mechanisms to explain the putative health effects of psychosocial intervention in breast cancer patients. One recent longitudinal study, which started with the intent of evaluating the intervention effect on not only psychological distress, but also immune function and survival, did show a survival advantage for intervention participants compared to comparison group participants. There is evidence now that psychological stress, via the HPA axis and SNS, can influence the course of tumor progression at almost every phase of the cancer continuum, from health behaviors to metastases. However, more systematic studies with large sample sizes and long-term follow-up effects are needed to provide conclusive evidence of any survival effects of these interventions. Potential psychosocial effects on biological mechanisms are depicted in Fig. 2.

Stepped Care Model of Psychosocial Intervention

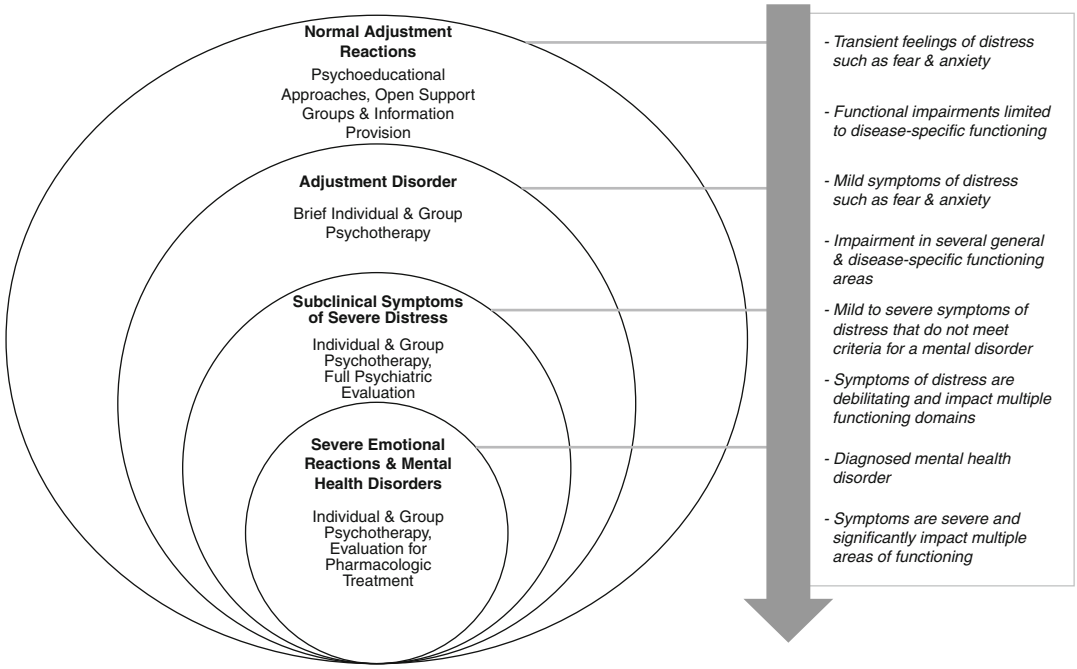
Several psychosocial treatments among cancer patients have shown promise in improving emotional well-being, and both general and disease-specific quality of life. Most intervention approaches involved group therapy interventions following cognitive behavioral, stress and coping, stress management, and supportive



Cancer: Psychosocial Treatment, Fig. 2 Development and progression of cancer and how/where psychological stress and interventions might influence the process

group environment theories and models. Some work has also provided psychoeducational interventions, engaged spouses/partners, or provided phone-based delivery of the interventions. Regardless of the intervention approach, it is important to consider the distress continuum among cancer patients to determine the most optimal level of care based on their needs (see Fig. 3).

Psychosocial intervention is not necessary for all patients and a stepped care model of intervention delivery is recommended. This involves a collaborative care approach to intervention efforts in which patients are involved in treatment planning and therapeutic resources are utilized based on systematic assessment and monitoring of patients' psychosocial well-being. Stepped care approaches require that treatments of different intensity are provided depending on the need of the individual. Treatments are initially implemented that are of minimal intensity but still likely to provide benefit and progress to more intensive interventions only if patients do not demonstrate improvement from simpler approaches or for those who can be reliably predicted to not likely benefit. An important feature of the stepped care model is that progress and decisions regarding intervention efforts are systematically monitored and changes in outcomes of interest are carefully assessed. A "step up" to a more comprehensive therapy is made only when there are no significant gains in the targeted outcomes. Stepped care may involve increasing intensity of a single therapeutic approach, transition to a different therapeutic approach, or using several therapeutic approaches additively. Likewise, different interventions may be applied to address different aspects of a patient's problem. Psychosocial needs also change as patients move from through their cancer experience and either transition to survivorship or face advanced disease and



Cancer: Psychosocial Treatment, Fig. 3 Psychological interventions' stepped approaches as a function of emotional reactions across the cancer distress continuum

end-of-life concerns. Utilizing a stepped care approach to promote adjustment and well-being at all phases of the cancer continuum may enhance intervention efficacy through more stringent assessment methods and appropriateness of intervention techniques, while also using the least amount of therapeutic resources.

The model in Fig. 3. proposes that treatment planning and intervention efforts must consider the distress continuum among cancer patients to determine the most optimal level of care based on their needs. Most cancer patients adjust relatively well to the cancer diagnosis and treatment. The majority of individuals experience some transient levels of distress characterized by mild symptoms of anxiety and depression, fear, and interpersonal disruption specific to disease-related functioning (e.g., sexual dysfunction). Because their emotional reactions are transient and significantly below clinical levels, these patients are likely to benefit from information provision or psychoeducational approaches that offer information on what to expect from prostate cancer treatment, the recovery process, available options for coping with treatment-related side effects (e.g., sexual aids), and communication skills to effectively navigate the medical system or voice concerns with the spouse/partner and family and friends.

A minority but yet significant number of cancer patients may experience emotional reactions that warrant a more structured approach at psychological care. Those lacking in social resources, presenting with high levels of perceived stress and enduring longstanding interpersonal dysfunction – likely driven by deficits in interpersonal skills and personality traits – are more likely to benefit from such interventions. Similarly, individuals with premorbid psychopathology and physical limitations, greater treatment-related functioning limitations, and recurrent disease are more likely to experience greater levels of distress and benefit the most from psychosocial interventions. Those who meet criteria for a mental health disorder are likely to be experiencing an adjustment disorder which is characterized by clinically significant symptoms of distress. In such cases, brief individual and group psychotherapeutic approaches can be useful in ameliorating

persistent symptoms of distress that among prostate cancer survivors are commonly related to treatment-related dysfunction. If untreated, these symptoms can interfere with multiple domains of health-related quality of life. Cancer patients who experience subclinical manifestations of mental health disorders such as anxiety, depression, and PTSD (i.e., experience severe symptomatology but not meeting diagnostic criteria) may benefit from a full psychiatric evaluation to determine the most appropriate level of care. For these survivors, individual and group psychotherapeutic approaches can positively impact mental health and health-related quality of life outcomes. Among the small number of patients who experience severe emotional reactions and are diagnosed with a mental health disorder, evaluation for pharmacologic treatment, in addition to individual and group psychotherapeutic approaches, is warranted.

Cross-References

- ▶ Intervention Theories
- ▶ Psychosocial Adjustment

References and Readings

Anderson, B. L., Yang, H. C., Farrar, W. B., Golden-Kreutz, D. M., Emery, C. F., Thornton, L. M., et al. (2008). Psychological intervention improves survival for breast cancer patients: A randomized clinical trial. *Cancer, 113*(12), 3450–3458.

Andrykowski, M. A., & Manne, S. L. (2006). Are psychological interventions effective and accepted by cancer patients? I. Standards and levels of evidence. *Annals of Behavioral Medicine, 21*(2), 93–97.

Burish, T. G., & Jenkins, R. A. (1992). Effectiveness of biofeedback and relaxation training in reducing the side effects of cancer chemotherapy. *Health Psychology, 11*, 17–23.

Dale, H. L., Adair, P. M., & Humphris, G. M. (2010). Systematic review of post-treatment psychosocial and behaviour change interventions for men with cancer. *Psycho-oncology, 19*(3), 227–237.

Daniels, J., & Kissane, D. W. (2008). Psychosocial interventions for cancer patients. *Current Opinion in Oncology, 20*(4), 367–371.

Falagas, M. E., Zarkadoulia, E. A., Ioannidou, E. N., Peppas, G., Christodoulou, C., & Rafailidis, P. I. (2007). The effect of psychosocial factors on breast cancer outcome: A systematic review. *Breast Cancer Research, 9*(4), 1–23.

Institute of Medicine. (2007). *Cancer care for the whole patient: Meeting psychosocial health needs*. Washington, DC: National Academies Press.

Jacobsen, P. B. (2010). Improving psychosocial care in outpatient oncology settings. *Journal of the National Comprehensive Cancer Network, 8*, 368–370.

Jacobsen, P. B., Donovan, K. A., Vadaparampil, S. T., & Small, B. J. (2007). Systematic review and meta-analysis of psychological and activity-based interventions for cancer-related fatigue. *Health Psychology, 26*, 660–667.

Jacobsen, P. B., & Jim, H. S. (2008). Psychosocial interventions for anxiety and depression in adult cancer patients: Achievements and challenges. *CA: A Cancer Journal for Clinicians, 58*(4), 214–230.

Manne, S. L., & Andrykowski, M. A. (2006). Are psychological interventions effective and accepted by cancer patients? II. Using empirically supported therapy guidelines to decide. *Annals of Behavioral Medicine, 21*(2), 98–103.

McGregor, B., & Antoni, M. H. (2009). Psychological intervention and health outcomes among women treated for breast cancer: a review of stress pathways and biological mediators. *Brain, Behavior and Immunity, 23*, 159–166.

Meyer, T. J., & Mark, M. M. (1995). Effects of psychosocial interventions with adult cancer patients: A meta-analysis of randomized experiments. *Health Psychology, 14*(2), 101–108.

Spiegel, D., Butler, L. D., & Giese-Davis, J. (2007). Effects of supportive-expressive group therapy on survival of patients with metastatic breast cancer. *Cancer, 110*(5), 1130–1138.

Stanton, A. L. (2006). Psychosocial concerns and interventions for cancer survivors. *Journal of Clinical Oncology, 24*(32), 5132–5137.

Zabora, J., Brintzenhofeszoc, K., Curbow, B., Hooker, C., & Piantadosi, S. (2001). The prevalence of psychological distress by cancer site. *Psycho-Oncology, 10*, 19–28.

Canonical Correlation

Stephanie Ann Hooker
Department of Psychology, University of Colorado, Denver, CO, USA

Definition

Canonical correlation is a multivariate statistical technique that specifies relationships between

two sets of variables. Researchers interested in understanding how two multidimensional constructs are related may find this technique useful. For example, someone interested in further understanding the relationships between the multidimensional constructs of personality and a healthy behavioral lifestyle might identify two sets of variables that measure those constructs. In the personality set, one might include factors like conscientiousness, openness to experience, and neuroticism, whereas in the healthy behavior set, one might include physical activity, healthy eating, sleep, or dental hygiene.

To use this technique, the researcher should identify two sets of measured variables. The variables selected for a set should measure different dimensions of the same construct (e.g., conscientiousness, openness to experience, and neuroticism would all be different facets of personality). Similar to exploratory factor analysis, canonical correlation identifies latent variables within each set. The canonical correlation (R^c) is the statistic that identifies the strength and directionality of the relationship between two latent variables (one from each set). Only statistically significant canonical correlations should be interpreted. The R^c is interpreted like the Pearson correlation coefficient, ranging from −1.0 to 1.0. A positive R^c indicates a positive relationship between the two latent variables and a negative R^c indicates a negative relationship between the two latent variables. R^c values closer to 1.0 (or −1.0) indicate stronger relationships.

Latent variables are interpreted using two statistics: standardized coefficients and canonical variate-variable correlations. Standardized coefficients indicate the extent to which each measured variable contributes to the latent variable. Canonical variate-variable correlations indicate the strength and directionality of the relationship between the measured variable and the latent variable. Stevens (2009) suggests examining both the standardized coefficients and the canonical variate-variable correlations to include the measured variable in the interpretation of the latent variable. Many of the measured variables may correlate highly with the latent variable, but the standardized coefficient identifies which variables may be redundant in the interpretation. Once the researcher determines which measured variables contribute to each latent variable, he or she names the latent factor and interprets the meaning of the canonical correlation.

Cross-References

▶ Latent Variable

References and Readings

Stevens, J. P. (2009). Canonical correlation. In *Applied multivariate statistics for the social sciences* (pp. 395–411). New York: Routledge.

Capacity Assessment

▶ Functional Versus Vocational Assessment

Capsaicin

Barbara Resnick
School of Nursing, University of Maryland, Baltimore, MD, USA

Synonyms

Pepper

Definition

Capsaicin is the ingredient found in different types of hot peppers, such as cayenne peppers, that makes the peppers spicy hot. You can eat it raw or as a dried powder placed in food. It is also available as a dietary supplement, topical cream, or via a high dose dermal patch (trade name Qutenza). Capsaicin, in any of these forms, is used to relieve the pain of peripheral neuropathy

from postherpetic neuraligia caused by shingles and for temporary musculoskeletal pain and has been used to treat psoriasis (to decrease itching and inflammation). Capsaicin works by first stimulating and then decreasing the intensity of pain signals in the body. Capsaicin stimulates the release of a compound believed to be involved in communicating pain between the nerves in the spinal cord and other parts of the body. To be effective, the cream needs to be used four to five times a day. At the time of use, the skin may burn or itch, although these sensations decrease over time. It is important to wash your hands thoroughly after each use and to avoid getting the cream in your eyes or places in which there are moist mucous membranes such as the mouth or vaginal or rectal areas. Contact with these areas will cause burning. The cream should also not be used on areas of broken skin.

Capsaicin has also be used as a supplement to improve digestion, eliminate infections, prevent heart disease by lowering blood cholesterol levels and blood pressure, and prevent clotting and atherosclerosis. Theoretically, capsaicin acts as an antioxidant and protects the cells of the body from the damage of free radicals. In so doing, health benefits can be derived. Lastly, capsaicin makes mucus thinner and thus may improve pulmonary function among those with chronic obstructive pulmonary disease or chronic bronchitis.

Capsaicin is generally considered safe when taken orally or used as a cream. As noted, it can cause some unpleasant effects. If this occurs, the best way to alleviate further pain is to remove the exposure via removing clothing if it has been contaminated and washing off the skin with soap, shampoo, or other types of detergents. Water, vinegar, and bleach are all ineffective at removing capsaicin. Applications of cool compresses may help with the burning sensations experienced with capsaicin.

An allergic reaction to capsaicin is possible. If you are just beginning to use capsaicin, either as fresh or prepared food or in powder form, start with small amounts. If you use a topical cream, you should first apply it to a small area of skin to test for an allergic reaction.

References and Readings

Bode, A. M., & Dong, Z. (2011). The two faces of capsaicin. *Cancer Research, 71*(8), 2809–2814.

Fraenkel, L., Bogardus, S. T., Concato, J., & Wittink, D. R. (2004). Treatment options in knee osteoarthritis: The patient's perspective. *Archives of Internal Medicine, 164*, 1299–1304.

Johnson, W. (2007). Final report on the safety assessment of capsicum annuum extract, capsicum annuum fruit extract, capsicum annuum resin, capsicum annuum fruit powder, capsicum frutescens fruit, capsicum frutescens fruit extract, capsicum frutescens resin, and capsaicin. *International Journal of Toxicology, 26*(Suppl. 1), 3–106.

Carbohydrates

James Turner
School of Cancer Sciences, The University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

CHO; Saccharide

Definition

A carbohydrate is an organic compound, or in other words, a compound containing a carbon atom. In addition to carbon, all carbohydrates also comprise the atoms hydrogen and oxygen, and share the common formula

$$C_nH_{2n}O_n$$

where n is any whole number.

The name "carbohydrate" is derived from the bonding of a water molecule to a carbon atom, thus carbohydrates are hydrates of carbon.

Carbohydrates can be classified into several categories. Monosaccharides are the most basic units, and when two monosaccharides are chemically bonded, a disaccharide carbohydrate is formed. Oligosaccharides are generally considered to be carbohydrates with three to ten

monosaccharides, and polysaccharides are carbohydrates with more than ten of these basic units. In nutrition, carbohydrates are often categorized into "simple" and "complex" forms. Simple carbohydrates include monosaccharides and disaccharides (sugars) whereas complex carbohydrates are oligosaccharides and polysaccharides (starches). Carbohydrates, despite being nonessential dietary constituents, function primarily as a source of energy, and are a particularly important fuel for high-intensity exercise.

Cross-References

- ▶ Glucose
- ▶ Insulin

References and Readings

Bender, D. A. (2002). *Introduction to nutrition and metabolism* (3rd ed.). London: Taylor & Francis.

McArdle, W. D., Katch, F. I., & Katch, V. L. (2001). *Exercise physiology. Energy, nutrition and human performance* (5th ed.). Baltimore: Lippincott Williams & Wilkins.

Carcinogens

Elizabeth Franzmann
Department of Otolaryngology/Division of Head and Neck, Miller School of Medicine, University of Miami, Miami, FL, USA

Synonyms

Mutagen

Definition

Substances that cause cancer

Description

It is well established that cancer initiation and progression occurs through complex genetic and environmental interactions (Pfeifer & Hainaut, 2011). Completely genetically induced tumors are rare (Pfeifer & Hainaut, 2011). Most malignancies occur as a result of exposure to internal or environmental agents that cause genetic damage (Pfeifer & Hainaut, 2011). However, susceptibility to these environmentally induced mutations can be inherited (Pfeifer & Hainaut, 2011). Environmental factors in a very broad sense can include physical and chemical agents, dietary factors, behavioral exposures such as tobacco and alcohol and microenvironmental factors such as infection and inflammation. Any such factor that causes cancer is a carcinogen.

Sir Percivall Pott was the first to report that a malignancy could be caused by an environmental carcinogen when he described "the chimney-sweepers" cancer in 1775 (Cogliano, 2010; Stone, 2003). This work concluded that scrotal cancer was caused by soot that became wedged in the scrotum and also marks the first time that an occupational cancer was linked to a specific cause (Cogliano, 2010; Stone, 2003). As a result of this type of work, it is now understood that tobacco, including that found in second-hand smoke, causes lung cancer (Stone, 2003). It is also known that mesothelioma, frequent in shipyard workers, is due to asbestos exposure (Cogliano, 2010; Pfeifer & Hainaut, 2011; Siemiatycki et al., 2004; Stone, 2003), and leukemia, frequent in the shoe-production industry, is related to benzene (Cogliano, 2010; Siemiatycki et al., 2004). Similarly nickel refining, smelting, and welding are associated with cancers of the lung, nasal cavity, and sinuses and ionizing radiation is associated with bone, leukemia, lung, liver, and many other types of cancer (Siemiatycki et al., 2004). Certain viruses such as human papillomavirus (HPV) and hepatitis C virus are also carcinogenic (Stone, 2003).

Following Pott's example, public health agencies such as the United States National Toxicology Program and International Agency for Research on Cancer (IARC) have worked to identify and educate the public about additional carcinogens (Cogliano, 2010; Siemiatycki et al., 2004). In the case of the IARC, agents, mixtures,

or exposure circumstances are selected for evaluation if humans are known to be exposed and there is reason to suspect they may cause cancer (Siemiatycki et al., 2004). At regular intervals, the IARC meets as a working group consisting of 15–30 experts from related fields (Siemiatycki et al., 2004). These experts identify a concerning agent and review the epidemiological, animal, and other laboratory studies to help determine whether a substance of interest is carcinogenic (Siemiatycki et al., 2004). Epidemiologic evidence is generally considered the most important determinant (Siemiatycki et al., 2004). This evidence stems from associations between suspected causal agents and presence or absence of cancer in populations. The second most important determinant is the direct laboratory animal evidence of carcinogenicity (Siemiatycki et al., 2004). Other laboratory evidence such as genotoxicity, mutagenicity, metabolism, cytotoxicology, or mechanisms are also considered important (Siemiatycki et al., 2004). Based on the combination of these different types of data, the IARC develops a consensus and then classifies the substance as carcinogenic, probably carcinogenic, possibly carcinogenic, not classifiable, or probably not carcinogenic (Siemiatycki et al., 2004). Results of the working group meetings are published in the IARC monographs which provide important information for determining research priorities and preventing cancer (Siemiatycki et al., 2004).

Despite Pott's work, incidence of scrotal cancer in England did not decrease until the 1950s when counteractive measures such as improved chimney-cleaning, alternative heating methods, and protective clothing were put in place (Cogliano, 2010). Even today, exposure to many of the hundreds of common and suspected carcinogens occurs in industry (Cogliano, 2010). Further education and preventive measures are needed to fully educate and protect the public.

References and Readings

Cogliano, V. J. (2010). Identifying carcinogenic agents in the workplace and environment. *The Lancet Oncology, 11*, 602.

Pfeifer, G. P., & Hainaut, P. (2011). Next-generation sequencing: Emerging lessons on the origins of human cancer. *Current Opinion in Oncology, 23*, 62–68.

Siemiatycki, J., Richardson, L., Straif, K., Latreille, B., Lakhani, R., Campbell, S., et al. (2004). Listing occupational carcinogens. *Environmental Health Perspectives, 112*, 1447–1459.

Stone, M. J. (2003). History of the Baylor Charles A. Sammons cancer center. *Proceedings (Baylor University Medical Center), 16*(1), 30–58.

Carcinoma

Elizabeth Franzmann
Department of Otolaryngology/Division of Head and Neck, Miller School of Medicine, University of Miami, Miami, FL, USA

Synonyms

Malignant neoplastic disease

Definition

Carcinoma includes malignancies that begin in the lining or covering of organs.

Description

According to the National Cancer Institute (National Cancer Institute [NCI], 2011), carcinoma is the most common category of cancer and includes malignancies that begin in the lining or covering of organs. Other cancer categories include sarcomas that begin in connective or supportive tissue such as muscle and bone, leukemias which start in blood-forming tissues, lymphoma and myelomas which originate in the immune system, and central nervous system tumors which include malignancies that start in the brain and spinal cord (NCI, 2011). Nonmelanoma skin cancer, including basal cell and squamous cell, are the most common carcinomas in the United States with more 1,000,000 diagnosed annually (NCI, 2011). Other

carcinomas involving the prostate, breast, lung, and colon are the next most common (NCI, 2011).

Carcinoma, like any cancer, is characterized by dysregulated growth and uncontrolled dissemination of abnormal cells which, left unchecked, can result in death (American Cancer Society [ACS], 2011). Exposure to environmental factors such as tobacco, viruses, chemicals, and radiation can cause cancer and such agents are known as carcinogens (ACS, 2011; NCI, 2011). Internal factors such as inherited mutations, immunodeficiency, and mutations arising from metabolic dysfunction can also give rise to cancer (ACS, 2011). Because most malignancies initiate and progress through a combination of environmental and internal factors, only about 5% of cancers are felt to be familial (ACS, 2011). Decades can pass between exposure to external factors and detectable cancer (ACS, 2011). Some individuals who are exposed to known carcinogens, such as tobacco, for many years may never develop cancer (ACS, 2011). Individual factors such as ability to repair DNA damage, remove carcinogens, and destroy abnormal cells all play a role in determining who will go on to develop cancer (NCI, 2011).

Depending on site, stage, and specific pathology of the carcinoma, treatment includes combinations of surgery, radiation, and chemotherapy (ACS, 2011; NCI, 2011). Other therapies such as hormone therapy, biological therapy, and targeted therapy may also be used (NCI, 2011). Treatment can be very toxic, resulting in long-term morbidity especially for late stage disease which is primarily determined by its size and whether it has spread to lymph nodes or other areas of the body (NCI, 2011). Even with the most aggressive therapy, sometimes cure cannot be attained. For these reasons, programs to prevent and detect cancers early are imperative.

The American cancer Society (ACS) recommends regular screening for breast, colorectal, and cervical cancer as screening programs have resulted in decreased mortality for these cancers (Smith et al., 2011). Though evidence in favor of prostate cancer screening is increasing, there is still some controversy whether risks outweigh benefits (Smith et al.). The ACS recommends that men aged 50 or over and with at least a 10-year life expectancy should have an opportunity to make an informed decision with their health care provider about prostate cancer screening after receiving counseling as to the risks, benefits, and uncertainties associated with such screening (Smith et al.).

Prevention of carcinoma focuses on decreasing tobacco use, increasing nutritional awareness, and limiting exposure to known carcinogens. Tobacco use is leading risk factor for carcinoma and the most preventable cause of death worldwide, responsible for the deaths of half of long-term users (ACS, 2011). Furthermore, it has also been estimated that one-third of cancer deaths in the United States each year are due to poor nutrition, physical inactivity, and excess weight (ACS). In addition, environmental exposures other than tobacco use can increase risk of carcinoma. These exposures include infectious agents, excessive sun exposure, and exposures to carcinogens that exist in air, food, water, and soil (ACS). The United States' National Toxicology Program and the International Agency for Research on Cancer work to identify carcinogens and provide information to the public and other regulatory agencies in an effort to decrease the burden of human cancer (ACS, 2011).

Cross-References

▶ Carcinogens

References and Readings

American Cancer Society. (2011). *Cancer facts & figures 2011*. Atlanta, GA: Author.

National Cancer Institute. (2011). What is cancer? Accessed July 17, 2011, from http://www.cancer.gov/cancertopics/cancerlibrary/what-is-cancer

Smith, R. A., Cokkinides, V., Brooks, D., Saslow, D., Shah, M., & Brawley, O. W. (2011). Cancer screening in the United States, 2011: A review of current American Cancer Society guidelines and issues in cancer screening. *CA: A Cancer Journal for Clinicians, 61*, 8–30.

Carcinoma of the Prostate

► Cancer, Prostate

Cardiac Arrhythmia

► Arrhythmia

Cardiac Cachexia

► Cachexia (Wasting Syndrome)

Cardiac Death

Ana Victoria Soto[1] and William Whang[2]
[1]Medicine – Residency Program, Columbia University Medical Center, New York, NY, USA
[2]Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Synonyms

Sudden cardiac death

Definition

Cardiac death is defined as occurring when the rhythmic contractions of the heart cease and do not return spontaneously. Generally speaking, cardiac death may occur suddenly or non-suddenly. Sudden cardiac death is defined by death within 1 h of the onset of symptoms, in the absence of preceding evidence of severe heart failure. This definition is usually used to capture death due to cardiac arrhythmia. Non-sudden cardiac death generally encompasses death due to pump failure (Hinkle & Thaler, 1982).

References and Readings

Hinkle, L. E., Jr., & Thaler, J. T. (1982). Clinical classification of cardiac deaths. *Circulation, 65*, 457–464.

Cardiac Events

Siqin Ye
Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Synonyms

Coronary event; Major adverse cardiac and cerebrovascular event (MACCE); Major adverse cardiac event (MACE); Major adverse cardiovascular event (MACE)

Definition

The term cardiac event is used to denote the composite of a variety of adverse events related to the cardiovascular system.

Description

The exact definition for cardiac events often varies depending on the specific study. At the narrowest, it is synonymous with coronary event, which refers to adverse events caused by disease processes affecting the coronary arteries. These may include what are termed "hard" events such as deaths that are attributed to coronary artery disease and nonfatal myocardial infarctions but also occasionally "soft" events such as angina or revascularizations for progressive coronary artery disease (Kip, Hollabaugh, Marroquin, & Williams, 2008). More broadly, the term cardiac event is often used interchangeably with another loosely defined term, major adverse cardiac event or MACE. Common definitions of MACE include death (either all-cause or cardiac), nonfatal myocardial infarction, and revascularization (with optional additional specification of target vessel or lesion, i.e., if the revascularization occurred at the site of a previously identified diseased coronary vessel or atherosclerotic lesion, respectively); occasionally, stroke is also incorporated into MACE, and

the term is alternatively defined as major adverse cardiovascular event or major adverse cardiac and cerebrovascular event (MAACE). Finally, in some circumstances, nonfatal heart failure events (i.e., hospitalization for heart failure) are also considered cardiac events, though this is infrequent and occurs mainly in studies that focus on the prognosis and treatment of heart failure (Skali, Pfeffer, Lubsen, & Solomon, 2006).

Since cardiac events and the other related terms described above are composites of clinical events of varying significance, there remains considerable debate on what should constitute the most appropriate component endpoints and how to define them. Furthermore, it has been increasingly recognized that the wide variability in these definitions may significantly influence the results and impact of clinical trials and other studies. For instance, many have noted that less consequential but more frequently occurring endpoints such as revascularizations or heart failure exacerbations are often what drive the statistical significance or the lack there of for the results of many trials (DeMets & Califf, 2002; Lim, Brown, Helmy, Mussa, & Altman, 2008). Different component endpoints may also trend in opposing directions, rendering the interpretation and generalization of the primary result problematic (Wilcox, Kupfer, & Erdmann, 2008). These considerations have induced recent attempts to standardize the definitions of events that have the most clinical relevance, and guidelines such as the 2001 ACC Clinical Data Standards and the 2007 Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction Expert Consensus Document have outlined explicit definitions for terms such as cardiovascular death and myocardial infarction, with emphasis placed on objective findings that include ECG changes and the typical rise and fall of biomarkers such as cardiac troponins. In addition, most contemporary studies have begun routinely disclosing the results of individual endpoints as well as those of alternative composite measures. It is hoped that with these and other future efforts, the methodological challenges inherent in the use of composite endpoints such as cardiac events will finally be adequately addressed.

Cross-References

▶ Coronary Event

References and Readings

ACC Writing Committee for Acute Coronary Syndromes Clinical Data Standards & ACC Task Force on Clinical Data Standards. (2001). American college of cardiology key data elements and definitions for measuring the clinical management and outcomes of patients with acute coronary syndromes. *Journal of the American College of Cardiology, 38*(7), 2114–2130.

DeMets, D. L., & Califf, R. M. (2002). Lessons learned from recent cardiovascular clinical trials. *Circulation, 2002*(106), 746–751.

Kip, K. E., Hollabaugh, K., Marroquin, O. C., & Williams, D. O. (2008). The problem with composite end points in cardiovascular studies. *Journal of the American College of Cardiology, 51*(7), 701–707.

Lim, E., Brown, A., Helmy, A., Mussa, S., & Altman, D. G. (2008). Composite outcomes in cardiovascular research: A survey of randomized trials. *Annals of Internal Medicine, 149*, 612–617.

Skali, H., Pfeffer, M. A., Lubsen, J., & Solomon, S. D. (2006). Variable impact of combining fatal and nonfatal end points in heart failure trials. *Circulation, 2006*(114), 2298–2304.

Thygesen, K., Alpert, J. S., White, H. D., on behalf of the Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction. (2007). Universal definition of myocardial infarction. *European Heart Journal*, *28*(20), 2525–2538.

Wilcox, R., Kupfer, S., Erdmann, E., on behalf of the PROactive Study investigators (2008). Effects of pioglitazone on major adverse cardiovascular events in high-risk patients with type 2 diabetes: Results from PROspective pioglitAzone clinical trial in macrovascular events (PROactive 10). *American Heart Journal*, *155*(4), 712–717.

Cardiac Output

Simon Bacon
Department of Exercise Science, Concordia University, Montreal Behavioral Medicine Centre, Montreal, QC, Canada

Definition

Cardiac output (Q) is the volume of blood pumped out of the heart (specifically from the

right and left ventricles) per minute. It is generally calculated as a function of heart rate and stroke volume (cardiac output = heart rate × stroke volume). Average resting cardiac output is about 5 L/min (normal range 4–8 L/min) and tends to be slightly higher in men versus women. During acute exercise and mental stress, cardiac output increases. This increase can be as high as 35 L/min for exercise (in elite athletes) and 15 L/min for mental stress.

There are many methods of measuring cardiac output, which range from intracardiac catheterization (invasive) to arterial pulse tonometry (noninvasive). The Fick principle, which uses the measurement of oxygen consumption and the oxygen content of the arterial and venous blood, is considered the most accurate method of assessing cardiac output, though it is an invasive technique, which limits its utility. Great effort has been placed into finding accurate reliable noninvasive methods of assessing cardiac output, such as, dye dilution, ultrasound-based techniques, impedance cardiography, and, more recently, magnetic resonance imaging. Each one of these comes with both positives and negatives and the selection of one method over another needs to be made given the individual requirement for cardiac output measurement.

As cardiac output is driven by heart rate and stroke volume, the factors that control changes in these parameters also influence cardiac output. Specifically, parasympathetic and sympathetic activity and venous return influence cardiac output.

Cross-References

- ▶ Blood Pressure
- ▶ Heart Rate

References and Readings

Berne, R. M., & Levy, M. N. (2001). *Cardiovascular physiology* (8th ed.). St. Louis, MO: Mosby.

Hall, J. E. (2011). *Guyton and Hall textbook of medical physiology* (12th ed.). New York: Elsevier.

Cardiac Rehabilitation

Leah Rosenberg[1] and Sarah Piper[2]
[1]Department of Medicine, School of Medicine, Duke University, Durham, NC, USA
[2]Institute of Metabolic Science, Addenbrookes Hospital, Metabolic Research Laboratories, University of Cambridge, Cambridge, UK

Synonyms

Secondary prevention programs

Definition

Cardiac rehabilitation is a multidisciplinary program of secondary prevention measures that assist cardiovascular disease patients in their comprehensive recovery to previous functioning.

Description

Introduction

Cardiac rehabilitation is a comprehensive platform of pharmacologic, psychosocial, and behavioral secondary prevention measures that is typically provided to patients with a history of cardiovascular disease. Cardiac rehabilitation is designed with a multidisciplinary approach to patient care and requires a cohesive plan of various therapies and practitioners. Cardiac rehabilitation has been shown to improve outcomes and initiate a type of "re-conditioning" process for many patients (Clark et al. 2005). Targeted patient populations for cardiac rehabilitation include those individuals who have recently had an acute cardiovascular event (i.e., myocardial infarction or unstable angina), post-cardiac bypass patients, and those who have stable angina, heart failure, or other patients with cardiac disease who have become deconditioned for any reason.

History of Cardiac Rehabilitation Programs

In the 1930s, patients who had suffered a myocardial infarction were instructed to

observe strict bed rest, often up to 6 weeks in duration. Gradually, increasing levels of physical activity were added to the post-event regimen. In addition to the salutatory effects of cardiac rehabilitation for recovery of previous functional status, it was eventually recognized that there were significant benefits in avoiding the hazards of bed rest which included deconditioning, deep venous thrombosis, and even limb atrophy and contractures. Today, post-acute coronary syndrome patients are encouraged to return to physical activity soon after the event. Early intervention with physical therapy is now a hallmark of contemporary cardiovascular care.

Typical Components of Cardiac Rehabilitation

The United States Public Health Service (USPHS) defines cardiac rehabilitation programs as comprehensive, multidisciplinary efforts with the following components (Hamm et al. 2011). These are the broad categories that encompass both short- and long-term goals. Newly admitted cardiac rehabilitation patients must undergo risk stratification to identify their needs for supervision and particular exercise plan.

1. Medical evaluation
2. Exercise training
3. Secondary prevention efforts and risk factor reduction
4. Patient education and counseling

Evidence Supporting Cardiac Rehabilitation

There are several trials that have compared the efficacy of cardiac rehabilitation programs that focus primarily on risk factor reduction versus an approach that favors increasing exercise tolerance. An integrated approach is the most favorable for reducing morbidity and mortality after a cardiovascular event. Modification of depressive symptoms is an important target for a cohesive rehabilitation program (Milani et al. 2007). Other less-quantifiable benefits include the socialization and support that comes from working with a variety of clinicians and peer groups.

A growing body of evidence in the literature supports exercise-training programs for cardiac rehabilitation (Antman et al. 2008). All enrolled patients should undergo a thorough medical evaluation prior to initiating any program of physical exertion. This is particularly relevant for those who are survivors of an acute coronary syndrome or symptomatic heart failure. While there used to be a prevalent belief in the medical community that prolonged bed rest was the only safe activity level after a cardiac event, numerous studies have demonstrated the safety of medically supervised exercise programs (Franklin, 1998). These exercise programs not only improve the quality of life for cardiac patients but have actually been shown to increase life expectancy in some cases. Specifically, most individualized exercise programs should encompass aerobic activities for at least 2 days per week.

Guidelines for Cardiac Rehabilitation

The American Society of Cardiovascular and Pulmonary Rehabilitation have published guidelines outlining ten core competencies that practitioners must have to provide the highest standard of evidence-based care for patients. Briefly, the ten areas are patient assessment, nutritional counseling, weight management, blood pressure management, lipid management, diabetes management, tobacco cessation, psychosocial management, physical activity counseling, and exercise training evaluation. The guidelines encompass an array of skills that transcend the abilities of any single provider. Instead, they assume a collaborative and comprehensive approach to cardiac rehabilitation. The competencies are divided into discrete "knowledge" points and then "skills" without specific reference to the particular type of provider who will provide the services. To coordinate the broad variety of necessary services, they suggest a case management model for individual patients. In the position statement enunciating the ten core competencies, the Society emphasizes the extent to which individual providers need not be proficient in all facets of secondary prevention but rather be willing and able to identify particular patient needs. The focus on multidisciplinary care involves the participation of physicians, nurses, physical therapists, clinical nutritionists, social workers, and psychologists.

Of particular interest is the core competency of psychosocial management. The knowledge piece requires cardiac rehabilitation providers to become aware with the literature on the impact of psychological factors on the pathophysiology of cardiovascular event onset and the impediments that can prevent recovery. In particular, the competency requires specific attention to developing familiarity with the effect of major depression on adverse cardiovascular outcomes and worse adherence to treatments (Prochanska & DiClemente, 1983). This references the current research question of whether poorer outcomes among depressed post-heart attack patients are due to their non-adherence of rehabilitative therapies or rather a distinct pathophysiologic state.

Cross-References

- ▶ Cardiovascular Disease
- ▶ Physical Therapy
- ▶ Recovery
- ▶ Rehabilitation

References and Readings

Antman, E. M., Hand, M., Armstrong, P. W., Bates, E. R., Green, L. A., Halasyamani, L. K., et al. (2008). 2007 focused update of the ACC/AHA 2004 guidelines for the management of patients with ST-elevation myocardial infarction: A report of the American College of Cardiology/American Heart Association task force on practice guidelines: Developed in collaboration With the Canadian Cardiovascular Society endorsed by the American Academy of Family Physicians: 2007 Writing Group to review new evidence and update the ACC/AHA 2004 guidelines for the management of patients with ST-elevation myocardial infarction, writing on behalf of the 2004 writing committee. *Circulation, 117*(2), 296–329.

Clark, A. M., Hartling, L., Vandermeer, B., & McAlister, F. A. (2005). Meta-analysis: Secondary prevention programs for patients with coronary artery disease. *Annals of Internal Medicine, 143*(9), 659–672.

Franklin, B. A., Bonzheim, K., Gordon, S., & Timmis, G. C. (1998). Safety of medically supervised outpatient cardiac rehabilitation exercise therapy: A 16-year follow-up. *Chest, 114*(3), 902–906.

Hamm, L. F., Sanderson, B. K., Ades, P. A., Berra, K., Kaminsky, L. A., Roitman, J. L., et al. (2011). Core competencies for cardiac rehabilitation/secondary prevention professionals: 2010 Update: Position statement of the American Association of Cardiovascular and Pulmonary Rehabilitation. *Journal of Cardiopulmonary Rehabilitation and Prevention, 31*(1), 2–10.

Milani, R. V., & Lavie, C. J. (2007). Impact of cardiac rehabilitation on depression and its associated mortality. *American Journal of Medicine, 120*(9), 799–806.

Prochanska, J. O., & DiClemente, C. C. (1983). Stages and processes of self-change of smoking: Toward an integrative model of change. *Journal of Consulting and Clinical Psychology, 51*, 390–395.

Cardiac Risk Factor

▶ Heart Disease and Cardiovascular Reactivity

Cardiac Stress Test

▶ Maximal Exercise Stress Test

Cardiac Surgery

Leah Rosenberg
Department of Medicine, School of Medicine, Duke University, Durham, NC, USA

Synonyms

Cardiothoracic surgery; Cardiovascular surgery

Definition

Cardiac surgery is the subset of operative procedures focused on the heart and vasculature. Common examples of cardiac surgery include coronary artery bypass grafting (CABG), valvular repair, and the correction of congenital cardiac malformations. Cardiac surgery is a subspecialty of general surgery that requires additional training beyond a traditional 5-year residency. Advancements in anesthesiology have been critical to the development of cardiac surgery. For example,

the widespread use of heart-lung bypass machines since the 1990s have extended the possible duration and complexity of these procedures.

Surgeries on the heart and great vessels (e.g., aorta and vena cavae) are generally performed on seriously ill patients who have either tried or been deemed ineligible for less invasive measures such as medication-based or percutaneous interventions (Lie, Bunch, Smeby, Arnesen, & Hamilton, 2012). Increased rates of mood disorder such as depression or cognitive impairment have been noted in post-CABG patients, suggesting a possibly important role for behavioral therapies (Katon, Ludman, & Simon, 2008). To date, however, it is unknown whether depression treatment in post-CABG patients improves cardiovascular outcomes.

Cross-References

- ▶ Cardiovascular Disease
- ▶ Coronary Artery Disease
- ▶ Coronary Heart Disease

References and Readings

Katon, W., Ludman, E., & Simon, G. (2008). *The depression helpbook* (2nd ed.). Chicago: Bull Publishing.

Lie, I., Bunch, E. H., Smeby, N. A., Arnesen, H., & Hamilton, G. (2012). Patients' experiences with symptoms and needs in the early rehabilitation phase after coronary artery bypass grafting. *European journal of cardiovascascular nursing*, *11*(1), 14–24.

Cardiologist

Daichi Shimbo
Center for Behavioral Cardiovascular Health, Columbia University, New York, NY, USA

Synonyms

Cardiology expert; Cardiovascular medicine expert; Heart doctor

Definition

A cardiologist is a physician who has specialty training in the area of cardiology. Cardiologists are often MD trained, and typically had general training in internal medicine (or pediatrics if a pediatric cardiologist) prior to the completion of cardiology fellowship. Cardiologists are often confused with cardiac or cardiothoracic surgeons, who primarily perform operations on the heart. A "board-certified cardiologist" is a physician who trained in cardiology, met minimum training requirements, and also passed the cardiology board exams. After cardiology fellowship, physicians can choose to undergo additional training in a subspecialty of cardiology (e.g., echocardiography, nuclear cardiology, intervention, etc).

Cross-References

- ▶ Cardiology

References and Readings

Baughman, K. L., Duffy, F. D., Eagle, K. A., Faxon, D. P., Hillis, L. D., & Lange, R. A. (2008). Task force 1: Training in clinical cardiology. *Journal of the American College of Cardiology*, *51*(3), 339–348.

Cardiology

Daichi Shimbo
Center for Behavioral Cardiovascular Health, Columbia University, New York, NY, USA

Synonyms

Cardiovascular medicine

Definition

Cardiology is a medical specialty of the structure, function, and disorders of the heart.

Traditionally, cardiology has mainly focused on the heart; however, more recently, the field of cardiology has expanded into the study and disorders of the arteries and veins, as well as other organs such as the brain (i.e., stroke or transient ischemia attack) or kidney (i.e., cardiorenal syndrome). This is probably due to a common underlying pathophysiology of disease. As such, cardiology has recently involved areas of medicine typically associated with other specialties (such as neurologists, nephrologists, etc.).

Cross-References

▶ Cardiologist

References and Readings

Fuster, V., O'Rourke, R., Walsh, R., & Poole-Wilson, P. (2007). *Hurst's the heart* (12th ed.). New York: McGraw-Hill Professional.

Cardiology Expert

▶ Cardiologist

Cardiothoracic Surgery

▶ Cardiac Surgery

Cardiovascular Disease

▶ Acute Myocardial Infarction
▶ Angina Pectoris
▶ Cardiovascular Disease Prevention
▶ Congestive Heart Failure
▶ Coronary Heart Disease
▶ Heart
▶ Heart Disease
▶ Heart Failure
▶ Hypertension
▶ Hypertrophy

Cardiovascular Disease (CVD)

▶ Heart Disease and Smoking

Cardiovascular Disease Prevention

Stephanie Ann Hooker
Department of Psychology, University of Colorado, Denver, CO, USA

Synonyms

CVD prevention

Definition

Cardiovascular disease is a group of chronic diseases (e.g., myocardial infarction, chronic heart disease, stroke) of the heart and blood vessels. Cardiovascular disease prevention is composed of the various early actions taken to thwart the onset of cardiovascular disease.

Description

Cardiovascular diseases (CVD) claim the lives of thousands of individuals every year. In 2007, the top cause of death in the United States was heart disease (616,067 deaths), second was cancer (562,875 deaths), and third was stroke (or cerebrovascular disease, in many cases a form of cardiovascular illness; 135,952 deaths) (U.S. Centers for Disease Control and Prevention [CDC], 2010). Although there is a general inheritability for CVD, the formation of these diseases is seen as largely preventable. In a unique interpretation of the causes of death in the year 2000, Mokdad and colleagues (Mokdad, Marks, Stroup, & Gerberding, 2004) found that the top three actual causes of death were due to three modifiable behavioral risk factors: (1) smoking (435,000 deaths; 18.1% of total deaths in the year

2000); (2) poor diet and physical inactivity (400,000 deaths; 16.6%); and (3) alcohol consumption (85,000 deaths; 3.5%). All three of these behaviors are risk factors for CVD and are considered preventable.

Primary prevention of CVD, like other chronic, noncommunicable diseases, is seen as more cost-effective than treatment of the disease, which is usually long term and expensive (Probst-Hensch, Tanner, Kessler, Burri, & Künzli, 2011). Practitioners of behavioral medicine can encourage their patients and the community at large to make lifestyle modifications to prevent the onset of CVD. These can include behavioral changes such as (1) getting regular health screenings; (2) limiting tobacco exposure; (3) engaging in regular physical activity; (4) eating a heart-healthy diet; (5) maintaining a healthy weight; (6) limiting alcohol use; and (7) reducing stress and negative emotionality. These seven behavioral changes are described in detail below.

Get Regular Health Screenings

Beginning at age 20, adults should get screenings for CVD risk factors at least every 2 years. These include blood pressure, body weight, waist circumference, and pulse (as a screen for atrial fibrillation). At least every 5 years (or less if at higher risk), blood lipids (either fasting serum lipoproteins or total and high-density lipoproteins [HDL] if fasting unavailable) and fasting blood glucose should be recorded to monitor risk for hyperlipidemia and diabetes (Pearson et al., 2002).

Blood pressure should be maintained at a level below 140/90 mmHg for the average individual whereas those with diabetes should maintain their blood pressure below 130/80 mmHg (Pearson et al., 2002). Individuals with hypertension can make behavioral modifications (e.g., limit salt intake, increase physical activity, and reduce alcohol intake). Blood pressure medications are recommended for individuals who have attempted lifestyle modifications but have not succeeded in controlling blood pressure.

Limit Tobacco Exposure

Individuals should avoid exposure to tobacco smoke as much as possible. Thus, they should not smoke cigarettes or other forms of tobacco, and those who do should quit. Exposure to secondhand smoke should be limited as well (Pearson et al., 2002). Tobacco use accounted for 18.1% of all deaths in the United States in the year 2000 and was the top behavioral risk factor for death (Mokdad et al., 2004). Cigarette smoking is one of the main risk factors for coronary heart disease, and women who smoke have a 25% greater risk of coronary heart disease than men after controlling for other risk factors (Huxley & Woodward, 2011).

Engage in Regular Physical Activity

The American Heart Association recommends that individuals engage in at least 30 min of moderate-intensity exercise most days of the week (Pearson et al., 2002). Exercise treats many CVD risk factors, including elevated blood pressure, insulin resistance, glucose intolerance, obesity, elevated triglycerides, and low HDL cholesterol (Thompson et al., 2003). Exercise also has short-term effects of reducing serum triglycerides for up to 72 h, introducing a spike in HDL, reducing systolic blood pressure for up to 12 h, and helping stabilize glucose levels (Thompson et al., 2003). Physical activity might also help individuals make other preferable behavior changes, including helping with smoking cessation (Ussher, Taylor, & Faulkner, 2008).

Eat a Heart-Healthy Diet

Individuals should eat a "heart-healthy" diet. This is a diet that is low in fat (saturated fat $<10\%$ of calories), cholesterol (<300 mg/day), and trans-fats (limit as much as possible), and salt (<6 g/day) and that is rich in assorted fruits, vegetables, whole grains, and low-fat dairy. Energy intake should match energy expenditure, i.e., intake should not exceed what is needed, and if necessary, intake should be reduced for weight loss (Pearson et al., 2002). Although there has been a major focus on *how much* individuals consume, there is evidence that *what* individuals eat is important to reduce CVD risk. In a meta-analysis of randomized clinical trials of dietary interventions in which patients were advised to

either (1) reduce total fat intake, (2) reduce saturated fat intake, (3) reduce dietary cholesterol, or (4) shift from saturated to unsaturated fat, modification in dietary fat intake reduced risk for cardiovascular mortality by 9% and reduced risk for subsequent cardiac events by 16% (Hooper et al., 2001).

Maintain a Healthy Weight

Body mass index (BMI; weight (kg)/height $(m)^2$) should be maintained in the normal range (18.5–24.9 kg/m^2) (Pearson et al., 2002). Waist circumference should be maintained at less than 40 in. in diameter for men and less than 35 in. in diameter for women (Pearson et al., 2002). In a meta-analysis of over 80,000 individuals, greater waist-to-hip ratio and waist circumference were associated with greater risk of CVD-related mortality, after controlling for other relevant CVD risk factors, over an average 98.7-month follow-up (Czernichow, Kengne, Stamatakis, Hamer & Batty 2011). However, BMI, the most commonly used measure of obesity, was not related to CVD-related mortality after controlling for other risk factors, suggesting that some (if not all) of the risk that higher BMI contributes to CVD mortality is subsumed by the other related risk factors (e.g., blood pressure, cholesterol).

Limit Alcohol Use

Alcohol use should be limited to $\leq$2 drinks/day for men ($\leq$14 drinks/week) and $\leq$1 drink a day for women ($\leq$7 drinks/week) (Pearson et al., 2002). In a meta-analysis of prospective cohort studies linking alcohol use to cardiovascular outcomes, including mortality and cardiovascular event morbidity, moderate consumption of alcohol (about 1 drink/day) was associated with a 14–25% reduced risk of all cardiovascular outcomes when compared to abstainers (Ronksley, Brien, Turner, Mukamal, & Ghali 2011). Conversely, consuming higher amounts of alcohol (>1 drink/day) was associated with higher probability of CVD-specific mortality and cardiac events.

Reduce Stress and Negative Emotionality

Individuals should limit their exposure to stress and promote positive rather than negative emotions. There is evidence that negative emotions (i.e., depression, anxiety, and anger) are related to the development of CVD. Multiple pathways have been proposed to explain the relation between negative affect and CVD risk, and these include engagement in more adverse health behaviors, greater stress exposure, greater physiological reactivity, lower heart rate variability, and greater inflammation (cf., Suls & Bunde, 2005, for a review). In particular, type D personality, a personality type comprised of negative emotionality and social inhibition, is positively related to cardiac events. A recent meta-analysis by O'Dell, Masters, Spielmans, and Maisto (2011) revealed that patients with type D personalities were three times more likely to experience myocardial infarction, coronary artery bypass grafting, percutaneous cardiac intervention, or cardiac mortality than non-type D individuals. Conversely, there is preliminary evidence that some psychosocial resources may provide a protective buffer against cardiovascular risk. Roepke and Grant (2011) reviewed 32 studies of personal mastery (i.e., the belief that one has some control over future life circumstances) and cardiometabolic health and revealed that the overwhelming majority of studies found that higher mastery was associated with a reduced risk for cardiovascular outcomes.

Conclusions

Cardiovascular disease is largely preventable when individuals practice a healthy lifestyle. This includes practicing health-promoting behaviors like engaging in regular physical activity, eating a heart-healthy diet, maintaining a healthy weight, and getting regular health screenings and avoiding health-compromising behaviors like risky alcohol use and tobacco use. Additionally, individuals can make attempts to reduce their experiences of stress and negative emotions and promote positive emotional experiences. The combination of these behaviors will help reduce the risk of CVD and promote longer, healthier lives.

Cross-References

► Cardiovascular Disease

References and Readings

Czernichow, S., Kengne, A., Stamatakis, E., Hamer, M., & Batty, G. (2011). Body mass index, waist circumference and waist-hip ratio: Which is the better discriminator of cardiovascular disease and mortality risk? Evidence from an individual-participant meta-analysis of 82 864 participants from nine cohort studies. *Obesity Reviews, 12*, 680–687.

Hooper, L., Summerbell, C. D., Higgins, J. P. T., Thompson, R. L., Capps, N. E., Smith, G. D., et al. (2001). Dietary fat intake and prevention of cardiovascular disease: Systematic review. *BMJ, 322*, 757–763 (Clinical Research Editions).

Huxley, R., & Woodward, M. (2011). Cigarette smoking as a risk factor for coronary heart disease in women compared with men: A systematic review and meta-analysis of prospective cohort studies. *Lancet, 6736*, 1–9.

Mokdad, A., Marks, J., Stroup, D., & Gerberding, J. (2004). Actual causes of death in the United States, 2000. *JAMA: The Journal of the American Medical Association, 291*, 1238–1245.

O'Dell, K., Masters, K. S., Spielmans, G. I., & Maisto, S. A. (2011). Does Type-D personality predict outcomes among patients with cardiovascular disease? A meta-analytic review. *Journal of Psychosomatic Research, 71*, 199–206.

Pearson, T. A., Blair, S. N., Daniels, S. R., Eckel, R. H., Fair, J. M., Fortmann, S. P., et al. (2002). AHA guidelines for primary prevention of cardiovascular disease and stroke: 2002 update: Consensus panel guide to comprehensive risk reduction for adult patients without coronary or other atherosclerotic vascular diseases. *Circulation, 106*, 388–391.

Probst-Hensch, N., Tanner, M., Kessler, C., Burri, C., & Künzli, N. (2011). Prevention: A cost-effective way to fight the non-communicable disease epidemic. *Swiss Medical Weekly, 141*, 1–8.

Roepke, S., & Grant, I. (2011). Toward a more complete understanding of the effects of personal mastery on cardiometabolic health. *Health Psychology, 30*, 615–632.

Ronksley, P., Brien, S., Turner, B., Mukamal, K., & Ghali, W. (2011). Association of alcohol consumption with selected cardiovascular disease outcomes: A systematic review and meta-analysis. *BMJ, 342*, 1–13.

Suls, J., & Bunde, J. (2005). Anger, anxiety, and depression as risk factors for cardiovascular disease: The problems and implications of overlapping affective dispositions. *Psychological Bulletin, 131*, 260–300.

Thompson, P., Buchner, D., Piña, I., Balady, G. J., Williams, M. A., Marcus, B. H., et al. (2003). Exercise and physical activity in the prevention and treatment of atherosclerotic cardiovascular disease: A statement from the Council on Clinical Cardiology (Subcommittee on Exercise, Rehabilitation, and Prevention) and the Council on Nutrition, Physical Activity, and Metabolism (Subcommittee on Physical Activity). *Arteriosclerosis, Thrombosis, and Vascular Biology, 8*, e42–e49.

US Centers for Disease Control and Prevention. (2009). *Heart disease prevention*. Retrieved August 28, 2011, from http://www.cdc.gov/heartdisease/prevention.htm

US Centers for Disease Control and Prevention. (2010). Deaths: Final data for 2007. *National Vital Statistics Report, 58*. Accessed March 21, 2011, from http://www.cdc.gov/NCHS/data/nvsr/nvsr58/nvsr58_19.pdf

Ussher, M. H., Taylor, A., & Faulkner, G. (2008). Exercise interventions for smoking cessation (Review). *Cochrane Database of Systematic Reviews, 4*, 1–37.

Cardiovascular Medicine

► Cardiology

Cardiovascular Medicine Expert

► Cardiologist

Cardiovascular Psychophysiology: Measures

► Cardiovascular Recovery
► Psychophysiology: Theory and Methods

Cardiovascular Recovery

William Gerin
The College of Health and Human Development, University Park, PA, USA

Definition

Cardiovascular recovery refers to the extent to which elevations in blood pressure (BP) or heart rate (HR) due to a stressor persist after the stressor is no longer present.

Description

Recovery of cardiovascular prestress resting levels following a stressor has been of interest to researchers for many years, going back to the original cardiovascular reactivity studies by Hines and Brown (1936), who noted that not only did hypertensive patients show greater responses to the cold pressor than normotensive controls but also recovered more slowly. Since then, evidence from several studies suggests that recovery may provide prognostic information concerning the development of cardiovascular diseases, such as hypertension and coronary heart disease (CHD) (Fredrickson & Matthews, 1990).

Cardiovascular reactivity (the magnitude of the acute BP or HR response to a stressor) has been implicated as a risk factor in the development of cardiovascular-related diseases (i.e., the "reactivity hypothesis"). However, this measure does not take into account the duration of the response (i.e., recovery; the time BP or HR takes to return to prestress baseline levels from elevations due to a stressor that is no longer present). There is strong evidence that sustained elevation of BP is a cause of target organ damage, e.g., left ventricular hypertrophy, and, over time, of essential hypertension (HTN) (Fredrickson & Matthews, 1990). To the extent that we may regard slow recovery periods, observed in the laboratory, as analogous to sustained elevated BP in the natural environment, this measure provides information over and above that of the magnitude of the response. However, there is not nearly the body of evidence examining the causes and effects of recovery as there is for reactivity.

Much of the evidence that does exist for recovery comes from cross-sectional comparisons, examining variables that are related to the development of cardiovascular diseases and including comparisons based on normotensive-hypertensive status; family history of hypertension; and ethnicity. For example, children of normotensive parents show more rapid recovery than children of hypertensives (Linden, Earle, Gerin, & Christenfeld, 1997). It is noteworthy that in this review, differences in *reactivity* were *not* observed among the groups. Hines & Brown (1936) found that hypertensive subjects showed longer recovery times than normotensives. Finally, studies examining race have found that Black women and men had slower recovery rates than White women and men (Linden et al., 1997).

Thus, several important risk factors for hypertension and CHD appear to influence BP and HR return to prestress levels following a stressor. The recovery data are important, especially given findings showing that in a sample of borderline hypertensives, a strong predictor of future stable hypertension was the recovery of diastolic BP following a mental arithmetic task (Borghi, Costa, Bocshi, Mussi, & Ambrosioni, 1986). In fact, these researchers found recovery a more useful predictor than reactivity.

There are in theory many reasons why recovery should be poorer in one individual than in another. In general, the mechanisms could be central or peripheral. An example of the former would be a persistence of the autonomic arousal, or an inability to "unwind" following exposure to a stressor. A second mechanism could be an impairment of baroreflex sensitivity. The function of the baroreflex is to buffer acute changes of blood pressure, and an insensitive reflex would result in an enhanced and protracted pressor response to a stimulus. A third, and peripheral, mechanism is changes in the vasculature, such as hypertrophy and remodeling, which could result in delayed relaxation of vascular smooth muscle following exposure to a vasoconstrictor stimulus.

There is evidence that recovery and reactivity represent independent dimensions. For example, in one review paper, reactivity changes were weakly correlated with recovery scores (Linden et al., 1997). In addition, Haynes, Gannon, Orimoto, O'Brien, and Brandt (1991) reported that, across a total of 65 studies, of the 81 statistical analyses (out of 180) that indicated *nonsignificant stressor effects* (i.e., reactivity) for a variable (e.g., between group, between phases), significant effects *were* found during the *recovery* phase for 74% of the same variables. Conversely, of the 74 statistical analyses that indicated *significant* stressor effects for a variable, nonsignificant effects were found during the *recovery*

phase for 42% of those same variables. This is important because it suggests that the physiological mechanisms underlying the two processes of reactivity and recovery must be considered separately and that the information contained in *both* measures may provide greater insight into the cardiovascular mechanisms underlying the stress response than either measure alone. These considerations have led a number of researchers to suggest that causal explanations of biobehavioral disorders and the design of clinical interventions may be well served by studying psychophysiological recovery.

Early Theories of Stress and Recovery

As early as the 1930s, the seminal theories of stress, and the optimal ways to respond to stress, were proposed. For example, Seyle (1936) proposed that stress has three phases: activation, resistance, and exhaustion. When the body is initially challenged by a stressor, it responds with physiological activation of a defense system to deal with the immediate stressor, what is often referred to as the fight-or-flight response. A resistance (or coping) phase follows during which the body begins to suffer from the effects of heightened activity, but continues to function to ward off the stress-inducing stimuli. If the resolution of stress is unsuccessful the body may experience exhaustion. Activation that endures beyond the resistance stage (i.e., does not lead to swift resolution) is presumed to contribute to disease. At about the same time, Freeman (1939) posited that psychological recovery from experimental loads may be useful in estimating an individual's ability to withstand conflict in ordinary life situations. Such early suggestions that quick recovery from stress-induced arousal reflects particularly effective coping laid the foundation for later theories of the stress-disease linkage.

Subsequent work has thus refined these theories, and posited biological mechanisms for how stress impacts the body and contributes to disease. Important to refinement was the altering of Seyle's notion from a ubiquitous, "whole-system" response to challenge, to one that distinguished at least two axes of physiological responding. The sympatho-adrenal axis reflects activation due to motor and cognitive effort, including rises in epinephrine, norepinephrine, muscle tension, plasma free fatty acid levels, and blood pressure due to cardiac output. This activation when accompanied by adrenocortical hormone suppression has also been described as a "positive stress reaction" because it is short-lived and permits adaptive responding with maximal strength (De La Torre, 1994). In contrast, the hypothalamic-pituitary axis (HPA) is thought to reflect affective distress and be the result of chronic, unresolved strain (De La Torre), and may be the most indicative bodily response during delayed recovery. HPA axis activity is associated with increased release of free fatty acid into circulation, suppression of immune function, increased glucose and urea production, and increased blood pressure due to vasoconstriction (i.e., total peripheral resistance); HPA activation is inferred from the measurement of cortisol and its precursor adrenocorticotropic hormone (ACTH).

Cognitive-Affective Determinants of Poorer Recovery

A fundamental question that arises concerns how acute stressors can have lasting effects for some but not others, or put another way, why some individuals have poorer recovery than others. Brosschot, Gerin, and Thayer (2005) have proposed that the tendency to relive stressors in one's mind (i.e., ruminate and worry) causes repeated HPA activation and results in negative health outcomes, such as sustained hypertension. In other words, people do not need an external stressor to be present to experience stress. Rather, stress can have longer durations and impacts on the body, simply through thinking about and remembering negative emotions and having persistent thoughts about the negative experiences. Furthermore, it is those individuals who continually experience the mental representation of stress that have poor recovery and ultimately poor health. This focus on the cognitive-affective determinants of poorer recovery has begun to be seen as an important area of study, augmenting reactivity models.

Conclusion

In sum, the duration of time it takes for an individual's cardiovascular system to return to resting levels is a key determinant of that person's health. Furthermore, the duration of experienced stress (i.e., recovery) is an independent and often more important predictor of future health than the magnitude of the stress response (i.e., reactivity). Current models of hypertension and cardiovascular disease are beginning to focus on delayed recovery as an essential variable to consider. This work, and future explorations, will need to consider the role that perseverative cognitions, such as rumination, play in delaying cardiovascular recovery. While the effects of the arrangement of stress in one's environment cannot be ignored, how stress is arranged in one's mind appears to be as important a factor to determining one's health.

Cross-References

- ▶ Blood Pressure Reactivity or Responses
- ▶ Cardiovascular Disease
- ▶ Hypertension
- ▶ Perseverative Cognition
- ▶ Psychophysiologic Recovery
- ▶ Rumination

References and Readings

Borghi, C., Costa, F. V., Bochi, S., Mussi, A., & Ambrosioni, E. (1986). Predictors of stable hypertension in young borderline subjects: A five year follow-up study. *Journal of Cardiovascular Pharmacology, 8*, S138–S141.

Brosschot, J. F., Gerin, W., & Thayer, J. F. (2005). The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *Journal of Psychosomatic Research, 60*, 113–124.

De La Torre, B. (1994). Psychoendocrinologic mechanisms of life stress. *Stress Medicine, 10*, 107–114.

Fredrickson, M., & Matthews, K. A. (1990). Cardiovascular responses to behavioral stress and hypertension: A meta-analytic review. *Annals of Behavioral Medicine, 12*, 30–39.

Freeman, G. L. (1939). Toward a psychiatric Plimsoll mark: Physiological recovery quotients in experimentally induced frustration. *Journal of Psychology, 8*, 247–252.

Gerin, W. (2010). Laboratory stress testing methodology. In A. Steptoe (Ed.), *Handbook of behavioral medicine: Methods and applications*. New York: Springer.

Haynes, S. N., Gannon, L. R., Orimoto, L., O'Brien, W. H., & Brandt, M. (1991). Psychophysiological assessment of poststerss recovery. *Journal of Consulting and Clinical Psychology, 3*, 356–365.

Hines, E. A., & Brown, G. E. (1936). The cold pressor test for measuring the reactibility of the blood pressure: Data concerning 571 normal and hypertensive subjects. *American Heart Journal, 11*, 1–9.

Linden, W., Earle, T. L., Gerin, W., & Christenfeld, N. (1997). Physiological stress reactivity and recovery: Conceptual siblings separated at birth? *Journal of Psychosomatic Research, 42*, 117–135.

Obrist, P. A. (1981). *Cardiovascular psychophysiology: A perspective*. New York: Plenum Press.

Seyle, H. (1936). A syndrome produced by diverse nocuous agents. *Nature, 138*, 32.

Turner, J. R. (1994). *Cardiovascular reactivity and stress: Patterns of physiological response*. New York: Plenum Press.

Cardiovascular Response/Reactivity

- ▶ Blood Pressure Reactivity or Responses

Cardiovascular Risk Factors

Rachel S. Rubinstein and Richard J. Contrada
Department of Psychology, Rutgers, The State University of New Jersey, Piscataway, NJ, USA

Synonyms

Protective factors; Psychosocial factors

Definition

A cardiovascular risk factor is a predictor of one or more diseases of the heart or circulation.

Description

A "risk factor" is a variable that bears an empirical association with one or more diseases or other

medical conditions. "Cardiovascular disease" and "heart disease" refer to a set of specific disorders that affect the heart and circulation. Therefore, a cardiovascular risk factor is a correlate of one or more cardiovascular diseases. A distinction is sometimes made between risk and protective factors as a way to capture the direction of the relationship. For example, elevated cholesterol is a risk factor, whereas social integration has been studied for its possible protective effects.

The identification of risk factors for medical conditions is a major goal of the field of epidemiology. The term "risk factor" was introduced in the context of cardiovascular epidemiology, a field that underwent great expansion during the twentieth century. Certain acute infectious conditions that had been the major sources of death in the early 1900s came under control as a result of advances in the fields of public health and biomedicine. As a consequence, several multiply determined chronic disorders became more prevalent. Chronic illnesses, and diseases of the heart and blood vessels in particular, became the leading sources of death in the United States and in other industrialized nations. This continues to be the case despite reductions in cardiovascular death over the past several decades. Many risk factors for cardiovascular disorders have been identified. They may be categorized in a number of different ways, for example, in terms of the particular form(s) of cardiovascular disease with which they are related or on the basis of characteristics of the risk factors themselves.

Major Cardiovascular Disorders

Among the various forms of cardiovascular disease (CVD), coronary heart disease (CHD) is a major contributor to cardiovascular morbidity and mortality. Also referred to as ischemic heart disease, CHD occurs when the heart is inadequately supplied with oxygenated blood. It has several clinical manifestations including angina pectoris (a syndrome involving chest pain and other symptoms), myocardial infarction (MI) or "heart attack" (death of a portion of the myocardium), and sudden cardiac death (death within minutes of symptom onset). Coronary heart disease can promote other cardiovascular disorders, such as when damage due to myocardial infarction leads to congestive heart failure, a condition in which the pumping action of the heart cannot adequately meet the demands of the body for oxygen and nutrition. Atrial fibrillation is one of several forms of CVD that involve a disturbance in heart rhythm and may be a consequence of MI or heart failure.

Clinical CHD is usually a consequence of coronary atherosclerosis, or coronary artery disease (CAD). Coronary atherosclerosis involves the accumulation of plaque, comprising fatty substances and other materials, which forms on the inner lining of the coronary arteries, the vessels that supply oxygenated blood to heart muscle. The buildup of atherosclerotic plaque, which reflects a number of metabolic, hemodynamic, inflammatory, and hematologic processes, culminates in CHD when blood vessel openings become narrowed to the point at which blood flow to the heart muscle is obstructed and can no longer meet its metabolic demands. The triggering event in CHD is often thrombosis (clot formation) leading to occlusion of an already narrowed coronary artery. The atherosclerotic process also may affect blood vessels of the brain, leading to cerebrovascular incidents commonly referred to as "stroke."

Still another form of cardiovascular disease is essential hypertension, a condition defined by sustained high blood pressure levels with no identifiable cause. Hypertension is often associated with vascular inelasticity, referred to as arteriosclerosis. Hypertension increases risk for CHD as well as for stroke, retinopathy, heart failure, and kidney disease.

Risk factors differ somewhat for different forms of CVD. For example, cigarette smoking is a well-established risk factor for MI and other forms of CHD, but its role in the development of essential hypertension is less clear. On the other hand, dietary intake of salt, a possible risk factor for essential hypertension in some segments of the population, has a less well-established relationship with other types of heart disease. Similarly, certain kinds of heart valve problems

more clearly operate as predisposing factors for heart rhythm disturbances than for other cardiovascular conditions.

Taken together, the multifaceted nature of cardiovascular diseases, complexities in interrelationships among its various forms, and their overlapping but nonidentical determinants complicate the description and classification of cardiovascular risk factors. However, clinical manifestations of CHD and other major forms of CVD reflect a common substrate, atherosclerosis and, in the aggregate, account for considerable morbidity and mortality. This provides an important focus for much research in this area. Moreover, although genetic and other biological risk factors may be in play from the time of birth, much of the burden of cardiovascular diseases reflects the operation of multiple aspects of lifestyle, as will be discussed further below. This raises the possibility that programs of primary prevention may bring about significant reductions in the burden of cardiovascular disease.

Attributes of Risk Factors

Risk factor status, by itself, does not imply causality. The case for a causal role requires several forms of evidence. For example, there should be an association with disease in well-designed studies that is consistent, strong, prospective (rather than merely cross-sectional), and independent of other possible risk factors. The putative risk factor also should show a dose–response relationship with disease outcomes and should plausibly be related to disease etiology and pathophysiology, and its removal should reduce the risk of disease. Although causal analysis often follows after a variable is identified empirically as a risk factor, in other cases a variable is examined as a possible risk factor only after a role in disease causation is first suggested in mechanistic research.

The strength of the relationship between a risk factor and disease is often expressed in terms of relative risk. Relative risk is the ratio of the probability of disease occurrence with and without the risk factor in question. For example, a relative risk of 2.0 would indicate that individuals with the risk factor are twice as likely (or, equivalently, 100% more likely) to develop the condition by comparison with those without the risk factor. Major risk factors for one or more cardiovascular conditions are those for which a significant relative risk is well established. Variables for which the evidence is less clear are sometimes referred to as contributing risk factors. The more risk factors that are present, and the higher the level of each one, the greater the risk for CVD.

In addition to its relative risk, attributes of a cardiovascular risk factor that determine its overall importance from a public health standpoint include its prevalence, the degree to which it can be readily modified, and the impact of its modification on CVD outcomes. Several risk factors, such as resting blood pressure and cholesterol level, can be modified, and their modification is associated with reductions in cardiovascular morbidity and mortality. In other cases, including that of many suspected psychosocial variables, there are questions either about the effects of interventions on the risk factor or about the impact of risk factor modification on CVD outcomes. Still other risk factors, such as gender and age, are clearly not modifiable, though it remains possible that some of the pathways through which they exert their effects are amenable to intervention.

Risk Factor Categories

In addition to distinctions in terms of the CVD outcomes with which they are associated, CVD risk factors may be distinguished on the basis of their intrinsic characteristics. One rather broad distinction is that between states or conditions of the person and those that describe the environment. Person factors include variables such as resting blood pressure, gender, and personality. Environmental factors may be defined geographically, in terms of regions, such as the Southeastern United States (where CVD is highly prevalent), or with respect to variables such as the average socioeconomic status of individuals residing in a particular community (which is inversely associated with CVD risk).

Risk factors also may be categorized in terms of the time point and chronicity of their influence on the natural history of CVD. The hypothesized pathogenic effect may be to promote progression of CAD, as in the case of variables related to blood cholesterol or sugar levels. Alternatively, a risk factor may be suspected of playing a role in the manifestation of clinical CHD, as where an acute stressor triggers an ischemic event in the context of previously asymptomatic CAD. Within these major disease phases, risk factors may exert their influence gradually, over the course of months and years, or more rapidly, within days, hours, or even minutes.

Risk factors also may be described at several levels of analysis. Many are biological, ranging from the molecular, for example, specific genetic polymorphisms and proinflammatory factors, to systemic physiological conditions such as high resting blood pressure. Some risk factors are behavioral, including cigarette smoking, physical inactivity, and various dietary practices, and some are psychological, including personality attributes like cynical hostility and mood and anxiety disorders like major depression and posttraumatic stress disorder. Still others are described at a social level of analysis, including social network characteristics and socioeconomic status.

Risk Factor Interactions

Risk factors for CVD do not operate in isolation from one another. Individual variables may share causal antecedents, influence one another directly, and exert additive or synergistic effects in the etiology and pathogenesis of disease. For example, CVD risk factors such as poor diet and exercise habits combine to promote obesity and high cholesterol levels, which are themselves CVD risk factors. Functional relationships among a set of multiple determinants of a single outcome suggest that it may be useful to consider them in combination. A case in point is cardiometabolic syndrome, a biological CVD risk factor defined as a cluster consisting of central obesity, hypertension, and dysregulation in glucose and fat metabolism. The disease-promoting effects of certain risk factors are thought to amplify those of others. In particular, cigarette smoking has been examined for its possibly interactive effects with other variables including genes, high blood pressure, and oral contraceptive use.

Traditional and Psychosocial Risk Factors

Historically, those risk factors that were identified early on or that fit within the original paradigm for understanding cardiovascular diseases have been referred to as "traditional" or "biomedical." Variables that exemplify this tradition and that are recognized by contemporary epidemiologists include cigarette smoking, resting blood pressure, cholesterol levels, diabetes, older age, male gender, specific genetic markers, and family history; also implicated are obesity, physical inactivity, a high fat, high carbohydrate diet, low levels of high-density lipoprotein cholesterol, high levels of triglycerides, and high levels of C-reactive protein and other inflammatory markers.

Beginning in the middle of the twentieth century and continuing today, a very large body of research has sought to identify additional types of risk factors for CVD, including a number of social and psychological variables. These efforts were stimulated by limitations in the predictive power of the more traditional risk factors, theoretical and empirical work concerning the effects of psychological stress and emotion on cardiovascular physiology, and informal observations by clinicians and empirical research findings that were suggestive of psychosocial influences on CVD. Many of the more recently identified risk factors (some of which are discussed below) are characteristics of the person and social context that are referred to as "psychosocial." Recognition of the potential importance of psychosocial CVD risk factors contributed significantly to the emergence and growth of the fields of health psychology, behavioral medicine, and behavioral cardiology.

Lifestyle as the Major Determinant of CVD

The designation of many of the traditional CVD risk factors as "biomedical" is something of a misnomer. For example, four major

cardiovascular risk factors identified in early epidemiological work, and still the target of considerable research, are cigarette smoking, resting blood pressure, cholesterol levels, and blood sugar problems including diabetes. Cigarette smoking is, of course, a behavior pattern, and although it is maintained, in part, by physiological processes of nicotine addiction, its initiation and natural history also reflect social and psychological influences. Similarly, blood pressure, cholesterol, and blood sugar are to some extent regulated by specific behaviors such as diet and exercise and also may reflect psychosocial influences such as stress and emotion.

Given that most forms of CVD take decades to develop, recognition that many of the traditional risk factors reflect aspects of lifestyle has important public health implications. One is that efforts to prevent CVD should begin early in life. Behavior patterns such as cigarette smoking and those involved in weight regulation and nutrition begin during or even before adolescence. Tobacco use has a devastating effect on health, including cancer and respiratory consequences as well as CVD, and recent trends involving the earlier emergence of obesity and, relatedly, diabetes mellitus, are alarming in light of their projected impact on trends in the prevalence of heart disease. When combined with possible psychosocial determinants of CVD, which in many cases also may begin to emerge in the earlier years of life, the need for a life span perspective on CVD risk reduction becomes quite clear. The promotion and maintenance of a healthy lifestyle in young people has the greatest potential for reducing the lifetime burden of cardiovascular diseases.

Stress and Emotional Dispositions

Several promising psychosocial risk factors for CVD involve the concept of psychological stress. Psychological stress entails (a) stressors, or environmental events and conditions that place demands and constraints on a person's adaptive resources; (b) psychological responses to stressors, including perceptual-evaluative (appraisal) processes that initiate stress and emotion processes, and cognitive and behavioral responses, including coping activity, that may counteract or exacerbate stressors and their impact; and (c) biological responses, including neuroendocrine, autonomic, cardiovascular, and immunological/inflammatory perturbations that are potentially damaging to cardiovascular health. Among stressors that have been linked empirically to CVD outcomes are major life events, occupational stress, and marital conflict.

Psychosocial factors that in some way interact with psychological stress also have received attention as possible CVD risk factors. One such construct, the type A behavior pattern (TABP), formed the foundation for contemporary work on psychosocial factors in CVD. Type A refers to a set of behaviors that include competitiveness and achievement striving, impatience and time urgency, hostility and anger, and vigorous speech and motor characteristics (Friedman & Rosenman, 1974). Type B refers to a more relaxed, less impatient, and less irritable pattern of behavior. Type A was conceptualized as the outcome of a person-situation interaction in which its defining features are displayed in response to stressful and challenging events and conditions in susceptible individuals. The TABP construct initially attracted considerable attention for a prospective association with CHD that was independent of traditional risk factors such as cholesterol levels, resting blood pressure, and cigarette smoking (Rosenman et al., 1975). Subsequent research did not fully confirm these findings, resulting in diminished interest in the TABP (Matthews, 1988).

About this time, evidence began to emerge to suggest that hostility and anger form the risk-enhancing components of the TABP. Prospective studies of hostility and anger constructs and different forms of anger expression have yielded promising findings (Kent & Shapiro, 2009). Much of this research has relied on the Ho Scale first described by Cook and Medley (1954). It appears that hostility, characterized by cynicism and interpersonal mistrust, may be related to CAD-related outcomes, although negative findings have been reported as well (Kent & Shapiro, 2009).

More recently, depression has been identified as a potentially potent independent predictor of

CHD in healthy populations and as a factor that may contribute to both the manifestation and worsening of CHD among coronary patients. In addition, depression is associated with several major cardiac risk factors (e.g., hypertension, physical inactivity). Various forms, severity levels, and symptoms of depression have been examined in this regard. Findings indicate that depressive symptoms and major depression are associated with increased cardiovascular morbidity and mortality, even after controlling for other risk factors (Kent & Shapiro, 2009). Further, major depression is associated more strongly with adverse cardiac events than is the presence of subclinical depressive symptoms (Rozanski, Blumenthal, Davidson, Saab, & Kubzansky, 2005).

In addition, depressed individuals are more likely than nondepressed individuals to have more than one risk factor for CVD, which may indicate that the association between depression and CVD is due, in part, to the combination of risk factors rather than to each risk factor considered independently (Joynt, Whellan, & O'Connor, 2003). As with hostility, there are some inconsistencies in this research. Nonetheless, the sheer volume of findings that support depression as a CHD risk factor builds a strong case in its favor (Kent & Shapiro, 2009).

A third emotional disposition that has been implicated as a possible CVD risk factor is anxiety. Research has revealed a link between anxiety and the development of CVD in physically healthy populations, but evidence for this association has been mixed (Suls & Bunde, 2005). Studies of populations with known CHD have also yielded inconsistent findings, with some reporting null effects and others finding an inverse relationship (Suls & Bunde). To assess anxiety, some researchers use diagnostic interviews and clinical criteria, whereas others use self-report measures. Generally, results supporting anxiety as a CVD risk factor are more consistent in samples of initially healthy individuals than in CVD patients. This may signify that negative emotions constitute a greater risk for the development of CVD than for its progression. Inconsistencies in the findings also may reflect difficulty in assessing anxiety in the context of a medical condition and hospitalization, and in differentiating a temporary state of anxiety from chronic anxiety (Suls & Bunde). As with depression, anxiety has been examined both as a subclinical dimension of individual differences and in terms of clinical conditions such as posttraumatic stress disorder.

Still another emotional disposition that may operate as a CVD risk factor, neuroticism, refers to individual differences in irritability, anger, sadness, anxiety, worry, hostility, self-consciousness, and vulnerability in response to threat, frustration, or loss. Initially, neuroticism was not thought to play a causal role in CVD. Instead, the association with CAD was thought to reflect effects on somatic complaints and health-care-seeking behaviors (Costa & McCrae, 1987). However, an expanding body of evidence implicates neuroticism as a possible causal agent in multiple mental and physical disorders, including CVD (Lahey, 2009). Neuroticism is thought to contribute to health risk through the experience of more stressors, less social support, and greater likelihood to engage in risky behaviors. Given that neuroticism incorporates anger, sadness, and anxiety, and in light of positive associations among these emotional dispositions when measured separately, questions have been raised about the independence of these variables and their possible interactions, especially since few studies have examined two or more of them simultaneously (Suls & Bunde, 2005).

Social Factors

In addition to these dispositional constructs, some CVD risk factors are present in an individual's social environment. One example is low social support (Krantz & McCeney, 2002). Social support refers to the availability of a variety of social contacts from whom to derive benefits. Such benefits include emotional support, tangible aid, feelings of belonging, and informational support. Social support is associated with other factors that are related to health such as socioeconomic status and medication compliance. Prospective studies have found an association between low social support and risk of CVD.

Particular emphasis has been placed on stress as a mechanism underlying the association between low social support and CVD, although relevant investigations have yielded divergent findings (Uchino, Cacioppo, & Kiecolt-Glaser, 1996). It appears social networks may be cardioprotective as a result of their stress-buffering effects, but they also may operate independently of stress, for example, by promoting healthy behaviors and discouraging unhealthy ones.

Another social contextual factor that has been identified as a CVD risk factor is low socioeconomic status (SES). SES has been defined in terms of a person's occupation, economic resources, social standing, and education. There is considerable support for the existence of an SES health gradient that affects many diseases, including CVD. Higher SES is associated with better general health, less chronic illness, and decreased mortality. That this association is evident throughout the SES spectrum suggests that its effects cannot be completely explained by the effects of poverty on access to affordable health care. Relevant mechanisms may include cognitive and emotional processes, as well as psychosocial factors including social support (Marmot et al., 1991).

Identification of possible psychosocial risk factors for CVD gave rise to research on explanatory mechanisms. These may be described in terms of three major categories, namely, stress-related physiological activity; behaviors that may promote CVD in initially healthy individuals, including other CVD risk factors such as cigarette smoking, sedentary lifestyle, and poor diet; and cognitive and affective responses to illness and its treatment once CVD has developed, including processes culminating in delay in health-care seeking and treatment noncompliance.

Of the many theoretical and empirical contributions to emerge from mechanism-focused work on psychosocial CVD risk factors, perhaps the most significant development was formulation of the reactivity hypothesis. Reactivity refers to changes in physiologic activity associated with psychological stress, including alterations in neuroendocrine, autonomic, hemodynamic, hematologic, and immunological/inflammatory processes. Beginning with research on TABP, findings began to emerge in which emotional attributes and social-contextual factors moderated the effects of psychological stress on one or more physiological response measures. In addition, accumulating evidence suggested that physiologic reactivity represents a dimension of individual differences that is consistent across different psychological stressors and stable over time. It appears related to or may even constitute a form of emotional volatility that runs through emotion constructs discussed above including anger/hostility, depression, anxiety, and neuroticism. These findings, in turn, have led to the hypothesis that physiological reactivity might operate as an independent CVD risk factor and to empirical observations linking reactivity to CVD outcomes including the development of CHD and essential hypertension and the precipitation of acute episodes of myocardial ischemia and other cardiac events.

Conclusion

Although the risk and protective factors described above have received considerable attention, they are not exhaustive of the constructs that have been examined as potential causes of CVD. Many other variables have been investigated, including macrosocial factors such as culture, political systems, and migration; additional forms of stress, such as racism; emotional dispositions such as Type D behavior (negative emotions accompanied by social inhibition); social and personal forms of religion and spirituality; specific behaviors such as alcohol consumption; and various infectious conditions and biomarkers. These efforts are fueled by the need to identify additional risk factors to account more completely for new cases of CVD and to improve the public health benefits of risk factor modification for this multiply determined set of chronic lifestyle disorders.

Cross-References

- ▶ Anxiety and Heart Disease
- ▶ Cardiovascular Disease Prevention
- ▶ Depression: Symptoms
- ▶ Epidemiology
- ▶ Fibrinogen
- ▶ Psychological Stress
- ▶ Social Inhibition
- ▶ Social Relationships
- ▶ Social Support
- ▶ Stress Vulnerability Models

References and Readings

Contrada, R. J., & Baum, A. (2011). *Handbook of stress science: Biology, psychology, and health*. New York: Springer.

Cook, W. W., & Medley, D. M. (1954). Proposed hostility and pharisaic-virtue scales for the MMPI. *Journal of Applied Psychology, 38*, 414–417.

Costa, P. T., & McCrae, R. R. (1987). Neuroticism, somatic complaints, and disease: Is the bark worse than the bite? *Journal of Personality, 55*, 299–316.

Friedman, M., & Rosenman, R. H. (1974). *Type A behavior and your heart*. New York: Knopf.

Joynt, K. E., Whellan, D. J., & O'Connor, C. M. (2003). Depression and cardiovascular disease: Mechanisms of interaction. *Biological Psychiatry, 54*, 248–261.

Kent, L. M., & Shapiro, P. A. (2009). Depression and related psychological factors in heart disease. *Harvard Review of Psychiatry, 17*, 377–388.

Krantz, D. S., & McCeney, M. K. (2002). Effects of psychological and social factors on organic disease: A critical assessment of research on coronary heart disease. *Annual Review of Psychology, 53*, 341–369.

Lahey, B. B. (2009). Public heath significance of neuroticism. *American Psychologist, 64*, 241–256.

Marmot, M. G., Stansfeld, S., Patel, C., North, F., Head, J., White, I., et al. (1991). Health Inequalities among British civil servants: The Whitehall II study. *Lancet, 337*, 1387–1393.

Matthews, K. A. (1988). Coronary heart disease and Type A behaviors: Update on and alternative to the Booth-Kewley and Friedman (1987) quantitative review. *Psychological Bulletin, 104*, 373–381.

Rosenman, R. H., Brand, R. J., Jenkins, C. D., Friedman, M., Straus, R., & Wurm, M. (1975). Coronary heart disease in the Western Collaborative Group Study. *Journal of the American Medical Association, 233*, 872–877.

Rozanski, A., Blumenthal, J. A., Davidson, K. W., Saab, P. G., & Kubzansky, L. (2005). The epidemiology, pathophysiology, and management of psychosocial risk factors in cardiac practice: The emerging field of behavioral cardiology. *Journal of the American College of Cardiology, 45*, 637–651.

Suls, J., & Bunde, J. (2005). Anger, anxiety, and depression as risk factors for cardiovascular disease: The problems and implications of overlapping affective dispositions. *Psychological Bulletin, 131*, 260–300.

Uchino, B. N., Cacioppo, J. T., & Kiecolt-Glaser, J. K. (1996). The relationship between social support and physiological processes: A review with emphasis on underlying mechanisms and implications for health. *Psychological Bulletin, 119*, 486–531.

Cardiovascular Stress Responses

- ▶ Blood Pressure Reactivity or Responses

Cardiovascular Surgery

- ▶ Cardiac Surgery

Care of Older Adults

- ▶ Geriatric Medicine

Care Recipients

Maija Reblin
College of Nursing, University of Utah, Salt Lake City, UT, USA

Synonyms

Patients

Definition

One who receives care; an individual with a medical condition or who requires support with activities of daily living and is in a relationship with a caregiver, such as a doctor, nurse, friend, or family member, who provides treatment,

assistance, or comfort (National Alliance for Caregiving & AARP. Caregiving in the U.S, 2009). Care recipients are not necessarily passive; action often must be taken to access, secure, and personalize care. This may involve navigation of the health-care and insurance system; decision making based on personal, family, or cultural values and beliefs; selecting, managing, and adhering to the treatment regime; emotional responses to and coping with the potential stress of receiving care; and managing communication with care providers (Holman & Lorig, 2000).

References and Readings

Holman, H., & Lorig, K. (2000). Patients as partners in managing chronic disease. Partnership is a prerequisite for effective and efficient health care. *BMJ, 320*(7234), 526–527.

National Alliance for Caregiving and AARP. Caregiving in the U.S. (2009). Available at: http://www.caregiving.org/data/Caregiving_in_the_US_2009_full_report.pdf

Caregiver Acts of Omission

► Child Neglect

Caregiver Burden

► Caregiver/Caregiving and Stress
► Stress, Caregiver

Caregiver Hassle

► Stress, Caregiver

Caregiver Strain

► Stress, Caregiver

Caregiver/Caregiving and Stress

Alyssa Parker
UTSW Health Systems, South western Medical Center, Dallas, TX, USA

Synonyms

Caregiver burden

Definition

Caregiving affects the quality of life of millions of individuals and is frequently associated with physical and psychological distress. Caregiving burden has been linked to decreased preventative health behaviors and perception of quality of life, which ultimately negatively impacts the care recipient. Additionally, the chronic stress of caregiving has been found to decrease immune functioning of the caregiver in general, including decrements in cellular immunity, higher risk for infectious disease, and slower wound healing. Multicomponent interventions have been helpful in coping both cognitively and behaviorally.

Description

Caregiving has become an issue of national public health. Due to advances in medicine and technology, a shortage of nurses and other health-care workers, and a movement since the 1960s away from institutionalization, caregiving, especially family caregiving, has become a necessity that affects the quality of life of millions of individuals (Family Caregiver Alliance, 2011). Caregiving is a diverse endeavor because the demands of caregiving differ with regard to age, developmental level, mental health needs, and physical health needs of both the caregiver and the care recipient. Those in the caregiving role become a critical agent between the recipient and a multitude of environments, including biological, psychological, social, cultural, physical, and

political (Perkins & Haley, 2010). Although the core of successful caregiving revolves around the caregiver's own physical and mental health, it is a situation that has been described as one filled with heartache, pain, and loss (George & Gwyther, 1986; Poulshock & Diemling, 1984).

Thrust into a role devoid of formal training, choice, or compensation, many family caregivers suffer physical and psychological distress related to their experiences. In an effort to provide care for their ill relatives, caregivers often neglect their own health. Some caregivers believe they are not entitled to time to themselves or time away from the recipient, which ultimately leaves them feeling fearful and guilty (Bedini & Guinan, 1996). Those who do participate in noncaring activities, such as socializing or discovering hobbies, may derive less positive experiences due to the spillover effect of distress resulting from care. As a result, subjective well-being, including positive affect, life satisfaction, and perceived quality of life, may be affected (Gilleard, et al., 1984; Kosberg & Cairl, 1986). Compared to matched controls, caregivers, especially spousal caregivers, have demonstrated uniformly negative changes in immune function due to chronic stress, including decrements in cellular immunity, higher vulnerability to infectious disease, and slower wound healing. These immunological consequences often persist at measurable levels even after cessation of caregiving tasks and may be the cause of morbidity and mortality in the elderly (Kiecolt-Glaser, Dura, Speicher, Trask & Glaser, 1991; Kiecolt-Glaser, 1999). Additionally, individuals who report strain are less likely to engage in preventative health behaviors such as getting enough sleep, taking time to recuperate, exercising, eating regular meals, and keeping medical appointments (Burton et al., 1997; Talley & Crews, 2007). Consequently, caregivers are at significant risk for experiencing health problems, depression, anxiety, and social isolation.

Risk for physical and mental health difficulties can be predicted to some extent by qualities present in both the caregiver and the care recipient. The dependency needs of the recipient, such as the number of hours of care needed or the degree to which activities of daily living can be completed independently, play an important role in caregiver burden. Those in the care position with the heaviest burden are more likely to report their health as fair or poor and are more likely to report physical strain as well as significant emotional strain (Caregiving in the US, 2004). Burden has also been linked to caregiver mood, caregiver's perceptions of the degree of recipient disability, and negative affectivity. Negative affectivity is the extent to which a person experiences negative mood states, including upset, anger, worry, guilt, fear, and disgust. Caregivers who rate high in negative affectivity often report distress, discomfort, and dissatisfaction over time, regardless of the situation (Blake et al., 2000). Higher negative affectivity has also been linked to less adaptive coping strategies and is a vulnerability factor in the development of anxiety and depression (Gunthert et al., 1999). Relationship with the recipient prior to illness or disability and availability of social support also play important roles in the extent to which the caregiver experiences strain.

Research on the differences between male and female caregivers has been mixed. Although men and women do not differ greatly in aspects of providing care, male caregivers report experiencing less burden and demonstrate more problem-focused coping strategies than female caregivers (Tiegs et al., 2006). One explanation is that women's involvement in the caregiving role tends to be more intensive and affective in nature than their male counterparts. Additionally, it has been suggested that women are more likely to carry out household tasks while caring for a family member (Miller & Cafasso, 1992; Parks & Pilisuk, 1991). Other research has shown no gender differences when controlling for protective factors, such as personality and social support.

Due to the associated risks, individuals caring for loved ones benefit from the development of a repertoire of both cognitive and behavioral strategies that enable them to defend against distress while continuing to provide effective care. Research to date on caregiver interventions has focused primarily on reducing depression and

strain via an emphasis on the following six intervention approaches: psychoeducational, supportive, respite/adult care, psychotherapy, improvements in care receiver competence, and multicomponent interventions (Sorenson et al., 2002). Intervention outcomes include the family caregiver's well-being, psychologic morbidity (stress, depression, perceived burden), beliefs (self-efficacy, control), cognitive behaviors and positive psychological outcomes (rewards, gains), and care recipient's function, behavior, and ability to avoid institutionalization (Gitlin et al., 2003). The most effective caregiver interventions to date have been multicomponent interventions that utilize a combination of cognitive behavioral approaches to reducing caregiver stress. Behaviorally, exercise and the utilization of social support have been the most valuable techniques in relieving stress associated with caregiving. Available social support and perceived social support can buffer caregiver vulnerability to stress and provide physical assistance when needed (Dean & Lin, 1977; O'Brien, 1993). Cognitively, the utilization of logical analysis and problem solving has been associated with higher levels of life satisfaction, better health, and lower depression in caregivers. A realistic appraisal and acceptance of the difficult situation is healthy and allows the caregiver to live his or her own life while accommodating the needs of the recipient. Less effective cognitive coping styles include avoidant-evasive, regressive, and an increased use of wishfulness and fantasizing by the caregiver, all of which have been related to higher levels of care burden (Hayley et al., 1987; Quayhagen & Quayhagen, 1988).

Despite the reality of care strain and its resulting physical and mental health risks, many caregivers persist for years in their roles and are able to report positive and reciprocal caregiving experiences (Pinquart & Sorenson, 2004). Long-term caregiving may result in the acquisition of skills and a sense of self-efficacy within the care role. Some individuals found that caregiving provided a sense of usefulness during a time in which they felt a loss of control. This sense of usefulness and improved self-esteem based on perceived abilities to handle difficult situation may provide some symptom relief from depression and anxiety (Konstam et al., 2003). Finding meaning through caregiving allows the caregiver to hold positive beliefs about one's self and one's caregiving experience.

Cross-References

- ▶ Care Recipients
- ▶ Chronic Disease or Illness
- ▶ Daily Stress
- ▶ Dementia
- ▶ Disability
- ▶ Disease Burden
- ▶ Elderly
- ▶ End-of-Life Care
- ▶ Family Assistance
- ▶ Family, Caregiver
- ▶ Home Health Care
- ▶ Lifestyle Changes
- ▶ Medical Decision-Making
- ▶ Stress, Caregiver
- ▶ Stress, Emotional

References and Readings

Bedini, L. A., & Guinan, D. M. (1996). If I could just be selfish. Caregivers' perceptions of their entitlements to leisure. *Leisure Sciences, 18*, 227–239.

Blake, H., Lincoln, N. B., & Clarke, D. (2003). Caregiver strain in spouses of stroke patients. *Clinical Rehabilitation, 17*(3), 312–317.

Burton, L. C., Newsom, J. T., Schulz, R., Hirsch, C. H., & German, P. S. (1997). Preventative health behaviors among spousal caregivers. *Preventative Medicine, 26*(2), 162–169.

Connell, C. (1994). Impact of spouse caregiving on health behavior and physical and mental health status. *American Journal of Alzheimer's Care Related Disorders Research, 9*, 26–37.

Dean, A., & Lin, N. (1977). The stress-buffering role of social support. *The Journal of Nervous and Mental Disease, 6*, 403–417.

Family Caregiving Alliance, (n.d.). *National Center on Caregiving: Family caregiving and public policy, principles for change*. Retrieved January 2011, from http://www.caregiver.org/caregiver/jsp/content_node.jsp?nodeid=788

George, K. L., & Gwyther, L. (1986). Families caring for elders in residence: Issues in measurement of burden. *Journal of Gerontology*.

Gilleard, C. J., Gilleard, K., Gledhill, K., & Whittick, J. (1984). Caring for the mentally infirm at home: A survey of the supporters. *Journal of Epidemiology and Community Health, 38*, 319–325.
Gitlin, L. N., Belle, S. H., Burgio, L. D., et al. (2003). Effect of multicomponent intervention on caregiver burden and depression: The REACH multisite initiative at 6-month follow-up. *Psychology and Aging, 18*(3), 371–374.
Gunthert, K., Cohen, L., & Armeli, S. (1999). The role of neuroticism in daily stress and coping. *Journal of Personality and Social Psychology, 77*, 1087–1100.
Hayley, W. E., Levine, E. G., Brown, S. L., Berry, J. W., & Hughes, G. H. (1987). Psychological, social, and health consequences of caring for a relative with senile dementia. *Journal of American Geriatrics Society, 35*, 405–411.
Kiecolt-Glaser, J. K. (1999). Stress, personal relationships, and immune function: Health implications. *Brain, Behavior, and Immunity, 13*, 61–72.
Kiecolt-Glaser, J. K., Dura, J. R., Speicher, C. E., Trask, O. J., & Glaser, R. (1991). Spousal caregivers of dementia victims: Longitudinal changes in immunity and health. *Psychosomatic Medicine, 53*, 345–362.
Konstam, V., Holmes, W., Wilczenski, F., Baliga, S., Lester, J., & Priest, R. (2003). Meaning in the lives of caregivers of individuals with Parkinson's disease. *Journal of Clinical Psychology in Medical Settings, 10*(1), 17–26.
Kosberg, J. I., & Cairl, R. E. (1986). The cost of care index: A case management tool for screening informal care providers. *Gerontologist, 26*, 273–285.
Miller, B., & Cafasso, L. (1992). Gender differences in caregiving: Fact or aritfact? *Gerontologist, 32*, 498–507.
Monahan, D. J., & Hooker, K. (1995). Health of spouse caregivers of dementia patients: the role of personality and social support. *Social Network, 40*(3), 305–314.
National Alliance for Caregiving/AARP. (2004). *Caregiving in the U.S*. Washington, DC: Author.
O'Brien, M. T. (1993). Multiple Sclerosis: Health-promoting behaviors of spousal caregivers. *Journal of Neuroscience Nursing, 25*(2), 105–112.
Parks, S. H., & Pilisuk, M. (1991). Caregiver burden: Gender and the psychological costs of caregiving. *The American Journal of Orthopsychiatry, 61*, 501–509.
Perkins, E. A., & Haley, W. E. (2010). Compound caregiving: When lifelong caregivers undertake additional roles. *Rehabilitation Psychology, 55*, 409–417.
Pinquart, M., & Sorenson, S. (2004). Associations of caregiver stressors and uplifts with subjective well-being and depressed mood: a meta-analytic comparison. *Aging & Mental Health, 8*(5), 438–449.
Poulshock, S. W., & Diemling, G. (1984). Families caring for elders in residence: Issues in measurement of burden. *Journal of Gerontology, 39*, 230–239.
Quayhagen, M. P., & Quayhagen, M. (1988). Alzheimer's stress: Coping with the caregiving role. *Gerontologist, 28*, 391–396.
Sorenson, S., Pinquart, M., & Duberstein, P. (2002). How effective are interventions with caregivers? An updated meta-analysis. *Gerontologist, 42*(3), 356–372.
Talley, R. C., & Crews, J. E. (2007). Framing the public health of caregiving. *American Journal of Public Health, 97*(2), 224–228.
Tiegs, T. J., Heesacker, M., Ketterson, T. U., et al. (2006). Coping by stroke caregivers: Sex similarities and differences. *Topics in Stroke Rehabilitation, 13*(1), 52–62.

Carpal Tunnel Syndrome

Daniel Gorrin
Department of Physical Therapy, University of Delaware, Newark, DE, USA

Definition

The carpal tunnel refers to the area of the wrist between the carpal bones and the overlaying fibrous band of connective tissue called the transverse carpal ligament or the flexor retinaculum. The median nerve passes through the carpal tunnel along with nine tendons of muscles providing finger and wrist flexion (flexor digitorum profundus, flexor digitorum superficialis, and flexor pollicis longus).

Carpal tunnel syndrome refers to an entrapment or compression of the median nerve at the wrist. The median nerve can become compressed under the flexor retinaculum. The etiology is unknown in most cases; however, carpal tunnel syndrome can result from a trauma such as a fracture or dislocation of the carpal bones at the wrist. Such trauma can lead to direct injury of the nerve and increased pressure within the carpal tunnel. Other potential causes of the condition include rheumatoid arthritis, renal disease, hypothyroidism, lupus, obesity, pregnancy, alcoholism, diabetes, and certain collagen diseases. If the underlying cause of the condition can determined and treated, the median nerve dysfunction could be resolved.

Symptoms of carpal tunnel syndrome include burning, numbness, and tingling in the region of the hand supplied by the median nerve (thumb, index finger, middle finger, and medial side of the ring finger) which can be exacerbated at night.

Increased symptoms at night can likely be attributed to the patient favoring wrist flexion during sleep. This position narrows the space within the carpal tunnel causing increased pressure on the nerve. In more severe cases, the patient may experience weakness and atrophy of the musculature controlling the thumb.

Electrodiagnostic tests and electromyographic studies can be used in conjunction with patient history and physical examination in order to diagnose carpal tunnel syndrome. Initially, treatment is intended to control inflammation and decrease stress on the nerve. Conservative treatment includes activity modification, splinting to decrease wrist flexion and pressure on the median nerve, and steroid injections to decrease inflammation within the tunnel. If the patient does not respond to conservative management, a surgical decompression of the median nerve may be indicated.

References and Readings

Drake, R. L., Wayne Vogl, A., & Mitchell, A. W. M. (2010). *Gray's anatomy for students* (2nd ed.). Philadelphia: Churchill Livingstone Elsevier.

Magee, D. J. (2008). *Orthopedic physical assessment* (5th ed.). St. Louis, MO: Saunders Elsevier.

Magee, D. J., Zachazewski, J. E., & Quillen, W. S. (2009). *Pathology and intervention in musculoskeletal rehabilitation* (1st ed.). St. Louis, MO: Saunders Elsevier.

Standring, S. (2008). *Gray's anatomy* (40th ed.). Philadelphia, PA: Churchill Livingstone Elsevier.

Case Fatality

► Mortality

Case Reports

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Case studies

Definition

A case report is a descriptive study that provides a detailed description of a case of a disease that is unusual, and therefore noteworthy, for some particular reason. It is usually written by a doctor, or perhaps by a group of doctors who have all become familiar with the case with each having something unique to contribute to the report. An extension of the case report is a case series, where the first report sparks interest and leads to reports on similar cases.

In the discipline of epidemiology, a more substantive investigative process often begins with a case report or case series (Webb, Bain, & Pirozzo, 2005). These reports provide detailed descriptions of an individual, or a small group of individuals, who share salient characteristics. The disease might not have been seen before, been noted in the literature before, or rarely been seen in that form before. Also, it may be noteworthy that a known disease occurred in a patient who would not normally be expected to have the disease, or in a geographic location where the disease is particularly rare.

Such reports are by nature selective: Doctors may or may not write a case report which may or may not be published and reach a large readership of other doctors. Additionally, they are not able to provide evidence of causality, and they cannot provide much evidence on the patterns of disease occurrence. For these reasons, they tend to appear toward the bottom of the "Hierarchy of Evidence," a tabular representation of the relative strengths of various investigational methodologies. Nonetheless, they can be very informative as the starting point for more extensive investigation.

Provocative case reports can certainly lead to important findings. A report of a series of five cases of *Pneumocystis carinii* pneumonia that occurred in young, previously healthy, homosexual men in three Los Angeles hospitals in a 6-month period during 1980–1981 is noteworthy (Webb et al., 2005). In this case, the disease had been seen before, but virtually always in patient populations with different characteristics: the elderly, patients who were severely

malnourished (and hence compromised when combating infection), and patients receiving chemotherapy for cancer who had developed compromised immune systems. The clustering of cases in the population of young homosexual men suggested a different disease. While the case reports, as noted previously, were not able to address causality or causal biological pathways, they did suggest the possibility of a relationship with the patients' sexual behavior. You may recognize that this disease is now known as HIV/AIDS (which is certainly not limited to young homosexual men).

Cross-References

- ▶ Clusters
- ▶ Hierarchy of Evidence

References and Readings

Webb, P., Bain, C., & Pirozzo, S. (2005). *Essential epidemiology: An introduction for students and health professionals*. New York: Cambridge University Press.

Case Studies

- ▶ Case Reports

Case-Control Studies

Jane Monaco
Department of Biostatistics, The University of North Carolina at Chapel Hill, Chapel Hill, NC, USA

Synonyms

Observational designs; Observational studies; Observational study

Definition

A case-control study is a study in which subjects are selected based on their outcome status, such as with disease or disease-free. Investigators select cases (subjects with the outcome of interest) and controls (subjects without the outcome of interest) and then compare the exposure (or risk factor) status in the two groups.

Description

Case-control studies are a very common observational study design within behavioral medicine research. Because the participants are selected based on their outcome status (commonly disease status), this study design is well suited for an outcome that is rare. For diseases with long latency periods (for example, melanoma or coronary heart disease), case-control studies can also be time efficient because the outcome has already occurred at the initiation of the study. When the exposure (or risk factor) is rare, a case- control study is often not practical.

Case-control studies determine the subjects' exposure retrospectively, commonly through historical records or self-report conducted after the exposure has occurred. Limitations of using retrospective data contribute to results from case-control studies being considered weaker than results from experimental designs that examine similar associations. Recall bias can occur when case subjects remember exposure differentially compared to controls. For example, a mother whose infant was born with a birth defect may differentially recall her use of medication during pregnancy compared to a mother of an infant without a birth defect (Rockenbauer, Olsen, Czeizel, Pedersen, & Sørensen 2001). The use of retrospective data, however, may facilitate study approval by ethical review boards, particularly, when the risk factor is illegal or known to be harmful, such as illicit drug use or tobacco use.

The selection of control subjects is critical in the design of a case-control study. Subjects chosen as controls should be as similar as possible to the case subjects except, potentially, with respect

to the exposure. Specifically, cases and controls should have had equal chance to be exposed to the risk factor. For this reason, cases and controls are often matched with respect to age, gender, ethnicity, and other factors.

In many case-control studies, the groups are compared by evaluating the odds ratio which is defined as the odds of exposure among the cases divided by the odds of exposure among the controls. In general, investigators cannot determine incidence rates of the disease since the subjects are selected based on disease (outcome) status. Thus, computing a relative risk directly is not possible. However, the relative risk can be approximated by the odds ratio when the outcome of interest is relatively rare.

In a typical behavioral medicine case-control example, Brent et al. (1993) investigated the association between adolescent suicide and multiple psychiatric risk factors. Sixty-seven adolescent suicide victims (cases) were matched to 67 controls with respect to age, gender, socioeconomic status, and county of residence. Investigators obtained information about the suicide victims through a "psychological autopsy protocol" in which parents, siblings, and friends were interviewed concerning the victim's risk factors. The controls' risk factor information was obtained from the participant and at least one parent. The study found that the suicide victims had significantly higher odds of major depression and substance abuse compared to the controls.

Some characteristics of case-control studies:

- Usually less expensive and less time consuming than cohort designs or experimental designs
- Often used when the outcome of interest is rare or has a long latency period
- Not practical when exposure is rare
- Sample size requirement is usually smaller than cohort or experimental designs
- Often used in initial investigations of an association due to logistical ease and relative lower cost
- May be used when exposure of participants to risk factor would be considered unethical in an experimental design
- Appropriate when studying multiple risk factors
- Usually can only address a single outcome (disease)
- Susceptible to recall bias (since exposure and outcome are determined retrospectively) and selection bias (which can occur when the controls are selected in such a way that they did not have same risk of exposure as the cases)
- Often considered weaker study design compared to cohort studies or randomized trials that study analogous associations

Cross-References

▶ Cohort Study
▶ Odds Ratio
▶ Retrospective Study

References and Readings

Brent, D. A., Perper, J. A., Moritz, G., Allman, C., Friend, A., Roth, C., et al. (1993). Psychiatric risk factors for adolescent suicide: A case-control study. *Journal of the American Academy of Child and Adolescent Psychiatry, 32*(3), 521–529.

Hennekens, C. H., Buring, J. E., & Mayrent, S. L. (1987). *Epidemiology in medicine*. Boston, MA: Lippincott Williams & Wilkins.

Kleinbaum, D. G., Sullivan, K. M., & Barker, N. D. (2007). *A pocket guide to epidemiology*. New York: Springer.

Rockenbauer, M., Olsen, J., Czeizel, A. E., Pedersen, L., & Sørensen, H. T. (2001). Recall bias in a case-control surveillance system on the use of medicine during pregnancy. *Epidemiology, 12*(4), 461–466.

Case-Crossover Studies

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

The innovative case-crossover study design is a hybrid design. Hybrid designs are those that combine the elements of two or more basic designs, or extend the strategy of one basic design through repetition (Kleinbaum, Sullivan, & Barker, 2007).

The case-crossover design represents an attempt to achieve the ideal, but unattainable, design of studying a group of subjects exposed to a particular event, activity, or influence, and also studying exactly the same subjects during the same period when not exposed to it. The design utilizes each subject as his or her own control. Exposure to the event in a defined (and likely fairly short) time period before the onset of disease is compared with typical exposure to it in a much longer period before disease onset, defined as the normal exposure.

Only a limited set of research topics are amenable to the employment of the case-crossover design (Rothman, Greenland, & Lash, 2008). The exposure must vary over time within individuals, rather than stay constant, and the exposure must have a short induction time and a transient effect. A classic example is the study reported by Maclure (1991), which used this design to study the effect of sexual activity on incident myocardial infarction. Several aspects make this design appropriate and informative in this case. First, the exposure to the factor of interest, sexual activity, is intermittent and presumed to have a short induction period for the hypothesized effect. Second, any increase in risk for myocardial infarction caused by sexual activity is presumed to be confined to a short time interval following the activity. Third, since myocardial infarctions are thought to be triggered by events close in time, this outcome is well suited to this type of study (Rothman et al., 2008).

Cross-References

► Case-Control Studies

References and Readings

Kleinbaum, D. G., Sullivan, K. M., & Barker, N. D. (2007). *A pocket guide to epidemiology*. New York: Springer.

Maclure, M. (1991). The case-crossover design: A method for studying transient effects on the risk of acute events. *American Journal of Epidemiology, 133*, 144–153.

Maclure, M., & Mittleman, M. A. (2000). Should we use a case-crossover design? *Annual Review of Public Health, 21*, 193–221.

Rothman, K. J., Greenland, S., & Lash, T. L. (2008). Case-control studies. In K. J. Rothman, S. Greenland, & T. L. Lash (Eds.), *Modern epidemiology* (3rd ed., pp. 111–127). Philadelphia: Lippincott Williams & Wilkins.

Webb, P., Bain, C., & Pirozzo, S. (2005). *Essential epidemiology: An introduction for students and health professionals*. New York: Cambridge University Press.

Casual Sex

► Sexual Hookup

CAT Scan

Mary Spiers
Department of Psychology, Drexel University, Philadelphia, PA, USA

Synonyms

Computed transaxial tomography; Computerized axial tomography; CT scan; X-ray computed tomography

Definition

A CAT scan is a structural imaging method based on the x-ray principle but is more sensitive to bone, tissue, and fluid density differences and employs a narrower beam, allowing for the segmentation of the imaged area into multiple transaxial images from many different angles. These images can be combined via computer technology to provide either 2D or 3D images. Enhanced CAT scans can reveal even greater contrast through injection of an intravenous dye. CAT scans are useful for imaging bones, soft tissue, blood vessels, and internal organs and particularly useful in imaging size and location of tumors and their relationship to normal tissue. CAT scans are also useful in imaging injuries to skeletal structures in relation to the surrounding tissue and the detection of vascular disorders in the body and brain. CAT scans are particularly useful in the identification of emboli (blood clots)

and aneurysms. Scans of the brain cannot reveal microscopic brain changes (e.g., axonal injury) but are useful in identifying lesions, tumors, and stroke (infarct) and particularly for differentiating hemorrhagic from nonhemorrhagic stroke. CAT scans are generally cheaper than magnetic resonance imaging (MRI) but provide poorer resolution. In relation to the brain, CAT scans also do not show the functioning of the brain as would be revealed with functional imaging methods such as (functional MRI (fMRI) or positron emission tomography (PET)).

Cross-References

- ▶ Cancer Screening/Detection/Surveillance
- ▶ Computerized Axial Tomography (CAT) Scan
- ▶ Functional Magnetic Resonance Imaging (FMRI)
- ▶ Magnetic Resonance Imaging (MRI)
- ▶ Neuroimaging

References and Readings

Weissleder, R., Wittenberg, J., & Harisinghani, M. G. (2007). *Primer of diagnostic imaging* (4th ed.). St. Louis: Mosby.

Zillmer, E. A., Spiers, M. V., & Culbertson, W. C. (2008). *Principles of neuropsychology* (2nd ed.). Belmont, CA: Wadsworth/Thompson Learning.

Catastrophizing/Catastrophic Thinking

Lara Traeger
Behavioral Medicine Service, Massachusetts General Hospital/Harvard Medical School, Boston, MA, USA

Synonyms

Arbitrary inference

Definition

Catastrophizing refers to the anticipation without evidence of extreme and terrible consequences or outcomes of an event. Catastrophizing is a characteristic type of cognitive distortion or error that may underlie a negative and inaccurate thought (Beck, Rush, Shaw, & Emery, 1979; Clark, Beck, & Alford, 1999). It can have negative health consequences for individuals who are managing a chronic illness. For example, a recent cancer survivor may interpret his fatigue as meaning that he will never recover his usual energy level and that he will have to give up all of his meaningful activities. This type of thinking can maintain negative emotions such as depression and lead to adverse or unhelpful behaviors such as poor medical adherence.

Cross-References

- ▶ Cognitive Distortions
- ▶ Negative Thoughts

References and Readings

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression*. New York: Guilford Press.

Clark, D. A., Beck, A. T., & Alford, B. A. (1999). *Scientific foundations of cognitive theory and therapy of depression*. New York: Wiley.

Catecholamines

George J. Trachte
Academic Health Center,
School of Medicine-Duluth Campus,
University of Minnesota, Duluth, MN, USA

Synonyms

Adrenaline; Epinephrine; Norepinephrine/Noradrenaline

Definition

Catecholamines are derivatives of the chemical dihroxyphenyl (catechol) ethylamine. They are found in the sympathetic nervous system, adrenal

medulla, and selected brain regions. The prominent naturally occurring catecholamines in humans are dopamine, norepinephrine, and epinephrine. These agents are intrinsic neurotransmitters of the sympathetic nervous system and mediate the "fight or flight" reactions to stressful situations. Examples of sympathetic responses include tachycardia, hypertension, pupillary dilation, sweating, and liberation of fuel sources. They also are prominent neurotransmitters in specific regions of the brain, typically being associated with pleasure, excitement, and movement.

Catecholamine synthesis involves conversion of the amino acid, tyrosine, to dihydroxyphenylalanine (DOPA) by adding a hydroxyl group. DOPA is converted to dopamine by removal of a carboxyl group. Dopamine is a neurotransmitter in the brain and in peripheral nerves. Dopamine also is a precursor of norepinephrine, requiring a conversion by dopamine ß hydroxylase. Norepinephrine is the major neurotransmitter of the sympathetic nervous system and also is a major neurotransmitter in the central nervous system. Norepinephrine can be converted to epinephrine by phenylethanolamine-N-methyl transferase, primarily in the adrenal medulla but also in the brain. Epinephrine is considered to be a neurotransmitter of the sympathetic nervous system although it functions more as an endocrine agent, circulating in the blood to promote various stress-related effects.

The physiological relevance of norepinephrine and epinephrine in the periphery is quite obvious because these agents mediate most of the responses to stressful situations. They have the following effects: activation of ß1 receptors on the heart to increase heart rate, force of contraction, and blood pressure; activation of α1 receptors on vascular smooth muscle, the eye, and sweat glands to raise blood pressure, dilate pupils, and promote sweating; and activation of α1, ß2, and ß3 receptors to liberate fuel stores for energy. Central actions of dopamine and norepinephrine are equally obvious. Dopamine is critically involved in movement, reward, emotion, memory, and cognition. Conditions related to dopamine deficiencies include Parkinson's disease. Conditions related to excessive stimulation of dopamine receptors include addiction, schizophrenia, psychoses, and learning deficits. Norepinephrine also is a critical central neurotransmitter. Augmentation of norepinephrine concentrations in nerve synapses is a frequent mechanism of action of antidepressants.

The synthesis of catecholamines is regulated primarily at the tyrosine to DOPA step. The release of catecholamines is regulated by activity of the sympathetic nervous system, involving acetylcholine as a neurotransmitter, or by activation of specific brain regions.

Dopamine is recognized as a critical neurotransmitter influencing movement, memory, cognition, emotion, and reward. As such, it influences movement and its absence is most easily noted in the symptoms of Parkinson's disease. Dopamine also is involved in a variety of psychological abnormalities such as addiction, schizophrenia, psychoses, and learning deficits. It also inhibits prolactin release, potentially indirectly elevating mood.

Norepinephrine has mood-elevating effects and a variety of antidepressants increase the concentrations of norepinephrine in neuronal synapses of the brain. The variety of antidepressants elevating norepinephrine concentrations includes monamine oxidase inhibitors, tricyclic antidepressants, and norepinephrine reuptake inhibitors.

Cross-References

- ▶ Central Nervous System
- ▶ Epinephrine
- ▶ Norepinephrine/Noradrenaline

Causal Diagrams

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Causal pathway diagram; Causal pathway model

Definition

A causal diagram is a more modern form of causal pathway models that have been used to summarize visually hypothetical relationships between variables of interest to the researcher. They represent a merger of graphical probability theory with path diagrams. This theory confers a powerful means of deducing the statistical associations implied by causal relations.

Once the rules for reading statistical associations from causal diagrams are mastered, they facilitate many tasks. These include understanding confounding and selection bias, choosing covariates for adjustment and for regression analyses, and understanding analyses of direct effects and instrumental-variable analyses (Glymour & Greenland, 2008).

Cross-References

- ▶ Bias
- ▶ Regression Analysis

References and Readings

Glymour, M. M., & Greenland, S. (2008). Causal Diagrams. In K. J. Rothman, S. Greenland, & T. L. Lash (Eds.), *Modern epidemiology* (3rd ed., pp. 183–209). Philadelphia: Wolters Kluwer/ Lippincott Williams & Wilkins.

Causal Pathway Diagram

▶ Causal Diagrams

Causal Pathway Model

▶ Causal Diagrams

Cause Marketing

▶ Social Marketing

Causes

▶ Attribution Theory

Celexa®

▶ Selective Serotonin Reuptake Inhibitors (SSRIs)

Cell Adhesion Molecule

▶ Adhesion Molecules

Cellular Theory of Aging

Emil C. Toescu
Division of Medical Sciences, The University of Birmingham, Edgbaston, Birmingham, UK

Definition

Cellular theories explain the aging process as originating in individual cells, either at the level of the genetic information or through changes in metabolism.

Description

The quest for understanding the process of aging is probably as long as human history, and its resolution is still far from clear or even assured. A major factor for this state of affairs is that aging is a complex, multifactorial process that develops during ontogeny gradually, at multiple levels, involving a certain degree of stochastic randomness. At a certain time (early 1990s), more than 300 various hypotheses were circulating for explaining aging, and, responding to a need for organizing such a vast catalogue, these

hypotheses were classified as *cellular theories* that explain the aging process as originating in individual cells, either at the level of the genetic information or through changes in metabolism; *system theories*, that propose that aging, while expressed at the level of individual cells, results from dysfunction in one or another of the general system that maintain overall body homeostasis (e.g., *the neuroendocrine theory of aging*); and *evolutionary theories*, that address the fundamental biological puzzle that aging, as a fundamentally deleterious process, should have been gradually eliminated during evolution since evolution aims to improve the adaptation of individuals and species to their environment.

Within the group of cellular theories, the various hypotheses can be further separated into those that invoke (a) changes in the genetic makeup (genome) of cells or (b) alterations and dysfunction in various metabolic pathways (overall, the "wear and tear" theories).

The *genome-related theories of aging* start from the fundamental fact that the whole of the genetic information that controls the identity, development, and status of a cell is contained within the DNA. Like anything else in nature, this molecule can be damaged either by random, stochastic agents or by specific factors or processes. Amongst other features, one of the unique properties of the DNA is that it is the only biological molecule that relies for maintenance on the repair of the same existing molecule, without the possibility of remanufacture. Apart from the implications for the importance and reliability of the DNA repair mechanisms, this fact also leads to the conclusion that DNA molecules accumulate damage over a lifetime since an error in DNA sequence information, once made during replication or recombination, becomes irreversible, due to the loss of the reference template. DNA integrity can be affected by several mechanisms. One is endogenous, represented by the cellular metabolism; activity in all cells will generate continuously reactive oxygen and nitrogen species (free radicals) that either directly, or secondarily, through generation of lipid peroxidation products, alkylating agents or protein carbonyl species, will damage DNA by inducing single-strand breaks and oxidation of various bases. The other category of damaging agents is exogenous, represented by chemical or physical (e.g., UV and other types of ionizing radiations) factors. It has been shown that DNA mutations/alterations and chromosomal abnormalities increase with age both in animals (e.g., rodents) and humans. In addition, the role of genetic mutation in inducing the aging phenotype is demonstrated by a number of syndromes of accelerated aging (progeria). Amongst them, the best known is the Werner's syndrome which is determined by an autosomal recessive mutation in a gene, WRN, that encodes for a protein with structural similarities with a DNA helicase (enzyme catalyzing DNA unwinding). Loss of WRN function results in a syndrome displaying the typical features of aging, but starting as early as the second decade of life: bilateral cataracts, graying of hair and alopecia, type 2 diabetes, atherosclerosis and hyperlipidemia, osteoporosis, etc. Another progeric manifestation is the Hutchinson-Gilford's syndrome, with a rather similar clinical manifestation but resulting from a point mutation in the gene encoding for a nuclear protein: lamin A/C (LMNA). Although the exact function of either protein is not fully established, recent experimental evidence point to the fact that they are involved in the process of DNA repair. The importance of maintaining a robust genomic stability led to the evolutionary development of powerful and flexible DNA repair systems that include mechanisms for dealing with both single-stand breaks (e.g., base excision repair and nucleotide excision repair) and double-strand breaks (e.g., homologous recombination or nonhomologous end joining). Although there are many reports of correlations between stability of DNA repair mechanisms and rate of aging in various animals (mammals) and, also, of an age-dependent functional decline in one or another DNA repair mechanism, other studies found no clear evidence for a drastic decline in DNA repair during aging, an observation taken simply to reflect the central role of genome stability for cell viability. In addition, accumulation of damage with age does not necessarily imply a decline in DNA repair – as any biological process,

genome maintenance systems are imperfect, and alterations can accumulate over time, particularly in animals with longer life spans.

A more recent line of investigation of the relationships between DNA damage and aging stems from the fact that genome maintenance involves not only the DNA repair systems but also the cellular responses triggered directly by the DNA damage. These responses include apoptosis, cellular senescence, and cell cycle arrest, known to cause age-related impairments in various tissues. Thus, one of the most ubiquitous response to unrepaired or improper repair double-strand breaks involves the ataxia-telangiectasia-mutated (ATM) kinase. Activated ATM, in addition to modulation of several cell cycle proteins DNA repair factors, targets p53, a central protein at the crossroad of several cell viability pathways. While p53 suppresses the onset of malignancy, having an indirect positive on lifespan, it also triggers cellular senescence and apoptosis. A strong theoretical argument for the involvement of such a universal and general cellular response in mediating the pro-aging effects of DNA damage is that the phenotype of aging is relatively constant from species to species and also, in general lines, from individual to individual whereas, with few exceptions, the exo- or endogenous induction of DNA damage is stochastic and should result in highly variable functional outcomes.

An important cellular theory of aging is the *cell senescence/telomere theory*. The idea of cell senescence was formulated in 1965, describing the fact that normal cells can undergo only a limited number of cell divisions (Hayflick's limit), after which the cells enter replicative senescence, remain quiescent, and then, after a period of time, die. Since the number of cell divisions varies from species to species (e.g., mouse cells divide roughly 15 times, while the cells for Galapagos tortoise divide 110 times), it has been proposed that this process of replicative senescence is an important regulator of life span and thus a contributor to aging (NB this senescence process, dependent on the cell replication, is different from the metabolic cellular senescence, that results from the accumulation with time of metabolic dysfunction, that result in functional impairment of various cellular activities, see below). It has been proposed that replicative senescence ultimately results from the loss of telomeres, which are specific chains of a repeating DNA sequences located at the ends of each linear chromosome. With each cell division, a small amount of DNA is necessarily lost on each chromosome end, resulting in ever-shorter telomeres, altered telomere structure, and, when the telomere is under a critical length, a stop of replication and eventual replicative senescence. Activation of the telomerase enzyme will regenerate telomeres, prevent replicative senescence, and immortalize human primary cell cultures. Importantly, in all cancer cells, there is an activation of telomerase or of an alternate pathway of telomere extension that avoids replicative senescence.

Although there is a wealth of correlative data (e.g., shorter telomeres in aged people or, more specifically, in individuals with neurodegenerative diseases, including Alzheimer's; induction of telomere shortening in condition of increased metabolic stress), a causal involvement of telomere reduction in aging is doubtful as telomerase-deficient mice do not age more rapidly. Instead, as with the other genetic theories of aging discussed above, it is more likely that replicative senescence influences aging through the various cellular responses it triggers. It has been described that senescent cells produce and secrete various degradative enzymes and inflammatory factors that alter the microenvironment and lead to disturbed tissue structure and function. Also, replicative senescence degrades and ultimately limits the regenerative potential of stem cell. The intracellular mechanism triggered by telomere shortening is the activation of the same tumor suppressor p53 protein. The type of p53-dependent cellular response (cell arrest, apoptosis, or senescence) is often cell type dependent and varies with the type of stimulus that triggers it and severity of stress that the cells are exposed to. Being a tumor suppressor protein, it is not surprising that mice mutated for p53 with loss of function have a dramatically increased incidence of cancer, while p53 signaling is altered in the majority of human cancers. However, if cellular

senescence, linked with p53 activation, acts to suppress tumor formation, how can it be explained that cancer is more prevalent with age when senescence is also increased? There is currently no generally accepted explanation, and it is likely that it results from subtle changes in the balance between several processes and factors, such that, due to its ample homeostatic and functional reserve, in the adult organisms, the functional and structural deleterious effects that senescent cells might cause to the tissues can be efficiently repaired by the normal tissue renewal processes. Thus, in the main, in the mature organisms, the main role of the p53-dependent senescence is to provide cancer protection. In contrast, in the aged organisms, the time-dependent accumulation of mutations (i.e., DNA damage), together with the unfavorable metabolic environment, and the decrease in the renewing capacity generate conditions suitable for cancer growth.

One of the most widely acknowledged theories of aging is the *Mitochondrial Free Radical Theory of Aging* (*MFRTA*), which has been presented in various guises, either as metabolic or as "wear and tear" theories, and linked to other hypotheses, such as the "rate of living" theory. The latter probably has the longest history, originating at the beginning of last century with the empirical observation of a relationship between metabolic rate, body size, and longevity, such that long-lived animals are, on average, larger. Further metabolic studies led to the proposal that the faster the metabolic rate of an animal, a standby for biochemical activity and for the effect of temperature, the faster the organism will age. In the mid-1950s, the mechanisms causing cell damage and death in response to ionizing radiation were becoming clearer: the production of free radicals, a highly reactive species of molecules characterized by the existence of a single unpaired electron in the outer layers of the atom. Due to their chemical properties, oxygen and nitrogen are the molecules most prone to become free radicals, and the instability of such a molecule renders them very reactive, generating chain redox reactions of sequential oxidation (loss of electrons) and reduction (gain of electrons) of a variety of cellular substrates. In many instances, such redox changes result in a modification of function of the target proteins, leading to loss of metabolic homeostasis and ensuing damage. If the free radicals attack is of limited intensity or duration, the cellular damage can be contained and either accumulate slowly over time or be repaired; more intense level of injury would result in cell death. The original form of the Free Radical Theory of Aging (FRTA) envisaged aging as resulting from the long-term accumulation of free radical–induced damage, affecting mainly nuclear DNA, which is very sensitive to the action of free radicals. An important development of this hypothesis came with the discovery that the free radicals can result not only from the effects of exogenous factors, such as irradiation, but are also a natural output of normal physiology. One of the reasons why this hypothesis of aging became so paradigmatic is that it linked with several previous views, such that a higher rate of metabolism would generate higher free radical loads and consequent damage, and lead to a higher rate of aging. In the mid-1980s, the FRTA was complemented with the mitochondrial perspective, with several observations contributing to this development. (1) The mitochondria are the major source of free radicals since two of the protein complexes that form the mitochondrial respiratory chain (aka, electron transport chain) generate stochastically, in an unregulated fashion, reactive oxygen species (i.e., oxygen free radicals). (2) Mitochondria possess specific mitochondrial DNA, that is, spatially located very near to the source of free radicals, in the mitochondrial matrix. (3) Mitochondrial DNA has limited repair capacity. (4) Mitochondrial DNA codes for some of the proteins in the respiratory complex, and DNA mutation could generate dysfunctional proteins, initiating a time (age)-dependent vicious circle of increased free radical producing. Thus, the strong formulation of the complete MFRTA flows along the following functional axis: (a) oxygen free radicals generated (mainly from mitochondria) as a function of metabolic rate cause cumulative oxidative damage, resulting in structural degeneration, functional decline, and age-related diseases, leading to (b) oxidative stress that is the

predominant cause of age-associated degenerative change, and thus (c) the mitochondrial free radicals are the cause of aging.

In the last few decades, a huge amount of experimental evidence accumulated to show that with age there is indeed an accumulation of mitochondrial oxidative damage and a progressive decline in mitochondrial function and performance. In many tissues, including the brain (which has a special position since the neurons are the only cell types in the body that are maintained in a postmitotic state, i.e., they do not divide), there is an age-dependent accumulation of global oxidative damage to proteins, DNA, and lipids. However, in the last few years, the availability of very powerful experimental models that allow genetic manipulations (full or conditional knock-in of proteins or knock-down of proteins, use of interference RNA as silencers of specific protein synthesis, etc.) led to the expression of serious reservations about the full validity of MFRTA. Thus, decreasing free radical levels with dietary antioxidants or by genetically induced overexpression of protein antioxidants, such as superoxide dismutase (SOD), that metabolizes the oxygen superoxide (a free radical) to hydrogen peroxide, or catalase, that metabolizes hydrogen peroxide to water and regenerates the gaseous oxygen, did not induce the expected significant increase in lifespan of the test animals. In contrast, inactivation of antioxidant activity while increasing the free radical levels did not determine a significant reduction of lifespan and even increased, in some instances, the lifespan.

It is worth assessing for a moment the reasons of the discrepancy between the two sets of data. The important point about most of earlier studies mentioned is that they were correlative, reporting that with age there is an increase in oxidative damage. However, correlation is not necessarily causation and implies the possibility that both aging and increased oxidation can be caused, at the same time, by another process(es), and, indeed, aging is viewed now as a multifactorial process. It also can be that oxidative stress might be the consequence of aging, with aging having some discrete cause, or causes, distinct from oxidative stress. Alternatively, oxidative stress might result from the failure of one particular maintenance system of the organism and thus participate in causing aging, but only as a factor amongst others. This perspective on the role of oxidative stress in actually causing aging has also practical implications, as it is still possible to advocate antioxidant therapies as being beneficial to health in counteracting the effects of free radicals, but not as a magic, blanket coverage anti-aging cure. In addition, each intervention should be critically evaluated, both because some antioxidant supplementation trials provided surprising results and because of an increasing number of studies showing the crucial roles of ROS in cellular signaling, and thus advocating against a too strong suppression of free radicals production.

Cross-References

▶ Neuroendocrine Theory of Aging

References and Readings

Bratic, I., & Trifunovic, A. (2010). Mitochondrial energy metabolism and ageing. *Biochimica et Biophysica Acta, 1797*(6–7), 961–967.

Chen, J. H., Hales, C. N., & Ozanne, S. E. (2007). DNA damage, cellular senescence and organismal ageing: Causal or correlative? *Nucleic Acids Research, 35*(22), 7417–7428.

Collado, M., Blasco, M. A., & Serrano, M. (2007). Cellular senescence in cancer and aging. *Cell, 130*(2), 223–233.

Garinis, G. A., van der Horst, G. T., Vijg, J., & Hoeijmakers, J. H. (2008). DNA damage and ageing: New-age ideas for an age-old problem. *Nature Cell Biology, 10*(11), 1241–1247.

Lapointe, J., & Hekimi, S. (2010). When a theory of aging ages badly. *Cellular and Molecular Life Sciences, 67*(1), 1–8.

Lombard, D. B., Chua, K. F., Mostoslavsky, R., Franco, S., Gostissa, M., & Alt, F. W. (2005). DNA repair, genome stability, and aging. *Cell, 120*(4), 497–512.

Mattson, M. P., Gleichmann, M., & Cheng, A. (2008). Mitochondria in neuroplasticity and neurological disorders. *Neuron, 60*(5), 748–766.

Shawi, M., & Autexier, C. (2008). Telomerase, senescence and ageing. *Mechanisms of Ageing and Development, 129*(1–2), 3–10.

Toescu, E. C. (2005). Normal brain ageing: Models and mechanisms. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences, 360*(1464), 2347–2354.
Viña, J., Borrás, C., & Miquel, J. (2007). Theories of ageing. *IUBMB Life, 59*(4–5), 249–254.

Center for Epidemiologic Studies Depression Scale (CES-D)

Whitney M. Herge, Ryan R. Landoll and Annette M. La Greca
Department of Psychology, University of Miami, Coral Gables, FL, USA

Definition

The Center for Epidemiologic Studies Depression Scale (CES-D Scale) is a 20-item self-report measure designed to assess depressive symptoms over the previous week (Radloff, 1977). The CES-D assesses multiple symptom clusters, including depressed affect, lack of hope, feelings of guilt and shame, and somatic symptoms (e.g., disrupted sleep or appetite) with an emphasis on negative affect (Radloff, 1977). Sample items include "During the past week, . . .I felt that I could not shake off the blues even with help from my family or friends," and ". . .I felt that everything I did was an effort," (Radloff, 1977). Four items are worded positively and reverse coded to (a) ensure the respondent is attending to each question and not answering carelessly and (b) measure the respondent's positive affect (Radloff, 1977). Each item is rated on a frequency scale (0 = Rarely or None of the Time, 1 = Some or a Little of the Time, 2 = Occasionally or a Moderate Amount of Time, 3 = Most or All of the Time; Radloff, 1977). Total scores can range from 0 to 60; higher scores represent more depressive symptoms (Radloff, 1977).

The CES-D is one of the most commonly used measures for assessing the presence of depressive symptoms in adults, as it has good psychometric properties (Sharp & Lipsky, 2002; Vahle, Andresen, & Hagglund, 2000). The internal consistency of this measure is strong in both the general adult population ($\alpha = 0.85$) and among clinically depressed adults ($\alpha = 0.90$; Radloff, 1977). Further, reliability of the CES-D, as measured by test-retest correlations over periods ranging from 2 weeks to 12 months, has generally been in the moderate range (0.45–0.67), indicating adequate stability (Radloff, 1977). With regard to validity, the CES-D is capable of discriminating between the general adult population and psychiatric inpatients, as well as between severity levels of clinical populations (Radloff, 1977). Among clinical populations, it has also been shown to correlate positively with other measures of depression, including nurse-clinician ratings (0.56; Craig & Van Natta, 1976), and self-rating scales (0.44–0.75; Radloff, 1977). The CES-D primarily has been used to screen for high levels of depressive symptoms in community populations (Radloff, 1977). Scores above 16 denote a level of depressive symptoms which may require follow-up investigation (Zich, Attkisson, & Greenfield, 1990). Research regarding age, gender, and ethnic differences in the underlying factor structure and use of the CES-D is inconclusive to date, and as such, a definitive statement cannot be made (e.g., Callahan & Wolinsky, 1994; Hertzog, Van Alstine, Usala, Hultsch, & Dixon, 1990; Liang, Van Tran, Krause, & Markides, 1989; Roberts, 1980).

Recently, the CES-D has been used as a depression screening tool for adolescents as young as 14 years of age (e.g., Charbrol, Montovany, Chouicha, & Duconge, 2002; Cuijpers, Boluijt, & van Straten, 2008; Sharp & Lipsky, 2002). The CES-D appears to be reliable for use with adolescents of high school age (M age = 17, SD = 1.4; Chabrol et al., 2002). With a community sample of adolescents, the reliability of the CES-D has been satisfactory ($\alpha = 0.85$; Chabrol et al., 2002). Further, the factor structure of the CES-D appears to function similarly in adults and adolescents (four factors: depressed affect, positive affect, somatic and retarded activity, and interpersonal; Chabrol et al., 2002; Radloff, 1977). Using a clinical cutoff score of 22, the CES-D has been shown to have a specificity indicator of 74.31 and a sensitivity indicator of 90.48 in adolescent

community samples (Cuijpers et al., 2008), although there is debate regarding the most appropriate cutoff score for use with adolescents (e.g., Roberts, Andrews, Lewinsohn, & Hops, 1990).

References and Readings

Callahan, C. M., & Wolinsky, F. D. (1994). The effect of gender and race on the measurement properties of the CES-D in older adults. *Medical Care, 32*(4), 341–356.

Chabrol, H., Montovany, A., Chouicha, K., & Duconge, E. (2002). Study of the CES-D on a sample of 1,953 adolescent students. *Encephale, 28*, 429–432.

Craig, T. J., & Van Natta, P. (1976). Recognition of depressed affect in hospitalized psychiatric patients: Staff and patient perceptions. *Diseases of the Nervous System, 37*(10), 561–566.

Cuijpers, P., Boluijt, P., & van Straten, A. (2008). Screening of depression in adolescents through the internet: Sensitivity and specificity of two screening questionnaires. *European Child & Adolescent Psychiatry, 17*(1), 32–38.

Hertzog, C., Van Alstine, J., Usala, P. D., Hultsch, D. F., & Dixon, R. (1990). Measurement properties of the Center for Epidemiological Studies Depression Scale (CES-D) in older populations. *Psychological Assessment: A Journal of Consulting and Clinical Psychology, 2*(1), 64–72.

Liang, J., Van Tran, T., Krause, N., & Markides, K. S. (1989). Generational differences in the structure of the CES-D Scale in Mexican Americans. *Journal of Gerontology, 44*(3), S110–S120.

Radloff, L. S. (1977). The CES-D scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement, 1*(3), 385–401.

Roberts, R. E. (1980). Reliability of the CES-D scale in different ethnic contexts. *Psychiatry Research, 2*, 125–134.

Roberts, R. E., Andrews, J. A., Lewinsohn, P. M., & Hops, H. (1990). Assessment of depression in adolescents using the Center for Epidemiologic Studies Depression Scale. *Psychological Assessment: A Journal of Consulting and Clinical Psychology, 2*(2), 122–128.

Sharp, L. K., & Lipsky, M. S. (2002). Screening for depression across the lifespan: A review of measures for use in primary care settings. *American Family Physician, 66*(6), 1001–1009.

Vahle, V. J., Andresen, E. M., & Hagglund, K. J. (2000). Depression measures in outcomes research. *Archives of Physical Medicine and Rehabilitation, 81*(12–2), S53–S62.

Wiegman Dick, R., Beals, J., Keane, E. M., & Manson, S. M. (1994). Factorial structure of the CES-D among American Indian adolescents. *Journal of Adolescence, 17*, 73–79.

Zich, J. M., Atkisson, C. C., & Greenfield, T. K. (1990). Screening for depression in primary care clinics: The CES-D and the BDI. *International Journal of Psychiatry in Medicine, 20*(3), 259–277.

Center for Scientific Review

Lee Ellington
Department of Nursing, College of Nursing, University of Utah, Salt Lake City, UT, USA

Basic Information

The Center for Scientific Review (CSR) resides within the National Institutes of Health (NIH) and is charged with the review of the scientific merit of NIH grant applications. The mission of CSR is to ensure that investigators' applications receive fair, constructive, and timely feedback, resulting in the goal of NIH to fund sound yet innovative research. A primary responsibility of CSR is to convene experts in the field to conduct peer review of grant applications. The CSR receives all grant applications for NIH and some applications from the U.S. Department of Health and Human Services, resulting in well over 110,000 applications per year. In 2009, CSR worked with 25,000 peer reviewers. The CSR consists of the director, referral officers, integrated review group chiefs, scientific review officers, and related administrative personnel.

When an application arrives at NIH, a CSR referral officer examines the application and routes it to the integrated review group that best fits the scope of the application. Within the integrated review group there are study sections, which are essentially peer review groups. Each study section is managed by a scientific review officer (SRO) and typically includes 20 or more scientists. The SRO assigns two to four peer reviewers for each application. Reviewers provide written critiques and provisional impact scores for each application and then attend an in-person review meeting. Approximately half of the applications are discussed by the reviewers and other members of the study section as a function of the provisional scoring process. The assigned reviewers present their critiques and then the discussion is open to the entire review group. After the general discussion, the assigned reviewers revisit initial overall impact scores and state their final score. The remainder of the study

section members record their scores privately. A few days after the review meeting, priority scores and percentile rankings are posted on NIH Commons and can be accessed by the principal investigator for each application. Whether the application was discussed by the full group or not, there will be written critiques and scores.

The CSR is independent from the NIH institutes or centers (IC) that make funding decisions. That is, CSR is concerned with scientific merit outside the context of funding priorities at the various institutes. After written critiques and scores are available, a second level of peer review is performed by the IC advisory councils. These councils consider the scientific merit of the application from CSR in conjunction with their institute's funding priorities to determine which grant applications will be funded. Applications that are not funded may be resubmitted a second time to CSR for peer review.

Major Impact on the Field

Behavioral medicine research is often funded by the NIH, and a number of study sections review behavioral science research applications. These study sections include scientists from the multiple disciplines represented within the Society of Behavioral Medicine and ensure that applications examining behavioral influences on health are fairly evaluated. The CSR website provides review group descriptions. Some examples of study sections which are well suited for reviewing specific behavioral science applications include behavioral and social consequences of HIV/AIDS, psychosocial risk and disease prevention, and social sciences and population studies. The SCR referral officer evaluates applications to find the most appropriate study section. The assignment is posted on ERA Commons for the principal investigator to assess prior to review.

Cross-References

► National Institutes of Health

References and Readings

http://cms.csr.nih.gov/
http://nih.gov/icd/

Centers for Disease Control and Prevention

► Behavioral Sciences at the Centers for Disease Control and Prevention

Central Adiposity

Simon Bacon
Department of Exercise Science, Concordia University, Montreal Behavioral Medicine Centre, Montreal, QC, Canada

Synonyms

Abdominal obesity; Apple shaped; Visceral adiposity

Definition

Central adiposity is the accumulation of fat in the lower torso around the abdominal area. Central adiposity is a function of both subcutaneous fat, which sits under the skin, and visceral fat, which surrounds the internal organs in the peritoneal cavity. Currently, it would seem that the toxic component of central adiposity is the visceral fat.

High levels of central adiposity have been associated with an increased risk of a number of diseases, including type 2 diabetes, hypertension, heart disease, and dementia. Of note, it would seem that central adiposity is independent of body mass index (a proxy of total adiposity) as a predictor of disease (even though the two are highly correlated). This increased risk is thought to be due to the hormonal action of visceral fat, which actively excretes adipokines, most of which impair glucose tolerance.

Central adiposity is most often measured as waist circumference (though the point of measurement varies across studies). However, there are other measures such as waist-to-hip ratio, waist-to-height ratio, and CT- and MRI-based visceral and subcutaneous fat.

While the causes of obesity and increased body weight are quite clear (an imbalance between energy intake and expenditure), the exact causes of individual increases in central adiposity are not known, i.e., why some people can have high total adiposity but not central adiposity and vice versa.

Cross-References

► Obesity

References and Readings

Lee, C., Huxley, R., Wildman, R., & Woodward, M. (2008). Indices of abdominal obesity are better discriminators of cardiovascular risk factors than BMI: A meta-analysis. *Journal of Clinical Endocrinology and Metabolism, 61*(7), 646–653.

Misra, A., & Vikram, N. K. (2003). Clinical and pathophysiological consequences of abdominal adiposity and abdominal adipose tissue depots. *Nutrition, 19*(5), 457–466.

Rexrode, K. M., Carey, V. J., Hennekens, C. H., Walters, E. E., Colditz, G. A., Stampfer, M. J., Willett, W. C., & Manson, J. E. (1998). Abdominal adiposity and coronary heart disease in women. *JAMA, 280*(21), 1843–1848.

Central Nervous System

Moritz Thede Eckart
General and Biological Psychology, Department of Psychology, University of Marburg, Marburg, Germany

Synonyms

Brain and spinal cord

Definition

The vertebrate nervous system is divided into the central nervous system (CNS) and the peripheral nervous system (PNS). The CNS consists of two parts: the brain (located in the skull) and the spinal cord (located in the spine). The PNS is the division of the nervous system that is located outside the skull and spine consisting of two types of neurons: afferent (sensory) neurons which relay impulses toward the CNS and efferent (motor) neurons which relay nerve impulses away from the CNS (Breedlove, Watson, & Rosenzweig, 2010; Pinel, 2006).

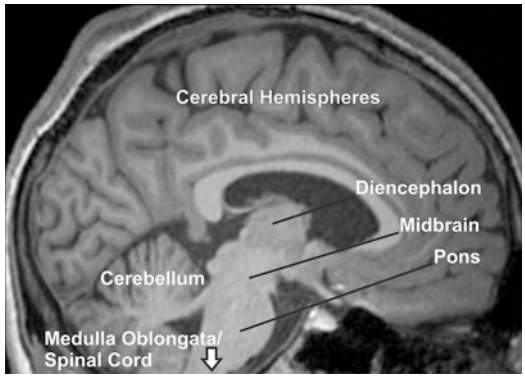
The CNS integrates the sensory information that it receives from the PNS (via the afferent nerves) and coordinates the behavior of the organism and the activity of all parts of the body (via the efferent nerves) (Pinel, 2006). Furthermore, the brain is processing not only simple motor behaviors or physical actions like walking or digestion but also all the complex cognitive, motivational, and emotional processes like affect, learning and memory, and especially those actions that are believed to be quintessential to humans like thinking, speaking, or creativity (Kandel, Schwartz, & Jessell, 2000).

Research on CNS functioning – neuroscience – is a multidisciplinary field that analyzes the biological basis of behavior and psychological processes. The term "neuroscience" was introduced in the mid-1960s, signaling the beginning of an era in which multiple disciplines – neuroanatomy, psychology, biology, medicine, pharmacology, and others – would work together cooperatively, sharing a common language, concepts, and goal, to understand the structure and function of the normal and abnormal brain. Currently, neuroscience is still one of the most rapidly growing areas of science (Squire et al., 2003).

Description

Anatomy

The CNS is the most protected organ of the body: It is encased by bone and covered by



Central Nervous System, Fig. 1 Sagittal MRI scan of a human brain with main structures: Cerebral hemispheres, diencephalon, midbrain, pons, and cerbellum. Medulla oblongata and spinal cord would continue ventrally from the Pons. (Courtesy of the working group "Brainimaging," medicine department, Philipps-University of Marburg.)

three protective membranes (1. dura mater, 2. arachnoid membrane/subarachnoid space, 3. pia mater). Also the cerebrospinal fluid has a protecting function: it supports and cushions the CNS. Additionally, the blood–brain barrier protects the brain from toxins that could enter the brain via the bloodstream. For instance, the degree to which psychoactive drugs influence psychological processes depends on their ease of penetrating the blood–brain barrier (Pinel, 2006).

The CNS is a bilateral and essentially symmetrical structure with seven main parts (see Fig. 1): (1) spinal cord, (2) medulla oblongata, (3) pons, (4) midbrain, (5) cerebellum, (6) diencephalon, and (7) cerebral hemispheres (consisting of cerebral cortex, basal ganglia, hippocampus, and amygdaloid nuclei) (Kandel et al., 2000). Other common nomenclatures for the parts of the CNS are as follows: spinal cord, myelencephalon (medulla), metencephalon (pons and cerebellum), mesencephalon (midbrain), and diencephalon and telencephalon (cerebral hemispheres) (Pinel, 2006, an integrated overview over both nomenclatures can be found in Breedlove et al. (2010)). An integration of both nomenclatures is summarized in Table 1.

The *spinal cord* is the most caudal part of the CNS. It receives and processes sensory information from the PNS: the skin, joints, and muscles of the limbs and trunk and controls movements of the limbs and the trunk. The spinal cord continues rostrally as the brain stem, which consists of the medulla oblongata, pons, and midbrain. The 12 cranial nerves are the only nerves of the PNS projecting directly into the brain rather than via the spinal cord.

The *medulla oblongata*, which lies directly above the spinal cord, includes several centers responsible for vital autonomic functions (digestion, breathing, control of heart rate).

The *pons*, which lies above the medulla oblongata, conveys information about movement from the cerebral hemispheres to the cerebellum.

The *midbrain*, which lies rostral to the pons, controls many sensory and motor functions like eye movement and coordination of visual and auditory reflexes.

Medulla oblongata, pons, and midbrain are often summarized as the *brain stem*. The brain stem receives sensory information from the skin and muscles of the head and provides motor control of the head via the cranial nerves. It also conveys information from the brain to the spinal cord and vice versa. Furthermore, the brain stem plays an important role in the regulation of arousal and awareness.

The *cerebellum* lies behind the pons and is crucially involved in the modulation of the force and range of movement, learning of motor skills and movement patterns, coordination, and tuning.

The *diencephalon* lies rostral to the midbrain and contains two structures: the thalamus, which processes most of the information reaching the cerebral cortex from the rest of the nervous system (and is thus often seen as the "gateway" to the cortex), and the hypothalamus, which is involved in the regulation of autonomic, endocrine, and visceral functions.

The *cerebral hemispheres* consist of a heavily wrinkled outer layer – the cerebral cortex (synonym in mammals: neocortex or isocortex) – and three deep-lying structures: the basal ganglia, the hippocampus, and the amygdaloid nuclei. The basal ganglia participate in regulating motor performance, the hippocampus plays a major role in the consolidation of the declarative memory, and

Central Nervous System, Table 1 A schematic view of the common nomenclatures of the brain, divided by main structures and substructures

Peripheral Nervous System (PNS)	Central Nervous System (CNS)											
	Spinal Cord	Brain										
		Myel-encephalon (medulla oblongata)	Met-encephalon		Mes-encephalon (midbrain)	Di-encephalon		Tel-encephalon (cerebral hemispheres)				
			Cerebellum	Pons		Thalamus	Hypothalamus	Cerebral cortex	Basal ganglia	Hippocampus	Amygdaloid nuclei	

the amygdaloid nuclei coordinate the autonomic and endocrine response of emotional states.

The *cerebral cortex* is divided into four anatomical distinct lobes: frontal, parietal, temporal, and occipital. The frontal lobe is involved in planning and executive functions, the parietal lobe in somatic sensation, the occipital lobe in vision, and the temporal lobe in hearing (and speech in humans).

Cell Types

There are two main classes of cells in the nervous system: nerve cells (neurons) and glial cells (from Greek *glia*, meaning glue). Glial cells far outnumber neurons – there are between 10 and 50 times more glia than neurons in the vertebrate CNS (Breedlove et al., 2010; Kandel et al., 2000).

Glial cells are support cells that provide the brain with structure and sometimes insulate neural groups and synaptic connections from each other. Also, they can communicate with each other and with neurons, and they directly affect neuronal functioning by providing neurons with raw materials and chemical signals that alter neuronal structure and excitability. Further important functions (like the myelination of neurons) are summarized in Kandel et al. (2000), Chap. 2 or Breedlove et al. (2010), Chap. 2.

Nerve cells are the main signaling units of the nervous system. A typical neuron has four morphologically defined regions: the cell body (soma), dendrites, the axon, and presynaptic terminals. The cell body is the metabolic center of the brain. Dendrites branch out in treelike fashion and are the main apparatus for receiving signals from other neurons. The axon extends away from the cell body and is the main conducting unit for carrying signals (action potentials: all or none impulses) to other neurons. Action potentials constitute the signals by which the brain receives, analyzes, and conveys information.

Near its end, the axon divides into fine branches that form communication sites with other neurons – the synapses. The nerve cell transmitting a signal is called the presynaptic cell, the signal receiving cell the postsynaptic cell. Between both cells lies the synaptic cleft. When an action potential reaches a synaptic terminal, neurotransmitters are released into the postsynaptic cleft as the neurons output signal. The number of released neurotransmitters is determined by the number and frequency of the action potentials in the presynaptic terminals. The released neurotransmitters act on the receptors of the postsynaptic neuron either in an excitatory (increasing the likelihood of an action potential of the postsynaptic neuron) or in an inhibitory (reducing the likelihood of an action potential of the postsynaptic cell) manner. Whether the effect is excitatory or inhibitory

does not depend on the type of released neurotransmitter but on the type of receptor in the postsynaptic neuron. One estimate puts the human brain at about 100 billion (10^{11}) neurons and 100 trillion (10^{14}) synapses. For details on nerve cell functioning see Kandel et al. (2000), Chap. 2; Squire et al. (2003), Chap. 3; Pinel (2006), Chap. 4; or Breedlove et al. (2010), Chap. 2.

Neurotransmitter Systems

This section will focus on the main neurotransmitter systems: ▶ dopamine (DA), norepinephrine (NE), ▶ serotonin (5-HT), glutamate, and gamma-aminobutyric acid (GABA), their organization, function, and dysfunction (Meyer & Quenzer, 2005).

Dopamine

DA is metabolized from the precursor DOPA. There are five main subtypes of DA receptors organized into D_1-like (D_1 and D_5) and D_2-like (D_2, D_3, and D_4) families. The dopaminergic system can be divided into three major pathways:

1. The nigrostriatal pathway, which originates in the substantia nigra (located in the midbrain) and innervates the striatum (part of the basal ganglia)
2. The mesolimbic pathway, which originates in the ventral tegmental area (located in the midbrain) and innervates various limbic structures, such as amygdala, nucleus accumbens, or hippocampus (all located in the deep lying structures of the cerebral hemispheres)
3. The mesocortical pathway, which also originates in the ventral tegmental area and innervates the cerebral cortex, particularly the prefrontal area

DA is also found in the hypothalamus, where it is involved in hormone secretion and in sensory structures.

Function and Dysfunction of the Dopaminergic System

The nigrostriatal pathway plays a crucial role in voluntary control of movement. ▶ Parkinson's disease, first described by the physician James Parkinson in 1817 as the "shaking palsy" causes a degeneration of dopaminergic neurons in the substantia nigra. The major symptoms of Parkinson's disease involve movement – tremor, rigidity, bradykinesia (poverty or slowing of movement) – and postural disturbances, but also cognitive dysfunctions.

The mesolimbic and mesocortical pathways are involved in motivated behavior, reinforcement of learning and emotional appetitive states (Alcaro, Huber, & Panksepp, 2007). That is why the dopaminergic system also plays a crucial role in drug abuse addiction. Most dopaminergic agonists, like amphetamine or cocaine, are addictive drugs because of their rewarding properties and the induced positive affective states. Furthermore these pathways are closely related to the GABA and opioid system, which is important for understanding the highly addictive potential of GABA agonists (like benzodiazepines and most probably alcohol) and opioid agonists (like morphine or heroin).

Also, a dysfunction of the dopaminergic system is observed in schizophrenia. The dopamine imbalance hypothesis suggests that schizophrenic symptoms are due to reduced dopaminergic function in the mesocortical neurons, along with excess dopaminergic function in mesolimbic dopaminergic neurons, resulting in impaired prefrontal cortex function. Also, the reduction of schizophrenic symptoms by DA antagonists (like Haloperidol, a typical antipsychotic, or Risperidone, an atypical antipsychotic, see below) supports the hypothesis that dopamine is crucially involved in schizophrenic symptoms. But is has to be pointed out that not all symptoms occurring in schizophrenia can be explained by dysfunctions of the DAergic system. For example, also dysfunctions and volume reductions of the hippocampus seem to play a crucial role. Also 5-HT seems to be involved in the development of schizophrenia (see below).

Norepinephrine

The central nervous noradrenergic system originates in the locus coeruleus, a small area of the pons, which projects to almost all areas of the cerebral hemispheres, thalamus, hypothalamus, cerebellum, and spinal cord. Noradrenergic

neurons play an important role in vigilance, arousal, and behavioral functions like hunger/eating, sexual behavior, fear and anxiety, and pain and sleep.

Serotonin

5-HT is synthesized from tryptophan, which comes from proteins in our diet. Pharmacologists have identified at least 14 5-HT receptor subtypes (Saulin, Savli, & Lanzenberger, 2011). The 5-HT system originates from a cell cluster called raphe nuclei (located in medulla, pons, and midbrain) which projects to virtually all structures of the cerebral hemispheres, thalamus, and hypothalamus.

Function and Dysfunction of the 5-HT System

The 5-HT system is involved in food intake, reproductive behavior, pain sensitivity, anxiety, learning and memory, and facilitation of motor output. In psychology, psychiatry, and pharmacology, serotonergic drugs are commonly used in treating depression. There are three major classes of antidepressants, which enhance the amount of 5-HT in the postsynaptic cleft in different ways: monoamine oxidase inhibitors, tricyclic antidepressants, and selective serotonin reuptake inhibitors (SSRIs). Although the pharmacological mechanisms of these drugs are well known, it is still not clear which of their neurochemical actions are responsible for their effectiveness in treating depression – especially regarding the fact that the pharmacological effects of the drugs occur within hours whereas antidepressant effects require weeks of chronic treatment.

Also, in pharmacological treatment of schizophrenia, blockade of 5-HT receptors has become a major topic of research in the past years, since the very effective atypical antipsychotics like Risperidone act not exclusively on DA but also on 5-HT receptors.

Another class of drugs that act on the 5-HT system are hallucinogens like LSD (the abbreviation LSD comes from the German chemical name for the substance: *l*ysergs*ä*ure*d*iethylamid; English: lysergic acid diethylamide) or psilocybin (found in "magic mushrooms"), which became temporarily popular in "psychological experiments" in Harvard in the working group of Timothy Leary in the 1960s and 1970s (Leary, Wilson, & Koopman, 1977).

Glutamate

Glutamate neurotransmitters have potent excitatory effects on neurons throughout the CNS. *N*-Methyl-D-aspartic acid or *N*-methyl-D-aspartate (NMDA) receptors are the main target site of glutamate.

Glutamate and, especially, NMDA receptors are thought to play a crucial role in learning and memory, particularly long-term potentiation. Especially the hippocampus has a very high density of NMDA receptors. NMDA receptor agonists impair the acquisition of various learning tasks.

Gamma-aminobutyric Acid

GABA is synthesized from glutamate. Many main areas of the brain are rich in GABA, including the cerebral cortex, hippocampus, basal ganglia, and cerebellum.

Function and Dysfunction of the GABAergic System

GABA is the main inhibitory neurotransmitter of the brain. Because of GABA's widespread inhibitory effect on neural excitability, treatment with GABA antagonists leads to seizures.

The effect of GABA on the GABA receptor is enhanced by CNS-depressant drugs such as benzodiazepines, barbiturates, and ethanol (alcohol). Due to their anxiolytic effects, benzodiazepines and barbiturates are often prescribed to treat anxiety disorders, although these substances have severe side effects. Among others, the sleep architecture is altered (reduced REM sleep), they are highly addictive, and can cause coma and death by respiratory depression (especially at high doses or with combined alcohol consumption). Another medicinal use of benzodiazepines is as anticonvulsants in the treatment of epilepsy.

Brain Circuits

Often, functionally related structures of the brain are integrated into one circuit such as the limbic system which is mainly associated with

emotional processes (Kandel et al., 2000, Chap. 50) or the basal ganglia. In the following, the basal ganglia will be described exemplarily

The *basal ganglia* comprise of striatum (putamen and caudate nucleus), pallidum, substantia nigra, and the subthalamic nucleus.

The basal ganglia are – beside the cerebellum – one of the largest subcortical motor systems. Cerebellum and basal ganglia appear to influence (via thalamus) the same cortical motor systems. While the basal ganglia output is inhibitory, the cerebellar output is excitatory. Discharge of many basal ganglia neurons correlates with movement and lesions or degenerations (like in Parkinson's disease, chorea Huntington, obsessive-compulsive disorder, or Tourette syndrome) cause severe movement abnormalities: slow voluntary movements or involuntary postures and movements. In order to distinguish the basal ganglia from the "pyramidal" corticospinal motor system, the basal ganglia are often termed "extrapyramidal" motor system.

However, beside motor control, the basal ganglia are also involved in nonmotor function and cognitive aspects of movement (Squire et al., 2003, Chap. 31).

Chances and Limitations of Neuroscience

The new research methods of neuroscience enhanced the knowledge of how mental phenomena are linked to processes in the brain, which allows, for instance, the mapping of mental processes to specific regions of the brain.

Nowadays it is possible to investigate by functional magnetic resonance imaging (fMRI) which brain regions are activated during the presentation of emotionally salient stimuli or cognitive tasks. However it has to be pointed out that research on the biological bases of mental phenomena does not per se enhance the understanding of psychological processes: it is a misunderstanding that biological processes can explain psychological phenomena.

On the contrary, mainly neuroreductionist conceptions of psychological processes (often favoring investigations of input–output relations) may abridge the development of complex theories on mental phenomena. No matter how precisely the brain is investigated – by microscope, imaging techniques, or in the future by even more exact methods – always the same physical objects will be found: neurons, synapses, neurotransmitters, ions, electrons, and protons, but not mental processes. "Granted that a definite thought, and a definite molecular action in the brain occur simultaneously, we do not possess the intellectual organ, nor apparently any rudiment of the organ, which would enable us to pass by a process of reasoning from the one phenomenon to the other. They appear together but we do not know why" (Mausfeld, 2010).

Cross-References

- ▶ Brain, Cortex
- ▶ Brain, Regions
- ▶ Dopamine
- ▶ Neurotransmitter
- ▶ Norepinephrine/Noradrenaline
- ▶ Parkinson's Disease
- ▶ Serotonin

References and Readings

Alcaro, A., Huber, R., & Panksepp, J. (2007). Behavioral functions of the mesolimbic dopaminergic system: An affective neuroethological perspective. *Brain Res Rev, 56*(2), 283–321 & Source.

Breedlove, S. M., Watson, N. V., & Rosenzweig, M. R. (2010). *Biological psychology* (Vol. 6). Sunderland, MA: Sinauer.

Kandel, E. R., Schwartz, J. H., & Jessell, T. M. (2000). *Principles of neural science* (Vol. 4). New York: McGraw-Hill.

Leary, T., Wilson, R. A., & Koopman, G. A. (1977). *Neuropolitics: The sociobiology of human metamorphosis*. Los Angeles: Starseed/Peace Press.

Mausfeld, R. (2010). Psychologie, Biologie, kognitive Neurowissenschaften: Zur gegenwärtigen Dominanz neuroreduktionistischer Positionen und zu ihren stillschweigenden Grundannahmen [Psychology, biology, cognitive neurosciences. On the current predominance of neuroreductionist approaches and their tacit assumptions]. *Psychologische Rundschau, 61*(4), 180–190.

Meyer, J. S., & Quenzer, L. F. (2005). *Psychopharmacology: Drugs, the brain and behavior*. Sunderland: Sinauer.

Pinel, P. J. (2006). *Biopsychology* (6th ed.). Boston: Pearsons Education.
Saulin, A., Savli, M., & Lanzenberger, R. (2011). Serotonin and molecular neuroimaging in humans using PET. *Amino Acids*.
Squire, L. R., Bloom, F. E., McConnell, S. K., Roberts, J. L., Spitzer, N. C., & Zigmond, M. J. (2003). *Fundamental neuroscience* (2nd ed.). San Diego: Elsevier Science.

Central Tendency

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

Certain descriptive statistics provide concise yet meaningful summaries of large amounts of data. One category of such statistics is measures of central tendency. They provide a measure of a group's central value. Three measures of central tendency are the arithmetic mean, median, and mode, each of which has an entry in the encyclopedia.

Another way to conceptualize central tendency is to say that these measures provide an indication of the location of the data points. Imagine a set of data points ranging from 1 to 100. If the mean is 89, for example, this provides an indication that, overall, the data points are located toward the top of the range rather than toward the bottom. Conversely, if the mean is 27, this indicates that, overall, the data points are located toward the bottom of the range.

Measures of central tendency (consider here the arithmetic mean) are often presented along with measures of spread, or dispersion, of the individual values around the mean. It is possible to have a group of 100 numbers that range from 10 to 90, for example, and have a mean of 50. It is equally possible to have a group of 100 numbers that range from 45 to 55 and that also have a mean of 50. While the measure of central tendency, the mean, is the same in both cases, the dispersion is clearly greater in the former group of hypothetical numbers than in the latter group. Thus, the overall nature of the groups of numbers will differ despite their means being identical.

Cross-References

- ▶ Dispersion
- ▶ Median
- ▶ Mode

CER

- ▶ Comparative Effectiveness Research

Cerebrum

- ▶ Brain, Cortex

Cervical Adenocarcinoma

- ▶ Cancer, Cervical

Cessation Intervention (Smoking or Tobacco)

Mark Vander Weg
Department of Internal Medicine, The University of Iowa and Iowa City VA Health Care System, Iowa City, IA, USA

Synonyms

Nicotine dependence and nicotine addiction

Definition

Cessation interventions refer to treatments designed to assist individuals with stopping the

use of a particular substance, in this case tobacco. Cessation interventions may involve either behavioral or pharmacological treatment approaches or some combination of the two.

Description

Background

Tobacco use remains the single greatest preventable cause of morbidity and premature mortality in the United States (USA). Each year, cigarette smoking is responsible for more than 440,000 deaths, five million years of potential life lost, and $190 million in excess health-care expenditures and productivity losses in the USA alone. Worldwide, tobacco use accounts for more than five million deaths annually, with the total expected to increase to eight million by the year 2030 (World Health Organization, 2008).

In the USA, rates of cigarette smoking steadily declined in the years following the publication of the first Surgeon General's Report on Smoking and Health in 1964. In fact, since the mid-1960s, the prevalence of cigarette smoking has dropped by more than 50%. Rates of tobacco use have stabilized in recent years, however, with levels remaining essentially unchanged since 2005. Currently, 20.6% of the adult population smokes cigarettes (Centers for Disease Control and Prevention [CDC], 2010b). Among high school students, the prevalence of current (past 30-day) cigarette smoking is 17.2% (CDC, 2010a). As with adults, the rate of smoking has been relatively stable in recent years following a period of significant decline during the 1990s.

A variety of sociodemographic factors are associated with cigarette use (CDC, 2010b). Overall, men (23.5%) are more likely to smoke cigarettes than women (17.9%). Adults aged 25–44 years of age are most likely be cigarette smokers (24.0%), while those aged 65 and older are the least likely to smoke (9.5%). The prevalence of cigarette smoking is also inversely associated with both educational attainment and income. Smoking rates among those with less than a high school education are 28.5% compared with 11.1% for those with an undergraduate college degree and 5.6% for those with a graduate degree. The prevalence of smoking is also elevated among those who live below the poverty line (31.1%) relative to those at or above the poverty level (19.4%). With regard to geographic variations in cigarette use, rates are lowest for those living in the West (18.8%) and highest for those residing in the Midwest (23.1%). Rates of cigarette smoking are elevated among other subgroups as well. In particular, individuals with a history of psychiatric disorders and nonnicotine substance abuse tend to smoke at very high rates relative to the general population.

Although the onset of cigarette smoking continues through young adulthood, the vast majority of regular tobacco users initiate smoking prior to the age of 18. In the early stages of smoking, use tends to be episodic and is often isolated to specific social or environmental contexts (e.g., with friends, at a party). As tolerance to nicotine develops, however, the frequency and intensity of use increase. Following a period of regular use, many smokers become nicotine dependent, which is characterized by *tolerance* (the need for greater amounts of tobacco to achieve the same effect), *compulsive use* (difficulty controlling cigarette use), and nicotine *withdrawal* (a reversible and substance-specific syndrome of behavioral, cognitive, and physiological changes brought on by the cessation or reduction of tobacco use that causes distress or impairment in functioning) (American Psychiatric Association [APA], 2000). Signs of nicotine withdrawal include dysphoria or depressed mood, insomnia, irritability, frustration, or anger, anxiety, difficulty concentrating, restlessness, decreased heart rate, and increased appetite or weight gain (APA, 2000). Although it was once assumed that nearly all regular smokers were nicotine dependent, it is now recognized that a sizeable proportion of cigarette smokers do not meet formal criteria for nicotine dependence (Hughes, Helzer, & Lindberg, 2006). Unfortunately, for those who do become nicotine dependent, cigarette use tends to follow a chronic course lasting years or decades, often characterized by multiple relapse episodes.

Treatment

A variety of effective behavioral and psychopharmacological approaches are available for the treatment of tobacco use and dependence. The range of efficacious interventions for tobacco use includes public health-based approaches such as screening and brief advice and health policy initiatives. The present review, however, will emphasize clinical approaches involving behavioral and pharmacologic treatment strategies.

Behavioral treatments have long played an important role in the treatment of tobacco use and dependence. Behavioral approaches range from brief advice lasting just a few minutes to intensive group or behavioral counseling conducted over a period of weeks. A variety of different behavioral treatments have been applied to smoking cessation including aversive therapies such as rapid smoking and smoke holding, nicotine fading, problem solving and skills training, contingency management, relaxation training, and strategies emphasizing enhanced social support. Surprisingly, little is known, however, about the relative efficacy of the individual strategies or components. This is due, in part, to the fact that most treatment programs combine a variety of different behavioral strategies and tend to be evaluated as a whole rather than according to individual components. Nevertheless, sufficient evidence is available to support the efficacy of certain behavioral strategies. In the 2008 Update to the Clinical Practice Guideline for Treating Tobacco Use and Dependence, Fiore et al. identified two specific types of behavioral interventions and counseling that have proven effective for promoting smoking cessation. These included practical counseling and the provision of intratreatment social support. Practical counseling refers to general problem solving and behavioral skills training (e.g., setting a quit date, identifying high-risk situations, developing coping skills, and providing basic information about smoking and successful quitting). Intratreatment social support simply involves providing encouragement to smokers during their quit attempt, communicating caring and concern about the smoker, and encouraging them to talk about issues related to the quitting process, such as concerns they might have about quitting and experiences they encountered during prior quit attempts.

In addition to variability in content, behavioral interventions also differ with regard to ways of administering treatment. Evidence supports the use of several different formats for the delivery of behavioral treatment for tobacco use. Both individual and group counseling have been shown to be effective strategies for treating nicotine dependence. Proactive telephone counseling, in which an initial assessment is followed by a series of scheduled sessions initiated by the clinician, is another empirically supported mode of delivery. Self-help materials, while advantageous from cost and wide-scale dissemination perspectives, have demonstrated relatively modest success as a treatment strategy. Emerging data also suggest that of computer- and Internet-based cessation programs hold promise, although clear evidence regarding the characteristics and content of programs that are most effective is currently lacking.

Recent trends in the delivery of behavioral treatment for smoking cessation have emphasized the delivery of brief behavioral counseling for purposes of widespread dissemination. Such an approach is sound from a public health perspective in that it facilitates implementation and increases potential reach. Nevertheless, evidence strongly supports a dose–response association between treatment intensity and cessation outcomes. The number of treatment sessions and total amount of contact time are both positively associated with cessation outcomes such that more intensive interventions tend to be associated with a greater likelihood of cessation.

The most effective tobacco cessation interventions are those that combine behavioral and pharmacological treatment strategies. Indeed, treatments involving medication and behavioral counseling are significantly more effective than those using only one strategy or the other. There are currently seven medications considered to be first-line pharmacotherapies for smoking cessation based on their demonstrated safety and effectiveness in the general population. Five of these agents are forms of nicotine replacement therapy

(NRT): transdermal nicotine patch, nicotine gum, nicotine lozenge, nicotine nasal spray, and nicotine inhaler. The patch, gum, and lozenge are all available over the counter in the USA, while the spray and inhaler require a prescription. The two other first-line medications are bupropion hydrochloride (trade name Zyban®), an atypical antidepressant, and varenicline (trade name Chantix®), an $\alpha_4\beta_2$ nicotinic acetylcholine (ACh) receptor partial agonist. Each of these seven agents has strong empirical evidence to support their efficacy, with no single medication demonstrating clear superiority over the others. All are associated with an approximate doubling of the odds of successful quitting relative to placebo. Medication strategies combining bupropion with the nicotine patch as well as the nicotine patch with short-acting NRT (gum or nasal spray) have also been found to improve cessation rates relative to monotherapy comprised of either agent alone. Two other medications (the antihypertensive clonidine and the antidepressant nortriptyline) are considered to be second-line pharmacotherapies for smoking cessation. Although there is considerable evidence to support their efficacy for aiding smoking cessation, neither has yet been approved by the US Food and Drug Administration (FDA) for treating tobacco use and dependence. In addition, these agents tend to have a less favorable side effect profile than most of the first-line agents. For that reason, it is recommended that they be considered primarily among those for whom the first-line agents are contraindicated or who have not been successful at quitting using those medications. To date, there is insufficient evidence to support the efficacy of pharmacotherapy for use with pregnant women, light smokers, and adolescents. For that reason, guidelines recommend that treatment focus on behavioral strategies and counseling.

Despite the range of effective behavioral and pharmacological options for assisting with tobacco cessation, the vast majority of smokers do not use an empirically supported treatment during a given quit attempt. An estimated 65–80% of smokers who attempt to quit smoking do so without the aid of behavioral or pharmacological therapies (Shiffman, Brockwell, Pillitteri, & Gitchell, 2008; Zhu, Melcer, Sun, Rosbrook, & Pierce, 2000). Behavioral interventions are especially underutilized, with less than 10% of smokers using this form of treatment during any single quit attempt (Shiffman et al., 2008; Zhu et al., 2000). Furthermore, when smokers do make use of nonpharmacological treatment approaches, they tend to use self-help materials rather than strategies with greater empirical support such as individual, group, or telephone counseling (Shiffman et al., 2008). A variety of factors appear to contribute to the underutilization of effective smoking cessation treatments including a lack of awareness of available treatment options, a preference to quit smoking on one's own, perceived inconvenience, cost, and, in the case of pharmacotherapy, concerns about side effects.

Relapse Prevention

Nicotine dependence is becoming increasingly conceptualized as a chronic and refractory condition. Even among those who do receive evidence-based treatment for smoking cessation, most who attempt cessation eventually resume smoking following a given quit attempt. Although relapse can occur months or even years after an individual quits smoking, the vast majority occurs within the first 2 weeks. The long-term cessation rates for even the most successful interventions rarely exceed 30–35% (By comparison, for those who attempt to quit on their own without assistance, 1-year abstinence rates tend to be less than 5%). Perhaps surprisingly in light of the variety of new (primarily pharmacological) treatments that have become available over the past two decades, abstinence rates among participants in clinical trials have actually decreased over time (Inrvin & Brandon, 2000; Inrvin, Hendricks, & Brandon, 2003), leading to the speculation that those who continue to smoke, though fewer in number, are more likely to be nicotine dependent and to have comorbid psychiatric and substance use disorders that make it more difficult for them to successfully quit (Inrvin & Brandon, 2000).

Given the high rates of relapse among once abstinent smokers, much attention has been given

to trying to prevent tobacco users from resuming tobacco use following a successful quit attempt. Most of this work is based on the model originally developed by Marlatt for the treatment of alcohol use disorders (Marlatt & Donovan, 2005). The approach focuses on helping individuals to identify situations in which they may be especially tempted to smoke cigarettes (e.g., when consuming alcohol, during situations of elevated stress or dysphoria, when exposed to other smokers). Once these high-risk situations are identified, smokers can be taught to avoid them (at least in the short term) or to develop alternative coping strategies to help them manage the situation without smoking. Although conceptually appealing, relapse prevention interventions based on enhancing coping skills have generally not been shown to be effective for cigarette smoking. Other psychosocial and pharmacological approaches have similarly failed to reduce relapse rates in most cases. Methodological limitations associated with this literature, however, limit the conclusions that can be drawn regarding the relative effectiveness (or ineffectiveness) of different intervention strategies. Given the high rates of relapse, new strategies for helping to maintain abstinence over the long term are clearly needed.

Addressing Smokers Who Are Not Interested in Quitting

The treatment approaches described above apply primarily to those who express interest in quitting smoking. However, despite the fact that the vast majority of smokers indicate that they would like to quit, the proportion of tobacco users who express readiness to quit smoking at any given point in time is relatively small. Therefore, it is important to identify strategies for approaching the large number of smokers who indicate that they do not presently wish to make a quit attempt.

Historically, approaches to address tobacco use among cigarette smokers who express reluctance to quit focused on providing education about the harms of smoking and attempting to persuade them to quit. Such strategies tended to be paternalistic and proscriptive in style and based on the assumption that those who continued smoking did so primarily due to a lack of knowledge about the significant health risks. However, while health education does play an important role in smoking cessation and advice to quit from one's health or mental health-care provider is frequently cited as an important factor in motivating a quit attempt, treatment strategies that rely on confrontation and which solely emphasize the clinician's role as the health expert who knows what is best for the client typically meet with little success.

One approach that has been particularly influential in the field of health behavior change, and in the treatment of addictions in particular, is motivational interviewing (MI) (Miller & Rollnick, 2002). Motivational interviewing is a directive, client-centered approach to counseling that seeks to promote behavior change by helping people to explore and resolve ambivalence. The MI approach recognizes that the majority of smokers have mixed feelings about their tobacco use. While nearly all tobacco users acknowledge the health risks and can identify other negative consequences of smoking, most also perceive it as positively reinforcing and as playing an important functional role in their lives (e.g., negative affect reduction, stress management). Helping clients to recognize and resolve their ambivalence about quitting smoking is central to the MI approach. Rather than using direct persuasion in an attempt to enforce change externally, MI takes the perspective that the individual already possesses the motivation and skills necessary to make a change. Instead of viewing motivation as something an individual does or does not have, it is seen as fluid and susceptible to movement in either direction. The goal is to elicit and strengthen the motivation and commitment through the use of "change talk," in which the individual (rather than the clinician) makes his or her own argument for quitting smoking.

Four general principles help to guide the MI approach. The first involves *expressing empathy*, which entails making an attempt to view things from the perspective of the client. The second principle is to help the client to *develop discrepancy* between his or her values/goals and their current behavior. For example, individuals who

place being a good role model for their children and being available to support their family and friends in high regard can be helped to see how smoking is incongruent with these values. The third principle involves *rolling with resistance*. It is very common for individuals faced with decisions about modifying a health behavior such as tobacco use to demonstrate resistance to change, particularly if they feel their autonomy is being threatened. Rather than try to confront or fight the client's resistance, the MI approach contends that it can be much more productive to shift strategies and use this as an opportunity to further explore their views about the behavior. The final principle focuses on helping to *support self-efficacy*. An individual's belief that they are able to successfully make a change in their behavior is strongly associated with their likelihood of doing so. Therefore, fostering one's sense of their own self-efficacy by eliciting examples of past successes or providing illustrative cases of others who have made similar behavior changes can be very beneficial. In order to help facilitate these principles and resolve their ambivalence, MI utilizes interaction techniques such as open-ended questions, reflective listening, and providing positive affirmations.

Considerable evidence now supports the use of MI for helping individuals to change their smoking behavior. Although treatment effects tend to be modest, MI has been shown to successfully increase the likelihood of smoking cessation. The approach appears to be particularly effective for those expressing low motivation to quit. Due in large part to its collaborative and nonconfrontational style which respects an individual's ability to make their own decisions about when, how, and whether to change their behavior, MI also tends to be popular among both clinicians and clients.

Summary and Conclusions

Although public health policy initiatives and treatment advances have helped to reduce the proportion of the population that smokes cigarettes, tobacco use remains the leading cause of morbidity and premature mortality in our society. Several evidence-based behavioral and pharmacologic treatments have been found to significantly improve a smoker's chances of quitting successfully. However, most smokers fail to utilize effective interventions during any given quit attempt. Even among those who do receive empirically supported treatment, relapse rates remain very high. In order to continue progress in reducing rates of cigarette smoking, it is important to identify and implement strategies for increasing the use of evidence-based treatment for tobacco use and dependence, as well as for helping to reduce high rates of relapse among those who do attempt to quit.

Cross-References

- ▶ Motivational Interviewing
- ▶ Substance Use Disorders

References and Readings

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Arlington, VA: American Psychiatric Association.

Centers for Disease Control and Prevention. (2010a). Tobacco use among middle and high school students – United States, 2000–2009. *Morbidity and Mortality Weekly Report, 59*, 1063–1068.

Centers for Disease Control and Prevention. (2010b). Vital signs: Current cigarette smoking among adults aged ≥18 years – United States, 2009. *Morbidity and Mortality Weekly Report, 59*, 1135–1140.

Fiore, M. C., Jaén, C. R., Baker, T. B., Bailey, W. C., Benowitz, N., Curry, S. J., et al. (2008). *Treating tobacco use and dependence: 2008 update*. Rockville, MD: US DHHS, Public Health Service.

Hughes, J. R., Helzer, J. E., & Lindberg, S. A. (2006). Prevalence of DSM/ICD-defined nicotine dependence. *Drug and Alcohol Dependence, 85*, 91–102.

Inrvin, J. E., & Brandon, T. H. (2000). The increasing recalcitrance of smokers in clinical trials. *Nicotine & Tobacco Research, 2*, 79–84.

Inrvin, J. E., Hendricks, P. S., & Brandon, T. H. (2003). The increasing recalcitrance of smokers in clinical trials II: Pharmacotherapy trials. *Nicotine & Tobacco Research, 5*, 27–35.

Marlatt, G. A., & Donovan, D. M. (2005). *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors* (2nd ed.). New York: Guilford Press.

Miller, W. R., & Rollnick, S. (2002). *Motivational interviewing: Preparing people for change* (2nd ed.). New York: Guilford Press.

Shiffman, S., Brockwell, S. E., Pillitteri, J. L., & Gitchell, J. G. (2008). Use of smoking-cessation treatments in the United States. *American Journal of Preventive Medicine, 34*, 102–111.

World Health Organization. (2008). *WHO report on the global tobacco epidemic, 2008: The MPOWER package*. Geneva, Switzerland: World Health Organization.

Zhu, S., Melcer, T., Sun, J., Rosbrook, B., & Pierce, J. P. (2000). Smoking cessation with and without assistance: A population-based analysis. *American Journal of Preventive Medicine, 18*, 305–311.

CF

► Cystic Fibrosis

Changing

► Aging

Character Traits

Jonathan A. Shaffer
Department of Medicine/Division of General Medicine, Columbia University Medical Center, New York, NY, USA

Synonyms

Personality; Psychosocial traits

Definition

Character traits generally refer to the temporally stable and cross-situationally consistent individual patterns in how people think, act, and feel.

Description

Associations between character traits, health behaviors, and health outcomes have been well documented (Booth-Kewley & Vickers, 1994; Ozer & Benet-Martinez, 2006). Hostility and dominance, two components of the Type A behavior pattern, have been related to asymptomatic atherosclerosis, incident coronary heart disease, and cardiac-specific and all-cause mortality (Smith, 2006). Each of the five character traits that comprises the Five Factor Model, which has been recommended as a culturally robust framework by which to guide investigations of the association between personality and health outcomes (Taylor et al., 2009), has been linked to health behaviors, including wellness behaviors, accident control, traffic risk taking, and substance risk taking (Booth-Kewley & Vickers, 1994). These broad traits of the Five Factor Model include neuroticism (e.g., anxiety, hostility, and depression), extraversion (e.g., warmth, assertiveness, and positive emotions), conscientiousness (e.g., self-discipline, order, and achievement striving), agreeableness (e.g., altruism, trust, and compliance), and openness to experience (e.g., fantasy, esthetics, and feelings). Neuroticism has additionally been linked to both distress-relevant aspects of health (DeNeve & Cooper, 1998) and disease incidence (Friedman, Kern, & Reynolds, 2010). It has likewise been shown to predict, over more than four decades, subjective well-being, physical health, and longevity (Friedman et al., 2010). Other research has shown that extraversion and conscientiousness predict longevity, low agreeableness (trait hostility) and negative affectivity predict poorer physical health and earlier mortality, and creativity predicts health and is associated with resiliency (Ozer & Benet-Martinez, 2006). Moreover, a meta-analytic review has identified optimism as a significant predictor of positive physical health outcomes with regard to all-cause mortality, survival, cardiovascular outcomes, cancer outcomes, outcomes related to pregnancy, physical symptoms, immune functioning, and pain (Rasmussen, Scheier, & Greenhouse, 2009).

Although researchers initially considered whether single diseases (such as coronary heart disease) were associated with single character traits or personality types (such as hostility and Type A personality), Friedman and Booth-Kewley

(1987) offered evidence in contradiction to this paradigm. In their meta-analysis of five emotional facets of personality (including depression and anxiety) and five chronic diseases (including coronary heart disease) thought to be affected by psychosomatic factors, they identified a pattern of associations between multiple predictors and multiple disease outcomes. Friedman and Booth-Kewley's research pointed to a broader "disease-prone personality," and suggested the importance of assessing multiple character traits and multiple health outcomes in the same study (Friedman et al., 2010). Recent studies of the associations of the five traits of the Five Factor Model with health outcomes reflect this paradigm shift. For instance, Taylor and colleagues studied whether character traits from the Five Factor Model were associated with all-cause mortality in a general adult population in Scotland and found that high conscientiousness and openness were protective against all-cause mortality in men (Taylor et al., 2009).

Current research on character traits and physical health attempts to identify mechanisms by which personality gives rise to subsequent health outcomes, and a variety of mechanistic models have been proposed (Smith, 2006). Health behavior models suggest that character traits are associated with health behaviors, which in turn elicit health outcomes. An interactional stress moderation model posits that character traits contribute to appraisal and coping, which in turn lead to physiological responses and health outcomes. A transactional stress moderation model expands the interactional model by including the bidirectional effect of personality on exposure to stressful life circumstances and availability of stress-reducing resources. Finally, the constitutional predisposition model proposes that genetic or other psychobiologic factors underlie both character traits and the development of health outcomes.

Cross-References

- ▶ Dispositional Optimism
- ▶ Heart Disease and Type A Behavior
- ▶ Neuroticism
- ▶ Personality
- ▶ Trait Anger
- ▶ Trait Anxiety
- ▶ Type A Behavior
- ▶ Type D Personality

References and Readings

Booth-Kewley, S., & Vickers, R., Jr. (1994). Associations between major domains of personality and health behavior. *Journal of Personality, 62*(3), 281–298.

DeNeve, K. M., & Cooper, H. (1998). The happy personality: A meta-analysis of 137 personality traits and subjective well-being. *Psychological Bulletin, 124*, 197–229.

Friedman, H. S., & Booth-Kewley, S. (1987). The "disease-prone personality:" A meta-analytic view of the construct. *The American Psychologist, 42*, 539–555.

Friedman, H. S., Kern, M. L., & Reynolds, C. A. (2010). Personality and health, subjective well-being, and longevity. *Journal of Personality, 78*, 179–215.

Ozer, D., & Benet-Martinez, V. (2006). Personality and the prediction of consequential outcomes. *Psychology, 57*(1), 401–421.

Rasmussen, H. N., Scheier, M. F., & Greenhouse, J. B. (2009). Optimism and physical health: A meta-analytic review. *Annals of Behavioral Medicine, 37*, 239–256.

Smith, T. (2006). Personality as risk and resilience in physical health. *Current Directions in Psychological Science, 15*(5), 227–231.

Taylor, M. D., Whiteman, M. C., Fowkes, G. R., Lee, A. J., Allerhand, M., & Deary, I. J. (2009). Five factor model personality traits and all-cause mortality in the Edinburgh artery study cohort. *Psychosomatic Medicine, 71*, 631–641.

Characteristics

- ▶ Job Diagnostic Survey

Chemical Dependency Treatment

- ▶ Substance Abuse: Treatment

Chemo, Cancer Chemotherapy

- ▶ Chemotherapy

Chemokines

► Cytokines

Chemotherapy

Yu Yamada
Department of Psychosomatic Medicine, Kyushu University, Fukuoka, Japan

Synonyms

Chemo, Cancer chemotherapy

Definition

Chemotherapy is a treatment of diseases using chemical agents or drugs, particularly the treatment of cancer by cytotoxic and other drugs. In a non-oncological setting, the term may also refer to the administration of antibiotics against microorganisms. Here, only cancer chemotherapy is discussed.

The main purpose of chemotherapy is to systemically kill cancer cells in the body. Most traditional drugs that are used in chemotherapy interfere with the ability of cells to grow and multiply. The variety of chemotherapy drugs are classified based on how they work. For example, alkylating agents, like cyclophosphamide, kill cells by directly attacking DNA. Antimetabolites, like methotrexate, interfere with the production of DNA and the growth and multiplication of cells. Topoisomerase-interacting agents, antimicrotubule agents, and miscellaneous chemotherapeutic agents are traditional chemotherapy drugs. These drugs target not only cancer cells but also normal cells in the body. In contrast, there has been a recent emergence of targeted therapy, which involves drugs that block the growth of only cancer cells by interfering with specific targeted molecules needed for carcinogenesis and tumor growth. Small-molecule tyrosine kinase inhibitors, like imatinib mesylate, and monoclonal antibodies, like trastuzumab, are used in targeted therapy.

Chemotherapy drugs can be administered orally, by injection, through a catheter or port, or topically. Chemotherapy drugs are most often administered in combination, based on the known biochemical actions of available anticancer drugs. To achieve superior outcome with combined cancer chemotherapy, drugs which function through separate cytotoxic mechanisms and have different dose-limiting adverse effects are administered together at full dosages. Patients may undergo chemotherapy at regular intervals, i.e., once a week and once a month, depending on the type of cancer and drug therapy.

As most chemotherapy drugs are toxic to cancer cells as well as normal healthy cells, they can cause a variety of side effects, including hair loss, anemia, loss of appetite, nausea, and vomiting.

Several behavioral medicine studies have also suggested impairment of cognitive functions, such as memory and attention, in some patients who receive chemotherapy, mostly as adjuvant treatment for breast cancer. This impairment is referred to as "chemo-brain" or "chemo-fog." Despite increasing research in this area, the mechanisms behind chemotherapy-induced cognitive impairment remain largely unknown. Future studies are expected to shed light on both the prevention and treatment of "chemo-brain."

Cross-References

► Cancer Treatment and Management
► Cancer, Types of

References and Readings

Ahles, T. A., & Saykin, A. J. (2007). Candidate mechanisms for chemotherapy-induced cognitive changes. *Nature Reviews Cancer, 7*(3), 192–201.

DeVita, V. T., & Lawrence, T. S. (2008). *DeVita, Hellman, and Rosenberg's Cancer (Cancer: Principles and Practice)*. Philadelphia: Lippincott Williams and Wilkins.

Kennedy, B. J. (1999). Medical oncology: Its origin, evolution, current status, and future. *Cancer, 85*(1), 1–8.
Tannock, I. F., Ahles, T. A., Ganz, P. A., et al. (2004). Cognitive impairment associated with chemotherapy for cancer: Report of a workshop. *Journal of Clinical Oncology, 22*(11), 2233–9.

Chesney, Margaret

Margaret A. Chesney
Department of Medicine & Center for Integrative Medicine, University of California,
San Francisco, CA, USA

Biographical Information

Margaret Chesney was born in Baltimore, Maryland. She graduated from Whitman College in 1971 and received her PhD in Clinical and Counseling Psychology from Colorado State University in 1975. She received postdoctoral training in psychiatry from the Western Pennsylvania Psychiatric Institute where she studied behavioral approaches to improving psychological and physical health. In 1976, she joined Stanford Research Institute (SRI) to carry out research on stress and health. In 1978, she became Director of the new Department of Behavioral Medicine at SRI. In 1987, she moved her research to the Department of Medicine, University of California San Francisco (UCSF), to contribute behavioral medicine perspectives to the prevention and treatment of HIV/AIDS.

From 2000 to 2003, while at UCSF, Chesney pursued policy studies as a Senior Fellow at the Center for the Advancement of Health in Washington, DC, and served as a Scientific Advisor to the Office for Research on Women's Health at the National Institutes of Health (NIH). In 2003, she became Deputy Director of the National Center for Complementary and Alternative Medicine (NCCAM) and a Senior Advisor to the Director of the Office of Behavioral and Social Sciences Research at NIH. In 2010, Margaret returned to UCSF as Professor in Residence in the Department of Medicine, the Osher Foundation Distinguished Professor in Integrative Medicine, and Director of the Osher Center for Integrative Medicine at UCSF.

Throughout her career, Chesney has chaired and served on numerous advisory groups for the NIH and the State of California, covering topics including health promotion and disease prevention, living with and beyond chronic illness, women's health, and health-care policy. She is currently the Associate Editor of *Psychology, Health and Medicine*. Chesney has been President of the Academy of Behavioral Medicine Research, as well as President of the American Psychosomatic Society and President of the Division of Health Psychology of the American Psychological Association. She received the Distinguished Scientist Award from the Society of Behavioral Medicine in 2011, the Director's Award for work in Mind-Body Medicine from the NIH in 2005, the Charles C. Shepard Science Award, from the Centers for Disease Control and Prevention in 1999, and the President's Award from the Academy of Behavioral Medicine Research in 1987. In 2001, she was elected to the Institute of Medicine. She received an honorary doctorate from her alma mater, Whitman College, in 2008.

Major Accomplishments

Chesney has been engaged in clinical practice and research in the areas of stress, mind-body

interactions, and health. Her earliest studies involved the use of relaxation-based exercises as an alternative to medication for managing pain. Extending this approach to coronary heart disease, Chesney carried out a number of studies to identify the coronary-prone features of the Type A behavior pattern. With colleagues at SRI, she reported that hostility, competitiveness, and depressed mood are characteristics associated with increased risk of coronary events. She followed this work with trials investigating lifestyle interventions designed to promote health, prevent disease, and enhance well-being in both women and men.

In the late 1980s, Chesney was invited to join the Center for AIDS Prevention Studies at UCSF to develop behavioral interventions for persons living with HIV/AIDS. With Susan Folkman, Chesney carried out research on stress and coping among caregivers of persons with HIV/AIDS and developed a cognitive behavioral intervention, Coping Effectiveness Training (CET), for persons with HIV/AIDS, based on stress and coping theory. Shown to be effective with HIV, CET has been successfully applied to enhance coping with other chronic conditions including spinal cord injury and cancer. In addition, Chesney developed measures of adherence and led randomized trials of behavioral strategies to increase adherence to the complex treatment regimens for HIV/AIDS. She was also one of two leaders of a San Francisco community-based study to encourage persons infected with HIV to seek immediate treatment within the first days of infection, a study that led NIH to create a network investigating treatment of "primary HIV infection."

Her interest in policy level interventions for health promotion brought Chesney to Washington, DC, and NIH where she became familiar with the emerging field of integrative medicine. Returning to UCSF, her current research is investigating breathing, a core feature of many behavioral or integrative medicine interventions. With David Anderson, she is investigating whether breathing patterns may be a mechanism by which mind-body interventions influence blood pressure, a major risk factor for cardiovascular disease.

Cross-References

- ▶ Coping
- ▶ Integrative Medicine
- ▶ Stress Management

References and Readings

Anderson, D. E., & Chesney, M. A. (2002). Gender-specific association of perceived stress with inhibited breathing pattern. *International Journal of Behavioral Medicine, 9*, 216–277.

Anderson, D. E., & Chesney, M. A. (2009). Gender differences in the role of stress and emotion in cardiovascular function and disease. In M. J. Legato (Ed.), *Principles of gender-specific medicine* (2nd ed., Vol. 2, pp. 186–199). New York: Elsevier.

Chesney, M. A. (2006). The elusive gold standard: Future perspectives for HIV adherence assessment and intervention. *Journal of Acquired Immune Deficiency Syndrome, 43*(Suppl. 1), S149–S155.

Chesney, M. A., Black, G. W., Swan, G. E., & Ward, M. M. (1987). Relaxation training for essential hypertension at the worksite. The untreated mild hypertensive. *Psychosomatic Medicine, 49*, 250–263.

Chesney, M. A., Chambers, D. B., Taylor, J. M., Johnson, L. M., & Folkman, S. (2003). Coping effectiveness training for men living with HIV: Results from a randomized clinical trial testing a group-based intervention. *Psychosomatic Medicine, 65*, 1038–1046.

Chesney, M. A., Darbes, L., Hoerster, K., Taylor, J., Chambers, D. C., & Anderson, D. E. (2005). Positive emotions: The other hemisphere of behavioral medicine. *International Journal of Behavioral Medicine, 12*, 50–58.

Chesney, M. A., Hecker, M. H. L., & Black, G. W. (1988). Coronary-prone components of Type A behavior in the WCGS: A new methodology. In B. K. Houston & C. R. Snyder (Eds.), *Type A behavior pattern: Research, theory and intervention* (pp. 168–188). New York: Wiley.

Chesney, M. A., Ickovics, J. R., Chambers, D. B., Gifford, A. L., Neidig, J., Zwickl, B., et al. (2000). Self-reported adherence to antiretroviral medications among participants in HIV clinical trials: The AACTG adherence instruments. *AIDS Care, 12*(3), 255–266.

Chesney, M. A., Koblin, B. A., Barresi, P. J., Husnik, M. H., Celum, D. L., Colfax, G., et al. (2003). An individually-tailored intervention for HIV prevention: Baseline data from the EXPLORE Study. *American Journal of Public Health, 93*, 933–938.

Chesney, M. A., Neilands, T. B., Chambers, D. B., Taylor, J. M., & Folkman, S. (2006). A validity and reliability study of the coping self-efficacy scale. *British Journal of Health Psychology, 11*, 421–37.

Chest Pain

Siqin Ye
Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Synonyms

Angina pectoris

Definition

Acute chest pain is the common symptom of a multitude of medical conditions, ranging from the life threatening, such as myocardial infarction, pulmonary embolism, pneumothorax, and aortic dissection; to the less serious, such as esophageal reflux, peptic ulcer disease, and gallbladder disease; to benign entities, such as pericarditis, costochondritis, and panic attacks. As such, it is also one of the most frequent causes for ER presentation in the United States, accounting for as many as seven million visits annually. Rapid triage and accurate diagnostic workup are thus cornerstones of care for these patients (Cannon & Lee, 2008; Lee & Goldman, 2000).

The evaluation of acute chest pain begins with a thorough history and physical that helps to distinguish the underlying etiology and guide testing. For instance, clinical features that are suggestive of myocardial infarction include prior history of coronary artery disease, pain or pressure radiating to the arm or jaw, and association with nausea, vomiting, or diaphoresis (Panju, Hemmelgarn, Guyatt, & Simel, 1998). Pain that is pleuritic (i.e., worse with deep inspiration) can be caused by pulmonary embolism, while chest pain caused by aortic dissection is typically described as excruciating and tearing or ripping in quality, often radiating to the back. Pain that is worse with manual palpation, on the other hand, suggests a chest wall process such as costochondritis and is often reassuring. Rapid ECG at time of presentation is recommended to rule out ST-elevation myocardial infarction, and other testing such as serial biomarkers (e.g., cardiac troponins or creatine kinase MB isoenzyme), chest X-ray, and CT angiography of chest and thorax can be obtained based on the clinical suspicion (Cannon & Lee, 2008; Lee & Goldman, 2000). Despite the availability of these tests, however, the challenge remains to balance the need to correctly diagnose patients with life-threatening conditions with avoidance of the harm that can occur from unnecessary testing of those who are truly at low risk.

Cross-References

▶ Angina Pectoris

References and Readings

Cannon, C. P., & Lee, T. H. (2008). Approach to the patient with chest pain. In P. Libby, R. O. Bonow, D. L. Mann, D. P. Zipes, & E. Braunwald (Eds.), *Braunwald's heart disease: A textbook of cardiovascular medicine* (pp. 1195–1205). Philadelphia: Saunders Elsevier.

Lee, T. H., & Goldman, L. (2000). Evaluation of the patient with acute chest pain. *The New England Journal of Medicine, 342*(16), 1187–1195.

Panju, A. A., Hemmelgarn, B. R., Guyatt, G. H., & Simel, D. L. (1998). Is this patient having a myocardial infarction? *Journal of the American Medical Association, 280*(14), 1250–1263.

Child Abuse

Melissa Merrick[1] and Jason Jent[2]
[1]Division of Violence Prevention, Centers for Disease Control & Prevention, Atlanta, GA, USA
[2]Department of Pediatrics, Mailman Center for Child Development, University of Miami, Miami, FL, USA

Synonyms

Acts of commission

Definition

Child abuse is defined as acts of commission that include the use of words or overt actions that cause harm, potential harm, or threat of harm to a child (Leeb, Paulozzi, Melanson, Simon, & Arias, 2008). These are deliberate and intentional acts of commission by a caregiver, whether or not harm to a child was the intended consequence.

A caregiver is defined as a person who at the time of the maltreatment is in a permanent (primary caregiver) or temporary (substitute caregiver) role. In a custodial role, the person is responsible for care and control of the child and for the child's overall health and welfare. A primary caregiver lives with the child at least part of the time and can include, but is not limited to, a relative or biological, adoptive, step-, or foster parent(s); a legal guardian(s); or their intimate partner. A substitute caregiver may or may not live with the child and can include coaches, clergy, teachers, relatives, babysitters, residential facility staff, or others who are not the child's primary caregiver(s).

Acts of omission, child neglect, are discussed in a separate entry.

Types

There are three different forms of child abuse that involve acts of commission: physical abuse, sexual abuse, and psychological/emotional abuse.

Physical abuse is defined as the intentional use of physical force against a child that results in, or has the potential to result in, physical injury (Leeb et al., 2008). Physical abuse includes physical acts that range from those which do not leave a physical mark on the child to those which cause permanent disability, disfigurement, or even death. Examples of physical abuse can include hitting, kicking, punching, beating, stabbing, biting, pushing, throwing, pulling, dragging, dropping, shaking, choking, smothering, burning, scalding, and poisoning.

Sexual abuse is defined as any completed or attempted sexual act, sexual contact with, or exploitation (i.e., noncontact sexual interaction) of a child by a caregiver (Leeb et al., 2008). Sexual acts include contact involving penetration, however slight, between the mouth, penis, vulva, or anus of the child and another individual. Sexual acts also include penetration, however slight, of the anal or genital opening by a hand, finger, or other object. Sexual acts can be performed by the caregiver on the child or by the child on the caregiver. A caregiver might also force or coerce a child to commit a sexual act on another individual (child or adult). Abusive sexual contact involves intentional touching, either directly or through the clothing, of the following: genitalia (penis or vulva), anus, groin, breast, inner thigh, and/or buttocks. Abusive sexual contact can be performed by the caregiver on the child or by the child on the caregiver. Abusive sexual contact can also occur between the child and another individual (adult or child) through force or coercion by a caregiver. Touching that is required for the normal care or attention to the child's daily needs does not constitute abusive sexual contact.

Noncontact sexual abuse can include any of the following: (a) exposing a child to sexual activity (e.g., pornography, voyeurism of the child by an adult, intentional exposure of a child to exhibitionism); (b) filming a child in a sexual manner (e.g., depiction, either photographic or cinematic, of a child in a sexual act); (c) sexually harassing a child (e.g., quid pro quo, creating a hostile environment because of comments or attention of a sexual nature by a caregiver to a child); and (d) prostituting a child (e.g., employing, using, persuading, inducing, enticing, encouraging, allowing, or permitting a child to engage in or assist any other person to engage in prostitution or sexual trafficking).

Psychological/emotional abuse includes intentional caregiver behavior that conveys to a child that he/she is worthless, flawed, unloved, unwanted, endangered, or valued only in meeting another's needs (Leeb et al., 2008). Psychological/emotional abuse can be continual or episodic (e.g., triggered by a specific context or situation). Psychologically/emotionally abusive behaviors often consist of blaming, belittling, degrading, intimidating, terrorizing, isolating, restraining, confining, corrupting, exploiting, or otherwise behaving in a manner that is harmful, potentially

harmful, or insensitive to the child's developmental needs or can potentially damage the child psychologically or emotionally.

Description

Prevalence

In 2008, US state and local child protective services (CPS) received 3.3 million reports of children being abused and/or neglected. CPS estimated that 772,000 (10.3 per 1,000) of these children had substantiated cases of child abuse and/or child neglect. Approximately three quarters of them had no history of prior victimization. Sixteen percent of the children were classified as victims of physical abuse, 9% as victims of sexual abuse, and 7% as victims of psychological/emotional abuse (USDHHS, 2010). The remaining children were classified as victims of child neglect. A recent national study estimated that 1 in 5 US children has experienced some form of child abuse or neglect in their lifetime, with a rate of 1 in 10 experiencing some form of child abuse or neglect in the past year (Finkelhor, Turner, Ormrod, & Hamby, 2009). In 2008, a CPS-based study found that African-American (16.6 per 1,000 children), American Indian or Alaska Native (13.9 per 1,000 children), and multiracial (13.8 per 1,000 children) children had higher rates of victimization than other racial groups, with slightly higher rates for girls (10.8 per 1,000 children) than boys (9.7 per 1,000 children) overall (USDHHS, 2010). Research has demonstrated similar negative sequelae for children who have substantiated CPS reports of abuse and for children who have alleged or suspected CPS reports of abuse (Hussey et al., 2005).

Etiology and Sequelae

A combination of individual, relational, community, and societal factors contributes to the risk of child abuse. Although children are not responsible for the harm inflicted upon them, certain characteristics have been found to increase their risk of being abused (Berliner, 2011; Centers for Disease Control and Prevention, 2009; Runyon & Urquiza, 2011). Individual child factors that increase a child's vulnerability include child age younger than 4 years and those children with special needs. Also, parents' lack of understanding of child development and parenting skills; parents' history of child abuse, substance abuse, and/or mental health issues; parental characteristics such as young age, low education, single parenthood, large number of dependent children, and low income; and nonbiological, transient caregivers in the home (e.g., mother's male partner) all seem to increase the risk of perpetration of child abuse in the home. Other risk factors for perpetration include poor social connections and support, family violence (e.g., intimate partner violence), poor parent-child relationships, parenting stress, community violence, and concentrated neighborhood disadvantage (e.g., high poverty and residential instability, high unemployment rates).

Extensive research demonstrates that child abuse can have devastating effects on physical and mental health. Abuse during infancy or early childhood can cause important regions of the brain to form and function improperly with long-term consequences on cognitive, language, and socioemotional development and mental health. Children may experience severe or fatal head trauma as a result of abuse. Nonfatal consequences of abusive head trauma include varying degrees of visual impairment (e.g., blindness), motor impairment (e.g., cerebral palsy), and cognitive impairments (Christian, Block, & The Committee on Child Abuse & Neglect, 2009). Also, the stress of chronic abuse may result in anxiety and may make children more vulnerable to problems such as posttraumatic stress disorder, conduct disorder, and learning, attention, and memory difficulties (Dallam, 2001; Perry, 2001). Studies have found abused children are more likely to be arrested or become involved in delinquent and violent behavior in adolescence and experience teen pregnancy, low academic achievement, and decreased high school graduation rates (Langsford et al., 2007). Abused children are also at increased risk for adverse health behaviors, such as smoking, alcoholism, drug abuse, and engaging in high-risk sexual behaviors, which often lead to certain chronic diseases as adults, including heart disease, cancer, chronic lung

disease, liver disease, obesity, high blood pressure, and high cholesterol (Runyan, Wattam, Ikeda, Hassan, & Ramiro, 2002). In one long-term study, as many as 80% of young adults who had been abused met the diagnostic criteria for at least one psychiatric disorder at age 21. These young adults exhibited many problems, including depression, anxiety, eating disorders, and suicide attempts (Silverman, Reinherz, & Giaconia, 1996). Abuse can also increase the likelihood of adult criminal behavior and violent crime (Widom & Maxfield, 2001). Finally, early child abuse can have a negative effect on the ability of both men and women to establish and maintain healthy intimate relationships in adulthood (Colman & Widom, 2004), which may also perpetuate the cycle of violence from one generation to the next.

Cross-References

► Family Violence

References and Readings

Berliner, L. (2011). Child sexual abuse. In E. John & B. Myers (Eds.), *The APSAC handbook on child maltreatment* (3rd ed., pp. 215–232). Thousand Oaks, CA: Sage.

Centers for Disease Control and Prevention. (2009). *Child maltreatment: Risk and protective factors*. Retrieved July 20, 2011, from http://www.cdc.gov/Violence-Prevention/childmaltreatment/riskprotectivefactors.html

Christian, C. W., Block, R., & The Committee on Child Abuse & Neglect. (2009). American academy of pediatrics policy statement: Abusive head trauma in infants and children. *Pediatrics, 123*, 1409–1411.

Colman, R., & Widom, C. (2004). Childhood abuse and neglect and adult intimate relationships: A prospective study. *Child Abuse & Neglect, 28*, 1133–1151.

Dallam, S. J. (2001). The long-term medical consequences of childhood maltreatment. In K. Franey, R. Geffner, & R. Falconer (Eds.), *The cost of child maltreatment: Who pays? We all do*. San Diego, CA: Family Violence & Sexual Assault Institute.

Finkelhor, D., Turner, H., Ormrod, R., & Hamby, S. (2009). Violence, abuse, and crime exposure in a national sample of children and youth. *Pediatrics, 124*, 1411–1423.

Hussey, J., Marshall, J., English, D., Knight, E., Lau, A., Dubowitz, H., et al. (2005). Defining maltreatment according to substantiation: Distinction without a difference? *Child Abuse & Neglect, 29*, 479–492.

Langsford, J. E., Miller-Johnson, S., Berlin, L. J., Dodge, K. A., Bates, J. E., & Pettit, G. S. (2007). Early physical abuse and later violent delinquency: A prospective longitudinal study. *Child Maltreatment, 12*, 233–245.

Leeb, R. T., Paulozzi. L., Melanson, C., Simon, T., & Arias, I. (2008). *Child maltreatment surveillance: Uniform definitions for public health and recommended data elements, version 1.0*. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control. Retrieved July 20, 2011, from http://www.cdc.gov/violenceprevention/pdf/CM_Surveillance-a.pdf

Perry, B. D. (2001). The neurodevelopmental impact of violence in childhood. In D. Schetky & E. Benedek (Eds.), *Textbook of child and adolescent forensic psychiatry* (pp. 221–238). Washington, DC: American Psychiatric Press.

Runyan, D., Wattam, C., Ikeda, R., Hassan, F., & Ramiro, L. (2002). Child abuse and neglect by parents and other caregivers. In E. Krug, L. L. Dahlberg, J. A. Mercy, A. B. Zwi, & R. Lozano (Eds.), *World report on violence and health* (pp. 59–86). Geneva, Switzerland: World Health Organization.

Runyon, M. K., & Urquiza, A. J. (2011). Child physical abuse: Interventions for parents who engage in coercive parenting practices and their children. In E. John & B. Myers (Eds.), *The APSAC handbook on child maltreatment* (3rd ed., pp. 195–214). Thousand Oaks, CA: Sage.

Silverman, A. B., Reinherz, H. Z., & Giaconia, R. M. (1996). The long-term sequelae of child and adolescent abuse: A longitudinal community study. *Child Abuse & Neglect, 20*, 709–723.

U.S. Department of Health and Human Services, Administration on Children, Youth and Families. *Child Maltreatment* 2008, Washington, DC: U.S. Government Printing Office, 2010. Retrieved from http://www.acf.hhs.gov

Widom, C. S., & Maxfield, M. G. (2001). *An update on the "cycle of violence."* Washington, DC: National Institute of Justice; 2001. Retrieved July 20, 2011, from http://www.ncjrs.gov/pdffiles1/nij/184894.pdf

Child Development

Debbie Palmer
Department of Psychology, University of Wisconsin-Stevens Point, Stevens Point, WI, USA

Synonyms

Adolescent psychology; Child psychology; Developmental psychology; Pediatric psychology

Definition

The field of child development is concerned with the scientific study of human growth and functioning across the early stages of development (i.e., the prenatal period through adolescence) and within the multitude of contexts of daily life. Areas of interest include – though are not limited to – biological, cognitive, physical, social, and emotional change across the early portions of life. In all cases, an emphasis is placed on understanding how normative functioning changes or remains constant across time as a result of maturation and/or experience (Lerner, 2006). Child development is one aspect of the broader field of Developmental Psychology, which examines human growth and functioning across the entire lifespan.

Description

The field of child development is concerned with the scientific study of human growth and functioning throughout the early portions of life, including the prenatal period through adolescence. The entire human lifespan includes the prenatal period, infancy, childhood (early, middle, and late), adolescence (early, middle, and late), emerging adulthood, and adulthood (early, middle, and late) (Arnett, 2004; Santrock, 2012; Steinberg, 2011). One way the earliest periods of human life may be contemplated is in terms of chronological age. From this perspective, the prenatal period spans the time of conception through birth and lasts approximately 9 months for a typical pregnancy. Infancy encompasses from birth through 18 or 24 months of age. Early childhood includes from 18 or 24 months through 5 or 6 years of age. Middle and late childhood runs from 5 or 6 years through approximately 11 years of age. Adolescence is from around 11 years of age through approximately 18 years of age.

Child development considers the multitude of contexts of daily life that humans encounter. Areas of interest include – though are not limited to – biological, cognitive, social, and emotional developments, with an emphasis on how normative functioning and processes either change or remain constant across time as a result of maturation and/or experience (Lerner, 2006). *Biological processes* in infancy and childhood entail the growth and maturation of the internal organ systems and observable increases in both height and weight, and how these connect to the development and refinement of advancing motor skills. Subsequent biological growth in adolescence is demonstrated by secondary sexual maturation through the process of pubertal development and the attainment of more adult-like stature and weight. *Cognitive growth* in infancy and childhood includes rapid gains in language processing, production, and comprehension. Memory capacity expands and more sophisticated strategies for retention and recall are demonstrated. Also, individuals gain enhanced understanding of logic related to concrete concepts in childhood and to abstract concepts in adolescence. *Social growth* in infancy and childhood involves dependent interactions and attachments to caregivers and expands to include peer and friendship relationships. Later in adolescence, social networks expand to include cliques and significant others as increasingly intimate relationships develop. Early *emotional growth* in infancy and childhood entails the presence of primary feelings and the development of self-conscious emotions. Subsequent growth in adolescence involves enhanced understanding of societal rules for the display and regulation of emotions and coping with life's challenges.

These different domains of growth and development do not occur independently, but are interrelated. That is, an infant who smiles at the appearance of his or her father requires biological functioning (the sensation of seeing), cognitive and social functioning (recognition of and feeling attached to a familiar caregiver), and emotional functioning (smiling) (Santrock, 2012). Child development explores not only how these different domains develop but also how their interrelated processes are manifested in milestones associated with each of the early periods of human life. Prenatal development entails the development from a single fertilized egg to

a fetus that is able to function outside the mother's womb. The growth across the approximate 9-month period of time prepares the organism for the life ahead of it. In early childhood, individuals are making rapid strides in autonomy development and gaining self-control, which gets manifested in a variety of milestone achievements, such as potty training and turn-taking (Berk, 2003). Children are also being prepared to enter the formal education system at this time. In middle childhood, children typically enter the formal educational system and attend elementary school. Emphasis in milestone achievement is usually placed on academic ability, with fundamentals such as reading, writing, and basic mathematical skills attained and refined during this time. The social world of children also expands to include more peers and adults beyond family members. Extracurricular activities such as those involving sports and other cultural aspects of society (e.g., dance, music) are often initiated during this time (Santrock, 2012). During adolescence, an emphasis is often placed on the future, with preparation for later education, careers, and relationships being stressed. Increased time is spent with peers away from the family unit in less supervised settings. Increasingly, and across numerous contexts, responsibility is gained, along with enhanced expectations from others for self-reliant behavior and maturity. Rapid biological changes occur as result of pubertal development, which enables the adolescent to become capable of reproduction and leads to changes in social relationships. Self-images become more complex to incorporate sexual and identity development. More influence is sought within family functioning, which necessitates adjustments in how parents and siblings relate to the adolescent (Steinberg, 2011).

Theoretical approaches in child development can adhere to continuous or discontinuous conceptualizations. Continuous approaches cast development as gradually changing across time and experience, while discontinuous approaches cast development as being qualitatively distinct across each life stage. A scenario that illustrates the continuous approach is offered by Berk (2003), who suggested that babies and preschoolers may respond to the world in a manner very similar to how adults respond. That is, a child's thinking may be just as logical and well organized as that of an adult. He or she may demonstrate the ability to successfully sort objects in to different categories (e.g., clothes are separate from toys), show understanding when there are different quantities or amounts present (e.g., more cookies are in the jar than are on the plate), and retain information for long periods of time (e.g., go straight to where the DVDs are stored at grandma's house after not visiting for weeks). However, a child's thinking may be limited by how little experience he or she has had in interacting with the world. From this perspective, a child possesses the same skills as an adult, but has simply not acquired as much information or been able to refine these skills compared to adults. An example of a discontinuous approach to cognitive development includes the theoretical work of Jean Piaget, who emphasized the importance of adaptation to one's environment and increasing organization of knowledge across development (Piaget, 1954). He stated that cognition unfolded in an invariant manner, across four major stages: the sensorimotor period (experiencing the world through senses and actions), the preoperational period (representing things with words and images but lacking logical reasoning), the concrete operational period (thinking logically about concrete events, analogies), and the formal operational period (reasoning abstractly). According to Piaget, cognitive development could be described as occurring consistently across cultures and children were active agents and not merely passive recipients in their own development (Piaget, 1954).

Knowledge of child development is important to the field of behavioral medicine in numerous ways. Knowledge of normal child development can be extremely useful for parents, teachers, and health-care providers – as well as many others – who may encounter and interact with those who manifest diseases and/or deviations from normal development. Knowledge of typical development can aid in detecting and treating atypical development, and enables researchers and clinicians to develop the most appropriate care for children

with acute and chronic medical conditions, while also meeting children's developmental needs. Programs crafted for adult patients to educate or alter behaviors impacting health may not be appropriate for younger patients. Likewise, the effectiveness of health-care treatment of diseases and disorders in childhood can be directly impacted by biological or other developmental processes. For instance, changes in pubertal hormones among adolescents with type 1 diabetes can dysregulate glucose metabolism. By anticipating these biological changes, clinicians may be able to forewarn adolescent patients with type 1 diabetes and their parents and develop possible strategies to minimize deterioration in illness self-management during adolescence (Halvorson, Yasuda, Carpenter, & Kaiserman, 2005).

Child development research can also reveal periods of risk, when primary or secondary prevention efforts may be most effective. Health and health risk behaviors that affect morbidity and mortality in later life are established early in life. For instance, food selection choices (e.g., fast food versus more nutritionally balanced items) and decisions to be physically active may become consistent behaviors during childhood and adolescence. Similarly, high-risk behaviors (e.g., sexual experimentation, tobacco and alcohol use) often become relevant concerns during adolescence (Williams, Holmbeck, & Greenley, 2002). Thus, interventions to promote healthy behaviors and to prevent health risk behaviors may be most effective during childhood and adolescence. Other known periods of risk occur at important developmental transitions such as when rapid autonomy development among adolescents with pediatric conditions may conflict with the efforts of parental or family caregivers. Researchers in behavioral medicine/pediatric psychology have demonstrated the need to consider autonomy in the management of chronic conditions such as spina bifida and type 1 diabetes (Buchbinder, 2009; Friedman, Holmbeck, DeLucia, Jandasek, & Zebracki, 2009). Another period of risk that has gained recent attention for youth with chronic conditions is that of emerging adulthood, the time between late adolescence and the establishment of one's identity as an adult (ages 18–25 years). This transition is risky partially because it involves a transition from a pediatric to an adult health-care system (i.e., pediatrician may treat an individual until he or she is 18; age limits on parents' health insurance policy), which generally needs to be addressed during the earlier adolescent years. Unfortunately, the transition from adolescence to emerging adulthood or young adulthood remains a significant challenge for caregivers and their patients and the health-care system (Huang et al., 2011).

There are numerous professional organizations that support practice and research at the interface of child development and behavioral medicine. The Child and Family Health Special Interest Group of the Society of Behavioral Medicine is an interdisciplinary forum for researchers and clinicians to promote child health and development, prevent childhood illness and injury, and foster family adjustment to chronic illnesses. Other relevant organizations include the Society of Pediatric Psychology (Division 54 of the American Psychological Association), Society for Research on Child Development, Society for Research on Adolescence, and Eunice Kennedy Shriver National Institute of Child Health & Human Development.

Cross-References

- ▶ Diabesity in Children
- ▶ Family, Caregiver
- ▶ Health Behaviors
- ▶ Health Risk (Behavior)
- ▶ National Children's Study
- ▶ Prevention: Primary, Secondary, Tertiary
- ▶ Society of Behavioral Medicine

References and Readings

Arnett, J. (2004). *Emerging adulthood: The winding road from the late teens through the twenties*. New York: Oxford University Press.

Berk, L. E. (2003). *Child development* (6th ed.). Boston: Allyn and Bacon.

Buchbinder, M. (2009). The management of autonomy in type 1 diabetes: A case study of triadic medical

interaction. *Health: An Interdisciplinary Journal for the Social Study of Health, Illness and Medicine, 13*(2), 175–196. doi:10.1177/1363459308099683.
Friedman, D., Holmbeck, G., DeLucia, C., Jandasek, B., & Zebracki, K. (2009). Trajectories of autonomy development across the adolescent transition in children with spina bifida. *Rehabilitation Psychology, 54*(1), 16–27. doi:10.1037/a0014279.
Halvorson, M., Yasuda, P., Carpenter, S., & Kaiserman, K. (2005). Unique challenges for pediatric patients with diabetes. *Diabetes Spectrum, 18*(3), 167–173. doi:10.2337/diaspect.18.3.167.
http://www.cfw.tufts.edu/
http://www.healthychildren.org/
http://www.nlm.nih.gov/medlineplus/teendevelopment.html
http://www.srcd.org/
http://www.zerotothree.org/
Huang, J. S., Gottschalk, M., Pian, M., Dillon, L., Barajas, D., & Bartholomew, L. K. (2011). Transition to adult care: Systematic assessment of adolescents with chronic illnesses and their medical teams. *Journal of Pediatrics, 159*(6), 994–998. doi:10.1016/j.jpeds.2011.05.038.
Lerner, R. M. (2006). Developmental science, developmental systems, and contemporary theories of human development. In R. M. Lerner, W. Damon, & R. M. Lerner (Eds.), *Handbook of child psychology* (Theoretical models of human development 6th ed., Vol. 1, pp. 1–17). Hoboken, NJ: Wiley.
Piaget, J. (1954). *The construction of reality in the child*. New York: Harcourt Brace Jovanovich.
Santrock, J. W. (2012). *A topical approach to life-span development* (6th ed.). New York: McGraw-Hill.
Steinberg, L. (2011). *Adolescence* (9th ed.). New York: McGraw-Hill.
Williams, P. G., Holmbeck, G. N., & Greenley, R. N. (2002). Adolescent health psychology. *Journal of Consulting and Clinical Psychology, 70*(3), 828–842. doi:10.1037//0022-006X.70.3.828.

Child Neglect

Jason Jent[1] and Melissa Merrick[2]
[1]Department of Pediatrics, Mailman Center for Child Development, University of Miami, Miami, FL, USA
[2]Division of Violence Prevention, Centers for Disease Control & Prevention, Atlanta, GA, USA

Synonyms

Caregiver acts of omission

Definition

Child neglect is defined as acts of omission by a caregiver that include failure to provide for a child's basic physical, emotional, or educational needs and/or failure to protect a child from harm or potential harm (Leeb, Paulozzi, Melanson, Simon, & Arias, 2008). The resultant harm to a child may or may not be the intended consequence of the act of omission, but still represents neglect. Neglect typically consists of a chronic pattern of acts of omission by a caregiver that result in actual or potential harm to a child. However, there are specific singular instances where failure to supervise can result in significant harm to a child (e.g., injury, death).

A caregiver is defined as a person who at the time of the maltreatment is in a permanent (primary caregiver) or temporary (substitute caregiver) role. In a custodial role, the person is responsible for care and control of the child and for the child's overall health and welfare. A primary caregiver lives with the child at least part of the time and can include, but is not limited to, a relative or biological, adoptive, step-, or foster parent(s); a legal guardian(s); or their intimate partner. A substitute caregiver may or may not live with the child and can include coaches, clergy, teachers, relatives, babysitters, residential facility staff, or others who are not the child's primary caregiver(s).

Acts of commission, child abuse, are discussed in a separate entry.

Types

A caregiver's failure to provide a child's basic needs may result in specific types of neglect including: physical neglect, emotional neglect, medical neglect, and educational neglect (Barnett, Manly, & Cicchetti, 1993).

Physical neglect is defined as a caregiver's failure to provide a child adequate nutrition, hygiene, or shelter; or caregiver fails to provide clothing that is adequately clean, appropriate size, or adequate for the weather (Leeb et al., 2008).

Emotional neglect occurs when a caregiver ignores the child or denies emotional responsiveness or adequate access to mental health care

(e.g., pervasive failures by a caregiver to interact with a child that include consistently not responding to infant cries or to an older child's attempts to interact with the caregiver) (Barnett et al., 1993).

Medical neglect includes a failure by a caregiver to provide a child adequate access to medical, vision, and/or dental care, when access to care is available or when a caregiver fails to seek timely medical attention for a child when needed (Leeb et al., 2008). Medical neglect is also indicated when a caregiver fails to follow through with medical recommendations and/or treatment regimens (e.g., not consistently administering prescribed medications to a child), in which the caregiver's failure to follow through may result in harm to the child.

Educational neglect refers to a caregiver's failure to ensure that a child regularly attends school, which results in excessive absences (e.g., 25 or more days in 1 academic year) with no acceptable excuses (e.g., physician's note; Leeb et al., 2008). Educational neglect is also defined as the failure of a caregiver to enroll and maintain a child in school up until the age of 16.

With respect to a child's emotional and developmental level, failure to supervise a child's safety within and outside of the home is categorized as specific types of neglect including inadequate supervision and exposure to violent environments.

Inadequate supervision refers to the failure of a caregiver to ensure that the child engages in safe activities and uses appropriate safety devices so that the child is not exposed to unnecessary hazards and/or that the child is being supervised by an adequate substitute caregiver when the primary caregiver(s) is not available (Leeb et al., 2008). Inadequate supervision can also include circumstances where a caregiver knowingly fails to protect a child from maltreatment perpetrated by another caregiver. Under such conditions, the primary caregiver's behavior would be considered neglectful only if the maltreatment was recognized and allowed to occur. Regardless of the primary caregiver's knowledge of the maltreatment, the substitute caregiver's behaviors would be considered maltreatment.

Exposure to violent environments includes knowingly failing to take appropriate measures to protect a child from being exposed to pervasive violence (e.g., domestic violence) or dangerous conditions (e.g., selling drugs out of the home) within the home, neighborhood, or community (Leeb et al., 2008). Of course, such situations are an ethical challenge for the child protection field because in many circumstances, if one parent is being battered, he or she may be ill equipped and even unable to prevent his or her children from witnessing violence in the home.

Description

Prevalence

The most recent estimates of the prevalence of child abuse and/or neglect indicate that approximately 772,000 children (10.3 per 1,000 children in the population) are substantiated victims annually (USDHHS, 2010). Of the various forms of child abuse and neglect, approximately 552,000 children experience either neglect and/or medical neglect annually. Neglect is the most prevalent form of child maltreatment, with 71% of all substantiated cases of maltreatment being classified as neglect (USDHHS, 2010). However, neglect is often the most difficult form of maltreatment to recognize because physical evidence is rare unless a family's home environment is physically inspected or the neglect has resulted in an injury, specific medical problem (e.g., failure to thrive), or an exacerbated chronic medical condition (e.g., sickle cell disease).

Etiology and Sequelae

It is clear that no singular risk factor can adequately explain why children are neglected. Rather, a combination of individual, relational, community, and societal factors contributes to the risk of a child being neglected (Centers for Disease Control and Prevention, 2009; Cicchetti & Lynch, 1993; Erickson & Egeland, 2011). Although children are not responsible for caregivers' neglectful behaviors, certain characteristics have been found to increase their risk of being neglected (Centers for Disease Control

and Prevention, 2009). Child factors that increase vulnerability for neglect include being younger than 4 years old and having special needs (e.g., developmental disabilities, chronic medical conditions). A number of caregiver-specific risk factors contribute to an increased risk for the perpetration of neglect, including caregiver poor prenatal and postnatal medical care, a caregiver's lack of understanding of child development and parenting skills, lack of parental nurturance, substance abuse, caregiver mental health issues, poor parent-child relationships, and parenting stress (Stith et al., 2009). Other caregiver characteristics such as the caregiver's own history of maltreatment as a child, young age, low education, single caregiver household, and a large number of dependent children all have been linked to increased risk of child neglect. Other risk factors include low family income, poor social connections and support, family conflict and violence (e.g., intimate partner violence), community violence, and concentrated neighborhood disadvantage (e.g., high poverty and residential instability, high unemployment rates).

Child neglect has been found to have serious negative implications on children's cognitive, physical, and socioemotional development. However, the consequences of individual cases of child neglect vary and are impacted by a combination of factors, including the child's age and developmental status when neglected; the types of abuse and/or neglect experienced; the frequency, duration, and severity of the neglect; and the relationship of the perpetrator to the victim child (English et al., 2005; Chalk, Gibbons, & Scarupa, 2002). If children's needs for physical touch, emotional attachment to a caregiver, and caregiver-child interactions are neglected during infancy or early childhood, long-term consequences have been found in children's cognitive and socioemotional development. Research on neglected infants has demonstrated reduced brain wave activity and enlarged brain ventricles due to decreased brain growth (Perry, 1997, 2002). Neglected infants and young children are at increased risk for developmental delays, expressive and receptive language problems, decreased positive affect, emotion regulation difficulties, impulse control problems, physical aggression, noncompliance, anxious attachment to their caregivers, restricted positive views of the self, and social withdrawal (Dubowitz, Papas, Black, & Starr, 2002; Hildyard & Wolfe, 2002). Many of the problems observed in children who are neglected in early childhood remain in school-aged children including continued cognitive problems (e.g., poor performance on academic achievement tests and increased referrals for special education services), negative mental representations of the self and others, avoidance of peer interactions, limited social skills, and an increased prevalence of internalizing problems (e.g., depression, anxiety, peer rejection).

The effects of child neglect have also been implicated in adult functioning. Adults who have experienced neglect as a child are at increased risk for psychiatric disorders, substance abuse, violent behaviors, and intimate partner violence (Erickson & Egeland, 2011; Horwitz, Widom, McLaughlin, & White, 2001; Mersky & Reynolds, 2007; White & Widom, 2003; Widom, Marmorstein, & White, 2006).

Cross-References

- ▶ Child Abuse
- ▶ Family Violence

References and Readings

Barnett, D., Manly, J. T., & Cicchetti, D. (1993). Defining child maltreatment: The interface between policy and research. In D. Cicchetti & S. Toth (Eds.), *Child abuse, child development, and social policy* (pp. 7–73). Norwood, NJ: Ablex.

Centers for Disease Control and Prevention. (2009). *Child maltreatment: Risk and protective factors*. Retrieved on July 20, 2011 from http://www.cdc.gov/ViolencePrevention/childmaltreatment/riskprotectivefactors.html

Chalk, R., Gibbons, A., & Scarupa, H. J. (2002). *The multiple dimensions of child abuse and neglect: New insights into an old problem*. Washington, DC: Child Trends. Retrieved on July 20, 2011 from www.childtrends.org/Files/ChildAbuseRB.pdf

Cicchetti, D., & Lynch, M. (1993). Toward an ecological/transactional model of community violence and child maltreatment: Consequences for children's development. *Psychiatry, 56*, 96–118.

Dubowitz, H., Papas, M. A., Black, M. M., & Starr, R. H., Jr. (2002). Child neglect: Outcomes in high-risk urban preschoolers. *Pediatrics, 109*, 1100–1107.
English, D. J., Upadhyaya, M. P., Litrownik, A. J., Marshall, J. M., Runyan, D. K., Graham, J. C., et al. (2005). Maltreatment's wake: The relationship of maltreatment dimensions to child outcomes. *Child Abuse & Neglect, 29*, 597–619.
Erickson, M. F., & Egeland, B. (2011). Child neglect. In E. John & B. Myers (Eds.), *The APSAC handbook on child maltreatment* (3rd ed., pp. 103–124). Thousand Oaks, CA: Sage.
Hildyard, K. L., & Wolfe, D. A. (2002). Child neglect: Developmental issues and outcomes. *Child Abuse & Neglect, 26*, 679–695.
Horwitz, A. V., Widom, C. S., McLaughlin, J., & White, H. R. (2001). The impact of childhood abuse and neglect on adult mental health: A prospective study. *Journal of Health and Social Behavior, 42*, 184–201.
Leeb, R. T., Paulozzi, L., Melanson, C., Simon, T., & Arias, I. (2008). *Child maltreatment surveillance: Uniform definitions for public health and recommended data elements, Version 1.0*. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control. Retrieved on July 20, 2011 from http://www.cdc.gov/violenceprevention/pdf/CM_Surveillance-a.pdf
Merksy, J. P., & Reynolds, A. J. (2007). Child maltreatment and violent delinquency: Disentangling main effects and subgroup effects. *Child Maltreatment, 12*, 246–258.
Perry, B. (1997). Incubated in terror: Neurodevelopmental factors in the "cycle of violence." In J. D. Osofsky (Ed.), *Children in a violent society* (pp. 124–149). New York: Guilford Press.
Perry, B. (2002). Childhood experience and the expression of genetic potential: What childhood neglect tells us about nature and nurture. *Brain and Mind, 3*, 79–100.
Stith, S. M., Liu, T., Davies, L. C., Boykin, E. L., Alder, M. C., Harris, J. M., et al. (2009). Risk factors in child maltreatment: A meta-analytic review of the literature. *Aggression and Violent Behavior, 14*, 13–29.
U.S. Department of Health and Human Services, Administration for Children and Families, Administration on Children, Youth and Families, Children's Bureau. (2010). *Child maltreatment 2008*. Retrieved on July 20, 2011 from http://www.acf.hhs.gov/programs/cb/stats_research/index.htm#can
White, H., & Widom, C. (2003). Intimate partner violence among abused and neglected children in young adulthood: The mediating effects of early aggression, antisocial personality, hostility, and alcohol problems. *Aggressive Behavior, 29*, 332–345.
Widom, C., Marmorstein, N., & White, H. (2006). Childhood victimization and illicit drug use in middle adulthood. *Psychology of Addictive Behaviors, 20*, 394–403.

Child Psychology

▶ Child Development

Childhood Obesity

▶ Diabesity in Children

Childhood Origins of Cardiovascular Disease

▶ Bogalusa Heart Study

CHO

▶ Carbohydrates

Cholesterol

Barbara Smith
School of Nursing, University of Maryland, Baltimore, MD, USA

Synonyms

Sterol

Definition

Cholesterol is a steroid. It is essential to the proper functioning of cell membranes and the synthesis of many hormones critical to normal physiologic processes and health. When cholesterol is manufactured primarily by the liver, and to a lesser degree by the intestines and other cells in the body it is known as *endogenous*

cholesterol. On the other hand, cholesterol which one consumes and is absorbed into the blood stream via the gastrointestinal tract is known as *exogenous cholesterol*.

Description

Despite the essential role cholesterol plays in cell wall permeability and the synthesis of steroid hormones excessive amounts of circulating cholesterol and the low-density lipoprotein (LDL) subfraction of total cholesterol have been associated with an increase in cardiovascular morbidity and mortality. This increase in morbidity and mortality is likely related to the development of atherosclerosis, a disease of the large and intermediate arteries where plaques form on the lining of the artery. At some point the plaques may obstruct or at least impede the flow of blood through the vessel. High-density lipoprotein (HDL) subfraction of total cholesterol protects against the development of atherosclerosis by a less well-understood mechanism.

Clinical trials support the hypothesis that aggressive lowering of the LDL subfraction of cholesterol reduces CHD risk. Lifestyle modifications that include losing weight, reducing saturated fats and the intake of exogenous cholesterol, and increased physical activity can reduce the LDL subfraction in healthy as well as chronically ill populations; however, adherence to long-term dietary changes and increased physical activity can be difficult. If efforts to reduce LDL cholesterol using only lifestyle modification do not reduce LDL sufficiently, drug therapy should be considered. Efforts to reduce CHD risk by raising the HDL subfraction are not as promising.

The *Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel of Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III or ATP III)* provides an update of the panel's earlier clinical guidelines (ATP I and ATP II) for cholesterol testing and measurement. ATP I presented an approach to primary prevention of coronary heart disease (CHD) in persons with high LDL ($\geq$160 mg/dL) or borderline high LDL (130–159 mg/dL) and multiple risk factors. ATP II set a new optimal LDL level of $\leq$100 mg/dL for people with CHD. ATP III focuses on intensive LDL reduction in those with multiple risk factors.

Cross-References

► Lipoprotein

References and Readings

Expert Panel of Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. (2001). Executive summary of the third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). *Journal of the American Medical Association, 285*, 2486–2497.

Hall, J. E. (2011). *Guyton and hall textbook of medical physiology* (12th ed.). Philadelphia: Saunders (Elsevier).

Chromosomes

Rany M. Salem[1] and Laura Rodriguez-Murillo[2]
[1]Broad Institute, Cambridge, MA, USA
[2]Department of Psychiatry, Columbia University Medical Center, New York, NY, USA

Definition

Chromosomes are self-replicating structures found within cells, containing and organizing cellular DNA. The DNA contains the nucleotide base sequence encoding the hereditary genetic information. In most *prokaryotes*, the entire genome is carried on a single circular strand of DNA comprising one chromosome. In e*ukaryotic* cells (cells with a nucleus), the genome is organized across multiple chromosomes.

Each eukaryotic organism has its own specific number of chromosomes. Humans are diploid and have 23 pairs of chromosomes: one set of two sex chromosomes and 22 pairs of autosomal chromosomes for a total of 46 chromosomes. Not surprisingly, given their name, sex chromosomes determine sex. Females have two X chromosomes, and males an X and a Y chromosome. Humans are diploid, which means that they have two copies of each chromosome. Other species have different numbers. In humans, autosomal chromosomes are identified by the numbers 1 through 22.

Chromosomes can be seen under a light microscope and individual chromosomes can be differentiated using special stains to band the chromosomes based on A/T vs. G/C content. The staining pattern results in a chromosome-specific karyotype, which was used in early genetic studies to identify major chromosomal abnormalities (loss/extra chromosomes, translocations, deletions, and breaks) associated with disease. For example, Down's syndrome, a genetic condition that causes physical and cognitive impairment, can be diagnosed via karyotyping to identify trisomy 21, the presence of three copies of chromosome 21 (Korenberg et al., 1994). However, most genetic variance occurs at a much smaller scale, at individual nucleotides such as in single nucleotide polymorphism (SNP) or groups of nucleotides, in microsatellites, and insertions-deletions.

Cross-References

- ▶ DNA
- ▶ Gene

References and Readings

Korenberg, J. R., Chen, X. N., Schipper, R., Sun, Z., Gonsky, R., Gerwehr, S., et al. (1994). Down syndrome phenotypes: the consequences of chromosomal imbalance. *Proceedings of the National Academy of Sciences of the United States of America, 91*(11), 4997–5001.

Lewis, R. (2005). *Human genetics. Concepts and applications* (7th ed.). Boston: McGraw-Hill Science/Engineering/Math.

Strachan, T., & Read, A. P. (2003). *Human molecular genetics* (3rd ed.). London/New York: Garland Science/Taylor & Francis Group.

Chronic Bronchitis

- ▶ Chronic Obstructive Pulmonary Disease
- ▶ Emphysema

Chronic Care

- ▶ Disease Management

Chronic Depression

Kim Lavoie
Department of Psychology, University of Québec at Montreal (UQAM); Montreal Behavioural Medicine Centre, Montréal, QC, Canada
Division of Chest Medicine, Hôpital du Sacré-Coeur de Montréal; Research Centre, Montreal Heart Institute, Montréal, QC, Canada

Definition

Depression is a negative mood state that is generally characterized by feelings of sadness, discouragement, and hopelessness (American Psychiatric Association, 2000). Brief or transient feelings of depression (i.e., feelings lasting several minutes to several hours) are relatively common and are likely to be experienced by just about everyone at some point in their lives. However, more chronic forms of depression are less common and may be associated with significant interpersonal difficulties and functional impairments.

There now exist widely accepted, standardized diagnostic criteria that distinguish "normal" from "abnormal" forms of depression, the latter of which have been classified as "mood disorders" in the Diagnostic and Statistical Manual of Mental Disorders-4th Edition Revised

(DSM-IV-R) (American Psychiatric Association, 2000). Although chronic depression has been classified as a "mood disorder," it is important to recognize that mood disorders, which include such disorders as major and minor depressive disorder, dysthymia, cyclothymia, and bipolar disorders, actually represent syndromes, which are clusters of symptoms, only one of which is an abnormality of mood. However, chronic forms of depression also feature *vegetative* symptoms, including sleep, appetite, weight, and libido disturbances; *cognitive* symptoms, including decreased ability to concentrate, memory disturbances, decreased frustration tolerance, low self-esteem, and cognitive distortions; *impulse control* symptoms such as suicidal behavior; *behavioral* symptoms, including decreased motivation and interest in engaging in pleasurable activities, decreased ability to feel pleasure, and decreased energy; and *somatic* symptoms, including increased psychomotor agitation, nonspecific aches and pains, and headaches.

Chronic depression is a major cause of morbidity worldwide and represents the 4th most important contributor to the global burden of disease, accounting for 4.4% of all cases of premature mortality (Kastrup & Ramos, 2007). Lifetime prevalence rates of chronic depression vary greatly according to geographical location, with the lowest rates found in Japan (3%) and the highest rates found in the United States (17%) (Kessler, Chiu, Demler, & Walters, 2005; Kessler, Demler, Frank et al., 2005; WHO, 2001; Andrade & Caraveo, 2003; Kessler, Beglund & Demler, 2003; Kessler, Berglund, Demler, Jin, Merikangas, & Walters, 2005; Murphy, Laird, Monson, Sobol, & Leighton, 2000). On average, most countries report an average lifetime prevalence of about 10% (WHO, 2001; Andrade & Caraveo, 2003). Population studies have consistently shown major depressive disorder to be about twice as common among women relative to men, though the reasons for this remain unclear. The peak age of onset of major depressive disorder is between 20 and 40 years and is 1.5 to three times more prevalent among individuals with first degree relatives with a history of depression (American Psychiatric Association, 2000; Kessler et al., 2005).

The most common, widely accepted, and empirically validated treatments for chronic depression include pharmacotherapy (e.g., antidepressant medications including selective serotonin reuptake inhibitors [SSRIs] or selective serotonin and norepinephrine reuptake inhibitors [SSNRIs]), psychotherapy (e.g., cognitive-behavioral therapy [CBT] and interpersonal therapy [IPT]), or some combination of the two (American Psychiatric Association, 2000; Kessler, Demler et al., 2005). However, most major depressive episodes resolve spontaneously over time, irrespective of whether or not they are treated. The ▶ median duration of a major depressive episode has been estimated to be about 23 weeks, with the highest rates of recovery occurring within the first 3 months (Posternak, Solomon & Leo, 2006; Fava, Park, & Sonino, 2006). Research has shown that 80% of those suffering from their first major depressive episode will suffer from at least one more over the course of their life, averaging four episodes over their lifetime. However, the morbidity associated with *untreated* chronic depression has been compared to that of coronary artery disease, with mortality due to suicide affecting 30,000–35,000 individuals each year (Posternak et al., 2006). There are also enormous personal and societal costs associated with chronic depression, including higher rates of chronic illness (e.g., cardiovascular disease), decreased productivity, absenteeism and job loss, substance abuse, family dysfunction, and reduced overall quality of life (American Psychiatric Association, 2000; Posternak et al., 2006).

Cross-References

▶ Dysthymia

References and Readings

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders*: (DSM-IV-R), (4th Rev. ed.). Arlington, VA: American Psychiatric Press.

Andrade, L., & Caraveo, A. (2003). Epidemiology of major depressive episodes: Results from the International Consortium of Psychiatric Epidemiology

(ICPE) surveys. *International Journal of Methods in Psychiatric Research, 12*(1), 3–21.
Fava, G. A., Park, S. K., & Sonino, N. (2006). Treatment of recurrent depression. *Expert Review of Neurotherapeutics, 6*(11), 1735–1740.
Kastrup, M. C., & Ramos, A. B. (2007). Global mental health. *Danish Medical Bulletin, 54*, 42–43.
Kessler, R. C., Berglund, P., & Demler, O. (2003). The epidemiology of major depressive disorder: Results from the National Comorbidity Survey Replication (NCS-R). *JAMA: The Journal of the American Medical Association, 289*(203), 3095–3105.
Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry, 62*(6), 617–627.
Kessler, R. C., Chiu, W. T., Demler, O., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry, 62*(6), 617–627.
Kessler, R. C., Demler, O., Frank, R. G., et al. (2005). Prevalence and treatment of mental disorders, 1990 to 2003. *The New England Journal of Medicine, 352*(24), 2515–2523.
Murphy, J. M., Laird, N. M., Monson, R. R., Sobol, A. M., & Leighton, A. H. (2000). A 40-year perspective on the prevalence of depression: The Stirling County Study. *Archives of General Psychiatry, 57*(3), 209–215.
Posternak, M. A., Solomon, D. A., & Leon, A. C. (2006). The naturalistic course of unipolar major depression in the absence of somatic therapy. *The Journal of Nervous and Mental Disease, 194*(5), 324–329.
World Health Organization. (2001). *The world health report 2001 – Mental health: New understanding, New hope*. Geneva: WHO.

Chronic Depressive Disorder

► Dysthymia

Chronic Disease Management

Lara Traeger
Behavioral Medicine Service, Massachusetts General Hospital/Harvard Medical School, Boston, MA, USA

Synonyms

Chronic disease prevention and management

Definition

Chronic disease management refers to a variety of models to improve patient care for individuals affected by chronic disease.

Description

Chronic diseases typically require ongoing medical care and may limit activities of daily living. Examples include diabetes, hypertension, heart diseases, mood disorders, and asthma. Chronic diseases impact all countries, with increasing prevalence due to several factors (e.g., increased life expectancy, treatment advances, and changes in lifestyle behaviors; Singh, 2008). In the USA, over 40% of the population is living with at least one chronic disease (Centers for Disease Control and Prevention [CDCP], 2010). Historically, primary care practices were designed for the provision of acute care. In contrast, patients with chronic diseases typically require long-term treatment planning, symptom management, and regular follow-up with providers.

Chronic Disease Management Models

There is no single optimal approach to chronic disease management. Common components of chronic disease management models include care coordination across medical disciplines; regular monitoring and medication management; and tools to increase patients' self-efficacy for managing the daily challenges of their disease(s). Several strategies have been suggested to be both effective and applicable in patient care settings, including those with limited resources. Examples include tools for self-care (e.g., patient education), information collection (e.g., screening tools and disease registries), and service provision in community settings (Singh, 2008). A primary goal of most models is to help individuals become informed and active participants in their disease management, to reduce the duration and/or severity of disease-related disability. Mental health care is an important part of this process, as mental health problems such as depression can present significant barriers to patient self-care.

Current Approaches to Chronic Disease Management

Chronic disease management is an increasingly popular term used in health care policy and industry communications as a reference point for both cost containment and quality improvement. Current chronic disease management programs include both targeted models which focus on case management for patients who account for the most medical care utilization, and broader approaches which are based on assumptions that all chronically ill patients may benefit from regular assessment and tools for promoting self-care. Some programs are carved out to commercial vendors whereas others are integrated within managed care institutions. There are wide variations in program quality, content, type of communication with patients, and extent to which physician practices are involved.

Challenges of Patient Care

Meeting the needs of chronically ill patients is one of the greatest challenges facing current healthcare systems. The 2001 Institute of Medicine report, "Crossing the Quality Chasm: A New Health System for the 21st Century," emphasized that enduring improvements in disease management require multi-level changes to the environment in which healthcare organizations and providers function (Institute of Medicine [IOM], 2001). To date, healthcare delivery is complex and largely fragmented, with implications for care quality, efficiency, and safety. Several factors have been suggested to facilitate chronic disease management practices within existing primary care systems. These include practice reorganization to support regular follow-up appointments; incorporation of empirically supported strategies for enhancing patient self-care; and provider education, provider incentives, and information technology to support changes.

Cross-References

► Disease Management

References and Readings

Centers for Disease Control and Prevention. (2010). *Chronic diseases and health promotion*. Accessed October 1, 2011, from http://www.cdc.gov/chronicdisease/index.htm

Institute of Medicine. (2001). *Crossing the quality chasm: A new health system for the 21st century*. Washington, DC: National Academy Press.

Singh, D. (2008). *How can chronic disease management programmes operate across care settings and providers?* Copenhagen, Denmark: World Health Organization.

Chronic Disease or Illness

Tyler Clark
School of Psychology, The University of Sydney, Sydney, NSW, Australia

Definition

Chronic disease or illness is any disease or illness which is both long lasting and permanent. Chronic diseases normally cannot be prevented through vaccination nor are they curable through either medicine or time. For a disease to be classified as chronic, it must persist for a minimum of 6 weeks.

Description

Chronic diseases or illnesses are the leading cause of mortality in the world and are estimated by the WHO to represent 60% of all deaths (World Health Organization [WHO], 2010). Chronic diseases are mostly characterized by complex causality, multiple risk factors, long latency periods, a prolonged course of illness, and functional impairment or disability (Pencheon, Guest, Melzer & Gray, 2006). While the term chronic disease technically incorporates all long-lasting, permanent diseases, classification confusion may arise for diseases such as herpes zoster or seasonal asthma, which occur intermittently throughout the lifespan and fulfill

the technical requirements of the definition, but are typically categorized with those diseases which are not permanent, but fail to resolve and respond to treatment, such as chronic bronchitis (Last, 2007). Ten major chronic diseases include:

- Coronary heart disease
- Stroke
- Hypertension
- Hypothyroidism
- Diabetes
- Mental health problems
- Chronic obstructive pulmonary disease
- Asthma
- Epilepsy
- Cancer

The prevalence of chronic diseases increases across the lifespan and is often comorbid with other chronic diseases, with the average person aged >65 years having more than one chronic disease. Chronic disease is prevalent in both wealthy and poor countries, but is correlated with low socioeconomic status. The chronically ill constitutes an extremely large percent of home care visits, as much as 90% in the United States, as well as the majority of prescription drug use, days spent in hospital, doctor visits, and hospital emergency room admittance.

As chronic diseases persevere throughout the lifespan, they are accompanied by a high burden of disease: a measure of potential years lost, quality of life lost, and disability attributed to a disease (Broemeling, Watson, & Black, 2005). This burden of disease may include financial costs of chronic disease as well, such as the primary and tertiary health care costs of disease management and loss of workforce participation.

Risk Factors

Risk factors for chronic diseases such as coronary heart disease, stroke, and certain cancers include high cholesterol, high blood pressure, and low fruit and vegetable intake (MedicineNet.com, 2004). Chronic disease development is also associated with physical inactivity, obesity, alcohol, and tobacco use. Risk factors often co-occur and can operate synergistically, as well as with some psychosocial factors (e.g., hostility and family history; Gidron, Berger, Lugasi, & Ilia, 2002).

Chronic Disease Management

Chronic diseases exist across the lifespan and require long-term treatment and support. Treatment therefore focuses on disease management, which serves to decrease the duration or severity of impairment and disability associated with the disease. This management manifests in a variety of forms including but not limited to occupational or physical therapy and rehabilitation, psychological counseling and stress management, and self-management strategies, depending on the type and severity of the chronic disease. Many governments provide assistance or financial incentive programs to individuals with qualifying disabilities.

Chronic disease may have psychological and emotional ramifications such as denial, anxiety, and depression following diagnosis not only for the affected individual, but for family and friends as well, and these consequences may also affect prognosis. As a result, much effort in behavior medicine has focused on developing adaptive strategies for coping with chronic diseases.

Cross-References

- ▶ Coping
- ▶ Disease Management
- ▶ Lifestyle, Healthy
- ▶ Multiple Risk Factors
- ▶ Quality of Life
- ▶ Self-Management

References and Readings

Broemeling, A., Watson, D., & Black, C. (2005). *Chronic conditions and co-morbidity among residents of British Comlumbia*. Vancouver: Centre for Health Services of British Columbia. Available at www.chspr.ubc.ca. Accessed January 8, 2011.

Gidron, Y., Berger, R., Lugasi, B., & Ilia, R. (2002). Interactive effects of family history with psychological factors in relation to CAD. *Coronary Artery Disease, 13*, 205–208.

Last, J. M. (2007). *A dictionary of public health*. Oxford/New York: Oxford University Press.

MedicineNet.com (2004, June). Definition of Chronic Disease. Available at: http://www.medterms.com/script/main/art.asp?articlekey=33490. Accessed December 18, 2010.

Pencheon, D., Guest, C., Melzer, D., & Gray, J. A. M. (2006). *Oxford handbook of public health practice* (2nd ed.). Oxford/New York: Oxford University Press.
Taylor, S. E. (2009). *Health psychology* (7th ed.). New York: McGraw Hill. International Edition.
World Health Organization. (2010). Available at http://www.who.int.com. Accessed January 2011.

Chronic Disease Prevention and Management

▶ Chronic Disease Management

Chronic Fatigue

▶ Fatigue

Chronic Fatigue Syndrome

Urs M. Nater
Department of Psychology, University of Marburg, Marburg, Germany

Definition

Chronic fatigue syndrome (CFS) is defined by unexplained disabling fatigue of at least 6 months duration, accompanied by at least four out of eight of the following symptoms: impaired memory or concentration, sore throat, tender glands, aching or stiff muscles, multijoint pain, new headaches, unrefreshing sleep, and postexertional fatigue.

Description

Chronic fatigue syndrome (CFS) is a complex illness defined by unexplained disabling fatigue as its core feature and a combination of other accompanying symptoms, such as diffuse pain, subjective cognitive impairment, and sleep problems. The first formal case definition of the illness was published in the USA in 1988 (Holmes et al., 1988). In 1994, an international collaborative group published the current CFS research case definition (Fukuda et al., 1994). The 1994 case definition requires at least 6 months of persistent fatigue; this fatigue cannot be substantially alleviated by rest, is not the result of ongoing exertion, and is associated with substantial reductions in occupational, social, and personal activities. In addition, at least four out of eight of the following symptoms must occur with fatigue in a 6-month period: impaired memory or concentration, sore throat, tender glands, aching or stiff muscles, multijoint pain, new headaches, unrefreshing sleep, and postexertional fatigue. Medical conditions that may explain the prolonged fatigue as well as a number of psychiatric diagnoses exclude a patient from the diagnosis of chronic fatigue syndrome (Reeves et al., 2003). Consequently, a thorough medical history and physical assessment is required before the diagnosis can be formally established.

Comorbidity

There is a considerable overlap between CFS and psychiatric disorders. Recent data indicate that almost 60% of CFS cases in the population suffer from at least one comorbid psychiatric condition (Nater et al., 2009). Consistent with the fact that fatigue is a common symptom in depressive disorders, a substantial overlap in diagnoses of CFS and depression has been reported. However, there are also distinct symptoms, such as suicidal ideation, which are not more frequently present in CFS patients than in the general population. There are many patients with CFS who do not meet the current criteria for any other psychiatric disorder, indicating that CFS is not merely a psychiatric epiphenomenon.

Chronic fatigue syndrome often co-occurs with other medically unexplained syndromes such as fibromyalgia or irritable bowel syndrome. These disorders have in common with CFS the fact that they are defined as disorders that, after appropriate medical assessment, cannot be explained in terms of a conventionally defined medical disease (Barsky & Borus, 1999).

Taken together, CFS co-occurs and shares core symptoms with a variety of conditions, suggesting that similar pathways may be involved in the etiology and development of these pathological states.

Prevalence

Chronic fatigue syndrome is relatively common in the community, in primary care, and in hospital settings. The overall prevalence of CFS in the general population is reported to be between 0.1% and 2.5%. Prevalence rates vary significantly across studies, probably as a result of differences in diagnostic criteria and design. It is estimated that 2.2 million Americans suffer with CFS and that the disorder is more common in rural than urban populations (Bierl et al., 2004). Rates for CFS in primary care are higher than rates seen in the general population. Large community-based epidemiological studies in the USA indicate that CFS is equally or more common in African Americans, Hispanics and Native Americans, and in individuals who make less than $40,000 per year. However, in all these groups women are two to four times more likely than men to have CFS.

Pathophysiology

The pathophysiology of CFS is complex and far from being fully understood. Despite mixed findings in the vast literature of potential pathophysiological processes in CFS, tentative conclusions can be drawn concerning physiological systems that may be abnormal in at least some patients. It is important to remember, however, that it remains unknown whether any given abnormality represents a cause or a consequence of CFS.

Early etiological theories of the disorder focused on the immune system and infection with Epstein Barr and other latent viruses. Although cases of CFS may follow such infections, most studies have shown that infections are not a primary cause for the disorder.

A variety of immune system abnormalities have also been reported, including decreases in natural killer cell activity and increases in proinflammatory cytokines (Lorusso et al., 2009). Cytokines, such as IL-6 and TNF-alpha, have been implicated in the pathogenesis of fatigue and somnolence. Specifically, IL-6 participates in the pathogenesis of excessive daytime somnolence and post-exertional fatigue. In addition, alterations in the gene expression involved in immunity have been detected. Thus, immune factors seem to play an important role in CFS, although the exact mechanisms have not been fully established yet.

Patients with CFS also may have decreased functioning of the hypothalamic-pituitary-adrenal (HPA) axis, one of the body's primary stress response systems, which is also contributing to the peripheral and central causes of chronic pain and fatigue. Several studies report decreased levels of circulating cortisol and decreased adrenocortical reserve. This alteration may be associated with several symptoms typical for CFS, including fatigue, arthralgia, myalgia, exacerbation of allergic responses, feverishness, and changes in mood, cognition, and sleep. Cortisol exerts inhibitory effects on the secretion of cytokines, including IL-6, and helps return these cytokines to baseline levels after stress. Thus, alterations in the immune system have an impact on the endocrine system, and vice versa. In addition to decreased functioning, the HPA axis has also been reported to lose its normal diurnal rhythm in CFS patients (Nater et al., 2008). Clinical improvement has been associated with the normalization of this diurnal rhythm. The overall picture may be summarized as a relative hypoactivity of the HPA axis in CFS patients (Cleare, 2003).

It has been reported that abnormal autonomic nervous system (ANS) functioning may be common in patients with CFS, based on the fact that CFS includes typical autonomic symptoms, such as disabling fatigue, dizziness, diminished concentration, tremulousness, and nausea, and that at least some CFS patients demonstrate orthostatic intolerance when subjected to tilt table testing. Conversely, patients with postural orthostatic intolerance syndrome often manifest symptoms similar to those seen in CFS. Whereas there is some evidence for involvement of altered ANS functioning, it needs to be noted that some CFS symptoms, such as sore throat, myalgias, and

cognitive alterations, cannot be attributed to dysautonomia.

Also, studies of the central nervous system (CNS) in CFS have examined both structural and functional alterations. Various studies have pointed to subtle morphological changes in CFS, although these changes might not be specific for CFS. Functional studies have found potential explanations for some of the motor and cognitive dysfunctions typically described in CFS.

Finally, psychological and stress-related factors have been associated with CFS. Some authors consider CFS as the consequence of dysfunctional cognitive styles and maladaptive coping strategies. Many patients report an increase in life stress in the year prior to disease development. Recent findings from a prospective study indicated that stress levels prior to manifestation of CFS predicted the risk for developing CFS (Kato, Sullivan, Evengard, & Pedersen, 2006). In addition, adverse experiences early in life increased the risk of developing CFS in adulthood manifold and resulted in the above-mentioned hypoactivity of the endocrine stress system (Heim et al., 2009). Thus, stressful experiences seem to play an important role in triggering CFS symptoms. However, it is likely that stress interacts with other vulnerability factors. Ongoing or acute stressors might elicit physiological changes in the predisposed body, ultimately leading to pathophysiological changes associated with CFS.

Treatment

Numerous treatments have been applied to CFS patients with various results. Those with the best experimental data to support efficacy include graded exercise training and cognitive behavioral therapy (CBT) (White et al., 2011). CBT strategies for CFS typically involve organizing activity and rest cycles, initiating graded increases in activity, establishing a consistent sleep regimen, and attempting to restructure beliefs around self, as well as disease attributions (Malouff, Thorsteinsson, Rooke, Bhullar, & Schutte, 2008). Also, low dose corticosteroids have been reported to improve symptoms in two studies. However, these positive findings could not be replicated. Trials of antidepressants have yielded an equally confusing mix of positive and negative results, but in general these agents appear to be significantly less effective for CFS than for depression or anxiety disorders.

Cross-References

▶ Fatigue

References and Readings

Afari, N., & Buchwald, D. (2003). Chronic fatigue syndrome: A review. *The American Journal of Psychiatry, 160*(2), 221–236.

Barsky, A. J., & Borus, J. F. (1999). Functional somatic syndromes. *Annals of Internal Medicine, 130*(11), 910–921.

Bierl, C., Nisenbaum, R., Hoaglin, D. C., Randall, B., Jones, A. B., Unger, E. R., et al. (2004). Regional distribution of fatiguing illnesses in the United States: A pilot study. *Popululation Health Metrics, 2*(1), 1.

Cleare, A. J. (2003). The neuroendocrinology of chronic fatigue syndrome. *Endocrine Reviews, 24*(2), 236–252.

Fukuda, K., Straus, S. E., Hickie, I., Sharpe, M. C., Dobbins, J. G., & Komaroff, A. (1994). The chronic fatigue syndrome: A comprehensive approach to its definition and study. International Chronic Fatigue Syndrome Study Group. *Annals of Internal Medicine, 121*(12), 953–959.

Heim, C., Nater, U. M., Maloney, E., Boneva, R., Jones, J. F., & Reeves, W. C. (2009). Childhood trauma and risk for chronic fatigue syndrome: Association with neuroendocrine dysfunction. *Archives of General Psychiatry, 66*(1), 72–80.

Holmes, G. P., Kaplan, J. E., Gantz, N. M., Komaroff, A. L., Schonberger, L. B., Straus, S. E., et al. (1988). Chronic fatigue syndrome: A working case definition. *Annals of Internal Medicine, 108*(3), 387–389.

Kato, K., Sullivan, P. F., Evengard, B., & Pedersen, N. L. (2006). Premorbid predictors of chronic fatigue. *Archives of General Psychiatry, 63*(11), 1267–1272.

Lombardi, V. C., Ruscetti, F. W., Das Gupta, J., Pfost, M. A., Hagen, K. S., Peterson, D. L., et al. (2009). Detection of an infectious retrovirus, XMRV, in blood cells of patients with chronic fatigue syndrome. *Science, 326*(5952), 585–589.

Lorusso, L., Mikhaylova, S. V., Capelli, E., Ferrari, D., Ngonga, G. K., & Ricevuti, G. (2009). Immunological aspects of chronic fatigue syndrome. *Autoimmunity Reviews, 8*(4), 287–291.

Malouff, J. M., Thorsteinsson, E. B., Rooke, S. E., Bhullar, N., & Schutte, N. S. (2008). Efficacy of

cognitive behavioral therapy for chronic fatigue syndrome: A meta-analysis. *Clinical Psychology Review, 28*, 736–745.

Nater, U. M., Lin, J. M., Maloney, E. M., Jones, J. F., Tian, H., Boneva, R. S., et al. (2009). Psychiatric comorbidity in persons with chronic fatigue syndrome identified from the Georgia population. *Psychosomatic Medicine, 71*(5), 557–565.

Nater, U. M., Youngblood, L. S., Jones, J. F., Unger, E. R., Miller, A. H., Reeves, W. C., et al. (2008). Alterations in diurnal salivary cortisol rhythm in a population-based sample of cases with chronic fatigue syndrome. *Psychosomatic Medicine, 70*, 298–305.

Prins, J. B., van der Meer, J. W., & Bleijenberg, G. (2006). Chronic fatigue syndrome. *The Lancet, 367*(9507), 346–355.

Reeves, W. C., Lloyd, A., Vernon, S. D., Klimas, N., Jason, L. A., Bleijenberg, G., et al. (2003). Identification of ambiguities in the 1994 chronic fatigue syndrome research case definition and recommendations for resolution. *BMC Health Services Research, 3*(1), 25.

White, P., Goldsmith, K., Johnson, A., Potts, L., Walwyn, R., Decesare, J., et al. (2011). Comparison of adaptive pacing therapy, cognitive behaviour therapy, graded exercise therapy, and specialist medical care for chronic fatigue syndrome (PACE): A randomised trial. *The Lancet, 377*, 823–836.

Chronic Inflammatory Polyarthritis

► Degenerative Diseases: Joint

Chronic Kidney Disease (CKD)

► End-Stage Renal Disease

Chronic Obstructive Pulmonary Disease

Akihisa Mitani
Department of Respiratory Medicine, Mitsui Memorial Hospital, Chiyoda-ku, Tokyo, Japan

Synonyms

Chronic bronchitis; Emphysema

Definition

Chronic obstructive pulmonary disease (COPD), one of the leading causes of morbidity and mortality worldwide, is a chronic disease of the lung that is characterized by decreased air flow and associated abnormal inflammation of the lungs. The disease results from interaction between individual risk factors (like alpha1-antitrypsin deficiencies) and environmental exposures to toxic agents (like cigarette smoking). The main mechanisms that may contribute to airflow limitation in COPD are fixed narrowing of small airways, emphysema, and luminal obstruction with mucus secretions (American Thoracic Society, 1995; Global Initiative for Chronic Obstructive Lung Disease (GOLD), 2006; The COPD Guidelines Group of the Standards of Care Committee of the BTS Thorax 1997; Petty & Nett, 2001).

The definition does not use the terms chronic bronchitis and emphysema, although most patients with COPD have them. Chronic bronchitis is diagnosed based on the clinical presentation, such as a chronic cough and sputum production. The diagnosis of emphysema, which is the term used to describe damage to the air sacs in the lung, is made from a pathological and/or morphological standpoint.

The respiratory symptoms of COPD are dyspnea, chronic cough, and sputum production. The dyspnea may initially be noticed only during exertion. Patients with a COPD exacerbation complain of increased cough and sputum, wheezing, and dyspnea, with or without fever.

Most patients with COPD have a history of cigarette smoking or other inhalant exposure. Therefore, when a person with a history of exposure to risk factors, especially smoke, has dyspnea, chronic cough, and sputum production, a diagnosis of COPD should be considered. Measurements of lung function are essential for the diagnosis of COPD. It is also used to determine the severity of the airflow obstruction and follow disease progression. Spirometry measures forced vital capacity (FVC) and forced expiratory volume in 1 s (FEV1.0). An FEV1.0/FVC ratio less than 70% generally indicates airway obstruction.

The overall goals of treatment of COPD are to prevent further deterioration in respiratory function, relieve symptoms, improve quality of life, and reduce mortality (Global Initiative for Chronic Obstructive Lung Disease (GOLD), 2006).

First of all, reduction of risk factors is needed. All COPD patients with smoking habit should be encouraged to quit smoking. Even a few minutes counseling could be effective. Pharmacotherapy, such as nicotine replacement and varenicline, is also recommended. Preventive care is also very important, and all patients should be recommended to get an immunization, including influenza and pneumococcal vaccines.

The mainstay drugs of COPD are bronchodilators, and inhaled therapy is preferred. Beta agonists, anticholinergics, and methylxanthines are given alone or in combination depending upon the severity of disease and each patient's individual response to therapy. Inhaled glucocorticoids can reduce the frequency of the acute exacerbation, although it cannot improve lung function. Systemic glucocorticoids are not recommended for a long-time treatment. Mucolytic drugs might be beneficial for selected patients.

Non-pharmacological treatment is equally important for managing COPD. It includes pulmonary rehabilitation and oxygen administration. Pulmonary rehabilitation has been shown to improve exercise capacity, decrease dyspnea, and improve quality of life and should be considered as an addition to medication therapy for the patients at all stages of disease. Long-term oxygen therapy improves survival and quality of life in the patients with hypoxemia.

Cross-References

- ▶ Lung Function
- ▶ Pulmonary Disorders, COPD: Psychosocial Aspects

References and Readings

American Thoracic Society. (1995). Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease. *American Journal of Respiratory and Critical Care Medicine*, *152*, S77.

Global Initiative for Chronic Obstructive Lung Disease (GOLD). (2011). Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: Revised 2011. Retrieved 10 April 2012 from http://www.goldcopd.org.

Petty, R. L., & Nett, L. M. (2001). *COPD: Prevention in the primary care setting*. The National Lung Health Education Program.

The COPD Guidelines Group of the standards of care committee of the BTS. (1997). BTS guidelines for the management of chronic obstructive pulmonary disease. *Thorax*, *52*(Suppl. 5), S1.

Chronic Pain

▶ Arthritis: Psychosocial Aspects

Chronic Pain Patients

Stuart Derbyshire
School of Psychology, The University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Persistent pain

Definition

Chronic pain is typically defined as pain that continues in excess of 3–6 months regardless of the cause of the pain. Less commonly, chronic pain is defined as pain that persists beyond the point of any possible healing or any other useful function such as the enforcement of rest.

Description

Major advances in the understanding of pain began with the observations of the physician Henry Beecher during World War 2. Beecher noted that seriously wounded soldiers brought from the front line requested less-pain medicine

and reported less pain than he was used to seeing in his civilian patients. Beecher inferred that pain is not simply a response to physical injury or disease but also includes a cognitive and emotional component. Twenty years later, Canadian psychologist Ronald Melzack and British physiologist Patrick Wall published their gate control theory. Gate theory proposed that noxious and non-noxious sensory information interact in the spinal cord with descending influence from the brain. The theory explains pain experience as dependent upon that interaction rather than just the strength of a noxious stimulus. The precise details of the theory are less important than the dramatic impact gate control had on the understanding of pain. Gate control theory ended simplistic ideas of pain based on an isolated dedicated pathway from the periphery to the brain. It provided the first plausible physiological explanations for the influence of psychological states on pain experience through a brain-spinal cord loop. Most importantly, gate control theory shifted attention away from the stimulus and toward the spinal cord, brain, and the subjective experience of pain. After the gate it became increasingly apparent that pain cannot be reliably judged based upon an objective measure of injury or receptor activation and so assessment of pain requires subjective report – the "what it is like" to be in pain.

The shift in focus away from the noxious stimulus that triggers pain and toward the psychological experience of pain was particularly important for the understanding of chronic pain. Chronic pain conditions are often characterized by the lack of a stimulus that can explain the pain. Patients with phantom limb pain, for example, feel pain in a limb that has been amputated. Patients with causalgia suffer severe burning pain at a site of injury long after the injury has healed. Even in diseases where there is an ongoing trauma, such as patients with cancer or arthritis, the pain is typically difficult to predict based on objective measures of disease activity and continues beyond any period when cessation of activity and rest might facilitate healing. Thus, the understanding of chronic pain is not helped by a focus on injury or disease but by a focus on the experience of pain. Chronic, persistent pain is a distinct medical entity, syndrome, or disease in its own right, but it is not a disease that can be defined by objective markers such as provided by X-rays or histological tests; chronic pain is a disease defined by the subjective experience of pain. In short, chronic pain is a problem because it feels bad.

This understanding of chronic pain is further reflected in the international association for the study of pain (IASP) definition of pain, which states that pain is "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage… pain is always subjective. Each individual learns the application of the word through experiences related to injury in early life." This definition recognizes a number of important facts about pain: (1) It is a multidimensional experience. (2) It is subjective. (3) It may or may not be associated with tissue damage.

The somewhat complex understanding of pain provided by the IASP is perhaps not especially important when considering acute pain. If someone hits their hand with a hammer, it is patently obvious that the pain was caused by the hammer and it is reasonable to assume that the pain will subside once the injury heals. Although it may be theoretically correct to point out that the pain is in the patient's mind, not their hand, and that the experience derives from psychology, and not the hammer, such points would be overly pedantic. When faced with an obvious injury it is reasonable to depersonalize the experience as a consequence of external forces, which rapidly lose their influence with healing. For patients with chronic pain, however, there is either no external force to blame or the external force never loses its influence. Either way, the experience is deeply personal and subjective.

The personal and subjective nature of chronic pain makes treatment difficult. Traditional treatment approaches involving periods of rest and analgesic medication use are typically unsuccessful in resolving chronic pain. Physicians and patients can easily become disillusioned when multiple treatments, used sequentially or in combination, fail to provide pain relief. In many

cases, physicians are left frustrated and patients dissatisfied with chronic, unremitting symptoms. Treatment approaches that focus on the patient's experience, what they feel and how they manage their feelings, are usually more successful. Cognitive behavioral therapy, for example, aims to modify the reciprocal relationships between sensation, cognition, emotion, and behavior so as to improve mood and decrease the disability associated with the pain. Cognitive behavioral therapy emphasizes the teaching of coping skills and the active role patients have in modifying how they think, feel, and believe. The aim is to reduce the negative impact of their pain even if the pain itself is not directly reduced.

Among adults, the prevalence of chronic pain where an identifying cause is difficult to find ranges between 2% and 40% depending on the study. Unsurprisingly, chronic pain substantially reduces quality of life and also generates considerable costs. In the Netherlands, for example, the cost of back pain alone equals 1.7% of the gross national product and in the UK, back pain results in the loss of over 150 million workdays annually. There is also evidence that the problem may be increasing. In the USA, the rate of disability claims associated with low back pain has increased over the rate of population growth by 1,400% since the early 1970s. Understanding chronic pain so as to address this increase and provide better treatments remains a considerable challenge.

Cross-References

► Stress

References and Readings

Loeser, J. D. (2006). Pain as a disease. In F. Cervero & T. J. Jensen (Eds.), *Handbook of clinical neurology* (pp. 11–20). Edinburgh: Elsevier.

McMahon, S., & Koltzenburg, M. (2005). *Wall and melzack's textbook of pain* (5th ed.). Edinburgh: Churchill Livinstone.

Melzack, R., & Wall, P. D. (1996). *The challenge of pain*. London: Penguin.

Chronic Pain, Types of (Cancer, Musculoskeletal, Pelvic), Management of

Michael J. L. Sullivan and Tsipora Mankovsky
Department of Psychology, McGill University, Montreal, QC, Canada

Definition

Intervention approaches to improve function and promote successful adaptation to chronic pain.

Description

This entry briefly reviews non-pharmacological approaches to the management of pain-related health conditions and pain-related disability. The review is selective as opposed to exhaustive, with emphasis on interventions that have been systematically evaluated. Where possible, references to clinical manuals are provided for readers who are interested in learning more about the specific intervention techniques described.

Psychological Treatment of Pain

By the mid-1960s, mounting clinical and scientific evidence was calling for a model of pain that would consider both the physiological and psychological mechanisms involved in pain perception. The call was most compellingly answered by Melzack and Wall's gate control theory of pain. From an applied perspective, the work of Melzack and Wall evolved into behavioral conceptualizations of pain (Fordyce, Fowler, Lehmann, & De Lateur, 1968), contributing ultimately to the development of biopsychosocial models of pain (Gatchel, Peng, Peters, Fuchs, & Turk, 2007). Biopsychosocial models propose that a complete understanding of pain experience and pain-related outcomes requires consideration of physical, psychological, and social factors (Gatchel et al., 2007).

Behavioral/Operant Programs

The first programs that specifically targeted the psychological aspects of pain-related disability were based on the view that pain-related disability was a form of "behavior" that was maintained by reinforcement contingencies. In the 1960s and 1970s, Wilbert Fordyce and his colleagues applied the concepts of learning theory to the problem of chronic pain (Fordyce et al., 1968; Fordyce, 1976). The focus of Fordyce's approach to treatment was not on reducing the experience of pain but on reducing the overt display of pain. The targets selected for treatment were pain behaviors such as distress vocalizations, facial grimacing, limping, guarding, medication intake, activity withdrawal, and activity avoidance.

The first behavioral approaches to the management of pain and disability were conducted within inpatient settings that permitted systematic observation of pain behaviors, as well control over environmental contingencies influencing pain behavior (Fordyce, 1976). Staff were trained to monitor pain behavior and to selectively reinforce "well behaviors" and selectively ignore "pain behaviors." Results of several studies revealed that the manipulation of reinforcement contingencies could exert powerful influence on the frequency of display of pain behaviors (Fordyce, Roberts, & Sternbach, 1985). The manipulation of reinforcement contingencies was also applied to other domains of pain-related behavior and shown to be effective in reducing medication intake, reducing downtime and maximizing participation in goal-directed activity.

A number of clinical trials on the efficacy of behavioral treatments for the reduction of pain and disability yielded positive findings (Sanders, 1996). However, given the significant resources required to implement contingency management interventions, issues concerning the cost-efficacy of behavioral therapy for pain and disability were raised. Concern was also raised over the maintenance of treatment gains since reinforcement contingencies outside the clinic setting could not be readily controlled. In order to increase access and reduce costs, behavioral treatments were modified to permit their administration on an outpatient basis. This change in delivery format compromised to some degree the control over environmental contingencies and required greater reliance on self-monitoring and self-report measures (Sanders, 1996).

Back Schools

Although back schools were first developed in the late 1960s, the first published reports of the benefits of "back schools" only appeared in the literature in the early 1980s (Zachrisson-Forsell, 1981). The structure and content of back schools reflected the prevailing view of the time that "information" or "knowledge" could be powerful tools to effect change in behavior (e.g., pain-related disability) (Heymans, van Tulder, Esmail, Bombardier, & Koes, 2004).

Back schools vary widely in terms of content, duration, and the intervention disciplines used to administer the program. The duration of back school interventions has ranged from a single information session to a 2-month inpatient program. Back school interventions have tended to use group formats with a didactic format where participants might be exposed to information about biomechanics, posture, ergonomics, exercises, nutrition, weight loss, attitudes, beliefs, and coping. As a function of the type of information being provided, the interventionist might be a physician, physiotherapist, occupational therapist, nurse, or psychologist (Linton & Kamwendo, 1987).

A recent review of randomized clinical trials of back school programs concluded that (a) back schools yielded benefit relative to treatment-as-usual interventions, (b) the treatment effect size was small, and (c) that back school programs implemented within occupational settings appeared to yield the most positive outcomes (Heymans, van Tulder, Esmail, Bombardier, & Koes, 2005).

Cognitive-Behavioral Programs

Cognitive-behavioral programs for the management of pain and pain-related disability began to appear in the 1980s (Turk, Meichenbaum, & Genest, 1983). CBT programs incorporated concepts drawn from earlier behavioral approaches as well as information-based approaches used in

back schools. The objective of many CBT programs is to equip individuals with the psychological "tools" necessary to adequately meet challenges of persistent pain (Turk et al., 1983).

Cognitive-behavioral interventions are currently considered the psychological treatment of choice for individuals coping with chronic pain and disability, (Gatchel et al., 2007). A number of clinical trials have demonstrated that these types of interventions can lead to clinically significant decreases in pain and emotional distress (Williams et al., 1996).

It is important to note that the term cognitive behavioral does not refer to a specific intervention but, rather, to a class of intervention strategies. The strategies included under the heading of cognitive-behavioral interventions vary widely and may include self-instruction (e.g., motivational self-talk), relaxation or biofeedback, developing coping strategies (e.g., distraction, imagery), increasing assertiveness, minimizing negative or self-defeating thoughts, changing maladaptive beliefs about pain, and goal setting (Turk et al., 1983). A client referred for cognitive-behavioral intervention may be exposed to varying selections of these strategies. The goals of CBT programs might also differ across settings and may include pain reduction, distress reduction, increased activity involvement, or return to work (Gatchel et al., 2007).

Stress Management Programs

Stress management programs represent a special case of cognitive-behavioral intervention. Stress management programs proceed from the view that, unless properly managed, chronic stresses can lead to a depletion of the individual's physical and psychological resources and, in turn, increase the individual's susceptibility to physical or psychological dysfunction (Lazarus & Folkman, 1984). Stress management approaches are considered separately from cognitive-behavioral pain management programs since the focus of stress management programs is not necessarily on managing pain symptoms or disability. Furthermore, while CBT programs are typically used for individuals who are work-disabled due to their pain condition, stress management programs have been used as preventive interventions for individuals who are experiencing symptoms of persistent pain but are still working. The primary focus of stress management interventions might be on stresses within the workplace or the individual's personal stresses (Feuerstein et al., 2004).

Problem-solving therapy is a variant of stress management programs that has recently been applied to individuals who are work-disabled due to musculoskeletal pain conditions (D'Zurilla, 1990; Smeets et al., 2008). Problem-solving therapy proceeds from the view that life stresses can be minimized if the individual is able to use appropriate problem-solving strategies to deal with difficult situations that might be encountered at the work place or in daily life. Problem-solving intervention programs will typically span several weeks (8–10 weeks) and might involve didactic lectures, group discussion, and homework assignments. The limited research that has addressed the efficacy of this form of intervention indicates that the addition of problem-solving therapy to usual treatment might improve return to work outcomes in individuals with disabling musculoskeletal pain (Smeets et al.).

Acceptance and Commitment Therapy

Acceptance and commitment therapy, also referred to as contextually based cognitive-behavior therapy, is a type of cognitive therapy that has evolved from Stephen Hayes' work on acceptance and adaptation (Hayes, Strosahl, & Wilson, 1999; McCracken, 2005). Proponents of ACT emphasize that they do use the term acceptance to refer to resignation but rather as a term to refer to the process of ceasing to struggle ineffectively against that which cannot be changed (Hayes et al., 1999). In the case of chronic pain, acceptance is viewed as a first step toward successful adaptation (McCracken, 2005). Acceptance is said to occur when the individual with chronic pain is willing to experience his or her pain without attempting to control it. Through treatment, individuals with chronic pain are taught to acknowledge their pain, observe it as a sensation, and then accept it as part of their

reality without judgment. Through treatment, individuals are also encouraged to focus on their values and to commit to activities consistent with their values, in spite of ongoing pain.

Several investigations have shown that ACT is effective in reducing pain intensity and self-reported disability (Vowles & McCracken, 2008). To date, ACT has only been used with individuals with long-standing chronic pain where the prospect of significant pain alleviation is realistic low. When symptom-focused treatment of the pain condition is unlikely to yield positive outcomes, acceptance-based interventions might represent a useful option for improving the quality of life of individuals with chronic pain. It is not clear whether ACT would be effective or even appropriate for individuals with recent onset pain where a substantive proportion of individuals would be expected to show significant recovery from their pain condition.

Risk-Factor-Targeted Interventions

Recent research on risk factors for prolonged pain and disability has prompted the development of risk-factor-targeted intervention programs (Sullivan, Feuerstein, Gatchel, Linton, & Pransky, 2005; Vlaeyen & Linton, 2000). The Progressive Goal Attainment Program (PGAP) was designed as a risk-factor-targeted intervention for individuals suffering from debilitating pain conditions (Sullivan, Adams, Rhodenizer, & Stanish, 2006). The primary goals of the PGAP are to reduce catastrophic thinking and fear of movement in order to promote reintegration into life-role activities, increase quality of life, and facilitate return to work. The intervention is typically delivered by occupational therapists, physiotherapists, or psychologists.

Since the PGAP is a risk-factor-targeted intervention, clients are only considered as potential candidates for the intervention if they obtain scores in the risk range on measures of catastrophic thinking, fear of movement, or disability beliefs. In the initial weeks of the program, the focus is on the establishment of a strong therapeutic relationship and the development of a structured activity schedule. The client is provided with a client workbook that serves as the platform for activity scheduling and contains the forms for various exercises that will be used through the treatment. Activity goals are established in order to promote resumption of family, social, and occupational roles. Intervention techniques are invoked to target specific obstacles to rehabilitation progress (e.g., catastrophic thinking, fear of movement, and disability beliefs). In the final stages of the program, the intervention focuses on activities that will facilitate reintegration into the workplace.

PGAP has been shown to be effective in reducing catastrophic thinking, fear of movement, and disability beliefs in individuals with whiplash injuries and work-related musculoskeletal injuries (Sullivan, Adams, Rhodenizer, & Stanish, et al., 2006). Research has supported the view that reductions in catastrophizing are significant determinants of treatment-related improvements in depressive symptoms, physical function, and return to work (Sullivan, Ward et al., 2005).

Graded Activity and Exposure

The premise underlying graded activity or exposure interventions is that disability can be construed as a type of phobic orientation toward activity (Vlaeyen & Linton, 2000). According to the fear-avoidance model, individuals will differ in the degree to which they interpret their pain symptoms in a "catastrophic" or "alarmist" manner. The model predicts that catastrophic thinking following the onset of pain will contribute to heightened fears of movement. In turn, fear is expected to lead to avoidance of activity that might be associated with pain (Vlaeyen & Linton, 2000). Prolonged inactivity is expected to contribute to depression and disability (Sullivan, Adams, Thibault et al., 2006). According to the fear-avoidance model, reducing fear of movement is a critical component of successful rehabilitation of individuals with debilitating pain conditions (Vlaeyen & Linton, 2000). Clients are typically only considered for exposure interventions if they obtain high scores on measures of fear of movement.

Graded activity or exposure to feared activities are treatment approaches that involve systematic exposure or engagement in activities that individuals avoid due to fears that they

might experience an exacerbation of their symptoms. Feared activities are initially identified and ranked hierarchically, from least to most feared activities. Beginning with the least feared activities, clients are systematically exposed to movements that comprise the activities that clients are currently avoiding. Clients are repeatedly exposed to specific movements until their fear of activity subsides. As clients overcome their fears associated with the least feared activities in their feared activities hierarchy, the exposure techniques are used on activities associated with higher levels of fear (Leeuw et al., 2007).

While graded exposure has been shown to be an effective intervention for reducing the fear of specific movements, its effects do not seem to generalize to un-targeted activities (Goubert, Francken, Crombez, Vansteenwegen, & Lysens, 2002). As such, the clinical significance of the intervention might depend on the degree to which important activities of daily living or occupational activities can be targeted. Graded activity and exposure interventions aimed at reducing fear of movement have been shown to be effective in reducing disability, reducing absenteeism, and facilitating return to work (Bailey, Carleton, Vlaeyen, & Asmundson, 2010).

Choosing Among Different Psychological Interventions

The intervention approaches described in this chapter differ in terms of their focus, structure, content, and objectives. With the range of potential intervention avenues currently available, the clinician might reflect on the question of which intervention approach might be most suitable for a particular client. Since little research has been conducting on matching client profiles to specific interventions, this question unfortunately cannot be addressed from an empirical standpoint. There are, however, various points of consideration that might assist the clinician in determining the most appropriate intervention for his or her client.

Few would question the importance of information provision in the management of chronic pain and disability. The more that clients understand about the nature of their pain condition, the more they will be able to play an active role in the management of their condition. As such, information-based approaches such as back schools might be an important element in the management of chronic pain. However, for most clients with chronic pain conditions, information alone is unlikely to yield clinically significant improvements in mood, suffering, or disability. Information-based techniques might best be viewed as important elements of a more comprehensive approach to treatment as opposed to stand-alone interventions.

For the greater part of the last two decades, psychosocial interventions were included primarily as part of tertiary care treatment for clients with long-standing chronic pain and disability. With little expectancy of clinical improvement of clients' pain conditions, the focus of many treatment programs was primarily on the alleviation of suffering. Cognitive-behavioral interventions that used distress reduction techniques such as relaxation, reappraisal, and cognitive restructuring were ideally suited to achieve reductions in suffering in clients with long-standing chronic pain (Morley, Eccleston, & Williams, 1999).

As research accumulated showing that psychological interventions yielded significant reductions in pain and emotional distress, there was greater interest in using psychological interventions for clients who were at earlier stages of chronicity (Sullivan, 2003). The term secondary prevention is used to describe interventions that are implemented for individuals considered "at risk" condition or chronic pain and disability but whose condition had not yet become chronic. With a less chronic population, treatment objectives of psychological interventions changed. Since many clients still had an employment-relevant skill set, and some might also have had a job to return to, there was an increased focus on return to function as a central objective of treatment, as opposed to a primary focus on reduction of suffering. Return to function is a central objective of interventions such as PGAP or graded exposure.

When treatment is initiated after a long period of chronicity, intervention strategies are more likely to address the consequences of pain and

disability (e.g., affective disorders, drug/alcohol overuse, family dysfunction) as opposed to risk factors for pain and disability. It is important for professionals working with clients with long-standing chronic pain and disability to have a background in mental health in order to be able to intervene on psychological conditions that might be compounding the client's pain condition. However, risk factors for chronicity are not necessarily psychological disorders nor would they necessarily be considered indices of dysfunction (in the absence of a pain condition). Nevertheless, their presence contributes to a higher probability that a pain condition will persist or worsen over time. The challenge to effective secondary prevention lies not only in the development of risk-factor targeted interventions but in developing mechanisms by which individuals at risk can be identified. Perhaps more so than is the case for psychological disorders, risk factors for chronicity may be particularly likely to go undetected during routine primary care. Treating physicians often become aware of psychological factors in pain and disability only once chronicity has developed and the client has become treatment resistant.

Since psychological risk factors for chronic pain and disability are not mental health conditions, the development of secondary prevention interventions opened the door for using professionals who were not mental health professionals to deliver psychological interventions for pain. Intervention programs like PGAP or graded exposure are more likely to use occupational therapists, physiotherapists, or kinesiologists as interventionists than psychologists. This should be viewed as a positive change since the shortage of psychologists involved in the treatment of pain severely limits access to psychological services for individuals with debilitating pain conditions.

Thus, chronicity and clinical complexity are two factors that will influence the type of psychological intervention that will be considered, the objectives of the intervention, and the training background of the professional that will be used to deliver the intervention. Undoubtedly, other psychological interventions will be added to the repertoire of psychological services offered to clients with debilitating pain conditions. It is paramount to consider the evidence base for psychological interventions for pain-related difficulties before offering them to clients with debilitating pain conditions. Offering interventions that are not evidence based increases the probability of treatment failure and is likely to contribute to further demoralization of a client already struggling with a heavy burden of distress and disability.

Cross-References

- ▶ Pain Management/Control
- ▶ Pain, Psychosocial Aspects
- ▶ Pain-Related Fear

References and Readings

Bailey, K., Carleton, N., Vlaeyen, J. W. S., & Asmundson, G. J. (2010). Treatments addressing pain-related fear and anxiety in patients with chronic musculoskeletal pain: A preliminary review. *Cognitive Behavior Therapy, 39*, 46–63.

D'Zurilla, T. (1990). Problem-solving training for effective stress management and prevention. *Journal of Cognitive Psychotherapy, 4*, 327–355.

Feuerstein, M., Nicholas, R., Huang, G., Dimberg, L., Ali, D., & Rogers, H. (2004). Job stress management and ergonomic intervention for work-related upper extremity symptoms. *Applied Ergonomics, 35*, 565–574.

Fordyce, W. (1976). *Behavioral methods in chronic pain and illness*. St. Louis: Mosby.

Fordyce, W., Fowler, R., Lehmann, J., & De Lateur, B. (1968). Some implications of learning in problems of chronic pain. *Journal of Chronic Diseases, 21*, 179–190.

Fordyce, W. E., Roberts, A. H., & Sternbach, R. A. (1985). The behavioral management of chronic pain: A response to critics. *Pain, 22*(2), 113–125.

Gatchel, R., Peng, Y. B., Peters, M. L., Fuchs, P. N., & Turk, D. C. (2007). The biopsychosocial approach to chronic pain: Scientific advances and future directions. *Psychological Bulletin, 133*, 581–624.

Goubert, L., Francken, G., Crombez, G., Vansteenwegen, D., & Lysens, R. (2002). Exposure to physical movement in chronic back pain patients: No evidence for generalization across different movements. *Behaviour Research and Therapy, 40*(4), 415–429.

Hayes, S. C., Strosahl, K. D., & Wilson, K. G. (1999). *Acceptance and commitment therapy: An experiential approach to behavior change*. New York: Guilford Press.

Heymans, M. W., van Tulder, M. W., Esmail, R., Bombardier, C., & Koes, B. W. (2004). Back schools for non-specific low-back pain. *Cochrane Database of Systematic Reviews, 4*, CD000261.

Heymans, M. W., van Tulder, M. W., Esmail, R., Bombardier, C., & Koes, B. W. (2005). Back schools for nonspecific low back pain: A systematic review within the framework of the Cochrane Collaboration Back Review Group. *Spine (Phila Pa 1976), 30*(19), 2153–2163.

Lazarus, R., & Folkman, S. (1984). *Stress, appraisal and coping*. New York: Springer.

Leeuw, M., Goossens, M. E., Linton, S. J., Crombez, G., Boersma, K., & Vlaeyen, J. W. (2007). The fear-avoidance model of musculoskeletal pain: Current state of scientific evidence. *Journal of Behavioral Medicine, 30*(1), 77–94.

Linton, S. J., & Kamwendo, K. (1987). Low back schools. A critical review. *Physical Therapy, 67*(9), 1375–1383.

McCracken, L. M. (2005). *Contextual cognitive therapy for chronic pain*. Seattle, WA: IASP Press.

Morley, S., Eccleston, C., & Williams, A. (1999). Systematic review and meta-analysis of randomized controlled trials of cognitive-behavior therapy and behavior therapy for chronic pain in adults, excluding headache. *Pain, 80*, 1–13.

Sanders, S. H. (1996). Operant conditioning of chronic pain: Back to basics. In R. Gatchel & D. C. Turk (Eds.), *Psychological approaches to pain management*. New York: Guilford.

Smeets, R. J., Vlaeyen, J. W., Hidding, A., Kester, A. D., van der Heijden, G. J., & Knottnerus, J. A. (2008). Chronic low back pain: physical training, graded activity with problem solving training, or both? The one-year post-treatment results of a randomized controlled trial. *Pain, 134*(3), 263–276.

Sullivan, M. J. L. (2003). Emerging trends in secondary prevention of pain-related disability. *The Clinical Journal of Pain, 19*, 77–79.

Sullivan, M. J. L., Adams, H., Rhodenizer, T., & Stanish, W. D. (2006). A psychosocial risk factor–targeted intervention for the prevention of chronic pain and disability following whiplash injury. *Physical Therapy, 86*(1), 8–18.

Sullivan, M. J., Adams, H., Thibault, P., Corbiere, M., & Stanish, W. D. (2006). Initial depression severity and the trajectory of recovery following cognitive-behavioral intervention for work disability. *Journal of Occupational Rehabilitation, 16*(1), 63–74.

Sullivan, M., Feuerstein, M., Gatchel, R. J., Linton, S. J., & Pransky, G. (2005). Integrating psychological and behavioral interventions to achieve optimal rehabilitation outcomes. *Journal of Occupational Rehabilitation, 15*, 475–489.

Sullivan, M. J. L., Ward, L. C., Tripp, D., French, D. J., Adams, H., & Stanish, W. D. (2005). Secondary prevention of work disability: Community-based psychosocial intervention for musculoskeletal disorders. *Journal of Occupational Rehabilitation, 15*(3), 377–392.

Turk, D., Meichenbaum, D., & Genest, M. (1983). *Pain and behavioral medicine: A cognitive-behavioral perspective*. New York: Guilford.

Vlaeyen, J. W., & Linton, S. J. (2000). Fear-avoidance and its consequences in chronic musculoskeletal pain: A state of the art. *Pain, 85*(3), 317–332.

Vowles, K. E., & McCracken, L. M. (2008). Acceptance and values-based action in chronic pain: A study of treatment effectiveness and process. *Journal of Consulting and Clinical Psychology, 76*(3), 397–407.

Williams, A. C., Pither, C. E., Richardson, P. H., Nicholas, M. K., Justins, D. M., Morley, S., et al. (1996). The effects of cognitive-behavioural therapy in chronic pain. *Pain, 65*(2–3), 282–284.

Zachrisson-Forsell, M. (1981). The back school. *Spine (Phila Pa 1976), 6*, 104–106.

Chronobiology

Tanja Lange
Department of Neuroendocrinology,
University of Luebeck, Lübeck, Germany

Definition

Chronobiology is the science of periodic changes in physiology and behavior of living organisms (Halberg, 1969). It describes these *biological rhythms* with statistical methods (*chronobiometry*) and elucidates the underlying molecular/biochemical mechanisms at a cellular and systemic level, the entrainment of these internal timing systems by external time cues, the effects of timed light and drug therapy (*chronotherapy*, *chronopharmacology*, *chronotoxicology*), as well as disturbances of biological rhythms that may lead to pathology (Dunlap, Loros, & DeCoursey, 2004; Foster & Kreitzman, 2004; Koukkari & Sothern, 2006; Redfern & Lemmer, 2007). In behavioral medicine, the most relevant biological rhythms show a period of about 24 h (*circadian*), 7 days (*circaseptan*), 30 days (*circatrigintan*), or 1 year (*circannual*).

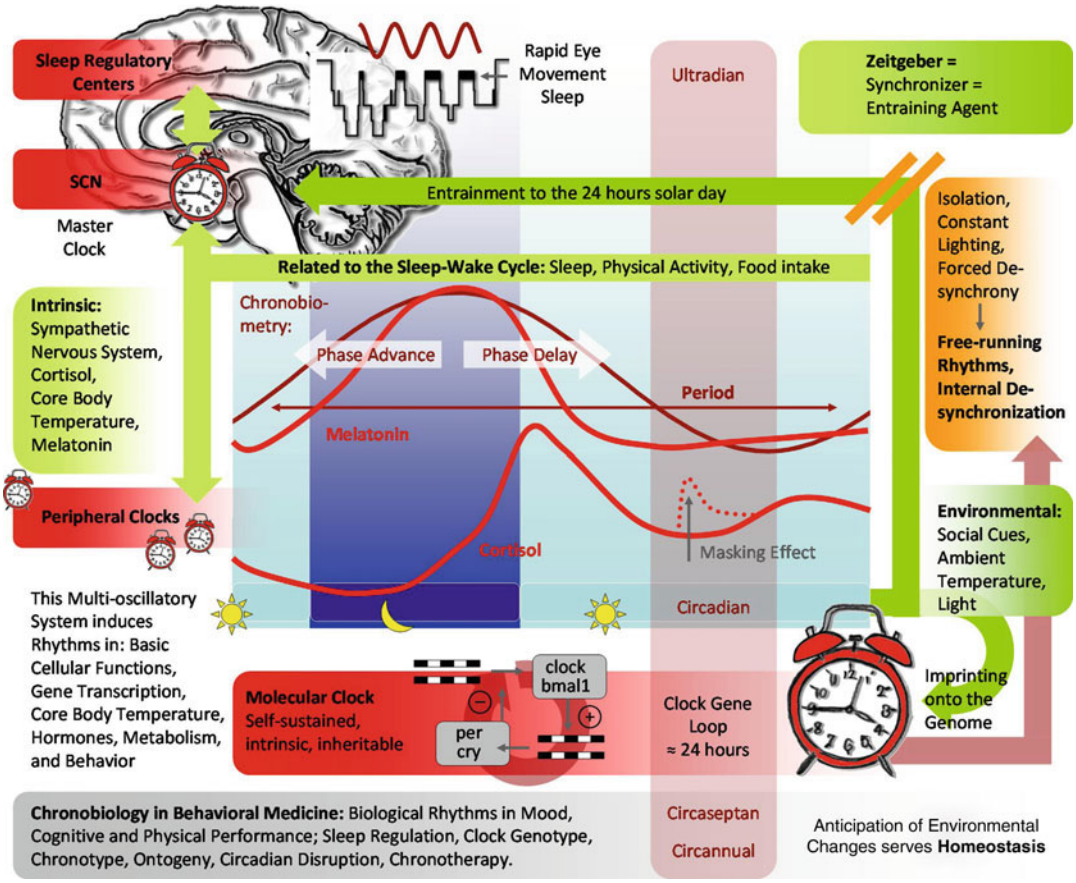
Description

Life is adapted to rhythms that are generated by movements of the Earth, the Moon, and the Sun

in relation to each other. Depending on the features of its habitat, every living organism on earth, including bacteria, plants, animals, and humans, shows rhythmic changes in physiology and behavior with different periods like that of tidal rhythms (~12 h, *circahemidian*), the daily light–dark cycle (~24 h, *circadian*), the weekly cycle (presumably stemming from alternations between spring- and neap-tides, ~7 days, *circaseptan*), the lunar cycle (~30 days, *circatrigintan*), and the seasons (~1 year, *circannual*). Periods that are longer than 1 day are called *infradian*, those that are shorter than 1 day are termed *ultradian*. Ultradian oscillations show very different periods and, like the 90 min of the non-REM-REM sleep cycle (REM: rapid eye movement), often lack an environmental counterpart.

Biological rhythms do not simply follow environmental changes but rather appear to anticipate them. They are still evident if an organism is deprived of any external time cue (*zeitgeber*) or in isolated cells in culture (e.g., white blood cells). Under such *free-running* conditions, a given rhythm period typically slightly deviates from the external cycle (e.g., 24.2 h instead of 24 h, hence the term "circa-dian") and this unmasked endogenous rhythm represents a trait with considerable interindividual differences (Aschoff, 1965). This indicates that environmental rhythms are adopted by inheritable internal time-keeping systems. It is assumed that endogenous timing mechanisms developed during evolution, with the goal of adapting the organism to relevant environmental changes (like the availability of food, exposure to predators, changes in ambient temperature, or periods of efficient reproduction) in an anticipatory manner. This anticipatory cycling is advantageous, e.g., with respect to energetic efficiency, and was therefore preserved by natural selection. The adaptation of living matter to environmental changes reflects a basic concept of physiology, i.e., *homeostasis* – the maintenance of the "internal milieu" of the organisms at a constant level (*setpoint*) despite external challenges. Core body temperature, e.g., is homeostatically regulated. In addition to the advantage of adaptation, more complex organisms might have benefited also from the separation of otherwise incompatible body and brain functions in time (e.g., encoding of new information during the active period and consolidation, i.e., the covert reactivation of "fresh" memory traces that is incompatible with active stimulus processing during the resting period).

To sum up, astronomically generated rhythms were evolutionary imprinted onto the genome of living organisms, creating anticipatory biological clocks and the organization of physiology and behavior in time (see Fig. 1). The underlying molecular machinery has been elucidated mainly for the circadian rhythm in the 1970s when the first *clock gene* was described in the fruit fly. By now many clock genes are discovered that are linked with their respective transcripts in an interlocked feedback loop that takes about 24 h for a full cycle. Activity in this feedback loop represents the molecular pendulum of the clock (Panda, Hogenesch, & Kay, 2002). In mammalian brain and peripheral organs, clock genes control basic cellular processes and up to 10% of the transcriptome in a tissue-specific manner. Though sophisticated, these self-sustained clocks are not precise – as it becomes evident under free-running conditions – and therefore have to be reset and synchronized (*entrainment*) by an external zeitgeber (*synchronizer, entraining agent*). The most important zeitgeber is light. Photic entrainment is provided by nonvisual retinal cells that convey the information about light and darkness to the hypothalamic *suprachiasmatic nuclei* (SCN), called the "master clock." In a hierarchic structure, the SCN signals to other brain centers (like sleep regulatory centers) and synchronizes clocks in peripheral tissues via the sympathetic nervous system and the hypothalamic-pituitary-adrenal axis. Apart from light, further external and internal synchronizers are ambient and core body temperature, sleep, physical activity, melatonin, food intake, and social cues. So, workdays and weekends are likely synchronizers for circaseptan rhythms, whereas circannual rhythms are assumed to be entrained by the length of the daily light span and changes in environmental temperature.



Chronobiology, Fig. 1 Environmental rhythms like the 24 h light–dark cycle were evolutionary imprinted onto the genome of living matter. Clock genes and their transcripts built up the molecular clock that ticks in the hypothalamic suprachiasmatic nuclei (SCN), but also in many if not all cells of the human body. As these molecular clocks are not precise they are synchronized (entrained) by zeitgeber. Environmental zeitgebers reset the phase of the SCN that itself signals to sleep regulatory centers and synchronizes peripheral clocks by intrinsic and activity-related factors. This multi-oscillatory system induces rhythms in physiology and behavior that are synchronized to environmental rhythms and therefore serve to anticipate external challenges (homeostasis). Two hormonal rhythms are depicted that can be statistically described (chronobiometry). Experimental procedures in chronobiology aim to dissect the effects of endogenous clocks from entraining and masking influences. Apart from circadian rhythms, chronobiology also describes oscillations with periods that are shorter than 1 day (like the ultradian rhythm of rapid eye movement sleep) and with periods of about 7 days (circaseptan) and about 1 year (circannual). Important aspects of chronobiology in behavioral medicine are summarized in the *grey box*

In animals and humans, this complex time-keeping system of clock genes and SCN regulates the sleep-wake cycle and induces rhythmic changes in cognitive and physical performance, core body temperature, hormone levels, and metabolism. Chronobiologists assess such rhythms with inferential statistical tools (*chronobiometry*) either under "natural," i.e., entrained, conditions or in experimental settings that allow to dissect the endogenous component of these rhythms from *masking* environmental factors or behaviors. Experimental designs use isolation procedures (cave or bunker experiments) or constant lighting conditions in humans and animals, respectively, to eliminate external time cues. Emerging free-running rhythms may then differ among parameters in terms of their period such that, e.g., the sleep-wake-cycle may

desynchronize from the rhythm of core body temperature (*internal desynchronization*) (Aschoff, 1965). The *forced desynchrony protocol* intentionally induces such an effect. It exploits the fact that an endogenous rhythm can only be entrained to periods that differ not too much from its own period (*range of entrainment*). If, therefore, the sleep-wake cycle is experimentally scheduled to 28 h, the rhythm of core body temperature runs out of phase with its own free-running period. Another elaborate approach used to dissociate the circadian rhythm from masking influences in humans is the *constant routine protocol*. In this protocol, the participants stay awake for more than 24 h under constant ambient light and temperature, in a supine position in bed with hourly isocaloric snacks and beverages. All these methods aim to scrutinize the contribution of multiple endogenous oscillators as well as entraining environmental, intrinsic, and activity-related factors to biological rhythms. In addition, they address the bidirectional interactions between the circadian system and sleep.

To unravel molecular mechanisms of biological rhythms chronobiologists study genetically manipulated animals (*knockouts* or *mutants* of certain clock genes in the whole genome or in individual organs), silence clock gene activity with *RNA interference* (RNA: ribonucleic acid) or couple clock genes with *luciferase* to allow continuous long-term monitoring of gene activity in cell cultures as well as in vivo (Panda et al., 2002). Human research focuses on twin studies and clock gene *polymorphisms*. *Clock genotypes* can then be set into relation to the circadian preference of individuals (*chronotype*, i.e., whether one is a "lark" or an "owl") that is assessed by investigating the phase of rhythms under "natural" entrained conditions by means of questionnaires, diaries, actigraphy, or *dim light melatonin onset* (DLMO). In this context, ontogenetic research elucidates the phase shifts that occur during lifetime, i.e., the phase delay in adolescence and the phase advance in the elderly (Phillips, 2009).

Chronobiology is an interdisciplinary science covering all fields of medical practice and research. Circadian, circaseptan, circatrigintan, and circannual rhythms are described in all disciplines of clinical medicine with respect to physiological functions, laboratory findings and the incidence of disease symptoms. In addition, efficacy and potential side effects of medical interventions show time dependency (*chronopharmacology, chronotoxicology*). Disruption of biological rhythms, as evident in shift workers, travels across time zones (*jet lag*), but also due to modern lifestyle, compromises mood, sleep, cognitive and physical performance, activates the stress axes, and may eventually lead to pathology and disorders like major depression, metabolic syndrome, obesity, immunosuppression, low grade systemic inflammation, and cardiovascular diseases. Conversely, sleep curtailment, chronic stress, high fat diet, many infections, and autoimmune diseases are associated with *circadian disruption* thus feeding into a vicious circle (Phillips, 2009). These relationships, however, also offer therapeutic options of re-entraining biological rhythms by means of zeitgebers (*chronotherapy, chronobiotics*), as it is done with bright light therapy in mood disorders and the administration of melatonin to prevent jet lag. The optimal timing of such interventions can be assessed by *phase-response curves* representing an important research tool of chronobiology. In addition, cognitive behavioral therapy can alleviate circadian and sleep disruption in psychiatric and neurologic diseases. As epidemiological data indicate that circadian disruption and associated sleep curtailments increase the incidence of metabolic and cardiovascular diseases and the risk of cancer, it is the goal of future research to elucidate if re-entrainment of biological rhythms can likewise be beneficial to prevent these diseases in shift workers and the elderly.

Cross-References

- ▶ Cardiovascular Disease
- ▶ Central Nervous System
- ▶ Circadian Rhythm
- ▶ Cognitive Behavioral Therapy (CBT)
- ▶ Cognitive Function
- ▶ Corticosteroids

- ▶ Cortisol
- ▶ Diurnal Mood Variation
- ▶ Homeostasis
- ▶ Hypothalamus
- ▶ Inflammation
- ▶ Life Span
- ▶ Lifestyle Changes
- ▶ Metabolic Syndrome
- ▶ Metabolism
- ▶ Mood
- ▶ Pathophysiology
- ▶ Physical Functioning
- ▶ Polymorphism
- ▶ Sleep
- ▶ Stress
- ▶ Sympathetic Nervous System (SNS)

References and Readings

Aschoff, J. (1965). Circadian rhythms in man. *Science, 148*, 1427–1432.

Dunlap, J. C., Loros, J. J., & DeCoursey, P. J. (2004). *Chronobiology: Biological timekeeping*. Sunderland, MA: Sinauer.

Foster, R. G., & Kreitzman, L. (2004). *Rhythms of life: The biological clocks that control the daily lives of every living thing*. London: Yale University Press.

Halberg, F. (1969). Chronobiology. *Annual Review of Physiology, 31*, 675–725.

Koukkari, W. L., & Sothern, R. B. (2006). *Introducing biological rhythms: A primer on the temporal organization of life, with implications for health, society, reproduction, and the natural environment*. New York: Springer.

Panda, S., Hogenesch, J. B., & Kay, S. A. (2002). Circadian rhythms from flies to human. *Nature, 417*, 329–335.

Phillips, M. L. (2009). Circadian rhythms: Of owls, larks and alarm clocks. *Nature, 458*, 142–144.

Redfern, P. H., & Lemmer, B. (2007). *Physiology and pharmacology of biological rhythms* (Vol. 125). New York: Springer.

Websites

Center for Chronobiology, University of California, San Diego. http://ccb.ucsd.edu

Howard Hughes Medical Institute. *Biological clocks, lecture series*. http://www.hhmi.org/biointeractive/clocks/lectures.html

Society for Research on Biological Rhythms. http://www.srbr.org

Zivkovic, B. *Clock tutorials*. http://borazivkovic.blogspot.com/2005/01/clock-tutorials.html

Church Attendance

- ▶ Religious Ritual

Church-Based Interventions

Marianne Shaughnessy
School of Nursing, University of Maryland, Baltimore, MD, USA

Synonyms

Faith community interventions; Faith-based interventions

Definition

Refers to any research, clinical, public health, or data collection initiative targeted to a faith-based organization or community.

Description

Academicians, clinicians, and researchers have partnered with church-based or faith-based organizations for the purposes of descriptive and interventional research, launching pilot programs and studying public health problems for years. There are multiple advantages to partnering with such populations for these purposes. These groups tend to be established communities, with an organized, recognized authority structure that provides a support network for all those within the group. This infrastructure is well suited to allow investigation of social and public health issues. Secondly, the groups share a common belief and value system, allowing for an assessment of how those beliefs affect behaviors. To the extent that health-related lifestyle behaviors are dictated by religious beliefs, studies of these populations can address health outcomes, such as

those explored in the Nun's study (University of Minnesota), or the influence of genetics on health, as in studies of the Old Order Amish (Hsueh et al., 2000). Finally, depending on the size of the faith community, it is possible to capture a large number of potential study subjects within one faith community or a network of faith communities.

Recognizing that church- and faith-based organizations could be significant partners in addressing social and health-related issues, President George W. Bush established the White House Office of Faith-Based and Community Initiatives in 2001 as a means to allow faith-based organizations to apply for federal funding to implement social service programs. Under criticism from the Americans United for the Separation of Church and State and the American Civil Liberties Union, safeguards were put into place that prevent these groups from advancing their religious agendas while administering programs using federal funds. In 2009, President Barack Obama changed the name of the organization to the White House Office of Faith-based and Neighborhood Partnerships. The Department of Health and Human Services now houses the Center for Faith-based and Neighborhood Partnerships. This center does not administer grants but provides information on building and sustaining partnerships for community-based programs. Several other US government departments currently host initiatives for faith-based and community partnerships, including the Substance Abuse and Mental Health Services Administration and the US Department of Agriculture.

Research interventions conducted within the context of church- or faith-based organizations can be effectively conducted only with careful consideration in advance of the church and community challenges, selection of the right faith community to meet the needs of the project, understanding of how to best implement the project without offense and skillful marketing strategies. Rev. Melvin Tuggle (2000) offers specific guidance on related principles and how to approach and interact with faith communities in inner-cities in "It is Well with My Soul: Churches and Institutions Collaborating for Public Health." In this book, Rev. Tuggle suggests the importance of approaching these collaborations as a true partnership and makes specific recommendations for ensuring a successful collaboration.

Churches and faith communities can also be starting points for interventions designed to be expanded to the community at large. By introducing a program, initiative, or intervention at a church, potential participants may observe the enthusiasm of those already engaged and create support for expansion of the project beyond the church group. With careful planning in advance and a thoughtful, respectful approach, it is possible to create a true partnership for research or clinical care projects to improve public health.

References and Readings

Hsueh, W. C., Mitchell, B. D., Aburomia, R., Pollin, T., Sakul, H., Gelder Ehm, M., et al. (2000). Diabetes in the old order Amish: Characterization and heritability analysis of the Amish Family Diabetes Study. *Diabetes Care, 23*(5), 595–601.

Tuggle, M. (2000). *It is well with my soul: Churches and institutions collaborating for public health*. Washington, DC: American Public Health Association.

University of Minnesota. *The nun study*. Accessed May 13, 2011, from https://www.healthstudies.umn.edu/nunstudy/

US Department of Health and Human Services. *Center for faith-based and neighborhood partnerships*. http://www.hhs.gov/partnerships/

Church-Based Support

▶ Religious Social Support

Cigarette

▶ Nicotine

Cigarette Advertising

▶ Tobacco Advertising

Cigarette Smoking

▶ Smoking Behavior

Cigarette Smoking and Health

▶ Smoking and Health

Cigarette Smoking Behavior

▶ Smoking Behavior

Cigarette Smoking Cessation

▶ Smoking Cessation

Circadian Clock

▶ Circadian Rhythm

Circadian Rhythm

Fumiharu Togo
Graduate School of Education, The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Synonyms

Circadian clock

Definition

A circadian rhythm is an approximately 24-h cycle of a biochemical, physiological, or behavioral process that is generated by internal biological clocks. In most animals, the intrinsic rhythm of the clock (cycle length) is slightly longer than 24 h, but normally the clock is synchronized to the 24-h day (entrainment) by environmental time signals (zeitgebers), the primary one of which is solar light. In the absence of timing signals (temporal isolation), circadian rhythms free-run on a non-24-h cycle, expressing the intrinsic rhythm of the clock. The process of synchronization involves daily, stimulus-induced adjustment (phase shifts) that compensate for the difference between the intrinsic period of the internal clock and the period of the environmental cycle. Light can induce phase shift that varies in magnitude and direction depending on the circadian phase of exposure. Light exposure in the subjective morning resets the internal clock to an earlier time, while light exposure in the early subjective night resets the clock to a later time. Intensity of the light, duration of the light pulse, and the spectral characteristics of the light determine the magnitude of a phase shift at any specific circadian phase. Blue light is an efficient wavelength to shift the circadian rhythms.

The suprachiasmatic nucleus (SCN), which is situated bilaterally in the hypothalamus, just above the optic chiasm, is of central importance in the generation and entrainment of mammalian circadian rhythms. Destruction of SCN disrupts a wide variety of circadian rhythms. Photic entrainment is thought to be largely mediated by retinal photoreceptors. Approximately one third of SCN cells are photically responsive which is believed to result from glutamatergic stimulation of N-methyl-D-aspartate receptors through the retinohypothalamic tract. Photic and glutamatergic stimulation of SCN cells in the early subjective night causes phase delay, whereas such stimulation late in the subjective night causes phase advance.

Circadian rhythms in some species can also be shifted and entrained by stimuli other than

light, such as exercise, social stimuli, or feeding. These so-called nonphotic zeitgebers may in some cases engage a circadian pacemaker system separate from that affected by light. Nonphotic influences on the clock phase appear to be mediated by the geniculohypothalamic tract, neuropeptide Y, and serotonergic pathways. Although the mechanism that constitutes exercise to promote phase shift in the human circadian clock is unclear, exercise during the late subjective day has been shown to produce a phase advance of the rhythm, whereas exercise during most of the subjective night produces phase delays.

Cross-References

► Neuropeptide Y (NPY)

References and Readings

Koukkari, W. L., & Sothern, R. B. (2006). *Introducing biological rhythms*. New York: Springer.
Refinetti, R. (2006). *Circadian physiology* (2nd ed.). Boca Raton, FL: CRC Press.

Citalopram

► Selective Serotonin Reuptake Inhibitors (SSRIs)

Classic Migraine

► Migraine Headache

Classical Conditioning

Annie T. Ginty
School of Sport and Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

Synonyms

Pavlovian conditioning

Definition

Classical conditioning is learning by association and focuses on what happens before an individual responds. It is often used in behavioral training. Perhaps, the most well-known example of classical conditioning is that of Pavlov's dogs. Pavlov measured salivation responses in dogs. Before conditioning, he rang a bell and noted that there was no increase in saliva from the dogs. Then, during conditioning, he rang a bell (unconditioned stimulus) and immediately put meat powder (conditioned stimulus) on the dogs' tongues which caused them to salivate (unconditioned response); he continued this several times. Finally, after conditioning, he rang the bell again but without food and the dogs salivated (conditioned response). Pavlov used classical conditioning so the dogs associated an unrelated stimulus (the bell) with food. Thus, they eventually produced the same saliva response they would for food with the bell. For further details, see Coon and Mitterer (2010) (Fig. 1).

Classical Conditioning, Fig. 1 Pavlovian conditioning

Cross-References

▶ Operant Conditioning

References and Readings

Coon, D., & Mitterer, J. O. (2010). *Introduction to psychology: Gateways to mind and behavior* (12th ed.). Wadsworth, CA: Wadsworth Cengage Learning.

Clinical Agreement

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Medical agreement

Definition

It is important in both clinical medicine and research to assess the extent to which different individuals (e.g., clinicians, observers) observe and report the same phenomenon (Jekel, Katz, Elmore, & Wild, 2007). Ideally, there would be perfect intraobserver agreement (the same person would always observe and report the same phenomenon in an identical manner), and perfect interobserver agreement (different people would observe and report the same phenomenon identically). However, these ideals are precisely that: they describe an ideal scenario, and real-life scenarios are often quite different. Elmore, Wells, Lee, Howard, and Feinstein (1994) studied both intraobserver and interobserver agreement among radiologists' interpretations of a specific mammogram, demonstrating that radiologists can differ, sometimes substantially, in their interpretations of mammograms and in their recommendations for management.

Quantifying the extent to which clinical agreement exists in a given situation is therefore important. Consider the following hypothetical data presented by Jekel et al. (2007) concerning clinical agreement between two clinicians regarding their diagnosis of the presence or absence of a cardiac murmur upon physical examination of 100 patients:

	Clinician number 1		
Clinician no. 2	Murmur present	Murmur absent	Total
Murmur present	30	7	37
Murmur absent	3	60	63
Total	33	67	100

These data show the following:

1. For 30 patients, the clinicians both determined the presence of a murmur.
2. For 60 patients, the clinicians both determined the absence of a murmur.
3. For 7 patients, Clinician number 2 determined the presence of a murmur while Clinician number 1 determined the absence of a murmur.
4. For 3 patients, Clinician number 1 determined the presence of a murmur while Clinician number 2 determined the absence of a murmur.

The maximum possible degree of clinical agreement is equal to the total number of patients, i.e., 100. This would occur when the two clinicians made the same determination for every patient. As already noted, this is an ideal but unlikely scenario. (Actually, the operationalization of the term "ideal" in this context has another aspect when making clinical judgments: Ideally, both clinicians make the same and CORRECT determination; it is a theoretical possibility that they could agree 100% of the time and also be wrong 100% of the time.) Various calculations can be conducted to quantify the degree of agreement.

The actual degree of agreement is 90 out of 100 cases. This value is typically presented as a percentage, which is 90% (the numbers here are deliberately chosen to facilitate straightforward calculations). However, purely by random chance, it is possible that the clinicians would agree sometimes. Imagine a scenario in which

the two clinicians were asked simply to write a list of 100 terms, each time choosing between "murmur present" and "murmur absent." Probabilistically, there would likely be some agreement. A key question therefore becomes: To what extent does the degree of clinical agreement between the two clinicians improve upon chance agreement alone?

The kappa test ratio provides an answer to this question. In this case, the mathematics (not presented here) lead to a kappa test ratio of 0.78, which is typically expressed in percentage terms, i.e., 78%. To put this in perspective, consider the arbitrary but useful divisions for the interpretation of kappa scores as presented by Sacket, Haynes, Guyatt, and Tugwell (1991):

1. Less than 20% represents negligible improvement in the degree of clinical agreement over chance alone.
2. 20–39% represents minimal improvement.
3. 40–59% represents fair improvement.
4. 60–79% represents good improvement.
5. 80% and above represents excellent improvement.

These hypothetical data yielded a kappa score of 78%, as noted already, meaning that this degree of improvement would fall in the "good improvement" category. With regard to real data, Jekel et al. (2007) stated that "the reliability of most tests in clinical medicine that require human judgment seems to fall in the fair or good range."

Cross-References

▶ Clinical Decision-Making
▶ Probability

References and Readings

Elmore, J. G., Wells, C. K., Lee, C. H., Howard, D. H., & Feinstein, A. R. (1994). Variability in radiologistis' interpretation of mamograms. *The New England Journal of Medicine, 331*, 1493–1499.

Jekel, J. F., Katz, D. L., Elmore, J. G., & Wild, D. M. G. (2007). *Epidemiology, biostatistics, and preventive medicine* (3rd ed.). Philadelphia: Saunders/Elsevier.

Sacket, D. L., Haynes, R. B., Guyatt, G. H., & Tugwell, P. (1991). *Clinical epidemiology: A basic science for clinical medicine* (2nd ed.). Boca Raton, FL: Little/Brown.

Clinical Decision-Making

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Decision analysis; Medical decision-making

Definition

Clinicians must make treatment decisions on a daily basis, and these decisions, or recommendations (final decisions are best made by the "health team" of a physician and his or her patient) should be based on the best available evidence. The terms "evidence-based medicine" and "evidence-based practice" have become part of the health lexicon, and "evidence-based behavioral medicine" is also an established term (see the ▶ Evidence-Based Behavioral Medicine (EBBM) entry in this encyclopedia for a detailed discussion).

While clinical decision-making relies on evidence, the evidence in the medical literature (with the exception of case reports) typically describes the experience of a population of patients rather than an individual patient. Evidence-based clinical decision-making, therefore, requires "the application of population-based data to the care of an individual patient whose experiences will be different, in ways both discernible and not, from the collective experience reported in the literature" (Katz, 2001). He also observed that "All of the art and all of the science of medicine depend on how artfully and scientifically we as practitioners reach our decisions. The art of clinical decision-making is judgment, an even more difficult concept to grapple with than evidence."

Decision analysis is a formalized approach to making complex clinical decisions that relies on plotting a "decision tree" containing the various options and then rating each in terms of

probability and utility. In this way, the clinician attempts to make explicit the quantitative principles upon which a given clinical decision will be based. Once these principles have been identified from the literature, both the clinician and the patient can consider them, challenge them as appropriate, and systematically eliminate treatment (or nontreatment) options until a clear preference emerges (Katz, 2001).

Cross-References

- ▶ Clinical Agreement
- ▶ Evidence-Based Behavioral Medicine (EBBM)
- ▶ Generalizability

References and Readings

Katz, D. L. (2001). *Clinical epidemiology and evidence-based medicine: Fundamental principles of clinical reasoning and research*. Thousand Oaks, CA: Sage.

Clinical Equipoise

- ▶ Principle of Equipoise

Clinical Ethics

- ▶ Ethical Issues

Clinical Guideline

- ▶ Clinical Practice Guidelines

Clinical Health Psychology

- ▶ Medical Psychology

Clinical Practice Guidelines

Karina Davidson[1] and Joan Duer-Hefele[2]
[1]Department of Medicine, Columbia University Medical Center, New York, NY, USA
[2]Columbia University, New York, NY, USA

Synonyms

Clinical guideline; Consensus guideline; Guideline; Practice guideline

Definition

Clinical practice guidelines "are systematically developed statements to assist practitioner and patient decisions about appropriate health care for specific clinical circumstances" (Field & Lohr, 1990). Good practice guidelines should be specific, comprehensive, and yet flexible enough to be useful (Field & Lohr, 1992).

Description

Clinical practice guidelines are needed because early reports suggested that less than 5% of medical treatment decisions were based on strong research evidence; about half were based on shared clinician beliefs that had minimal scientific support and half were based on personal opinion (Field & Lohr, 1990). Well-constructed clinical practice guidelines hold the promise of providing information to enable clinicians to choose the best treatments, diagnoses, or screening practices available, and regulators to set policy based on the current state of scientific knowledge. Practice guidelines can assist policymakers and public health advocates to be able to better determine which behavioral medicine practices should be reimbursed (Davidson, Trudeau, Ockene, Orleans, & Kaplan, 2004). Risk management – that is, the effort to lower or curb the number of poor outcomes and potential for malpractice litigation – is another possible

reason for developing clinical practice guidelines. They can also be used when considering accreditation and certification for education programs and individual clinicians. However, the primary reason for developing and then implementing clinical practice guidelines is the expectation that they will improve the patient, the public, and the community's health.

Each clinical practice guideline differs with respect to the practice it covers, the way in which the evidence is collected, the rules used to judge a practice as useful, and the way in which these guidelines are communicated to influence patient care. Essentially, some organization, whether a governmental body, a professional society, or an empanelled group of experts, reviews the evidence to support a specific screening, diagnostic, or treatment practice and then provides a guideline advising on the usefulness of that practice. *Evidence-based* practice guidelines are often distinguished from *consensus* practice guidelines that are informed by relevant research but not necessarily guided by systematic evidence reviews (Davidson et al., 2004).

American Heart Association (AHA), for example, has set out an explicit and formal review system for creating a guideline. The need for the guideline, the composition of the members of the writing group, and the approval process are all prespecified. Second, the criteria for searching for the evidence, the grade that will be given the evidence, and the summary statement similarly must also follow the prespecified system. For example, evidence for benefit of a drug or a preventive action is considered "A" if there are multiple randomized controlled trials or meta-analyses from multiple populations that all show benefit to the patient or the community. The AHA methodology handbook for creating systematic practice guidelines can be found as a pdf at: http://www.americanheart.org/presenter.jhtml?identifier=3039683.

Similarly, the United States Preventative Services Task Force has set up a system for objectively obtaining and reviewing all evidence for preventative services and releases guidelines advising practitioners and patients about the level of evidence to support or refute the use of certain interventions http://www.uspreventiveservicestaskforce.org/methods.htm. There are many other bodies who create clinical practice guidelines, such as the American Psychiatric Association http://www.psych.org/mainmenu/psychiatricpractice/practiceguidelines_1.aspx and the CDC-sponsored Community Guide (Briss et al., 2000) http://www.thecommunityguide.org/about/index.html.

A examination of where to find clinical practice guidelines revealed that most first look online and prefer governmental agency guidelines over others (Burgers, Cluzeau, Hanna, Hunt, & Grol, 2003). There is an excellent resource to look for any relevant clinical practice guidelines that is run by AHRQ – The National Guideline Clearing house – http://www.guideline.gov/.

There are some excellent educational references that explain how to locate, evaluate, and then, if relevant, use the information in practice guidelines (Hayward, Wilson, Tunis, Bass, & Guyatt, 1995; Wilson, Hayward, Tunis, Bass, & Guyatt, 1995). For a systematic approach to assessing guidelines, the AGREE instrument is available. http://www.agreecollaboration.org/

There is no formal accrediting body or a professional society in behavioral medicine that regularly produces practice guidelines; locations of guidelines that may be useful for behavioral medicine can be found in this citation (Davidson et al., 2004).

There is an optimistic assumption that the application of clinical practice guidelines results in better patient outcomes (Spring et al., 2005). However, rigorous program evaluation to support this assertion is only in its beginning stages. Grimshaw and others (Grimshaw & Russell, 1993) conducted a systematic review to address this question by examining evaluations of clinical guideline implementation for specific clinical conditions and preventative services. Of 59 papers, all but 4 detected significant improvements in the process of care following the introduction of guidelines. They concluded that explicit guidelines do improve clinical practice, but careful evaluation is always required. As few explicit behavioral medicine clinical practice

guidelines exist, little is known about the adoption of evidence-based guidelines and their use in other fields, and certainly, this is uncharted within behavioral medicine (Spring et al.).

It is also by no means certain that using practices recommended by practice guidelines will decrease health care costs; in medicine, it has sometimes increased them (Sackett, Rosenberg, Gray, Haynes, & Richardson, 1996). However, in the absence of evidence-based practice guidelines, managed care organizations and other policymakers may reimburse the most economical treatment (Pincus, 1994), a measure that would certainly restrict practice (Spring et al., 2005). The value of clinical practice guidelines for behavioral medicine is that, by making it possible to distinguish between effective and ineffective treatments, it encourages all of us to make informed decisions about training, about practice, and about reimbursement.

References and Readings

Briss, P. A., Zaza, S., Pappaioanou, M., Fielding, J., Wright-De Agüero, L., Truman, B. I., et al. (2000). Developing an evidence-based Guide to Community Preventive Services – Methods. The Task Force on Community Preventive Services. *American Journal of Preventive Medicine, 18*, 35–43.

Burgers, J. S., Cluzeau, F. A., Hanna, S. E., Hunt, C., & Grol, R. (2003). Characteristics of high-quality guidelines: Evaluation of 86 clinical guidelines developed in ten European countries and Canada. *International Journal of Technology Assessment in Health Care, 19*, 148–157.

Davidson, K. W., Trudeau, K. J., Ockene, J. K., Orleans, C. T., & Kaplan, R. M. (2004). A primer on current evidence-based review systems and their implications for behavioral medicine. *Annals of Behavioral Medicine, 28*, 226–238.

Field, M. J., & Lohr, K. N. (1990). *Clinical pratice guidelines: Directions for a new program. Committee to Advise the Public Health Service on clinical practice guidelines*. Washington, DC: Institute of Medicine.

Field, M. J., & Lohr, K. N. (1992). *Guidelines for clinical practice: From development to use*. Washington, DC: Committee on Clinical Practice Guidelines, Division of Health Care Services, Institute of Medicine.

Grimshaw, J. M., & Russell, I. T. (1993). Effect of clinical guidelines on medical practice: A systematic review of rigorous evaluations. *Lancet, 342*, 1317–1322.

Hayward, R. S., Wilson, M. C., Tunis, S. R., Bass, E. B., & Guyatt, G. (1995). Users' guides to the medical literature. VIII. How to use clinical practice guidelines. A. Are the recommendations valid? The Evidence-Based Medicine Working Group. *JAMA: The Journal of the American Medical Association, 274*, 570–574.

Pincus, H. A. (1994). Dialogue: Treatment guidelines: What are the risks? Risks are outweighed by the benefits. *Behavioral Healthcare Tomorrow, 3*, 40–41. 44–45.

Sackett, D. L., Rosenberg, W. M., Gray, J. A., Haynes, R. B., & Richardson, W. S. (1996). Evidence based medicine: What it is and what it isn't. *British Medical Journal, 312*, 71–72.

Spring, B., Pagoto, S., Kaufmann, P. G., Whitlock, E. P., Glasgow, R. E., Smith, T. W., et al. (2005). Invitation to a dialogue between researchers and clinicians about evidence-based behavioral medicine. *Annals of Behavioral Medicine, 30*, 125–137.

Wilson, M. C., Hayward, R. S., Tunis, S. R., Bass, E. B., & Guyatt, G. (1995). Users' guides to the medical literature. VIII. How to use clinical practice guidelines. B. what are the recommendations and will they help you in caring for your patients? The Evidence-Based Medicine Working Group. *JAMA: The Journal of the American Medical Association, 274*, 1630–1632.

Clinical Predictors

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

This term often refers to biomedical factors known to influence or predict health outcomes. These are taken into account in clinical practice, when estimating a patient's prognosis. Additionally, clinical predictors are considered in clinical research, when trying to test new etiological or prognostic factors, and there is a need to statistically control for known or previously established clinical predictors, which could possibly explain the role of the new tested factors. In behavior medicine, this is often the common approach, when testing the effects of a psychosocial factor on health outcomes. Often, it is crucial

to control for the effects of clinical predictors in behavior medicine, as clinical risk factors are either important in predicting prognosis, and since they may be associated with and partly explain the prognostic effects of psychosocial factors. In coronary heart disease, clinical risk factors can include left ventricular ejection fraction, number of occluded vessels, troponin levels, and comorbidities. In cancer, clinical risk factors can include performance level, tumor stage, and treatments. In surgery, clinical risk factors can include age, severity of surgery, and comorbidities.

Chida, Hamer, Wardle and Steptoe (2008), in their meta-analysis of over 160 studies, tested and found that psychosocial factors significantly predicted incidence and prognosis in cancer, and this was maintained also when statistically controlling for confounders, which included clinical predictors. One example in heart disease is the study by Denollet et al. (1996) showing that type D personality (high distress and social inhibition) predicted mortality from coronary heart disease, independent of clinical risk factors. Testing for such factors provides important strength to the claim that psychosocial factors affect health outcomes, independent of biomedical factors. This then justifies the need to consider and intervene in modifying psychosocial factors beyond targeting biomedical clinical predictors alone.

Cross-References

- ▶ Confounding Influence
- ▶ Risk Factors and Their Management

References and Readings

Chida, Y., Hamer, M., Wardle, J., & Steptoe, A. (2008). Do stress-related psychosocial factors contribute to cancer incidence and survival? *Nature Clinical Practice Oncology, 5*, 466–475.

Denollet, J., Sys, S. U., Stroobant, N., Rombouts, H., Gillebert, T. C., & Brutsaert, D. L. (1996). Personality as independent predictor of long-term mortality in patients with coronary heart disease. *Lancet, 347*(8999), 417–421.

Clinical Settings

Jeffrey Goodie
Department of Family Medicine, Uniformed Services University of the Health Sciences, Bethesda, MD, USA

Synonyms

Collaborative care; Integrated care; Primary care; Secondary care; Tertiary care

Definition

Clinical settings include primary, secondary, tertiary, and quaternary care settings. Primary care clinical settings are typically the first point of contact individuals have with the medical system. The majority of health care, including mental health care, is provided in the primary care setting. Family medicine (family practice), internal medicine, pediatric, and sometimes obstetrics and gynecological clinics are classified as primary care clinical settings. Primary care clinical settings are distinguished from other speciality care settings because no referral is needed for care and it is the source of care continuity and advocacy for the patient. The full spectrum of health care from health promotion, disease prevention, and assessment and treatment of acute and chronic medical conditions occurs in primary care (American Academy of Family Physicians, 2011; Shi & Singh, 2010).

The remaining clinical care settings are distinguished by the complexity of speciality care that can be provided (Shi & Singh, 2010). Secondary clinical settings include referral treatment facilities and specialists who do not typically have first contact with patients, but are not as specialized as those in tertiary care. Cardiology, dermatology, oncology, pulmonology, and urology clinics are examples of secondary clinical settings. Acute care provided in an emergency room and mental health care provided by

specialists (e.g., psychologists, psychiatrists), although they do not require referrals, are commonly classified as secondary care. Tertiary clinical settings use highly specialized facilities and providers to assess and treat referred patients. In tertiary clinical settings patients may receive complex surgeries (e.g., coronary artery bypass grafts) or intensive care when they are critically ill (e.g., intensive care units). Quaternary clinical settings offer unique, very highly specialized care, typically associated with regional, national, and/or academic health centers. Organ transplantation is one example of the care typically provided in a quaternary clinical setting.

Description

In the United States, the fastest growing segment of the population is older adults (i.e., 65 years and older) and the majority of their medical care will be provided in family and internal medicine primary care clinics. Concurrently, the Patient Centered Medical Home (PCMH) (American Academy of Family Physicians (AAFP), American Academy of Pediatrics (AAP), American College of Physicians (ACP), & American Osteopathic Association (AOA), 2007) concept and the National Center for Quality Assurance's accreditation process of the PCMH are reshaping the primary care clinical setting. The purpose of these efforts has been to facilitate the relationship between patients and their personal physicians. A key principle of the PCMH is the focus on the "whole person" and the biopsychosocial preventive care, acute care, chronic care, and end of life care needs of individuals at all stages of their lives.

Behavioral medicine is practiced across all clinical settings. Specialists in behavioral medicine commonly work in secondary (e.g., cardiology, chronic pain, oncology, and sleep) and tertiary clinical settings. In these clinics the behavioral medicine specialist works as part of a multidisciplinary, interdisciplinary, or transdisciplinary team to research, assess, and/or treat the biopsychosocial needs of patients. For example, behavioral medicine specialists may work with patients recovering from coronary artery bypass surgeries, patients diagnosed with cancer, or diabetes. Behavioral medicine specialists may conduct research, teach classes, or provide individual treatment related to managing the biopsychosocial factors (e.g., improving medication adherence, increasing social support, smoking cessation, stress management, weight management) associated with effective disease management. Sometimes specialized clinics are formed, such as a chronic pain or sleep clinic, where behavioral medicine specialists work with other secondary and tertiary providers to assess and treat the specific physiological, cognitive, and behavioral factors contributing to chronic pain and sleep disruption.

To meet the complex, chronic healthcare needs of the aging population, researchers and providers are continuing to focus on behavioral medicine in secondary and tertiary care settings. Increasingly behavioral medicine specialists are also collaborating with and integrating into the primary care clinical setting to assist the primary care team in meeting the complex health care needs of the elderly in a manner that brings evidence-based behavioral medicine assessment intervention to where the bulk of the elderly receive care. There are a variety of ways to describe how behavioral medicine services are integrated into primary care including co-location and embedding (American Academy of Family Physicians (AAFP), American Academy of Pediatrics (AAP), American College of Physicians (ACP), & American Osteopathic Association (AOA), 2007). Co-location may simply mean that the behavioral medicine services are offered in the same physical structure as the primary care clinic, but assessments and care are consistent with the standard of care typically followed by the behavioral medicine specialist (e.g., a psychologist seeing patients for 50-min hours) and maintaining separate records. An embedded behavioral medicine specialist is a primary care team member who follows the standard of care within primary

care (e.g., 15–30 min appointments) and documents all care within the primary care medical record. The Primary Care Behavioral Health model (Hunter & Goodie, 2010; Robinson & Reiter, 2007) is one of the most widely used examples of an embedded service. In contrast to the Primary Care Behavioral Health model is the Care Management model, which uses a specialist, often a nurse, to assist with the education and coordination of care of patients. The care manager helps to ensure that patients are getting the services they need from the medical system. Some clinics are blending Primary Care Behavioral Health and Care Management models to optimize the benefits of both care models.

Cross-References

- ▶ Primary Care
- ▶ Primary Care Providers

References and Readings

American Academy of Family Physicians. (2011). *Primary care*. Retrieved from http://www.aafp.org/online/en/home/policy/policies/p/primarycare.html#Parsys0002

American Academy of Family Physicians (AAFP), American Academy of Pediatrics (AAP), American College of Physicians (ACP), & American Osteopathic Association (AOA). (2007). *Joint principles of the patient centered medical home*. Retrieved from http://www.pcpcc.net/joint-principles

Hunter, C. L., & Goodie, J. L. (2010). Operational and clinical components for integrated-collaborative behavioral healthcare in the patient-centered medical home. *Family, Systems, and Health, 28*, 308–321.

Robinson, P., & Reiter, J. (2007). *Behavioral consultation and primary care: A guide to integrating services*. New York: Springer.

Shi, L., & Singh, D. A. (2010). *Essentials of the U.S. health care system*. Sudbury, MA: Jones and Bartlett.

Clinical Study Design

- ▶ Clinical Trial

Clinical Trial

Amy Jo Marcano-Reik
Department of Bioethics, Cleveland Clinic, Cleveland, OH, USA
Center for Genetic Research Ethics and Law, Case Western Reserve University, Cleveland, OH, USA

Synonyms

Clinical study design; Evidence-based medicine; Observational designs; Observational studies; Observational study; Randomized controlled trial

Definition

A clinical trial is a procedure in behavioral and biomedical research that is conducted to investigate potential treatments and effects of medical interventions. The public and US National Institutes of Health (NIH) service site, www.ClinicalTrials.gov, provides updated information on clinical trials regarding background and history, availability, results and outcomes, and links to other useful resources. Clinical trials may be designed to examine the effects of certain medications (e.g., different types of drugs or doses of drugs; Kahn et al., 2008, The Lancet) or behavioral interventions (e.g., a smoking cessation program; Moller, Villebro, Pedersen, & Tønnesen, 2002, The Lancet). There are many protocols and regulatory measures in place that must be adhered to for a clinical trial to be established. Once the clinical trial has been approved, researchers recruit healthy volunteers and/or patients to participate in the study. Patients may receive some benefit from the trial, such as access to a new medication; however, there are clinical trials where the patient/volunteer does not gain direct benefit from participating, such as serving in the control group (i.e., the placebo) or participating in a trial that includes a long-term design in which the treatments will not be available in the near/foreseeable future. These aspects will be different across clinical trials as the type, size, purpose, length, and location of trials will vary.

Cross-References

- ▶ Clinical Decision-Making
- ▶ Clinical Practice Guidelines
- ▶ Clinical Settings
- ▶ Medical Outcomes Study
- ▶ Randomized Clinical Trial
- ▶ Randomized Controlled Trial

References and Readings

Appel, L. J., Moore, T. J., Obarzanek, E., Vollmer, W. M., Svetkey, L. P., Sacks, F. M., et al. (1997). A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. *New England Journal of Medicine, 336*(16), 1117–1124.

Figueiredo, J. C., Grau, M. V., Haile, R. W., Sandler, R. S., Summers, R. W., Bresalier, R. S., et al. (2009). Folic acid and risk of prostate cancer: Results from a randomized clinical trial. *Journal of the National Cancer Institute, 101*(6), 432–435.

Kahn, R. S., Fleischhacker, W. W., Boter, H., Davidson, M., Vergouwe, Y., Keet, I. P., et al. (2008). Effectiveness of antipsychotic drugs in first-episode schizophrenia and schizophreniform disorder: An open randomised clinical trial. *The Lancet, 371*(9618), 1085–1097.

Moller, A. M., Villebro, N., Pedersen, T., & Tønnesen, H. (2002). Effect of preoperative smoking intervention on postoperative complications: A randomised clinical trial. *The Lancet, 359*(9301), 114–117.

Saposnik, G., Saposnik, G., Mamdani, M., Bayley, M., Thorpe, K. E., Hall, J., et al. (2010). Effectiveness of virtual reality exercises in stroke rehabilitation (EVREST): Rationale, design, and protocol of a pilot randomized clinical trial assessing the Wii Gaming System. *International Journal of Stroke, 5*(1), 47–51.

www.clinicaltrials.gov

Clusters

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

A cluster is a term used in environmental epidemiology. A disease cluster can be defined as "an unusual aggregation, in time or space or both, of occurrences of a disease" (Hertz-Picciotto, 2008). This means that an assessment of "usual" must occur. Usual rates of the disease can be determined from the distribution of occurrences in the same location in other time periods, in one or more similar locations at the same time period, or in larger areas than the specific locale of interest.

The theory of random sampling means that, at times, "chance clusters" will occur. Therefore, this possibility must be borne in mind when starting to investigate a particular cluster phenomenon. Consider the example of clusters of cancer in neighborhoods or small areas. It is of considerable importance to assess whether there is likely to be a specific environmental influence that is causing the cluster. If it is indeed likely, concerted efforts to identify the influence can be planned. However, if it appears particularly unlikely, such investigation (and the necessary resources to complete it) may not be advisable immediately.

Jekel, Katz, Elmore, and Wild (2007) discussed an instructive example concerning cancer. If the types of cancer in an identified cluster vary considerably, and are of the more common types (e.g., lung, breast, colon, prostate), it is probably the case that there is not a specific environmental hazard in the immediate locale. In contrast, if most of the cases in the cluster are of only one or a small number of cancers (especially leukemia, or brain or thyroid cancer), a more intensive investigation may be appropriate.

Reports to local, state, and federal agencies of perceived clusters are made frequently by concerned individuals or groups, physicians, and other health-care professionals. A balanced approach must be taken that balances public well-being with the acknowledgment that the majority of these reports do not lead to the identification of a common causal exposure. It is typically difficult to draw a conclusion from a single cluster, even one in which the aggregation of a disease seems particularly unusual.

Cross-References

- ▶ Cancer, Prostate
- ▶ Cancer, Testicular

References and Readings

Hertz-Picciotto, I. (2008). Environmental epidemiology. In K. J. Rothman, S. Grenland, & T. L. Lash (Eds.), *Modern epidemiology* (3rd ed., pp. 598–619). Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins.

Jekel, J. F., Katz, D. L., Elmore, J. G., & Wild, D. M. G. (2007). *Epidemiology, biostatistics, and preventive medicine* (3rd ed.). Philadelphia: Saunders/Elsevier.

Coagulation of Blood

Koji Miyazaki
Department of Hematology, Kitasato University School of Medicine, Sagamihara, Kanagawa, Japan

Synonyms

Hemostasis; Thrombosis

Definition

Normal Hemostasis

Hemostasis is a complex and highly regulated physiological process that maintains a balance between the liquid state of blood within the vasculature and the induction of blood clot formation following injury.

As such, it involves multisystem interactions between components of the vessel wall, blood cells (mainly platelets), and plasma proteins (Colman et al., 2005; Kaushansky et al., 2010).

The following events are involved in the hemostatic process:

1. Vasoconstriction: When the blood vessel ruptures, the wall of the vessel immediately contracts to reduce blood flow and thereby prevent blood loss.
2. Platelet activation: Platelets adhere to the vessel injuries via von Willebrand factor and aggregate with fibrinogen to form platelet plugs (primary hemostasis).
3. Blood coagulation: Clot formation occurs as a result of coagulation that is mediated by blood-clotting factors. Blood-clotting factors are inactive forms of proteolytic enzymes. When converted to active forms, they trigger a cascade of reactions that comprise the clotting process. At the initiation of coagulation, small amounts of thrombin are generated via FXa formation by the TF:FVIIa complex ("extrinsic pathway"). Large amounts of thrombin generation ("burst") follow; this process is dependent on FXa formation via FIXa- and FVIIIa-mediated complexes on an activated platelet surface. Generated thrombin converts fibrinogen to insoluble fibrin, which forms a meshwork cross-linked by factor XIIIa. The fibrin fibers subsequently enclose platelets, erythrocytes, leukocytes, and other plasma proteins to form blood clots (secondary hemostasis).
4. Fibrinolysis: The blood clot is dissolved when healing is complete, thereby assuring long-term vascular patency.

Disturbances of Blood Coagulation

The human hemostatic system is dependent on a delicate equilibrium between procoagulant and anticoagulant factors that interact with each other to ensure effective hemostasis at the sites of vascular injury. The procoagulant forces include platelet adhesion, aggregation, and fibrin clot formation, while the anticoagulant forces include the natural inhibitors of coagulation and fibrinolysis.

Any disruption in the balance between clot formation and clot dissolution can result in either thrombosis (due to hypercoagulation) or hemorrhage (due to hypocoagulation) (Colman et al. 2005).

Congenital disorders of coagulation include those conditions in which there are deficiencies or excessive amounts of either procoagulant or anticoagulant factors, manifesting as excessive clotting or bleeding. These disorders can have a profound effect on the overall health, well-being, and quality of life of affected children. Further, thrombotic tendencies can be related to acquired risk factors, including obesity, immobilization, diabetes, and hypertension.

Cross-References

- ▶ Aspirin
- ▶ Fibrinogen
- ▶ Fibrinolysis
- ▶ Obesity: Causes and Consequences

References and Readings

Colman, R. W., Marder, V. J., Clowes, A. W., George, J. N., Goldhaber, S. Z., et al. (2005). *Hemostasis and thrombosis* (5th ed.). Philadelphia: Lippincott Williams & Wilkins.

Kaushansky, K., Lichtman, M. A., Beutler, E., Kipps, T. J., Seligsohn, U., Prchal J. T., et al. (2010). *Williams hematology* (8th ed.). Hightstown, NJ: McGraw-Hill Professional.

Coding RNA

- ▶ RNA

Coffee

- ▶ Coffee Drinking, Effects of Caffeine

Coffee Drinking, Effects of Caffeine

Astrid Nehlig
U666, INSERM, Faculty of Medicine,
University of Strasbourg, Strasbourg, France

Synonyms

Age-related cognitive decline; Alertness; Alzheimer's disease; Anxiety; Attention; Caffeine; Coffee; Cognitive abilities; Concentration; Mood; Pregnancy; Sleep; Vigilance

Definition

Coffee is the drink most consumed by adults after water. Caffeine is the psychoactive substance contained in coffee, tea, soda, cocoa, and chocolate.

Description

In this entry, we will summarize the main effects known about coffee and caffeine consumption on health based on the numerous studies published over the last 10 years. The recent studies have reported the beneficial effects of moderate doses of coffee (3–4 cups per day) on alertness, vigilance, and cognitive abilities. However, coffee/caffeine can disturb sleep and generate anxiety. Its lifelong consumption slows down age-related cognitive decline and decreases the risk for developing Parkinson or Alzheimer's disease, as well as type 2 diabetes and numerous cancers (cancers of the digestive tract, breast, endometrial, and skin in particular). Coffee has no negative influence on cardiovascular health. However, coffee/caffeine should be consumed in moderation during pregnancy. The data summarized here come from both animal preclinical and human studies and in numerous cases originate in reviews and meta-analyses of the studies published in a given area. This large wealth of data allowed the evolution of the negative vision present in most minds that coffee was not good for health.

Coffee is the drink most consumed by adults. Caffeine is the psychoactive substance contained in coffee, tea, soda, cocoa, and chocolate (Table 1). It is also found in analgesic medications, energetic drinks, and over-the-counter slimming creams. The mean world consumption of caffeine, the major constituent of coffee (Table 2), is 1 mg/kg/day in adults from which about 80% come from coffee. It reaches 2.4–4.0 mg/kg/day in the USA and Canada, up to 7.0 mg/kg/day in Scandinavia. In 7–10-year-old children, caffeine consumption ranges from 0.5 to 1.8 mg/kg/day in developed countries, mainly from sodas and chocolate.

Low to moderate consumption of caffeine (50–250 mg, equivalent to a small to 2 large cups of coffee in one sitting) generates positive effects: feelings of well-being, relaxation, good mood, energy, increased alertness, and better concentration. The consumption of high to very high doses (400–800 mg, or 5–10 large cups of coffee in one sitting) leads to negative

Coffee Drinking, Effects of Caffeine, Table 1 Caffeine content of foods and drinks (Adapted from Debry (1994))

Foods and drinks	Volume or weight	Content of caffeine (mg) mean (extreme values)
Filtered coffee	150 mL	115 (60–180)
Espresso	30 mL	40 (40–60)
Instant soluble coffee	150 mL	65 (40–120)
Decaffeinated coffee	150 mL	3 (2–5)
Tea (leaves or bags)	150 mL	40 (30–45)
Iced tea	330 mL	70 (65–75)
Hot chocolate	150 mL	4 (2–7)
Regular soda	330 mL	30–48
Sugar-free soda	330 mL	26–57
Chocolate bar	30 g	20 (5–36)
Milk chocolate	30 g	6 (1–15)
Dark chocolate	30 g	60 (20–120)

Coffee Drinking, Effects of Caffeine, Table 2 Composition of medium-roasted coffee (Adapted from Debry (1994))

Constituents	Percentage of dry matter		Percentage of extraction by water at 100°C
	Arabica	Robusta	
Caffeine	1.3	2.4	75–100
Trigonelline	1.0	0.7	85–100
Minerals	4.5	4.7	90
Acids			
Chlorogenic	2.5	3.8	100
Quinic	0.8	1.0	100
Sugars			
Sucrose	0	0	100
Reducing sugars	0.3	0.3	
Polysaccharides	33	37	10
Lignin	2.0	2.0	–
Pectins	3.0	3.0	–
Proteins	10	10	15–20
Lipids	17	11	1
Caramelized products (e.g., melanoidins)	23	22.5	20–25
Volatile substances	0.1	0.1	40–80

Note that the content of caffeine in Robusta is twice as high as in Arabica

effects: nervousness, anxiety, aggressiveness, insomnia, tachycardia, and trembling. The moderate consumption of coffee and caffeine (3–4 cups/day) has no harmful effects on health (Table 3).

Caffeine absorption by the gastrointestinal tract reaches 99% in 45 min. Caffeine crosses all biological membranes, including the blood–brain barrier and brain concentration is close to plasma concentration. The half-life of caffeine ranges from 0.7 to 1.2 h in the rat and 2.5–4.5 h in humans. It is reduced by 30–50% in smokers, increased twofold by oral contraception, and considerably prolonged during the third trimester of pregnancy, as well as in the newborn and infant less than 6 months old.

Caffeine acts as an antagonist at adenosine receptors. Adenosine is a neuromodulator that regulates the release of neurotransmitters, mainly excitatory. Among the four types of adenosine receptors, A1, A2A, A2B, and A3, caffeine displays most of its biological effects by binding to A1 and A2A receptors. The antagonism at these receptors explains the stimulatory effects on caffeine on brain activity (Fredholm, Bättig, Holmén, Nehlig, & Zvartau, 1999).

Coffee/Caffeine and the Central Nervous System

Alertness and Sleep

The consumption of 1–4 cups of coffee (100–400 mg caffeine) daily increases alertness, proportionally to the quantity absorbed. This effect is particularly marked after sleep deprivation and when alertness is decreased as during the post-lunch dip, night and shift work, and regular cold.

A moderate consumption – 1–2 cups of coffee before bedtime – leads to difficulties and delays in going to sleep up to 3 h post intake. It also decreases the temporal organization of slow and REM sleep and the quality of deep sleep. The consequences are night awakenings, nightmares, difficulties to stand up, and sleepiness during the day. The effects vary and are more marked in elderly and occasional consumers. Moreover, the polymorphism of the gene coding for

Coffee Drinking, Effects of Caffeine, Table 3 Summary of the effects of coffee/caffeine on the cancer of different organs

Type of cancer	Number of studies	Effects of coffee	Doses
Colorectal	5 cohort; 15 case–control	24–60% risk reduction except in 3 cohorts	>3 cups/day
Liver	20 cohort; 11 case–control	30–55% risk reduction	From 1 to 2 cups/day dose-dependent effect
Stomach	23 studies	No effect	
Pancreas	37 studies	No effect	
Esophagus	17 studies	No effect	Risk increased in some studies because of the temperature of the drink
Upper aerodigestive tract	9 studies	39% risk reduction	4 cups/day
Breast	5 recent studies	No effect after menopause; 40% risk reduction before menopause even with increased genetic risk	4 cups/day
Ovary	11 studies	No effect	
Endometrial	5 studies	60% risk reduction	3 cups/day
Prostate	11 studies	No effect	
Kidney	26 studies	No effect	
Bladder	43 studies	No effect	<5 cups/day
		Increased risk	>5 cups/day
		Link with tap water	No dose-dependent effect
Skin	5 studies	Risk reduction if caffeine is applied topically	

the A2A adenosine receptor determines the interindividual sensitivity to the effects of caffeine on sleep (Rogers et al., 2010).

Sensory and Intellectual Abilities

A moderate consumption of coffee (1–4 cups per day) facilitates cognitive functions, while higher intake has rather negative effects on intellectual function. These effects depend on sex, age, time of the day, and whether consumption is chronic or not. Low caffeine consumption increases sensory and perceptive discrimination abilities. Attention is increased even at low levels of intake, 100 mg caffeine (1 cup of coffee), markedly in sleep-deprived subjects.

Up to 4 cups/day, coffee decreases reaction time. The effects are more prominent in suboptimal conditions, as at awakening, at night, in fatigued subjects, during long-lasting tasks, and in occasional consumers. The effects depend on dose and consumption habits. Caffeine does not directly improve learning and memory abilities. These effects seem rather indirect and linked to better concentration and capacity to focus attention (Nehlig, 2010).

Anxiety and Pain

Beyond 600 mg in one sitting, caffeine increases anxiety. The response largely differs between individuals and there is a link between the state of anxiety and two polymorphisms of the gene coding for A2A adenosine receptors (Rogers et al., 2010).

Moderate caffeine consumption reduces tension headache, migraine, dental and abdominal pain through its analgesic effects, directly via adenosine receptors and indirectly by the potentiation of the analgesic action of aspirin and ibuprofen (Nehlig, 2004).

Caffeine and Dependence

The abrupt cessation of caffeine intake may lead to moderate withdrawal symptoms but only in

about 10–20% of the population. These are mainly headaches, fatigue, lack of concentration, anxiety, irritability, and occasionally, nauseas. They start usually 12–24 h after abrupt caffeine cessation and last 2–3 days. They do not occur if caffeine consumption is reduced progressively. There is no tolerance to the central effects of caffeine.

Furthermore, caffeine does not activate the cerebral circuits of dependence, neither in humans after the consumption of 200 mg caffeine (2 cups of coffee) nor in rats at doses mimicking human levels of intake, i.e., 0.5–5.0 mg/kg (½ to 5 cups of coffee). Caffeine has rather reinforcing properties on its consumption. Doses of caffeine from tea or coffee (40–100 mg) appear sufficient to act as reinforcers (Nehlig, 2004).

Coffee/Caffeine and Cognition: Normal and Pathological Aging

Cognitive functions (reaction time, rate of perception, and treatment of information) remain stable until 60 and slow down between 60 and 80. Cognitive decline is accelerated by poor lifestyle, vascular diseases, genetic factors, oxidative stress, and inflammation.

Normal Age-Related Cognitive Decline

Lifelong caffeine consumption allows improving cognitive functions (reaction time, verbal and visuospatial memory) in elderly subjects. Some studies reported positive effects in both sexes while others only observed an effect in women. The positive effect of coffee/caffeine is most prominent in the oldest subjects, over 80. This association is not found with decaffeinated coffee, indicating the role of caffeine and is significant for consumptions as low as 2–3 cups of coffee/day. Thus, the usual consumption of coffee/caffeine over lifetime could increase the cognitive reserve of elderly subjects (Ritchie et al., 2007; Santos, Costa, Santos, Vaz-Carneiro, & Lunet, 2010).

Coffee and Alzheimer's Disease

Alzheimer's disease (AD) is the most frequent cause of dementia, leading to progressive cognitive decline. AD is characterized by elevated brain levels of b-amyloid peptide (Ab). The mean estimated risk between coffee/caffeine consumption and the development of AD is reduced by 23% for consumers compared to nonconsumers. The lowest risk to develop AD is found in consumers of 3–5 cups of coffee daily. The confirmation of the reduction of the risk of AD by coffee/caffeine consumption still needs prospective studies including more cases (Santos et al., 2010).

In transgenic mice developing AD, the chronic addition of caffeine to drinking water at a dose equivalent to 5 cups of coffee daily improves learning and memory, and reduces the concentrations of Ab peptide in hippocampus, the cerebral region that controls memory. Moreover, caffeine drinking in aged mice with AD allows reversing the working memory deficit and reducing cerebral Ab peptide concentration (Arendash & Cao, 2010).

Caffeine and Parkinson's Disease

The consumption of coffee and caffeine reduces the relative risk (RR) to develop Parkinson's disease (PD). There is a global 25% decreased risk of developing PD in coffee/caffeine consumers versus nonconsumers with risk reductions up to 80% for the intake of 4 cups of coffee daily. The preventive effect is linked to caffeine since regular coffee, tea, and caffeine decrease the risk while decaffeinated coffee does not (Costa, Lunet, Santos, Santos, & Vaz-Carneiro, 2010).

In women, data are less clear. In those not taking hormonal therapy, coffee is as preventive as in men. In women taking hormones, caffeine is preventive in low consumers while the risk is increased fourfold in those drinking 6 cups of coffee or more daily compared to nonconsumers (Ascherio et al., 2003). These differences could be linked to the polymorphism of the gene coding for one enzyme of caffeine metabolism (CYP1A2 rs762551) and to an interaction between caffeine and some forms of estrogen receptors (Palacios et al., 2010).

The mechanism involved in the preventive effect of caffeine in PD is its antagonism at A2A adenosine receptors. Caffeine improves parkinsonian symptoms and potentiates the effects of L-dopa, the classical treatment of PD.

Coffee and the Cardiovascular System

Coffee has negative effects on some biological markers of risk of coronary heart disease (CHD). Paradoxically, a high coffee consumption does not increase the risk of CHD. A recent meta-analysis of 21 prospective cohort studies showed that compared to low consumption (<1 cup/day in the USA and <2 cups/day in Europe), the combined relative risk (RR) of CHD for moderate coffee consumption (3–5 cups daily) is significantly reduced by 18% in women and 13% in men (Wu et al., 2009).

Likewise, in large populations with a long follow-up there is no influence of coffee (less than 5 cups/day) on the risk of heart failure (RR 0.87 for 2 cups/day) and RR 0.89–0.94 for all other levels (at least 3 cups/day) compared to men consuming less than 1 cup/day, confirming the lack of effect of moderate coffee consumption on heart failure (Ahmed, Levitan, Wolk, & Mittleman, 2009).

Furthermore, the consumption of coffee does not increase the risk of atrial fibrillation or flutter whatever be the dose. Even consumers of 1–3 cups or more than 4 cups of coffee daily reduce their risk of arrhythmias by 7 or 18%, respectively, compared to nonconsumers (Klatsky et al., 2010).

Coffee intake increases systolic and diastolic blood pressure by 1.2 and 0.5 mmHg, respectively. At an equivalent dosage, caffeine intake has a more marked hypertensive effect (4.2 and 2.4 mmHg, respectively). However, coffee is not considered a risk factor for arterial hypertension. Boiled coffee has the strongest effect, followed by filtered and instant coffee; decaffeinated coffee increases systolic blood pressure by 0.9 mmHg and decreases diastolic pressure by 0.15 mmHg (Noordzij et al., 2005).

Filtered, instant coffee, and espresso do not significantly modify lipid metabolism while unfiltered boiled coffee increases total cholesterol, mainly the low-density lipoproteins and triglycerides, and should be avoided (Thelle, 2005).

In conclusion, there is no apparent cardiovascular risk linked to coffee consumption, except possibly in some patients at risk that should also stop smoking, increase physical exercise, and improve their diet.

Coffee and Cancer

Cancers of the Digestive Tract

Lifelong consumption of coffee reduces the risk of developing liver cancer by 38 à 59% compared to nonconsumers. The underlying mechanisms remain to be clarified (Arab, 2010; Cadden, Partovi, & Yoshida, 2007; Nkondjock, 2009).

The risk of colorectal cancer is reduced by 17% in coffee consumers and up to 30% in highest consumers. This protection linked to coffee seems to involve the anticarcinogenic properties of the diterpenes and antioxidants of coffee, the stimulation of the secretion of biliary acids and neutral sterols in the colon, and the stimulation of colon motility (Galeone et al., 2010).

There is no association between coffee consumption and the risk of developing stomach or pancreas cancer. There is no evidence to support a harmful effect of coffee consumption on prostate cancer risk. Caffeine intake does not change the risk of esophagus or larynx cancer and reduces the risk of oral cavity or pharynx cancer by 39% for the consumption of 4 cups of coffee/day (Turati et al., 2011).

Breast, Ovary, and Endometrial Cancer

In postmenopausal women, there is usually no relation between caffeine/coffee intake and breast cancer. During premenopause, the risk reduction reaches 50% in women consuming at least 4 cups coffee daily compared to low consumers (1–2 cups/day). Also, in premenopausal women that carry the *BRCA1* or *BRCA2* mutation, which increases the risk of breast cancer, the risk is reduced by 25–70% by a consumption of 4–6 cups of coffee daily. This beneficial effect is limited to caffeinated coffee (Arab, 2010).

While there is no relation between coffee/caffeine intake and ovary cancer, coffee consumption of at least 3 cups daily reduces the risk of developing endometrial cancer by 60% (Arab, 2010).

Prostate, Kidney, Bladder, and Skin Cancer

Prostate cancer and kidney cancer are not influenced by the duration or quantity of coffee consumed (Arab, 2010; Park et al., 2010).

The most recent data on bladder cancer report a lack of association in women and 26% increased risk in men consuming coffee. However, a critical risk factor is linked to the type of water used to prepare coffee. Chlorinated tap water increases bladder cancer while mineral water does not. The results of most epidemiological studies allow now excluding a strong relation between coffee and bladder cancer. The major risk factors are smoking and other dietary factors (Arab, 2010; Pelluci et al., 2010).

In mice, caffeine added to drinking water or topically destroys skin cells damaged by UVB irradiation. Caffeine also doubles the mortality of human skin cells damaged by UVB, and hence could decrease the risk of skin cancer. The underlying molecular mechanism is similar in both species and leads to the hypothesis that caffeine applied topically could potentially protect human skin against the harmful effect of UVB (Heffernan et al., 2009; Lu et al., 2008).

Coffee and Type 2 diabetes

Since 2002, over 20 studies devoted to the relation between coffee consumption and the risk of developing type 2 diabetes reported a largely reduced risk linked to frequent coffee intake across diverse populations. It is similar in men and women, obese and nonobese subjects. Most studies suggest a dose–response curve with larger risk reductions for high coffee intake. In general, the consumption of at least 4 cups daily is associated to a 30–40% decreased risk of type 2 diabetes compared to nonconsumers. For lower intakes, the risk decreases by 7% for each additional coffee cup. This inverse association is observed with caffeinated and decaffeinated coffee, with or without sugar but not with caffeine alone.

Antioxidants from coffee, such as chlorogenic and quinic acids, are potential candidates for this preventive effect since they can act as regulators of carbohydrate metabolism (Huxley et al., 2009; Pimentel, Zemdegs, Theodoro, & Mota, 2009; van Dam et al., 2008).

Caffeine, Fertility, Pregnancy, Fetal and Neonatal Growth

The effects of coffee ingestion on various parameters of reproduction, pregnancy, and fetal development were reviewed recently (Peck, Leviton, & Cowan, 2010).

Effects on Fertility

In natural pregnancies, there is no link between caffeine consumption and reduction of fertility. Likewise, caffeine does not influence the number of ovocytes collected and fertilized, the number of embryos transferred and successfully reaching term in in vitro fecundations. For male fertility, there is no association between caffeine intake and the number, mobility, morphology, DNA status of spermatozoids, and the onset of pregnancy (Peck et al., 2010).

Effects on the Course of Pregnancy

Caffeine ingested by the mother is very rapidly absorbed, crosses the placental barrier, and distributes in all fetal tissues, including the central nervous system. The half-life of caffeine is dramatically increased in the fetus (over 100 h) deprived of the enzymatic equipment necessary for caffeine catabolism.

Most studies did not find any association between a daily caffeine intake lower than 300 mg (3 cups of coffee) and the risk of miscarriage. Moreover, when accounting for the severity of nauseas that often lead to a reduction in coffee/caffeine consumption, the RR for miscarriages drops from 1.5 to 1.7 for a daily caffeine consumption of 300–500 mg to 1.0–1.1. Recently, a RR of 2.2 for miscarriages was found at a caffeine intake higher than 200 mg/day. However, this study did not carefully control for confounding factors such as degree of smoking and duration of the nausea period. By caution, several associations advised women who wish to start a pregnancy to limit their caffeine intake to quantities lower than 200 mg/day, while others maintained the earlier limit of 300 mg/day.

The vast majority of studies did not find any association between caffeine and fetal growth whatever the dose. After adjustment for smoking

and alcohol, a few studies observed fetal growth retardation for caffeine intake ranging from 300 to 800 mg/day. Fetal growth is more sensitive to caffeine during the first than during the third trimester of pregnancy and intrauterine growth retardation is only significant over 600 mg/day caffeine. There is no consistent report of an association between total exposure to caffeine and the risk of early (≤34 gestational weeks) or late premature delivery (35–37 weeks) (Peck et al., 2010).

Animal data showed dose-dependent teratogenic effect of caffeine, only at very high doses, over 80 mg/kg (60–80 cups of coffee in one sitting). In humans, no study reported any increase in the incidence of congenital malformations in babies born from women consuming large quantities of caffeine (300–1,000 mg/day) during their whole pregnancy.

Effects on Postnatal Development

Caffeine enters maternal milk but has no consequence on its composition and stimulates its production. Hence, women are advised to consume their coffee after instead of before lactating.

Studies on psychomotor development are reassuring. The prenatal consumption of caffeine does not influence the Apgar score, suction reflex, weight, height, or psychomotor behavior assessed during the first year. No effect could be shown on the intellectual quotient, motor skills, or vigilance at 4 and 7 years (Nehlig & Debry, 1994).

In conclusion, a moderate caffeine consumption (lower than 200/300 mg/day), in all forms, does not seem to notably influence fertility and fetal growth. There is still some doubt for higher dosages and it is wise to recommend women that wish to start a pregnancy, or are pregnant, not to go over the reasonable limit of 200/300 mg/day caffeine.

Caffeine and Sports Activity

Most studies reported positive effects of caffeine on performance in endurance tests; the distance covered over a given time or speed in running and cycling are increased, the efficacy in final sprinting is improved and the delay before sensing pain or exertion is increased. Likewise, in team sports like rugby, soccer, and field hockey that alternate prolonged activity with bouts of intense activity, caffeine supplementation provides beneficial effects. Caffeine is also beneficial in long-distance swimming, rowing, and middle and distance running races. In brief, physical exercise involving strength and power such as lifts, throws, and sprints the effects of caffeine are less clear and variable. Women also benefit from caffeine in sports activities ranging from recreational activities to rowing competitions, mainly when trained and moderately active (Burke, 2008).

The effective dose of caffeine depends on the level of training, habituation to caffeine, and type of exercise. Usually, the efficacy of caffeine is optimal at doses of 1.5–4.5 mg/kg in nonconsumers, 3–6 mg/kg in moderate consumers, and 6.5–9.5 mg/kg in high consumers. The ergogenic effects of caffeine are more variable when caffeine is absorbed in a drink like coffee compared to the anhydrous form (capsules or tablets) (Burke, 2008; Astorino & Robertson, 2010).

The effects of caffeine on muscle metabolism are still unclear. Caffeine was suggested to mobilize fatty acids from adipose tissue to spare muscle glycogen. In reality, it seems that caffeine has rather a central effect central on fatigue or facilitates muscle function. Caffeine co-ingested with carbohydrates can enhance their absorption and oxidation during exercise. In endurance cycling, golf, and team sports, performance is more largely improved by caffeine + carbohydrates than by either constituent given alone. Caffeine reduces also pain in caffeine consumers as found in cycling, leg and arm muscle training, and other endurance activities (Goldstein et al., 2010).

Conclusion

The data presented here reflect a large number of studies performed over the last decade on coffee and health. This wealth of data allowed the evolution from the negative idea that coffee/caffeine could not be good for health because the consumer was enjoying these drinks too much. It is now widely accepted that

the moderate consumption of caffeine (3–4 cups coffee daily) in the context of a balanced diet has no negative impact on human health. In fact, on the basis of the data on normal cognitive decline, Parkinson's and Alzheimer's disease, type 2 diabetes, and cancer, the consumption of coffee appears even beneficial for human health.

Cross-References

- ▶ Aging
- ▶ Alzheimer's Disease
- ▶ Antioxidant
- ▶ Anxiety Disorder
- ▶ Arrhythmia
- ▶ Aspirin
- ▶ Atrial Fibrillation
- ▶ BRCA1 and BRCA2
- ▶ Breast Cancer
- ▶ Cancer and Diet
- ▶ Cancer, Bladder
- ▶ Cancer, Colorectal
- ▶ Cancer, Ovarian
- ▶ Cancer, Prostate
- ▶ Cancer, Types of
- ▶ Cardiovascular Disease
- ▶ Central Nervous System
- ▶ Cognitive Function
- ▶ Cognitive Impairment
- ▶ Coronary Heart Disease
- ▶ Dementia
- ▶ Diabetes
- ▶ Diastolic Blood Pressure (DBP)
- ▶ Elderly
- ▶ Estrogen
- ▶ Gender Differences
- ▶ Genetic Polymorphisms
- ▶ Heart Failure
- ▶ Hormone Treatment
- ▶ Hypertension
- ▶ Migraine Headache
- ▶ Neurotransmitter
- ▶ Pain Management/Control
- ▶ Parkinson's Disease
- ▶ Physical Activity
- ▶ Relative Risk
- ▶ REM Sleep
- ▶ Reproductive Health
- ▶ Risk Ratio
- ▶ Sex Differences
- ▶ Sleep
- ▶ Sleep Quality
- ▶ Slow-Wave Sleep
- ▶ Systolic Blood Pressure (SBP)
- ▶ Tachycardia
- ▶ Type 2 Diabetes mellitus

References and Readings

Ahmed, H. N., Levitan, E. B., Wolk, A., & Mittleman, M. A. (2009). Coffee consumption and risk of heart failure in men: An analysis from the Cohort of Swedish Men. *American Heart Journal, 158*, 667–672.

Arab, L. (2010). Epidemiologic evidence on coffee and cancer. *Nutrition and Cancer, 62*, 271–283.

Cadden, I. S., Partovi, N., & Yoshida, E. M. (2007). Review article: Possible beneficial effects of coffee on liver disease and function. *Alimentary Pharmacology & Therapeutics, 26*, 1–8.

Costa, J., Lunet, N., Santos, C., Santos, J., & Vaz-Carneiro, A. (2010). Caffeine exposure and the risk of Parkinson's disease: A systematic review and meta-analysis of observational studies. *Journal of Alzheimer's Disease, 20*(Suppl. 1), S221–S238.

Fredholm, B. B., Bättig, K., Holmén, J., Nehlig, A., & Zvartau, E. E. (1999). Actions of caffeine in the brain with special reference to factors that contribute to its widespread use. *Pharmacological Reviews, 51*, 83–133.

Nehlig, A. (2010). Is caffeine a cognitive enhancer? *Journal of Alzheimer's Disease, 20*(Suppl. 1), S85–S94.

Nkondjock, A. (2009). Coffee consumption and the risk of cancer: An overview. *Cancer Letters, 277*, 121–125.

Noordzij, M., Uiterwaal, C. S., Arends, L. R., Kok, F. J., Grobbee, D. E., & Geleijnse, J. M. (2005). Blood pressure response to chronic intake coffee and caffeine: A meta-analysis of randomized controlled trials. *Journal of Hypertension, 23*, 921–928.

Palacios, N., Weisskopf, M., Simon, K., Gao, X., Schwarzschild, M., & Ascherio, A. (2010). Polymorphisms of caffeine metabolism and estrogen receptor genes and risk of Parkinson's disease in men and women. *Parkinsonism & Related Disorders, 16*, 370–375.

Park, C. H., Myung, S. K., Kim, T. Y., Seo, H. G., Jeon, Y. J., Kim, Y., et al. (2010). Coffee consumption and risk of prostate cancer: A meta-analysis of epidemiological studies. *BJU International, 106*, 762–769.

Peck, J. D., Leviton, A., & Cowan, L. D. (2010). A review of the epidemiologic evidence concerning the reproductive health effects of caffeine

consumption: A 2000–2009 update. *Food and Chemical Toxicology, 48*, 2549–2576.
Pimentel, G. D., Zemdegs, J. C., Theodoro, J. A., & Mota, J. F. (2009). Does long-term coffee intake reduce type 2 diabetes mellitus risk? *Diabetology and Metabolic Syndrome, 1*, 6.
Santos, C., Costa, J., Santos, J., Vaz-Carneiro, A., & Lunet, N. (2010). Caffeine intake and dementia: Systematic review and meta-analysis. *Journal of Alzheimers Disease, 20*(Suppl. 1), S187–S204.
Wu, J. N., Ho, S. C., Zhou, C., Ling, W. H., Chen, W. Q., Wang, C. L., et al. (2009). Coffee consumption and risk of coronary heart diseases: A meta-analysis of 21 prospective cohort studies. *International Journal of Cardiology, 137*, 216–225.

Cognition

▶ Cognitive Factors
▶ Cognitive Function

Cognitions

Julia Allan
School of Medicine and Dentistry, University of Aberdeen, Foresterhill, Aberdeen, Scotland, UK

Synonyms

Ideas; Thoughts

Definition

Cognitions are internal mental representations best characterized as thoughts and ideas. Cognitions result from, and are involved in, multiple mental processes and operations including perception, reasoning, memory, intuition, judgment, and decision making.

As internal mental states, cognitions are not directly observable but are still amenable to study using the scientific method. Cognitions can be subjectively elicited on questioning or experimentally measured using reaction times, psychophysical responses, or real-time neuroimaging techniques to infer internal processing.

As cognitions play a fundamental role in determining behavior, the study of cognitive factors facilitates a better understanding of processes and outcomes in health, health behavior, illness, and disability. Examples of cognitions with particular relevance for behavioral medicine include illness perceptions (Leventhal, Diefenbach, & Leventhal, 1992); biases and distortions in decision making (Kahneman & Tversky, 1979); attitudes, beliefs, and perceptions of control (Ajzen, 1991); and the executive functions (Williams & Thayer, 2009).

Cross-References

▶ Beliefs
▶ Cognitive Factors
▶ Cognitive Function
▶ Cognitive Impairment
▶ Cognitive Mediators
▶ Cognitive Strategies

References and Readings

Ajzen, I. (1991). The theory of planned behaviour. *Organizational Behavior and Human Decision Processes, 50*, 179–211.
Conner, M., & Norman, P. (2005). *Predicting health behaviour: Research and practice with social cognition models* (2nd ed.). Buckingham: Open University Press.
Eysenck, M. W., & Keane, M. T. (2010). *Cognitive Psychology: A student's handbook* (6th ed.). London: Psychology Press.
Kahneman, D., & Tversky, A. (1979). Prospect theory: An analysis of decision under risk. *Econometrica, 47*, 263–292.
Leventhal, H., Diefenbach, M., & Leventhal, E. A. (1992). Illness cognitions: Using common sense to understand treatment adherence and affect cognition interactions. *Cognitive Therapy and Research, 16*, 143–163.
Williams, P. G., & Thayer, J. F. (2009). Executive functioning and health: An introduction to the special series. *Annals of Behavioral Medicine, 37*, 101–105.

Cognitive Abilities

▶ Coffee Drinking, Effects of Caffeine

Cognitive Appraisal

Tavis S. Campbell, Jillian A. Johnson and Kristin A. Zernicke
Department of Psychology, University of Calgary, Calgary, AB, Canada

Synonyms

Lazarus theory; Transactional model

Definition

The concept of cognitive appraisal was advanced in 1966 by psychologist Richard Lazarus in the book *Psychological Stress and Coping Process*. According to this theory, ▶ stress is perceived as the imbalance between the demands placed on the individual and the individual's resources to cope (Lazarus & Folkman, 1984). Lazarus argued that the experience of stress differs significantly between individuals depending on how they interpret an event and the outcome of a specific sequence of thinking patterns, called appraisals (Lazarus, 1991).

Cognitive appraisal refers to the personal interpretation of a situation that ultimately influences the extent to which the situation is perceived as stressful. It is the process of assessing (a) whether a situation or event threatens our well-being, (b) whether there are sufficient personal resources available for coping with the demand of the situation, and (c) whether our strategy for dealing with the situation is effective (Lazarus, 1991). This process can then be further subdivided into three categories: primary appraisal, secondary appraisal, and reappraisal:

- *Primary appraisal* refers to the initial evaluation of the situation, deemed as benign positive (positive), threatening (negative), or irrelevant (neutral). If the situation is appraised as being irrelevant or benign positive, no heightened physiological arousal occurs and the situation will not be perceived as stressful. If the situation is appraised as negative, the individual will make a secondary appraisal in regard to harm (harm-loss), threat, or challenge.
- *Secondary appraisal* refers to the evaluation of an individual's ability or resources to cope with a specific situation. Secondary appraisal interacts with primary appraisal to determine emotional reaction to a situation. A harm (harm-loss) appraisal is the assessment that damage has occurred as a result of the situation and the necessary resources to effectively cope with the situation may not be available. Threat appraisals occur when it is anticipated that the situation may result in loss or harm in the future and the resources to effectively cope with the situation may not be available. A challenge is perceived when a situation is demanding but ultimately can be overcome, resulting in the individual benefiting from the situation. Both harm and threat appraisals result in the situation being deemed as stressful, whereas a challenge appraisal does not.
- Reappraisal is the continuous reevaluation of a situation based on the availability of new information. This step of reappraisal takes place throughout the entire process and can change the way an individual perceives a situation.

References and Readings

Folkman, S., Lazarus, R. S., Dunkel-Schetter, C., DeLongis, A., & Gruen, J. (1986). Dynamics of a stressful encounter: Cognitive appraisal, coping and encounter outcomes. *Journal of Personality and Social Psychology, 50*(5), 992–1003.

Folkman, S., Lazarus, R. S., Gruen, J., & DeLongis, A. (1986). Appraisal, coping, health status, and psychological symptoms. *Journal of Personality and Social Psychology, 50*(3), 571–579.

Lazarus, R. S. (1991). *Emotion and adaptation*. New York: Oxford University Press.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.

Cognitive Behavior Therapy

▶ Cognitive Behavioral Therapy (CBT)

Cognitive Behavioral Therapy (CBT)

Lara Traeger
Behavioral Medicine Service, Massachusetts General Hospital/Harvard Medical School, Boston, MA, USA

Synonyms

Cognitive behavior therapy

Definition

Cognitive behavioral therapy is a classification of psychotherapies which integrate cognitive and behavioral theories and methods. CBT approaches share fundamental assumptions that cognitions mediate situational responses and that changes in cognitive activity can affect therapeutic changes in emotions and behaviors.

Description

Brief History of CBT

CBT interventions represent an integration of behavioral and cognitive theories and methods. Behavior therapy emerged in the 1950s and 1960s through research on clinical applications of classical and operant conditioning theories (e.g., systematic desensitization; Eysenck, 1966; Wolpe, 1958). Behavior therapy emphasizes the primacy of behaviors, and radical behaviorists view thoughts as a type of internal behavior. The primacy of thoughts in shaping situational responses appears in early philosophical traditions ranging from Stoicism to Buddhism (Wright et al., 2006). Formally, the cognitive underpinnings of CBT emerged in the 1960s and 1970s, largely through developments by Albert Ellis (rational emotive therapy; 1957) and Aaron T. Beck (cognitive therapy; 1963, 1964) and contributions from Alfred Adler, George Kelly, and behaviorists described above. Rational emotive therapy has been considered the first of the cognitive interventions to appear; it introduced a novel, directive approach to challenging patients' irrational beliefs. Beck's cognitive therapy also emphasized a primary role of cognitions in psychiatric problems, and he formally described the maladaptive cognitive biases associated with depression as targets for therapeutic change.

The coalescence of cognitive and behavioral therapies over the past few decades has been due to several factors, including the challenges of applying behavior theory to the complex range of human behaviors (for example, obsessional thinking), the introduction of a formalized cognitive therapy for depression, and the growing support for CBT interventions in both research and practice. Behaviorists view behavior change as the primary goal of therapy, whereas cognitive theorists view behavior strategies as means for affecting change. Yet both schools share a commitment to applying the scientific method to clinical problems and their treatments. Since the early works which focused primarily on depression and anxiety, CBT models have since been expanded to explain and treat a wide range of psychiatric disorders.

A number of membership organizations support CBT research and practice, and their histories reflect the history of CBT itself. For instance, in 1966, the Association for Advancement of Behavioral Therapies (AABT) was founded by behaviorists due to their dissatisfaction with the psychoanalytic model. The name was formally changed to the Association for Behavioral and Cognitive Therapies (ABCT) in 2005, to reflect the increasing influence of cognitive theory and methods. Similarly, the British Association for Behavioural Psychotherapy (BABP) was founded by behaviorists in 1972; its scope was broadened in 1992 when it became The British Association for Behavioural and Cognitive Psychotherapies (BABCP).

CBT Model of Clinical Symptoms

CBT emphasizes the role of individuals as active information processors. The meaning we apply to a situation shapes our emotional reactions to the situation and what we may do to cope with our emotions. Our behaviors, in turn, affect our

thought patterns and emotional responses. In other words, cognitions, emotions, and behaviors are intimately linked. These relationships are illustrated in the following scenario:

A supervisor had begun to criticize two employees, J.F. and A.B., for minor errors at work. J.F. interpreted the situation to mean that he was a poor performer and a liability to his department. This caused J.F. to feel dejected, which led him to increase his efforts to please the supervisor. J.F. began to work late at night; drink more coffee to stay awake; and consequently experience fatigue and anxiety the next day. This led him to make more errors at work, creating a self-fulfilling prophecy which strengthened his anxiety and negative beliefs about himself. Meanwhile, A.B. presumed that the supervisor was simply singling her out for criticism. This caused A.B. to feel irritated, which led her to act indifferently toward the supervisor while maintaining her current level of work performance. Her relationship with the supervisor deteriorated, reinforcing A.B.'s belief that people are generally disrespectful.

This scenario highlights that two different interpretations without clear evidence led to quite different emotional and behavioral consequences. These interpretations also could reinforce longstanding negative beliefs about the self (in the case of J.F.) or the world (in the case of A.B.). This is a key learning point for individuals during therapy. In the long term, entrenched patterns or styles of thinking and behaving can become associated with clinically significant distress. Indeed, psychiatric disorders are distinguished by distinct profiles of cognitive and behavioral bias. In his original work, Aaron T. Beck described depression as the result of negative thinking about the self, world, and future (1963, 1964). Other examples include phobias as the inaccurate perceptions of danger, and suicidality as the perception of hopelessness and deficits in problem-solving skills.

Applications of the CBT Model in Behavioral Medicine

The CBT model can be particularly useful in behavioral medicine, to capture biopsychosocial aspects of health promotion and disease management. Research evidence strongly supports links between cognitions, feelings, and health behaviors. For instance, many chronic medical conditions are associated with elevated risk for depression. Depressed individuals, in turn, have difficulties with motivation, interest, and problem solving, and are therefore less likely to practice self-care behaviors such as physical activity, healthy eating, and adherence to medical regimens. The following scenario illustrates these relationships:

S.P. had been prescribed a daily HIV medication for the past year. She did not believe that the medication did much to manage her condition. Every morning, she would dread looking at the medication bottle. It was a reminder that she was ill, and this reminder provoked other familiar thoughts that her life was over and that she would never find a romantic partner due to her HIV status. These thoughts, in turn, reminded her that she was profoundly alone. For S.P., it was easier to ignore the sight of the bottle and skip her medication dose, which she frequently did. However, the thoughts remained and often provoked painful depressed moods which decreased her motivation and energy to answer phone calls from her friends. S.P. spent most of her time at home alone, which reinforced her beliefs about being undesirable to others. Most recently, she missed her regular HIV primary care visit. It seemed too difficult to secure a ride to the clinic, and she thought, "What's the point anyway, this disease is not going away."

This scenario shows bidirectional relationships between depression and poor HIV self-care. In practice, the CBT case formulation would address how inaccurate cognitions, emotional distress, and coping behaviors are influencing each other in a perpetuating loop, which serves to maintain both depression and poor self-care. The case formulation would also help to highlight key areas for CBT intervention to break this loop. In developing the CBT treatment plan, a therapist may draw systematically from CBT strategies, including: (1) providing psychoeducation about depression, HIV, and HIV medications; (2) increasing engagement in activities

which promote enjoyment and sense of mastery; (3) challenging severe negative beliefs; and (4) problem-solving medical adherence. This approach highlights that all three domains (cognitions, emotions, and behaviors) are being addressed. Common CBT intervention elements are described further in the next section.

Common Elements of CBT Interventions

In CBT interventions, the therapist actively collaborates with the patient (i.e., "co-therapist"). They work together to identify and alter problematic patterns of thinking and behaving, and thereby help the patient manage negative emotions and improve quality of life. The therapist first collects information about the patient's presenting problems, and then shares and revises the CBT case formulation with the patient. This formulation directly informs the therapy. The therapist and patient work together to set a treatment plan and articulate goals at the outset of therapy, and to set agendas at each therapy session. During the course of CBT, the therapist may use Socratic questioning to guide patients in their own discovery of problematic patterns in their thinking and behaving. Sessions are problem oriented and typically focus on building skills which address these patterns. "Homework" assignments encourage the patient to rehearse and problem-solve the skills in real-life situations. Throughout treatment, progress is monitored using symptom inventories (for example, the Beck Depression Inventory [BDI] or the Hospital Anxiety and Depression Scale [HADS]) as well as informal feedback. Most CBT interventions are intended to be time limited; the ultimate goal is for patients to become increasingly independent in their use of the skills until the therapist is no longer needed.

The following is a sample of common CBT intervention strategies:

Psycho-education is used throughout CBT interventions. A critical component of CBT is to engage patients in understanding the CBT model, the rationale for treatment, and the therapeutic methods as applied to their clinical problems. In other examples, CBT for panic disorder includes information on physiologic activation, whereas a patient on long-acting pain medications may benefit from understanding the impact of missed or delayed medication doses.

Behavioral strategies are used to help patients break unhelpful behavior patterns such as fear avoidance or depressive inactivity. For example, exposure methods involve generating a hierarchy of situations that induce fear and avoidance, and conducting structured "experiments" which increase real-life or imaginal exposure to these situations. In behavioral activation, the patient is guided to increase activity level by generating a list of activities that promote enjoyment and sense of mastery, and then setting and monitoring daily or weekly activity goals.

Cognitive strategies are used to promote optimal thinking about difficult situations. As a primary example, cognitive restructuring is a framework for recognizing negative, inaccurate thoughts and replacing them with alternative ones that are more realistic and helpful. This may involve several steps: write down the situation; list negative thoughts that occurred during the situation; list emotions that arise when having these thoughts; identify cognitive distortions or errors that may underlie each thought; challenge each thought; and generate rational responses. The rational responses are self-statements that are used to reduce distress and view situations in a more helpful light.

Considerations for CBT in Behavioral Medicine Populations

CBT interventions have been incorporated into the American Psychiatric Association clinical practice guidelines for a wide range of psychiatric disorders. However, chronic medical conditions introduce some unique aspects to consider during CBT evaluation and delivery. Psychiatric symptoms can overlap with or mask disease symptoms and treatment side effects (for example, cancer-related fatigue, dyspnea, or uncontrolled pain), underscoring the importance of assessment and differential diagnosis for behavioral medicine patients. Also, health cognitions and emotional distress levels can be dynamic, changing over time in response to disease-related events (for example, receiving

medical test results), uncertain disease courses, or certain disease progression. For many medical conditions, disease symptoms fluctuate, influencing mobility, fatigue, and cognitive functioning. Adaptations to CBT protocols have been recommended to incorporate these factors. For instance, behavioral activation and homework assignments can be adapted so that patients modulate daily activities according to current level of energy (activity pacing). Cognitive restructuring can be supplemented with acceptance-based or problem-solving strategies when negative health cognitions reflect both realistic and unrealistic elements, and both controllable and uncontrollable stressors.

For the sample scenario of S.P., described above, a CBT intervention might proceed as follows:

The therapist worked with S.P. to generate a CBT model of her depression and problems with HIV self-care. Socratic questioning was used to help S.P. discover links between her thoughts (perceived impact of HIV on her value as a person); feelings (sadness and loneliness); and behaviors (medical non-adherence and self-isolation). The therapist and S.P. used this model to develop a treatment plan and set goals. S.P.'s main goal was to repair some of the meaningful relationships in her life. The therapist provided psycho-education about depression and HIV. S.P. began to internalize that self-care was a step toward improving relationships with others. Behavioral activation was introduced to help S.P. increase engagement in activities that she used to enjoy and that could give her opportunities to challenge her belief that others would reject her. Activities were modified on days when S.P. experienced fatigue or medication side effects. Cognitive restructuring helped S.P. develop healthier cognitions such as more neutral perceptions of HIV medications. Finally, problem solving was introduced to help S.P. organize her efforts toward increasing her adherence and enhancing her social support. While S.P. experienced setbacks, she increasingly began to recognize her tendency to make devaluing statements about herself during stressful situations, and she continued to work toward changing this pattern.

CBT Applications in Behavioral Medicine

There is growing evidence to support CBT interventions to improve health behaviors, enhance quality of life, and reduce psychological symptoms among individuals with medical comorbidities. For instance, Safren, Gonzalez and Soroudi (2008) developed a CBT intervention for depression and medical adherence (CBT-AD) in patients with chronic illness such as diabetes or HIV. Cognitive behavioral stress management (CBSM) is a group intervention developed by Antoni, Schneiderman and Ironson (2007) to improve quality of life in HIV-infected adults, which has since been adapted for cancer survivors (Penedo, Antoni, & Schneiderman, 2008). CBT strategies have also been adapted to treat or reduce disability associated with a range of specific medical concerns. Examples include nicotine dependence, obesity, chronic illness rehabilitation, hypertension, and various functional pain and fatigue conditions. For instance, CBT protocols for either nicotine dependence or obesity may include goal setting; psychoeducation; self-monitoring (recording number cigarettes or food intake per day); stimulus control (reducing contact with environmental cues that trigger smoking or overeating); and coping skills for relapse prevention.

Cross-References

- ▶ Behavior Change
- ▶ Behavioral Therapy
- ▶ Cognitions
- ▶ Cognitive Distortions
- ▶ Cognitive Restructuring
- ▶ Cognitive Strategies
- ▶ Problem Solving
- ▶ Systematic Desensitization

References and Readings

Antoni, M. H., Schneiderman, N., & Ironson, G. (2007). *Stress management for HIV: Clinical validation and intervention manual*. Mahwah, NJ: Lawrence Erlbaum Associates.

Beck, A. T. (1963). Thinking and depression. *Archives of General Psychiatry, 9*, 324–333.

Beck, A. T. (1964). Thinking and depression, II: Theory and therapy. *Archives of General Psychiatry, 10*, 561–571.
Dobson, K. S. (Ed.). (2010). *Handbook of cognitive-behavioral therapies* (3rd ed.). New York: Guilford Press.
Ellis, A. (1957). Rational psychotherapy and individual psychology. *Journal of Individual Psychology, 13*, 38–44.
Eysenck, H. J. (1966). *The effects of psychotherapy*. New York: International Science Press.
Kelly, G. (1955). *The psychology of personal constructs*. New York: WW Norton.
Moorey, S., & Greer, S. (2002). *Cognitive behaviour therapy for people with cancer*. Oxford/New York: Oxford University Press.
Penedo, F. J., Antoni, M. H., & Schneiderman, N. (2008). *Cognitive-behavioral stress management for prostate cancer recovery: Facilitator guide*. Oxford/New York: Oxford University Press.
Safren, S. A., Gonzalez, J. S., & Soroudi, N. (2008). *Coping with chronic illness: A cognitive-behavioral therapy approach for adherence and depression: Therapist guide*. Oxford/New York: Oxford University Press.
Wolpe, J. (1958). *Psychotherapy by reciprocal inhibition*. Stanford, CA: Stanford University Press.
Wright, J. H., Basco, M. R., & Thase, M. E. (2006). *Learning cognitive-behavioral therapy: an illustrated guide*. London/Washington, DC: American Psychiatric Publishing, Inc.

Cognitive Control

▶ Behavioral Inhibition

Cognitive Deficit

▶ Cognitive Impairment

Cognitive Disorder

▶ Cognitive Impairment

Cognitive Distortions

▶ Cognitive Behavioral Therapy (CBT)

Cognitive Evaluation Theory

▶ Self-determination Theory

Cognitive Factors

Eric Roy
Department of Kinesiology, University of Waterloo, Waterloo, ON, Canada

Synonyms

Cognition; Cognitive strategy; Cognitive style; Mental ability; Mental function

Definition

Cognitive factors refer to characteristics of the person that affect performance and learning. These factors serve to modulate performance such that it may improve or decline. These factors involve cognitive functions like attention, memory, and reasoning (Danili & Reid, 2006).

Cognitive factors are internal to each person and serve to modulate behavior and behavioral responses to external stimuli like stress. Performance on various activities of daily living has been found to be affected by these factors. Executive functions, for example, have been shown to predict ability to live independently in older adults such that those with poorer executive functioning are less able to live independently (Vaughn & Giovanello, 2010). Turning to behavioral responses to stress cognitive factors is known to play a role in posttraumatic stress disorder. The nature of the memory of the trauma may play a role in PTSD, that is, persistent PTSD is often associated with memories of the trauma that are poorly elaborated and not well integrated into the person's autobiographical memory (Dumore, Clark & Ehlers, 2001). More generally cognitive style may serve as an important cognitive factor. Messick (1994) refers to cognitive style as

characteristic modes of thinking, perceiving, problem solving, and remembering that may influence how a person approaches a problem or task.

Cross-References

- ▶ Cognitions
- ▶ Cognitive Function
- ▶ Cognitive Impairment
- ▶ Cognitive Strategy

References and Readings

Danili, E., & Reid, N. (2006). Cognitive factors can potentially affect pupils' test performance. *Chemistry Education Research and Practice, 7*, 64–83.

Dumore, E., Clark, D. M., & Ehlers, A. (2001). A prospective investigation of the role of cognitive factors in persistent posttraumatic stress disorder (PTSD) after physical or sexual assault. *Behavioral Research and Therapy, 39*, 1063–1084.

Messick, S. (1994). The matter of style: Manifestations of personality in cognition, learning, and teaching. *Educational Psychologist, 29*, 121–136.

Vaughn, L., & Giovanello, K. (2010). Executive function in daily living: Age related influences of executive processes on instrumental activities of daily living. *Psychology and Aging, 25*, 343–355.

Cognitive Function

Eric Roy
Department of Kinesiology, University of Waterloo, Waterloo, ON, Canada

Synonyms

Cognition; Mental ability; Mental function

Definition

Cognitive function derives from the term cognition which refers to the internal mental processes studied in a subdiscipline of psychology termed cognitive psychology. These internal mental processes underlie how people perceive, remember, speak, think, make decisions, and solve problems. Cognitive function is a general term used to describe many different functions such as memory and attention thought to be components of the mind (Benjafield, Smilek, & Kingstone, 2010).

Description

Cognitive functions are internal and are inferred from behavior using measures such as accuracy in performing a task like recalling a list of words or the time taken to find some word on a page of text. The study of cognitive functions derives from the information processing approach which argues that these functions involve operations occurring at various processing stages. The identification of these processing stages is typically based on a model of the cognitive function of interest. Using this model, a task thought to reflect the cognitive function of interest is manipulated in such a way as to place demands on the processing stages identified. If we use memory as an example, the task of recalling a list of words can be manipulated to place demands on two processing stages: encoding or putting words into memory or retrieval involving retrieving words from memory. The encoding stage is emphasized when demands are placed on just recognizing whether words presented were in the list, while the retrieval stage is emphasized when demands are placed on recalling the words from the list. The study of cognitive functions then involves the use of experimentation through manipulation of task demands. This use of the scientific method spawned the development of another subdiscipline of psychology termed cognitive science.

The study of cognitive functions involves not only identifying the processing stages but also the strategies used and the errors made. Turning again to memory function and word list recall as an example, one strategy used involves semantic clustering where the person creates groupings of words from the list based on the meaning category such as clothes or fruit. This clustering serves to improve recall of the words. With regard to errors, intrusions and false positive

errors in recalling words from the list provide insight into the integrity of memory. Intrusions are errors where the person recalls a word not on the list, while false positive errors occur when the person is read a list of words some of which were not on the recall list. A false positive error is one where the person endorses a word that was not on the list. Both of these errors indicate that the ability to discriminate in memory between words on the recall list from those not on the list is impaired.

This information on cognitive functions has been used in the development of psychological tests designed to examine cognitive functions (Hodges, 2007). These tests are administered to groups of people categorized based on factors such as age, sex, and years of education. Performance of these people is then used as normative data against which to compare performance of people who take the tests in the future. These comparisons involve determining the average and the standard deviation for each group in the normative sample. The mean and standard deviation are reference points to determine where relative to the mean a person taking the test falls. The distance the person's score falls relative to the mean is measured in standard deviation units. The number of units above or below the mean reflects the percentage of people in the normative sample who are above or below the mean. Thus, if we use the memory test as an example, a person with a score at one standard deviation unit above the mean would be at a point where 84% of people fall at or below this score. This approach to measurement termed psychometrics reveals the relative strengths of a person on various cognitive functions. This pattern of strengths is used in the subdisciplines of psychology called clinical psychology and educational psychology to direct people into education programs and work placements. The alternative to patterns of strengths is patterns of weakness in cognitive functions. Such patterns are used in a subdiscipline of psychology called clinical neuropsychology to identify cognitive impairments.

Another focus of study with regard to cognitive functions is the brain correlates of these functions. One approach called cognitive neuroscience uses functional neuroimaging and correlates patterns of brain activity to the processing stages in various cognitive functions. The other approach called clinical neuropsychology uses psychometrics alluded to above to identify patterns of impairment in cognitive functions arising from some type of brain damage and correlates these impairments with measures of brain damage using structural (e.g., MRI) and functional (e.g., fMRI) brain imaging.

Cross-References

▶ Assessment

References and Readings

Benjafield, J. G., Smilek, D., & Kingstone, A. (2010). *Cognition* (4th ed.). New York: Oxford University Press.

Hodges, J. (2007). *Cognitive assessment for clinicians*. New York: Oxford University Press.

Cognitive Impairment

Eric Roy
Department of Kinesiology, University of Waterloo, Waterloo, ON, Canada

Synonyms

Cognitive deficit; Cognitive disorder

Definition

Cognitive impairment refers to problems people have with cognitive functions such as thinking, reasoning, memory, or attention.

Description

Cognitive impairment can be present at any point in a person's lifespan (Kolb, & Whishaw, 2009).

Early in life, cognitive impairment may arise from, for example, genetic syndromes, prenatal drug and alcohol exposure, trauma, or oxygen deprivation during or after birth.

Cognitive impairment in childhood and adolescence may result from a number of conditions. Examples include malnutrition, heavy metal exposure, metabolic disorders, trauma to the brain, and side effects of drug treatments for cancer or Parkinson's disease (Ogden, 2005).

With age conditions such as traumatic brain injury, neurodegenerative disorders such as Alzheimer disease, stroke, brain tumors, and brain infections can cause cognitive impairment.

In some cases, cognitive impairment is reversible if the cause is identified and treated. For example, cognitive impairment arising from stroke due to a blockage of a blood vessel can be prevented if drugs designed to break up the blood clots are administered within hours of the formation of the clot. Similarly, cognitive impairments associated with metabolic disorders can be reversed with treatment of the disorder.

Cognitive impairment is defined as a disruption to some cognitive function such as memory (Lezak, Howieson, & Loring, 2004). Identifying a cognitive impairment requires a comparison of performance to some expected level of performance. In some cases, this expected performance is defined informally, for example, a person who is unable to remember the name of a life-long friend is thought to exhibit a cognitive impairment. In most cases, it is these cognitive impairments defined on the basis of informal expected level of performance which results in the person visiting a health-care practitioner for a more thorough investigation.

Such more thorough investigations identify cognitive impairments using more formal standards called norms which reflect expected performance on standardized tests of cognitive functions such as memory (Hebben, & Milberg, 2009). These tests are administered to groups of people categorized on factors such as gender, age, and years of education. Performance of these people forms normative data against which is compared performance of people who take the tests in the future (Strauss, 2006). These comparisons require determining the average and the standard deviation for each group in the normative sample. The mean and standard deviation are points of reference to determine where relative to the mean a person taking the test falls. The distance the person's score falls relative to the mean is measured in standard deviation units. The number of units above or below the mean reflects the percentage of people in the normative sample who are above or below the mean. Thus, if we use the memory test as an example, a person with a score at one standard deviation unit above the mean would be at a point where 84% of people in the normative sample fall at or below this score. This point is termed the 84th percentile. This approach to measurement reveals the relative strengths or weaknesses of a person on a cognitive function. A weakness is termed an impairment or deficit and reflects performance at one standard deviation unit below the mean at the 16th percentile. At this point, 84% of the people in the normative sample lie above this score.

This psychometric approach to identifying a cognitive impairment is often accompanied by a more qualitative approach where particular errors or strategies in test performance are of interest. For example, in the context of a memory impairment involving learning a list of words, the person may recall or recognize a word that was not on the list. This error reflects an impairment in discrimination in memory which provides some insight into the nature of the memory impairment.

Cross-References

- ▶ Assessment
- ▶ Brain Imaging
- ▶ Brain Injury
- ▶ CAT Scan
- ▶ Cognitive Function
- ▶ Cognitive Strategies
- ▶ Dementia
- ▶ Depression: Symptoms
- ▶ Disability
- ▶ False-Negative Error
- ▶ Neuroimaging
- ▶ Neuropsychology

- ▶ Psychometrics
- ▶ Traumatic Brain Injury

References and Readings

Hebben, N., & Milberg, W. (2009). *Essentials of neuropsychological assessment* (2nd ed.). New York: Wiley.
Kolb, B., & Whishaw, I. (2009). *Fundamentals of human neuropsychology* (6th ed.). New York: Worth Publishers.
Lezak, M., Howieson, D., & Loring, D. (2004). *Neuropsychological assessment* (4th ed.). New York: Oxford University Press.
Ogden, J. A. (2005). *Fractured minds: A case-study approach to clinical neuropsychology* (2nd ed.). New York: Oxford University Press.
Strauss, E. (2006). *A compendium of neuropsychological tests: Administration, norms and commentary* (3rd ed.). New York: Oxford University Press.

Cognitive Impairment Tests

- ▶ Screening, Cognitive

Cognitive Mediators

Linda D. Cameron[1] and Lana Jago[2]
[1]Psychological Sciences, University of California, Merced, Merced, CA, USA
[2]Department of Psychology, The University of Auckland, Auckland, New Zealand

Synonyms

Mediating cognitions

Definition

Cognitive mediators are mental processes or activities that take place between the occurrence of a stimulus and initiation of an associated response. Such processes can occur immediately following the stimulus (i.e., within microseconds), or they may be a more delayed response, taking days or weeks. These processes are delineated by mediation models of health experiences and behaviors which, in contrast to direct stimulus-response models, propose that events produce an effect on individual responses indirectly via cognitive mediators. Cognitive mediators include interpretation of information, information retrieval, judgments and evaluations, reasoning, and other mental processes. These processes may be conscious or nonconscious (i.e., automatically elicited outside of one's awareness), and they can be distinguished from affective mediators involving emotional processes.

Mediational models have been used to better understand the cognitive processes involved with a variety of health experiences, behaviors, and outcomes. Early behavioral medicine research on cognitive mediators examined their influence on pain perception and health status of individuals with medical conditions. More recently, research on cognitive mediators includes evaluations of their roles in the use of health-promoting behaviors such as smoking cessation, physical activity, dietary behavior, and behaviors for reducing skin cancer risk.

Cross-References

- ▶ Attitudes
- ▶ Beliefs
- ▶ Cognitions
- ▶ Cognitive Factors
- ▶ Cognitive Strategies
- ▶ Health Behaviors
- ▶ Health Outcomes Research
- ▶ Mediators
- ▶ Theory

References and Readings

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 51*, 1173–1182.
Gibbons, F. X., Gerrard, M., Lane, D. J., Mahler, H. I., & Kulik, J. A. (2005). Using UV photography to reduce

use of tanning booths: A test of cognitive mediation. *Health Psychology, 24*, 358–363.
Preacher, K. J., & Hayes, A. F. (2004). SPSS and SAS procedures for estimating indirect effects in simple mediation models. *Behavior Research Methods, Instruments, & Computers, 36*, 717–731.
Rucker, D. D., Preacher, K. J., Tormala, Z. L., & Petty, R. E. (2011). Mediation analysis in social psychology: Current practices and new recommendations. *Social and Personality Psychology Compass, 5*, 359–371.

Cognitive Reappraisal

► Cognitive Restructuring

Cognitive Restructuring

Lara Traeger
Behavioral Medicine Service, Massachusetts General Hospital/Harvard Medical School, Boston, MA, USA

Synonyms

Cognitive reappraisal

Definition

Cognitive restructuring is a strategy to recognize negative, inaccurate thoughts and replace them with alternative ones that are more realistic and helpful. This cognitive strategy, a key part of cognitive behavioral therapy, promotes optimal thinking about a stressful or overwhelming situation to reduce emotional distress. Cognitive restructuring may involve several steps: write down the situation; list negative thoughts that occurred during the situation; list emotions that arise when having these thoughts; identify cognitive distortions or errors that may underlie each thought; challenge each thought; and generate rational responses. The rational responses are self-statements that are used to feel better about the situation.

Cognitive restructuring may help individuals with a chronic illness to manage how the illness affects their perceptions of themselves, their relationships, and their future. For example, an individual may be experiencing persistent anxiety since his return to work following a myocardial infarction. The individual may be encouraged to identify a specific situation that is making him anxious ("My supervisor pointed out some errors in my work"); his negative thoughts ("I can't do anything right since I had my heart attack," and "I'll probably get fired"); and his resulting emotions (fear, despair). Through cognitive restructuring, the individual may work on challenging his thoughts and generating alternative responses: "I don't have any evidence that my supervisor is dissatisfied with my work in general. I have been taking care of myself since my heart attack. No one is perfect, and in the future, I can leave more time to check my work." The individual may then assess whether the rational responses help him to reduce his distress and view his situation in a more helpful light.

Cross-References

► Cognitive Behavioral Therapy (CBT)
► Cognitive Distortions
► Cognitive Strategies

References and Readings

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression*. New York: Guilford.
Clark, D. A., Beck, A. T., & Alford, B. A. (1999). *Scientific foundations of cognitive theory and therapy of depression*. New York: Wiley.

Cognitive Status Tests

► Screening, Cognitive

Cognitive Strategies

Linda D. Cameron[1] and Lana Jago[2]
[1]Psychological Sciences, University of California, Merced, Merced, CA, USA
[2]Department of Psychology, The University of Auckland, Auckland, New Zealand

Synonyms

Cognitive techniques; Mental strategies

Definition

Cognitive strategies are sets of mental processes that are consciously implemented to regulate thought processes and content in order to achieve goals or solve problems. Self-regulation theories of behavior focus on cognitive strategies as playing a critical role in guiding goal-directed behavior. Cognitive strategies are primary targets for numerous intervention approaches, including cognitive behavior therapy (CBT), mindfulness-based interventions, and acceptance and commitment therapy (ACT). Cognitive strategies include those directing attentional focus (e.g., attentional engagement or distraction), cognitive reframing or reinterpretation of distressing experiences, imagery techniques, and mental rehearsal of positive statements.

Within the health setting, pain management is one area in which cognitive strategies may be useful. Examples include distraction, where one diverts attention away from the painful stimulus and towards a non-painful alternative; imagery, such as imagining a favorite scene or other non-painful image; and redefinition, where pain cognitions related to threat or fear are replaced with constructive or nonthreatening thoughts. Cognitive strategies can also be of benefit to individuals with chronic illnesses who are experiencing psychological distress or difficulty managing their conditions. Within the context of health promotion, mental imagery techniques have been demonstrated to increase physical activity among sedentary adults.

Cross-References

- ▶ Cognitive Appraisal
- ▶ Cognitive Behavioral Therapy (CBT)
- ▶ Cognitive Factors
- ▶ Cognitive Restructuring
- ▶ Mindfulness
- ▶ Self-regulation Model

References and Readings

Chan, C. K. Y., & Cameron, L. D. (2011). Promoting physical activity with goal-oriented mental imagery: A randomized controlled trial. *Journal of Behavioral Medicine*. doi:10.1007/s10865-011-9360-6.

Hart, S. I., & Hart, T. A. (2010). The future of cognitive behavioral interventions within behavioral medicine. *Journal of Cognitive Psychotherapy, 24*, 344–353.

Kamholz, B. W., Hayes, A. M., Carver, C. S., Gulliver, S. B., & Perlman, C. A. (2006). Identification and evaluation of cognitive affect-regulation strategies: Development of a self-report measure. *Cognitive Therapy and Research, 30*, 227–262.

McCracken, L. M. (Ed.). (2011). *Mindfulness and acceptance in behavioral medicine: Current theory and practice*. Oakland, CA: Context Press/New Harbinger Publications.

Cognitive Strategy

- ▶ Cognitive Factors

Cognitive Style

- ▶ Cognitive Factors

Cognitive Techniques

- ▶ Cognitive Strategies

Cognitive-Behavioral Stress Management Training

- ▶ Williams LifeSkills Program

Cohort Study

Jane Monaco
Department of Biostatistics, The University of North Carolina at Chapel Hill, Chapel Hill, NC, USA

Synonyms

Follow-up study; Observational designs; Observational studies; Observational study

Definition

A cohort study is an observational study design in which subjects are usually selected based on their risk factor exposure and followed over time to evaluate whether they develop the outcome of interest (usually disease).

Description

Cohort studies are commonly used in behavioral medicine research to investigate associations in which experimental designs are unethical or too costly. In a cohort design, participants who have not experienced the outcome of interest are selected, usually based on whether or not they have been exposed to the risk factor of interest. Therefore, a cohort study design is efficient when the exposure is relatively rare but the outcome of interest is common. For example, a cohort design was used in a study of the association of prenatal polychlorinated biphenyl (PCB) exposure with behavior issues and cognitive disability (Lai et al. 2002). A cohort design is also appropriate when the exposure is common.

The most common type of cohort study, a prospective cohort study, identifies subjects without the outcome of interest (such as disease-free participants) at the outset of the study and then follows them forward through time to assess their outcome (or disease) status. Because the subjects have not experienced the outcome at the outset of the study, this prospective design is less susceptible to many types of bias compared to other observational study designs, such as case-control studies. Included in the prospective cohort study design are large studies such as the Framingham Study in which participants were selected for logistical reasons. By recruiting a large number of residents from the single community of Framingham, Massachusetts, follow-up was simplified, and investigators were able to study prospectively the associations between multiple risk factors and outcomes among the participants (Dawber, Kannel, & Lyell, 1963).

Not all cohort studies are conducted prospectively. In a retrospective cohort study, both the exposure and outcome may have occurred at the time of the initiation of the study. These retrospective, sometimes called historical, cohort studies are often conducted using data previously collected for other purposes. For example, pregnant women drivers involved in motor vehicle crashes were identified by linking Washington State Patrol records to birth and death certificates (Wolf et al., 1993). The exposure of interest, seatbelt use at the time of the crash, was determined using the police reports. Investigators determined pregnancy outcomes (including low birth weight and fetal death) based on the birth and fetal death certificate data. This retrospective cohort study found the risk of a low-birth-weight infant was higher among unrestrained female drivers compared to those wearing a seat belt at the time of the crash.

In a cohort study, investigators must follow both the exposed and unexposed subjects equally carefully to avoid detection bias. If the exposed subjects are followed more closely, then an excess number of outcomes may be detected within the exposed group resulting in an overestimate of the exposure effect. Also, loss to follow-up may result in biased results when that loss is associated with the exposure and outcome.

Some characteristics of cohort studies:

- Usually more expensive and time consuming than case-control designs; less expensive than an experimental design
- Often used when the exposure is rare

- Not practical when outcome of interest (disease) is rare or has a long-latency period
- Appropriate when studying multiple outcomes
- Usually can only address a single risk factor
- When information collected prospectively, reduces potential for bias
- Can be impacted by loss to follow-up
- Can compute incidence and relative risk of outcome directly
- Often considered stronger study design compared with case-control studies, but weaker study design compared to randomized trials that investigate analogous associations

Cross-References

▶ Bogalusa Heart Study
▶ Case-Control Studies
▶ Retrospective Study

References and Readings

Dawber, T. R., Kannel, W. B., & Lyell, L. P. (1963). An approach to longitudinal studies in a community: The Framingham study. *Annals of the New York Academy of Sciences, 107*(2), 539–556.

Hennekens, C. H., Buring, J. E., & Mayrent, S. L. (1987). *Epidemiology in medicine*. Philadelphia: Lippincott Williams & Wilkins.

Kleinbaum, D. G., Sullivan, K. M., & Barker, N. D. (2007). *A pocket guide to epidemiology*. New York: Springer.

Lai, T. J., Liu, X., Guo, Y. L., Guo, N., Yu, M., Hsu, C., et al. (2002). A cohort study of behavioral problems and intelligence in children with high prenatal polychlorinated biphenyl exposure. *Archives of General Psychiatry, 59*(11), 1061–1066.

Wolf, M. E., Alexander, B. H., Rivara, F. P., Hickok, D. E., Maier, R. V., & Starzyk, P. M. (1993). A retrospective cohort study of seatbelt use and pregnancy outcome after a motor vehicle crash. *The Journal of Trauma, 34*(1), 116.

Cold Pressor Task

▶ Cold Pressor Test

Cold Pressor Test

Laura A. Mitchell
Department of Psychology, School of Life Sciences, Glasgow Caledonian University, Glasgow, Scotland, UK

Synonyms

Cold pressor task

Definition

The cold pressor test is a widely used experimental technique for human pain or stress induction, involving immersion of the hand or forearm in cold water. First documented as a test of cardiovascular stress reactivity (Hines & Brown, 1936), its application in investigation of pain perception, mechanisms, and treatment is due to a gradually mounting painful sensation of mild to moderate intensity. As water temperatures used are within the range considered noxious (below 15°C), nociceptors (pain receptors) are activated and transmit an aversive signal to the CNS. While nociception-transduction ion channels involved have been identified, the exact mechanisms of cold pain are not fully elucidated (Basbaum, Bautista, Scherrer, & Julius, 2009).

Like other pain inductions, the cold pressor allows fast and precisely controlled evaluations not possible in a clinical context. Apparatus for the task is a tank of circulating water of temperature most often between 0°C and 5°C, with instruction to immerse the hand until too uncomfortable to continue. A maximum time limit per immersion of 3–5 min is normally applied. Quantitative measurement can then be made of pain threshold (point first perceived as painful), tolerance time, and perceived intensity and unpleasantness. The technique is regarded as safe for pain evaluations in children, usually at a slightly higher water temperature (von Baeyer, Piira, Chambers, Trapanotto, & Zeltzer, 2005).

Cross-References

► Pain Threshold

References and Readings

Basbaum, A. I., Bautista, D. M., Scherrer, G., & Julius, D. (2009). Cellular and molecular mechanisms of pain. *Cell, 139*, 267–284.

Hines, E. A., & Brown, G. E. (1936). The cold pressor test for measuring the reactibility of the blood pressure: Data concerning 571 normal and hypertensive subjects. *American Heart Journal, 11*, 1–9.

von Baeyer, C. L., Piira, T., Chambers, C. T., Trapanotto, M., & Zeltzer, L. K. (2005). Guidelines for the cold pressor task as an experimental pain stimulus for use with children. *The Journal of Pain, 6*(4), 218–227.

Colitis

► Inflammatory Bowel Disease

Collaborative Care

► Clinical Settings

Collaborator

► Co-workers

Colleague

► Co-workers

College Students

► Binge Drinking

Colorectal Cancer

► Cancer, Colorectal

Common Cold

Denise Janicki-Deverts and Crista N. Crittenden
Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA

Synonyms

Upper respiratory infection (mild)

Definition

The common cold is the familiar name for a mild upper respiratory infection (URI). Symptoms of the common cold include nasal congestion, mucus production, sneezing, cough, and sore throat (Eccles, 2005). The common cold is caused by any of a number of viruses, most often one of the rhinoviruses (see ► Common Cold: Cause).

URIs are responsible for 50% of all acute illnesses, with common colds accounting for most of that proportion. While symptoms are often mild, the common cold often confers a heavy burden on patients, healthcare providers, schools, and workplaces. Approximately 62 million cases of the common cold occur each year in the United States. In addition, 20 million school days are lost annually as well as 22 million work days due to the common cold (Adams, Hendershot, & Marano, 1999).

The incubation period for the common cold largely depends on the virus that causes it. On average, symptoms begin 2–3 days after virus exposure and infection. From the onset of the first symptoms, severity usually peaks within 2 days. Overall, the duration of the common cold is usually 7–10 days (Eccles, 2005).

Because only a proportion of people who are exposed to cold viruses actually develop symptoms, the common cold has become a fertile ground for studying psychosocial and behavioral

factors that influence vulnerability to infection. Viral challenge studies expose healthy individuals to common cold viruses and then keep them in quarantine over several days to assess who develops infection and symptoms and who does not. Prior to virus exposure, a plethora of behavioral and psychological measures are performed in order to assess the roles these factors may play in illness susceptibility.

Within these experimental studies, individuals are determined to have a cold if they (1) are infected with the challenge virus and (2) meet a set of predetermined criteria based on subjective symptom reports and objective physiological measurements. Infection is determined by the presence of viral shedding (i.e., replication of the virus in the host environment). Viral shedding is assessed by administering a nasal wash, which flushes the nasal cavity and sinuses, and then culturing a sample of the exposed wash solution for the presence of replicating virus. Presence and severity of cold symptoms, i.e., runny nose, sore throat, nasal congestion, etc., are assessed via observation and participant report. Objective measures of cold severity include mucus production and mucociliary clearance function. Mucus production is assessed by collecting used tissues from participants and measuring their weight; nasal mucociliary clearance, or how effective the body is in clearing mucus from the nasal passage, is assessed as the time it takes for a dye administered in the nostrils to reach the nasopharyx (Doyle, McBride, Swarts, Hayden, & Gwaltney, 1988). Additional measures, such as lung function, may also be taken.

Through these common cold studies, several behavioral and psychosocial factors have been found to greatly increase susceptibility to infection, including sleep patterns, social integration and stress. These topics are discussed further in ▶ Common Cold: Cause and ▶ Common Cold: The Stress Factor.

Cross-References

- ▶ Common Cold: Cause
- ▶ Common Cold: The Stress Factor
- ▶ Stress

References and Readings

Adams, P. F., Hendershot, G. E., & Marano, M. A. (1999). Current estimates from the national health interview survey 1996, National Center for Health Statistics. *Vital Health Statistics, 10*(200).

Doyle, W. J., McBride, T. P., Swarts, J. D., Hayden, F. G., & Gwaltney, J. M. (1988). The response of the nasal airway, middle ear and Eustachian tube to provocative rhinovirus challenge. *American Journal of Rhinology, 2*, 149–154.

Eccles, R. (2005). Understanding the symptoms of the common cold and influenza. *The Lancet Infectious Diseases, 5*, 718–725.

Common Cold: Cause

Denise Janicki-Deverts and Crista N. Crittenden
Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA

Synonyms

Upper respiratory infection (mild): cause

Definition

The common cold is a mild upper respiratory illness that results from infection with any of more than 200 viruses, most notably the rhinoviruses. The rhinovirus family is comprised of over 100 different viruses, with the relative prevalence of each being dependent on a number of factors, from geographical area to time of year. Overall, rhinoviruses make up approximately 30–50% of all acute respiratory illnesses, but in the fall season this proportion jumps to about 80%. Coronaviruses comprise another family of viruses that cause the common cold. Infections with coronaviruses are estimated to account for 7–18% of adult colds, and in contrast to rhinovirus infections, tend to be most prevalent during the winter and spring months. Additional cold viruses include parainfluenza, respiratory syncytial virus (RSV), the adenoviruses, and the enteroviruses which collectively account for

a comparatively small percentage of infections. In addition, 20–30% of common cold cases are of unknown origin (Heikkinen & Jarvinen, 2003).

Cold viruses are highly contagious, and interpersonal transmission of colds typically occurs in one of two ways: (1) inhaling viral particles that are released into the air in tiny droplets when infected persons cough, sneeze, or blow their nose; or (2) coming into contact with surfaces that have been contaminated by infected secretions (e.g., a doorknob that was touched by an infected person immediately after coughing into his or her hand) and then touching one's own eyes, nose, or mouth.

Several factors have been found to influence whether individuals become infected following exposure to a cold virus and/or the severity of their symptoms once infected. Most of these findings have derived from viral challenge studies wherein healthy individuals are exposed to cold viruses (most often rhinoviruses), placed under quarantine, and monitored by trained medical staff for objective signs and subjective symptoms of a cold (see ▶ Common Cold). Of all potential susceptibility factors, stress has been the most explored. Accordingly, the role of stress in cold susceptibility is discussed in a separate entry (see ▶ Common Cold: The Stress Factor). Stress, however, is far from being the only factor that has been found to influence who develops colds. For example, smokers are more likely than nonsmokers to become infected with the cold virus and, consequently, to develop illness symptoms (Cohen, Tyrrell, Russell, Jarvis, & Smith, 1993). Social relationship factors have been found to influence cold susceptibility as well. People who are high in trait sociability (which is thought to be an important determinant of quantity and quality of social interaction) and those with more diverse social networks are less susceptible to colds than their less sociable and less socially integrated counterparts (Cohen et al., 1997; Cohen, Doyle, Turner, Alper, & Skoner, 2003a). A third identified susceptibility factor is affect. Specifically, greater positive affect is associated in a dose-response manner with reduced likelihood of developing a cold (Cohen, Doyle, Turner, Alper, & Skoner, 2003b). Importantly, all of these factors remained associated with cold susceptibility even when controlling for age, sex, body weight, and season of exposure.

Cross-References

- ▶ Common Cold
- ▶ Common Cold: The Stress Factor
- ▶ Stress

References and Readings

Cohen, S., Doyle, W. J., Skoner, D. P., et al. (1997). Social ties and susceptibility to the common cold. *Journal of the American Medical Association, 277*, 1940–1944.

Cohen, S., Doyle, W. J., Turner, R. B., Alper, C. M., & Skoner, D. P. (2003a). Sociability and susceptibility to the common cold. *Psychological Science, 14*, 389–395.

Cohen, S., Doyle, W. J., Turner, R. B., Alper, C. M., & Skoner, D. P. (2003b). Emotional style and susceptibility to the common cold. *Psychosomatic Medicine, 65*, 652–657.

Cohen, S., Tyrrell, D. A. J., Russell, M. A., Jarvis, M. J., & Smith, A. P. (1993). Smoking, alcohol consumption, and susceptibility to the common cold. *American Journal of Public Health, 83*, 1277–1283.

Eccles, R. (2005). Understanding the symptoms of the common cold and influenza. *The Lancet Infectious Diseases, 5*, 718–725.

Heikkinen, T., & Jarvinen, A. (2003). The common cold. *Lancet, 361*, 51–59.

Common Cold: The Stress Factor

Denise Janicki-Deverts and Crista N. Crittenden
Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, USA

Synonyms

Upper respiratory infection (mild): the stress factor

Definition

One of the most consistent findings from viral challenge studies (see ▶ Common Cold) is that

the experience of stress is positively associated with susceptibility to the common cold. Here stress is defined as a psychological state resulting from outside factors or events placing demands on an individual that exceed his or her resources or ability to cope (Cohen, Kessler, & Gordon, 1995). Although stressful experiences such as bereavement and care giving have long been believed to suppress host resistance, the common cold studies were the first to demonstrate the role of the stress factor under prospective, controlled conditions.

Cohen, Tyrrell, and Smith (1991) conducted one of the first studies to explore the role of stress in susceptibility to the common cold. The authors assessed several stress factors, including life events and perceived stress in a sample of healthy adults, and then experimentally exposed these individuals to a cold virus or to a saline control. Despite controlling for several person and environmental factors, the researchers observed a dose-response association between stress and clinical colds: more stress was associated with an increased likelihood both of becoming infected and displaying clinical symptoms. Furthermore, they also found that long-lasting social stressors accounted for the greatest infection risk. These stress factor effects were all independent of potential mediators such as smoking, diet, alcohol use, and sleep quality. Cohen et al. (1998) further explored several types of stressors linked to the common cold and found that severe, chronic stressors – especially work and interpersonal stressors, lasting 1 month or longer – conferred a substantial risk of developing a clinical cold after virus exposure. Moreover, the longer the stress duration, the greater the relative risk of a cold. Again, these differences could not be completely explained by environmental, person-related, or behavioral factors.

An important feature of the common cold is that associated symptoms (sneezing, congestion, etc.) are caused by the body's immune response to the virus, not the virus per se. Most symptoms result from the production of pro-inflammatory cytokines that recruit other immune cells to fight the infection. Several "host" factors influence the immune system's response to infection and how severe resulting symptoms will be. These include age, general health, and past infection experience. However, the repeated finding of greater stress being associated with increased risk for colds independent of behavioral factors or health practices suggests that stress may be influencing the immune system, as well, by suppressing some resistance processes. For example, in influenza challenge studies, increased psychological stress was associated with higher pro-inflammatory cytokine concentrations, particularly interleukin (IL)-6 (Cohen, Doyle, & Skoner, 1999). In an experimental study in which stress was induced in a laboratory setting, Marsland, Bachen, Cohen, Rabin, and Manuck (2002) found that being exposed to a stressor was associated with increases in immune markers, such as circulating natural killer cells and cytotoxic T cells. These studies suggest that stress may be acting through major immunological pathways to increase symptoms of infectious illnesses.

Cross-References

- ▶ Common Cold
- ▶ Stress

References and Readings

Cohen, S., Kessler, R. C., & Underwood Gordon, L. (Eds.) (1995). Measuring stress: A guide for health and social scientists. New York: Oxford. Strategies for measuring stress in studies of psychiatric and physical disorders.

Cohen, S., Doyle, W. J., & Skoner, D. P. (1999). Psychological stress, cytokine production, and severity of upper respiratory illness. *Psychosomatic Medicine, 61*, 175–180.

Cohen, S., Frank, E., Doyle, W. J., Skoner, D. P., Rabin, B. S., & Gwaltney, J. M. (1998). Types of stressors that increase susceptibility to the common cold in healthy adults. *Health Psychology, 17*(3), 214–223.

Cohen, S., Tyrrell, D. A. J., & Smith, A. P. (1991). Psychological stress and susceptibility to the common cold. *The New England Journal of Medicine, 325*, 606–612.

Cohen, S., Tyrrell, D. A. J., & Smith, A. P. (1993). Life events, perceived stress, negative affect and susceptibility to the common cold. *Journal of Personality and Social Psychology, 64*, 131–140.

Marsland, A. L., Bachen, E. A., Cohen, S., Rabin, B., & Manuck, S. B. (2002). Stress, immune reactivity and susceptibility to infectious disease. *Physiology and Behavior, 77*, 711–716.

Common Disease-Common Variant

Jennifer Wessel
Public Health, School of Medicine, Indiana University, Indianapolis, IN, USA

Definition

The common disease-common variant (CDCV) hypothesis predicts that for any given common disease, the genetic risk will be due to common variants with high frequency in the population (Pritchard & Cox, 2002; Reich & Lander, 2001).

The allelic spectrum, i.e., the frequency of the allele in the population and the number of disease-predisposing alleles, of common diseases is still not well understood. The number of common variants contributing to any given common disease will most likely vary from less than 100 to several thousands, with many of these variants having a low effect on the disease (Padhukasahasram et al. 2010).

Genome-wide association studies (GWAS) have succeeded at identifying common variations, many with low effect sizes (odds ratio ~1.2–1.5). However, there are many more left to be identified. A number of reasons have been suggested as to why GWAS have not identified more variations. These include rare variations contributing to common diseases, phenotypic heterogeneity, sample size, or regions missed by single nucleotide polymorphism (SNP) microarrays.

Cross-References

- ▶ Allele
- ▶ Genome-Wide Association Study (GWAS)
- ▶ Single Nucleotide Polymorphism (SNP)

References and Readings

Padhukasahasram, B., Halperin, E., Wessel, J., Thomas, D. J., Silver, E., Trumbower, H., et al. (2010). Presymptomatic risk assessment for chronic non-communicable diseases. *PLoS One, 5*, e14338.

Pritchard, J. K., & Cox, N. (2002). The allelic architecture of human disease genes: common disease-common variant. . .or not? *Human Molecular Genetics, 11*, 2417–2423.

Reich, D. E., & Lander, E. S. (2001). On the allelic spectrum of dusease. *Trends in Genetics, 17*, 502–510.

Common Migraine

- ▶ Migraine Headache

Common-Sense Model of Self-regulation

Pablo A. Mora[1] and Lisa M. McAndrew[2]
[1]Department of Psychology, The University of Texas at Arlington, Arlington, TX, USA
[2]Department of Veterans Affairs, NJ Healthcare System, East Orange, NJ, USA

Synonyms

Illness representation model; Mental models of illness; Mental representations of illness

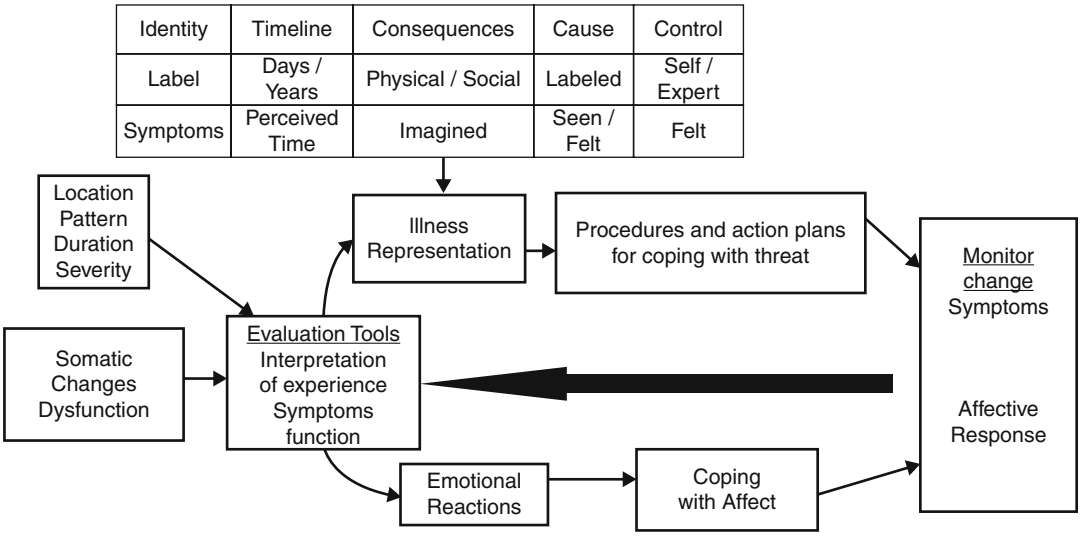
Definition

The common-sense model of self-regulation explains how individuals respond to and manage health threats. It proposes that people actively engage in problem-solving by developing mental models of health threats, subjective and objective treatment goals, and practices and procedures most likely to achieve those goals.

Description

Background

The origins of the common-sense model of self-regulation (CSM) can be traced to the parallel model proposed by Leventhal in the early 1970s to understand how individuals



Common-Sense Model of Self-regulation, Fig. 1 Individuals assess somatic changes based on the features of the changes by using evaluation tools (e.g., location, pattern, duration, or function) and comparing these features against illness prototypes. If deemed to be a health threat, the individuals develops cognitive and emotional representations of the threat. The cognitive representation consists of five bi-level domains. Cognitive and emotional representations guide the selection of coping procedures and the criteria for assessing effectiveness of these procedures (e.g., changes in symptom and/or affectivity). Evidence of success or failure to achieve desired changes will be used by the individual to re-assess the illness representation

respond to fear-arousing communications (Leventhal, 1970). Similar to the parallel model, the CSM posits that when a threat is perceived (e.g., physical symptoms or changes in function), individuals develop two parallel, yet interrelated, representations of the stimulus: cognitive and emotional (Leventhal et al., 1997). These representations and their content specify the actions (i.e., behaviors) in which individuals engage to remove the health threat. The CSM proposes that the processes involved in the self-regulation of health threats are regulated by a TOTE (Test-Operate-Test-Exit) system with both feedback and feed-forward loops (Miller, Galanter, & Pribram, 1960).

Common sense self-regulation is divided into three phases (Leventhal & Cameron, 1987). In the first stage, the perceptual stage, a discrepancy between a perceived input and a reference value is detected (i.e., health threat). At this stage, the individual develops a common sense illness representation of the potential health threat. In the second stage (i.e., the response stage), the individual selects and performs actions (i.e., coping procedures) to reduce the discrepancy. The illness representation developed during the perceptual stage will help select the types of coping procedures used by the individual during the response stage. In the last stage, the appraisal stage, the results of the action(s) aimed at reducing or eliminating the discrepancy are evaluated. If the actions are successful in dealing with the threat, the loop stops. If unsuccessful, the individual may reassess the representation and form a new illness representation, and/or select and perform new corrective actions. This loop will continue until the threat has been successfully removed or controlled.

Illness Representations

The CSM assumes that people are active problem-solvers who continuously assess the meaning of somatic sensations and/or changes in function by forming cognitive and emotional illness representations (see Fig. 1). Illness representations have a bi-level structure that includes an abstract level (i.e., disease labels and chronological time) and a concrete level

(i.e., symptoms and experienced time). Initially, the CSM identified five defining aspects of illness representation: identity, timeline, causes, consequences, and controllability. Abstract features of *identity* include the label applied to the health threat (e.g., diagnosis or name of the condition). Concrete features of *identity* refer to how the threat is experienced (e.g., symptoms and/or changes in function). *Timeline* refers to the objective (i.e., abstract: duration in minutes or hours) and perceived (i.e., concrete: perceived duration) temporal features of the health threat. *Causes* refer to the diagnosed (i.e., information conveyed by the doctor) and perceived (i.e., leaving home with wet hair) factors that caused the health threat. The causes of a threat can be grouped into external agents (e.g., virus, bacteria, or stress), internal susceptibilities (e.g., age, genetics), and behaviors (e.g., smoking). *Consequences* include both anticipated (i.e., abstract) and perceived and experienced (i.e., concrete) physical, psychological, social, and economic effects that the health threat will produce. Controllability refers to whether the person expects and perceives the health threat to be susceptible to control by experts (e.g., physician) and/or the self.

The existence of a sixth domain, "illness coherence," has been proposed by Weinman and collaborators (Moss-Morris et al., 2002). Illness coherence is a metacognition that refers to the extent to which an individual believes that the various features of an illness hang together. For example, someone who suffers from a cold would expect that symptoms such as a runny nose and a sore throat would last for a couple of weeks. However, if unusual symptoms are experienced or if the timeline of the symptoms is longer than expected, the individual will have trouble making sense of the health threat. Low coherence has been shown to have an impact on other dimensions of illness representations and on the appraisal and enactment of coping procedures.

Illness representations and their content serve as guides for the selection, performance, and evaluation of actions used to manage illness episodes. Feedback from these actions can reshape the representations and alter subsequent coping actions. For instance, actions taken to ameliorate a stomachache could involve drinking herbal tea or having a bland diet, whereas a headache might lead the person to take an over-the-counter pain reliever. The appraisal of the effectiveness of these actions will also vary depending on the specific content of the representations. For example, the expected timeline for ridding oneself of a stomachache is likely several hours to a day, while the expected timeline for determining that a pain reliever is effective in dealing with a headache could be to 1–2 h at most.

Appraisal of Health Threats. When a deviation from "normal function" is detected (e.g., a health threat such as somatic symptoms or declines in physical function), the individual will promptly engage in an automatic scanning process in which the properties of the health threat are assessed (Leventhal, Breland, Mora & Leventhal, 2010). These properties are compared against illness prototypes developed through prior personal experience, observation of others, and media exposure (cf. Fig. 1). Prototype checks are used evaluate somatic and/or functional changes with respect to features such as their location (e.g., head, stomach, chest), duration (e.g., perceived and clock time), rates of change (e.g., sudden onset or insidious), consequences (e.g., disrupts breathing or impairs walking), causes (e.g., exposure to sick people or perceived stress), and sensory properties (e.g., sharp or dull). If the features of the somatic or functional changes match an illness prototype, then a preliminary illness representation will be formed and lead the individual to engage in actions to remove or control the threat (i.e., coping procedures). A further appraisal of the threat will be conducted based on the perceived effects of these coping procedures. Feedback from coping procedures will provide critical information to either confirm or disconfirm the preliminary illness representation. For instance, a headache and a runny nose may be the result of a cold or of seasonal allergies. If the symptoms occur around spring, then the person may decide to take an antihistamine pill to help clear the symptoms. If after a few hours the symptoms

are not relieved, then the tentative "allergies" representation may be discarded, and a new health threat appraisal process will begin.

Lack of experience with a specific condition or unusual presentation of symptoms can create confusion during the matching and appraisal process and result in negative consequences such as delayed care seeking or poor illness management. For example, gastric pain caused by gallstones could be attributed to indigestion or stomach flu if a person has never been exposed to gallstones and the symptomatology associated with gallstones previously because the location is similar (i.e., abdominal area). This could lead individuals to engage in watchful waiting which, in turn, could increase the risk of serious consequences such as emergency hospitalization due to blockage of the pancreatic duct. Conditions that do not manifest according to the prototypes people have can result in inadequate management or control of the threat. For instance, the symptoms that people with congestive heart failure usually experience (e.g., swollen legs, breathing and sleeping problems) are not the symptoms that a person with a "heart" condition is supposed to experience (e.g., palpitations). This mismatch can result in poor adherence to medical treatment because the heart condition does not represent an immediate threat. Similarly, depressive symptomatology may not be properly identified and treated among older adults, because they are less likely to experience symptoms of dysphoria (i.e., depression without sadness, Gallo, Rabins, Gallo, & Rabins, 1999). Low negative affect will make the matching processes difficult for both the individuals who experience depressive symptoms and mental health professionals because the symptoms do not fit with a "depression prototype."

Treatment Representations

From the CSM perspective, treatment representations are conceptualized using the same framework as illness representations (Leventhal et al., 2010). That is, treatment beliefs are assumed to have an identity (e.g., "diuretic"), timeline (e.g., for how long one should take the medications or time for treatment to effect changes), causal factors (e.g., works by killing bacteria), control (e.g., cure and control of disease symptoms), and consequences (e.g., addiction or improved quality of life). Research directly examining these dimensions of treatment representations is limited. Several studies conducted by Leventhal and collaborators, however, have assessed some of these facets (e.g., Halm, Mora, & Leventhal, 2006). These assessments have focused on aspects such as triggers that initiate the use of medication (e.g., "I use medications when I have symptoms"), control (e.g., "My medicines protect me from becoming worse"), consequences ("My health in the future depends on my medications"), and emotional reactions ("How worried are you about the side effects of your medication?"). Evidence from these studies have shown that these aspects of treatment representations are strong predictors of illness self-management.

A different view of treatment representations has been put forth by Horne, Weinman, and Hankins (1999). Based on common sense regulation principles, Horne and collaborators identified commonly held beliefs about medications and medical treatments and grouped them into "general" and "specific" concerns about medications. "General concerns" encompass beliefs that medications, in general, are overprescribed by practitioners (i.e., overuse) and beliefs that medicines can be harmful and addictive (i.e., harm). "Specific concerns" address the beliefs that a prescribed medication is necessary for and efficacious in controlling a particular condition and concerns about the harmful effects of a medication prescribed for a specific illness.

Despite the apparent differences, both views are conceptually consistent. Further, the CSM could be used as a more general framework within which the two major domains (i.e., general and specific concerns) can be organized. For instance, specific beliefs include both cognitive and emotional aspects of treatment representations. Similarly, some specific beliefs about the necessity of medications can be categorized as beliefs about the consequences of treatment (e.g., my health in the future depends on medicines).

Measurement

Because of their central role in the CSM, most measurement efforts have focused on developing instruments to assess the content of illness and treatment representations. These efforts have been guided by two different approaches. In the first approach (i.e., domain-based approach), investigators develop measurement to assess content relevant to the specific illness condition under investigation. Researchers who use the second approach (i.e., instrument-based approach) prefer the use of basically the same instrument and items across domains.

The domain-based approach requires close familiarity with the health threat (i.e., illness condition) to be studied. Although illness representations of various conditions may share some features, they can be highly divergent in terms of how they are experienced by individuals, their consequences, and their management. Thus, to develop valid and relevant items, one needs to rely on the use of theory, pilot interviews with patients who suffer a given condition, and input from practitioners (Leventhal & Nerenz, 1985). Items developed by using this approach usually focus on very specific aspects of illness and treatment representations in order to gain a more detailed understanding of underlying psychological processes and mechanisms. The resulting instrument may consist of single-item subscales that may be unique for the illness condition being studied (e.g., Halm et al., 2006). The development of items to assess treatment representations is conducted in a similar fashion. When developing items to assess treatment representations, one must pay special attention to issues such as the cues used by individuals for initiating and evaluating action (e.g., Do symptoms or objective information such as blood glucose monitoring initiate self-management?), the expected time for observing effects, and the specific behaviors used to control or eliminate the health threat (e.g., complementary medicine, rest, and distraction).

The instrument-based approach is best represented by multi-item questionnaires developed to assess both illness and treatment representations. The Illness Perception Questionnaire (IPQ) assesses the five original domains of illness representations (i.e., identity, timeline, consequences, causes, and controllability), emotional representations, and illness coherence (Moss-Morris et al., 2002). The items do a good job of providing a snapshot of people's illness representations. Two versions of the IPQ are available: the IPQ-R which is the long version and consists of over 50 items and the brief IPQ which includes eleven questions (Broadbent, Petrie, Main, & Weinman, 2006). The IPQ questions are standard, though it is possible to make modifications to the items' wording and/or include a condition-specific symptom list, to reflect the illness condition being investigated. The IPQ has been successfully used in a wide range of studies examining various chronic conditions such as asthma, diabetes, cardiovascular disease, and rheumatic conditions.

To date, the only instrument that assesses treatment representations is the Beliefs about Medications Questionnaire (BMQ) developed by Horne et al. (1999). The BMQ is a multi-item instrument that comprises two scales that assess general and specific concerns about medicines. Similar to the IPQ, the wording of the items is standard but can be modified to reflect the different types of treatments. For example, pill can be substituted for inhaler. Research has shown that the aspects of treatment representations assessed by the BMQ predict self-management and medication adherence across various chronic conditions (e.g., Horne & Weinman, 1999).

Both approaches present different limitations and offer unique advantages. The domain-based approach has most often been criticized for relying on single-item measures to assess the various aspects of illness representations. The assumption underlying this criticism is that single items have low reliability. However, there is no evidence to suggest that single items do not make reliable assessments (Wanous, Reichers, & Hudy, 1997). The main advantage of this approach is that the development of domain-specific items can facilitate the theoretical understanding about the precise pathways linking illness and treatment representations, behaviors, and health outcomes within each

illness conditions. The downside of domain-based measures is that the uniqueness of items makes it difficult to compare findings across illness conditions.

Because the IPQ-R and the BMQ include multiple items to assess each construct, the estimation of reliability is not an issue. However, because there are several instances in each subscale where items are similarly worded, the multi-item nature of the scales does not result necessarily in a more precise instrument. In addition, as indicated by Broadbent, Petrie, Main, and Weinman (2006), the large number of items makes it difficult to use when resources are limited. Length of the instrument has been addressed by the development and validation of the brief IPQ. The main advantage of these instruments is that the wording of items is consistent across studies and illness conditions, which facilitates comparisons. It is important to note, however, that psychometric research is needed to determine whether these instruments are invariant across people with different illness conditions and across countries.

Combining both approaches will most likely have the greatest impact from both a research and an applied perspective. A set of items such as those from the brief IPQ would provide investigators with a core set of items that could be employed in all studies, thus enabling future comparisons. The addition of domain-specific items to the brief IPQ would allow investigators to delve into specific issues unique to the health threat under investigation.

Interventions and the CSM

A basic corollary of the emphasis the CSM puts on the content of illness and treatment representations and the actions specified by these representations is that successful interventions require an adequate model of the problem. This implies that interventions will vary depending on the health status of the target population (e.g., well vs. not well) and on the type of illness conditions individuals have if the target population for the intervention consists of people with chronic conditions. For instance, primary prevention interventions (e.g., screening or lifestyle changes) that target individuals who feel healthy require that investigators understand the factors that make them feel vulnerable to health threats (e.g., perceived risk based on age or family history). Interventions with persons suffering from chronic conditions require investigators to have a detailed understanding of the disease and its context. If the interventions are aimed at improving self-management, for instance, investigators need to know the cues that impede or facilitate behaviors (e.g., symptoms), beliefs about treatments (e.g., risk of addition), and the complexity of the treatment.

The context in which the intervention is delivered will also affect the foci and the delivery of interventions. For example, interventions to improve adherence to medical treatment may have different targets for change depending on whether they are delivered in the office of a primary care physician or in the community. Primary care physicians can focus on eliciting illness and treatment representations and negotiate with patients a representation that encourages adherence to medical treatment. For a condition such as asthma, if patients hold an acute view of the condition and, therefore, use inhaled corticosteroids only when having symptoms, then the primary care physician may provide them with appraisal tools to disconfirm this inaccurate belief. Such patients may be instructed to climb up one flight of stairs for a few days and take notice of their breathing, before beginning their daily regime of medications. This simple instruction should help patients realize that without their medication, their breathing becomes more difficult with minimal exercise, despite the absence of noticeable symptoms prior to the exercise. This could provide experiential evidence for the patient that asthma is present even when asymptomatic. To solidify this more accurate illness representation, these patients may then be instructed to repeat this exercise after following the inhaled corticosteroid regime as prescribed and notice improvements in their breathing after climbing up stairs. This second part of the intervention can allow patients to link improvements in breathing with the regular use of medications. Thereby, the reformulation of

their illness representation can be a life-saving tool. A community-based, population level intervention, unlike the physician-patient dyad interaction described above, is likely to deal with patients whose beliefs may differ widely. In this case, attempting to change each individual's representations may not be feasible. A more effective and efficient approach would focus on modifying management behaviors, such as how to incorporate the use of inhaled corticosteroids into one's daily routine, by, for example, leaving the inhaler on the kitchen or dining room table, the proper technique to use the inhaler, and incorporating abstract information (e.g., peak flow meter readings) into the monitoring of asthma. An intervention focusing on patient-doctor interaction has provided important evidence that physicians can successfully elicit patients' illness representations during medical visits (de Ridder, Theunissen, & van Dulmen, 2007). Unfortunately, this intervention did not test whether discussion of illness representations results in improved health outcomes.

Despite the consistent and robust findings that illness and treatment representations are powerful determinants of behaviors (Hagger & Orbell, 2003), interventions using principles from the CSM are sparse. The results of these interventions, nonetheless, are quite promising. Interventions with patients suffering from chronic conditions have demonstrated that changing illness representations has an important effect on health outcomes (e.g., Petrie, Cameron, Ellis, Buick, & Weinman, 2002). However, specific pathways through which changes in illness representations affect health are not yet clearly understood.

Concluding Remarks

Although the CSM is a sound and powerful theoretical framework, there is still much to do to improve the understanding of the cognitive and emotional determinants of health-related behaviors. Two key issues that need more research attention can be highlighted. First, enormous progress has been made in the study of illness and treatment representations, but less is known about how other aspects of the CSM such as appraisal tools, coping procedures, and criteria to appraise coping procedures interact to influence self-regulation. Not only is such knowledge necessary for better understanding of psychological phenomena but also for the design of more effective and potentially life-saving interventions. Emotional aspects of illness and treatment representations constitute another area that requires more research. Emotional representations have been shown to play an important role in care seeking and self-management (e.g., Mora, Robitaille, Leventhal, Swigar, & Leventhal, 2002); however, their interaction with illness representations in determining behaviors is not yet fully understood. Progress in these areas will greatly benefit from basic behavioral medicine research conducted in conjunction with intervention research. If properly designed and theoretically sound, intervention studies can provide strong evidence of causal relations. A comprehensive mapping of mechanisms, however, may require the use of nontraditional designs that focus on changing well-delimited processes in a sequential manner (i.e., tailored, stepwise interventions).

Theoretical progress should occur simultaneously with advances in measurement (Petersen, van den Berg, Janssens, & Van den Bergh, 2011). Testing the full CSM will require that researchers develop adequate measurement. Initiatives such as the NIH PROMIS provide a good model to follow (Cella, Gershon, Lai, & Choi, 2007). The creation of item banks based on theoretical and practical considerations would afford investigators both a common set of tools to allow comparisons and flexibility to address issues unique to the research problem. This will also require that researchers adopt current measurement strategies such as item response theory to examine the psychometric properties of their instruments.

Cross-References

- ▶ Coping
- ▶ Health Beliefs
- ▶ Illness Perceptions Questionnaire (IPQ-R)
- ▶ Self-regulation

References and Readings

Broadbent, E., Petrie, K. J., Main, J., & Weinman, J. (2006). The brief illness perception questionnaire (bipq). *Journal of Psychosomatic Research, 60*(6), 631–637.

Cameron, L. D., & Leventhal, H. (2003). *The self-regulation of health and illness behaviour*. New York: Routledge.

Cella, D., Gershon, R., Lai, J.-S., & Choi, S. (2007). The future of outcomes measurement: Item banking, tailored short-forms, and computerized adaptive assessment. *Quality of Life Research, 16*, 133–141. doi:10.1007/s11136-007-9204-6.

de Ridder, D. T., Theunissen, N. C., & van Dulmen, S. M. (2007). Does training general practitioners to elicit patients' illness representations and action plans influence their communication as a whole? *Patient Education and Counseling, 66*(3), 327–336. doi:http://dx.doi.org/10.1016/j.pec.2007.01.006.

Gallo, J. J., Rabins, P. V., Gallo, J. J., & Rabins, P. V. (1999). Depression without sadness: Alternative presentations of depression in late life. *American Family Physician, 60*(3), 820–826.

Hagger, M. S., & Orbell, S. (2003). A meta-analytic review of the common-sense model of illness representations. *Psychology & Health, 18*(2), 141–184.

Halm, E. A., Mora, P., & Leventhal, H. (2006). No symptoms, no asthma: The acute episodic disease belief is associated with poor self-management among inner city adults with persistent asthma. *Chest, 129*(3), 573–580.

Horne, R., & Weinman, J. (1999). Patients' beliefs about prescribed medicines and their role in adherence to treatment in chronic physical illness. *Journal of Psychosomatic Research, 47*(6), 555–567.

Horne, R., Weinman, J., & Hankins, M. (1999). The beliefs about medicines questionnaire: The development and evaluation of a new method for assessing the cognitive representation of medication. *Psychology & Health, 14*(1), 1–24.

Leventhal, H. (1970). Findings and theory in the study of fear communications. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol. 5, pp. 120–186). New York: Academic.

Leventhal, H., Benyamini, Y., Brownlee, S., Diefenbach, M., Leventhal, E. A., Patrick-Miller, L., et al. (1997). Illness representations: Theoretical foundations. In K. J. Petrie & J. A. Weinman (Eds.), *Perceptions of health and illness: Current research and applications* (pp. 19–45). Singapore: Source Harwood Academic Publishers.

Leventhal, H., Breland, J. Y., Mora, P. A., & Leventhal, E. A. (2010). Lay representations of illness and treatment: A framework for action. In A. Steptoe (Ed.), *Handbook of behavioral medicine: Methods and applications* (pp. 137–154). New York: Springer.

Leventhal, H., & Cameron, L. (1987). Behavioral theories and the problem of compliance. *Patient Education & Counseling, 10*(2), 117–138.

Leventhal, H., Leventhal, E. A., & Cameron, L. (2001). Representations, procedures, and affect in illness self-regulation: A perceptual-cognitive model. In A. Baum, T. Revenson, & J. Singer (Eds.), *Handbook of Health Psychology*. New York: Erlbaum.

Leventhal, H., & Nerenz, D. (1985). The assessment of illness cognition. In P. Karoly (Ed.), *Measurement strategies in health* (pp. 517–554). New York: John Wiley & Sons.

Miller, G. A., Galanter, E., & Pribram, K. H. (1960). *Plans and the structure of behavior*. New York: Holt.

Mora, P. A., Robitaille, C., Leventhal, H., Swigar, M., & Leventhal, E. A. (2002). Trait negative affect relates to prior week symptoms, but not to reports of illness episodes, illness symptoms and care seeking among older people. *Psychosomatic Medicine, 64*(3), 436–449.

Moss-Morris, R., Weinman, J., Petrie, K. J., Horne, R., Cameron, L. D., & Buick, D. (2002). The revised illness perception questionnaire (ipq-r). *Psychology & Health, 17*(1), 1–16.

Petersen, S., van den Berg, R. A., Janssens, T., & Van den Bergh, O. (2011). Illness and symptom perception: A theoretical approach towards an integrative measurement model.*Clinical Psychology Review, 31*(3), 428–439. doi:10.1016/j.cpr.2010.11.002.

Petrie, K. J., Cameron, L., Ellis, C. J., Buick, D., & Weinman, J. (2002). Changing illness perceptions after myocardial infarction: An early intervention randomized controlled trial. *Psychosomatic Medicine, 64*(4), 580–586.

Petrie, K. J., & John, W. (1997). *Perceptions of health and illness: Current research and applications*. Amsterdam: Harwood Academic Publishers.

Skelton, J. A., & Croyle, R. T. (1991). *Mental representation in health and illness*. New York: Springer.

Wanous, J. P., Reichers, A. E., & Hudy, M. J. (1997). Overall job satisfaction: How good are single-item measures? *Journal of Applied Psychology, 82*(2), 247–252.

Communication Skills

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

Communication skills are an essential medium through which physicians interact with patients, in order to diagnose and treat patients. According

to Ong, de Haes, Hoos, and Lammes (1995), doctor-patient communication has three main roles: (1) to create a positive interpersonal relationship, (2) exchange information, and (3) make treatment-related decisions. A positive interpersonal relationship includes facilitation of trust between the patient and a health professional that enables honest bidirectional expression of concerns and report of behaviors (e.g., risky behaviors, nonadherence). Exchange of information is the basis of the doctor-patient interaction, where information from patient to doctor enables the latter to arrive at more accurate diagnoses and more suitable and effective treatments. Similarly, exchange of information from physician to patient enables the doctor to inform the patient about risks of unhealthy behaviors (e.g., smoking) and benefit of others (e.g., self-monitoring of glucose levels or of physical activity). Finally, adequate communication helps physicians decide about patient-tailored treatments, suitable to their age, culture, levels of information-seeking, family history of an illness, comorbidities, etc.

According to research (e.g., Di Blasi, Harkness, Ernst, Georgiou, & Kleijnen, 2001; Ong et al., 1995), doctor-patient communication skills (e.g., information giving, listening, reassuring) affect patients' satisfaction from treatment, understanding and recall of the interaction with doctors, adherence to medical regimes, and actual health outcomes. The influence of communication skills on patient recall is important given that patients can at times recall very little of the information provided to them during consultations. A review on this topic found 14 studies on verbal variables and patient outcomes and showed that factors such as empathy, reassurance, "psychosocial talk," humor, and patient-centered talk correlated with positive health outcomes. The same review identified eight studies on nonverbal communication and patient outcomes and showed that factors such as head nodding, forward leaning, and less mutual gaze correlated with positive health outcomes (Beck, Daughtridge, & Sloane, 2002). Communications skills can be, and are taught, as part of medical education in many medical schools worldwide. Studies show that such training positively influences patients' health outcomes including blood pressure and glucose stability (Inui, Yourtee, & Williamson, 1976). Interestingly, doctors' communication skills also influence patients' decision making (van den Brink-Muinen et al., 2006), an important finding in an era where patients take a more active role in their health care.

Cross-References

- ▶ Communication, Nonverbal
- ▶ Education, Patient
- ▶ Empathy
- ▶ Empowerment

References and Readings

Beck, R. S., Daughtridge, R., & Sloane, P. D. (2002). Physician-patient communication in the primary care office: A systematic review. *Journal of the American Board of Family Practice, 15*, 25–38.

Di Blasi, Z., Harkness, E., Ernst, E., Georgiou, A., & Kleijnen, J. (2001). Influence of context effects on health outcomes: A systematic review. *Lancet, 357*, 757–762.

Inui, T. S., Yourtee, E. L., & Williamson, J. W. (1976). Improved outcomes in hypertension after physician tutorials. A controlled trial. *Annals of Internal Medicine, 84*, 646–651.

Ong, L. M., de Haes, J. C., Hoos, A. M., & Lammes, F. B. (1995). Doctor-patient communication: A review of the literature. *Social Science & Medicine, 40*, 903–918.

van den Brink-Muinen, A., van Dulmen, S. M., de Haes, H. C., Visser, A. P., Schellevis, F. G., & Bensing, J. M. (2006). Has patients' involvement in the decision-making process changed over time? *Health Expect, 9*, 333–342.

Communication, Nonverbal

Ross Buck
Communication Sciences and Psychology, University of Connecticut, Storrs, CT, USA

Synonyms

Body language

Definition

Communication involves three elements: sender, receiver, and message. In nonverbal communication, the message does not involve words, but rather employs *body language*. There are three major sorts of nonverbal communication. S*ymbolic nonverbal communication* is the intentional encoding of a message that is decoded by the receiver, the grammar and vocabulary of which must be learned by both sender and receiver. It is propositional in that it is capable of logical analysis (e.g., it can be false). Symbolic nonverbal communication includes sign language, finger spelling, and pantomime, as well as facial expressions and gestures associated with language. In Ekman and Friesen's (1969) analysis, the latter include *emblems* with specific "dictionary" definitions, *illustrators* of what is said, and *regulators* of interaction flow. Left hemisphere damage produces deficits in both linguistic and symbolic-nonverbal communication.

Spontaneous communication involves the display of a motivational-emotional state by the sender and a pickup of that display by the receiver. It is non-intentional, based upon innate displays and preattunements that coevolved, that is, that evolved simultaneously with the function of communication. Preattunements may be associated with mirror neuron systems that respond immediately and automatically to displays. The elements of spontaneous communication are *signs*, being inherent aspects of the referent (as smoke is a sign of fire). If the sign is present, the referent must be present by definition so that spontaneous communication is nonpropositional. Spontaneous displays include facial expressions, affective vocal prosody or paralanguage, postures and gestures, eye behaviors, touch (haptics), spatial behaviors (proxemics), and olfactory cues (e.g., pheromones). Right hemisphere damage produces deficits in communication via facial expression and affective prosody.

The third sort of nonverbal communication involves the intentional management of the display by the sender to manipulate the receiver (*deception*) or to follow *display rules*: learned rules about what displays are appropriate under what circumstances. Buck and van Lear (2002) termed this *pseudospontaneous communication*: it is symbolic on the part of the sender but spontaneous on the part of the receiver. The ability to influence others' emotions successfully is an important aspect of *charisma*. Ekman and Friesen (1975) identified expression management techniques: a person might *modulate* the intensity of the display, *qualify* a felt display by adding an additional display, and *falsify* the display in several ways: *neutralizing* and showing no display, *simulating* an unfelt display, or *masking* what one actually feels by showing a different, unfelt display.

Cross-References

▶ Emotional Expression

References and Readings

Buck, R. (1984). *The communication of emotion*. New York: Guilford Press.

Buck, R., & Duffy, R. (1980). Nonverbal communication of affect in brain damaged patients. *Cortex, 16*, 351–362.

Buck, R., & van Lear, C. A. (2002). Verbal and nonverbal communication: Distinguishing symbolic, spontaneous, and pseudo-spontaneous nonverbal behavior. *Journal of Communication, 52*, 522–541.

Ekman, P., & Friesen, W. V. (1969). Nonverbal leakage and cues to deception. *Psychiatry, 32*, 88–105.

Ekman, P., & Friesen, W. V. (1975). *Unmasking the face*. Englewood Cliffs, NJ: Prentice-Hall.

Ross, E. (1981). The aprosodias: Functional-anatomic organization of the affective components of language in the right hemisphere. *Archives of Neurology, 38*, 561–569.

Community Coalitions

Benjamin Hidalgo
Department of Psychiatry, Medical College of Wisconsin, Milwaukee, WI, USA

Synonyms

Community collaboration; Community partnership

Definition

The definition of community coalition can vary depending on the discipline of origin and different variables of interest (Gentry, 1987). However, a common definition used in community health is "a group of individuals representing diverse organizations, factions, or constituencies within the community who agree to work together to achieve a common goal" (Feighery & Rogers, 1990).

Description

Types of Coalitions

Historically, a diverse number of models have been conceptualized through which to understand the ways in which coalitions function in their communities. These include collaboration approaches, empowerment, asset-based approaches, constructions of risk and protective factors for intervention development, citizenship models promoting citizen participation, and promotion of community development (Francisco, Fawcett, Wolff, & Foster, 1996). Other approaches to understanding coalitions focus, more specifically, on optimal internal functioning of these organizations (e.g., Allen, 2005 and Foster-Fishman, Berkowitz, Lounsbury, Jacobson, & Allen, 2001).

Community Coalition Action Theory

While each of these emphasis and proposed mechanisms of action includes their own framework for understanding the successful development of a coalition, the single most comprehensive framework is the Community Coalition Action Theory as proposed by Frances Butterfoss and Michelle Kegler (2009). This examination of the structure and development of coalitions specifically in community change contexts was formulated through extensive research, practice, and review of the field. It is comprised of 14 major constructs each with its own set of theory propositions.

Construct 1: Stages of Development

In this construct, it is proposed that coalition building is cyclical. Coalitions develop in three general stages (formation, maintenance, and institutionalization), but these stages recycle as new members are recruited, plans change, or new issues are added. At each stage, specific factors unique to that stage and to that coalition enhance coalition function and progression to the next stage.

Construct 2: Community Context

Here it is proposed that contextual factors have a significant impact on the function and effectiveness of the coalition. These factors include but are not limited to geography, sociopolitical environment, social norms surrounding collaborative efforts, and timing.

Construct 3: Lead Agency/Convener Group

Coalitions form when a lead agency or convening organization responds to an opportunity, threat, or mandate. Through their review of the state of the field, the authors propose here that the formation of the coalition is more likely when the convener group provides support and resources during the formation stage (e.g., technical assistance, financial and material support, credibility, and networks and contacts). They also argue that enlisting community gatekeepers to develop credibility and trust with others in the community is a way to increase the success of coalition formation.

Construct 4: Coalition Membership

The authors propose, around membership, that coalitions begin by recruiting an initial core group of highly committed members. They also propose that effective coalitions eventually expand this established core group to include a broader constituency of partners that represent the more diverse needs, interests, and groups in the community.

Construct 5: Operations and Process

In order to ensure an effective internal process, five necessary components are proposed: open and frequent communication among staff members, shared and formalized decision making, effective conflict management, positive relationships among members, and the perception by members

that the benefits of participation outweigh the costs of participation.

Construct 6: Leadership and Staffing
Here it is proposed that effective coalition functioning, collaboration, and planning are improved by strong leadership and skilled, paid, staff.

Construct 7: Structure
The proposition in this construct is that having formalized rules, roles, structures, and procedures leads to routinized operations being better sustained and to overall coalition effectiveness.

Construct 8: Pooled Member and External Resources
Here it is proposed that that synergistic pooling of resources from members and from the community leads to effective assessment, planning, and implementation strategies.

Construct 9: Member Engagement
The authors propose that satisfied and committed members will participate more fully in the work of the organization.

Construct 10: Assessment and Planning
The proposition here points to evidence that shows that successful implementation of coalition efforts is more likely when comprehensive assessment and planning occur.

Construct 11: Implementation of Strategies
The proposition here is that community change is more likely to occur of coalitions direct their efforts at multiple levels.

Construct 12: Community Change Outcomes
This proposition highlights the fact that coalitions that can change community policies, practices, and environments are more likely to achieve long-term success and increases in community capacity to address future issues.

Construct 13: Health and Social Outcomes
Here the authors propose that the ultimate indicator of coalition effectiveness is improvement in health and social outcomes.

Construct 14: Community Capacity
The final construct in this model proposes that, as a result participating in a successful coalition, organizations, and community members, achieve increases in capacity and social capital that allow them to address health and social issues in the future.

Coalition Evaluation

Historically, there have been a wide variety of approaches to evaluating the effectiveness of coalitions. These evaluations have employed qualitative, quantitative, and mixed methodologies and typically have examined coalitions at one or more of the following levels: process and infrastructure, specific programs and interventions, health status or community change outcome, and extent of community capacity building (Butterfoss, 2007; Granner & Sharpe, 2004). Given the wide variety of coalitions, their reasons for existing, and the mechanism by which they propose to act on their communities, Berkowitz (2001) argues that this diverse set of evaluation strategies is necessary if evaluators are to effectively understand the degree to which coalitions are successful.

While acknowledging the necessary diversity of coalition evaluation strategies, Butterfoss (2007) proposes ten overall principles to guide coalition evaluation:

1. The evaluation should involve a process of partnership between coalition and evaluator.
2. The evaluation design should be informed by research, previous evaluations, and community wisdom.
3. The evaluation should actively include the participation of all stakeholders.
4. The evaluation process should be used to assess, reflect, improve, and inform.
5. Expectations for the evaluation should be made clear for all stakeholders.
6. Issues of power and privilege should be explicitly identified and addressed at the start.
7. The evaluation should constantly seek to foster positive relationships and trust among evaluators, community participants, practitioners, and funders.

8. The process of evaluation should be closely integrated into ongoing functions and activities.
9. The evaluation process, itself, should be periodically reevaluated to ensure that it continues to meet the coalition's needs and in order to apply findings to ongoing decision making and learning.
10. Findings should be shared frequently with all stakeholders in a format that is accessible to them.

Evaluations the follow these principles can help coalitions in a number of ways (Butterfoss, 2007; Butterfoss & Francisco, 2004): providing accountability to the community and funders for the actions of the coalition, determining whether coalition objectives are met, improving program implementation, increasing awareness and support of the coalition in the community, informing policy decisions, and contributing the empirical literature on best practices.

Cross-References

- ▶ Community-Based Health Programs
- ▶ Community-Based Participatory Research
- ▶ Health Promotion and Disease Prevention

References and Readings

Allen, N. E. (2005). A multi-level analysis of community coordinating councils. *American Journal of Community Psychology, 35*(1–2), 49–63.

Berkowitz, B., & Wolff, T. (1999). *The spirit of the coalition*. Washington, DC: American Public Health Association.

Berkowitz, B. (2001). Studying the outcomes of community-based coalitions. *American Journal of Community Psychology, 29*(2), 213–227.

Butterfoss, F., & Francisco, V. T. (2004). Evaluating community partnerships and coalitions with practitioners in mind. *Health Promotion Practice*, *5*, 108–114.

Butterfoss, F. D., Goodman, R. M., & Wandersman, A. (1996). Community coalitions for prevention and health promotion: Factors predicting satisfaction, participation, and planning. *Health Education Quarterly, 23*(1), 65–79.

Butterfoss, F. D. (2007). *Coalitions and partnerships in community health*. San Francisco: Jossey-Bass.

Butterfoss, F. D., Kegler, M. C., & Francisco, V. T. (2008). Mobilizing organizations for health promotion: Theories of organizational change. In K. Glanz, B. K. Rimer, & K. Viswanath (Eds.), *Health behavior and health education: Theory, research, and practice* (4th ed., pp. 335–361). San Francisco: Jossey-Bass.

Cramer, M. E., Atwood, J. R., & Stoner, J. A. (2006). Measuring community coalition effectiveness using the ICE© instrument. *Public Health Nursing, 23*(1), 74–87.

Downey, L. M., Ireson, C. L., Slavova, S., & McKee, G. (2008). Defining elements of success: A critical pathway of coalition development. *Health Promotion Practice, 9*(2), 130–139.

Butterfoss, F. D., & Kegler, M. C. (2009). The community coalition action theory. In R. J. DiClemente, R. A. Crosby, M. C. Kegler, R. J. DiClemente, R. A. Crosby, & M. C. Kegler (Eds.), *Emerging theories in health promotion practice and research* (2nd ed., pp. 237–276). San Francisco: Jossey-Bass.

Foster-Fishman, P. G., Berkowitz, S. L., Lounsbury, D. W., Jacobson, S., & Allen, N. A. (2001). Building collaborative capacity in community coalitions: A review and integrative framework. *American Journal of Community Psychology, 29*(2), 241–261.

Feighery, E., & Rogers, T. (1990). *Building and maintaining effective coalitions*. Palo Alto, CA: Health Promotion Resource Center, Stanford Center for Research in Disease Prevention.

Francisco, V. T., Fawcett, S. B., Wolff, T. J., & Foster, D. L. (1996). *Coalition typology: Toward a research-based typology of health and human service coalitions*. AHEC/Community Partners. Retrieved August 17, 2011, from Community Partners website: http://www.compartners.org/stacks/archive/hcm/coalition_typology.pdf

Gentry, M. E. (1987). Coalition formation and processes. *Social Work with Groups: A Journal of Community and Clinical Practice, 10*, 39–54.

Granner, M. L., & Sharpe, P. A. (2004). Evaluating community coalition characteristics and functioning: A summary of measurement tools. *Health Education Research, 19*(5), 514–532.

Kramer, J. S., Philliber, S., Brindis, C. D., Kamin, S. L., Chadwick, A. E., & Revels, M. L. (2005). Coalition models: Lessons learned from the CDC's community coalition partnership programs for the prevention of teen pregnancy. *Journal of Adolescent Health, 37*(S3), S20–S30.

Lentz, B. E., Imm, P. S., Yost, J. B., Johnson, N. P., Barron, C., Lindberg, M. S., et al. (2005). Empowerment evaluation and organizational learning: A case study of a community coalition designed to prevent child abuse and neglect. In D. M. Fetterman & A. Wandersman (Eds.), *Empowerment evaluation principles in practice* (pp. 155–182). New York: Guilford Press.

Wolff, T. (2001). The future of community coalition building. *American Journal of Community Psychology, 29*, 263–268.

Community Collaboration

► Community Coalitions

Community Health Advisors

► Promotoras

Community Health Representatives

► Promotoras

Community Health Workers (CHW)

► Promotoras

Community Partnership

► Community Coalitions

Community Sample

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

Community samples are used in community trials, or community intervention trials, i.e., trials in which the intervention is implemented at the community level. This contrasts with clinical trials, where intervention is implemented at the level of the individual subject.

Consider the example of testing the dental health advantages of adding fluoride (fluoridation) to drinking water. Realistically, a study investigating the influence of fluoridation would need large community samples. A classic study was reported by Ast and Schlesinger (1956) in which the drinking water for one town in New York State was fluorinated and the water for a second town in the state was not. The towns were chosen to be as similar as possible so that any difference in dental health could reasonably be attributed to the influence of interest, i.e., presence or absence of fluoride in the water. The study provided compelling evidence that fluoridation is both effective in reducing dental caries and a safe public health practice.

Exposure status in community trials, therefore, is assigned to an entire community rather than to individuals. Typical outcomes of interest include the risk of disease or the frequency of a health behavior (Hartge & Cahill, 2008). Since the unit of observation is the community, the assessment of potential confounders can also occur at the community level, and thus appropriate care in that regard is needed.

Cross-References

► Bias
► Clinical Trial
► Randomization

References and Readings

Ast, D. B., & Schlesinger, E. R. (1956). The conclusion of a ten-year study of water fluoridation. *American Journal of Public Health, 46*, 265–271.

Hartge, P., & Cahill, J. (2008). Field methods in epidemiology. In K. J. Rothman, S. Greenland, & T. L. Lash (Eds.), *Modern epidemiology* (3rd ed., pp. 492–510). Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins.

Community-Based Health Programs

► Community-Based Participatory Research

Community-Based Participatory Research

Lee Sanders
Center for Health Policy and Primary Care Outcomes Research, Stanford University, Stanford, CA, USA

Synonyms

Community-based research

Definition

Community-based participatory research (CBPR) is a set of principles and techniques designed to involve community members as collaborators in every aspect of the research process, including design, funding, implementation, and dissemination (Higgins, Maciak, & Metzler, 2001; Israel et al., 1998). Fully realized, CBPR includes shared expertise between researcher and community, shared decision making, and mutual ownership of the research enterprise and its results. Effective CBPR normally results from a long-standing, trusting relationship between an academic research team and a community-based organization (CBO) (Israel et al., 1998; Viswanathan et al., 2004). CBPR is of particular value to health researchers, public health professionals, and community leaders attempting to address health disparities influenced by social determinants (e.g., socioeconomic status, race, ethnicity, literacy, nutrition, environmental health). CBPR also holds relevance for policymakers attempting to turn community-needs assessments into evidence-based action or to translate basic and clinical research findings into population-wide practice.

Research Process

CBPR adheres to the same high-quality research standards that apply to health and behavioral research designs, including observational studies, cohort studies, and randomized controlled trials. CBPR distinguishes itself, however, by involving community members (through needs assessments, iterative community-based meetings, and other opportunities for comment) in every stage of the research process. Beginning with the research question, community members help define the health outcomes, behaviors, and environmental factors to be addressed by the research proposal. A community advisory committee, normally chaired by a community-based stakeholder, is often an integral part of the research process. In the spirit of mutual expertise and collaboration, CBPR research protocols often employ community residents as members of the research team, and they may include support for research facilities and research materials housed inside a community-based facility (Stratford et al., 2003; Vander Stoep, Williams, Jones, Green, & Trupin, 1999). All study interventions, research trainings, survey materials, informed-consent documents, and other materials include input and guidance from community members. Measures employed in CBPR usually include social determinants of health and cost-effectiveness variables from the community perspective. Study results are normally shared with the community advisory committee or other community members for feedback and interpretation before they are shared with outside audiences. With attention to community standards and research ethics, results are also disseminated across the community. In the case of interventions determined to be effective, sustainability planning that includes community leaders is a critical element of the CBPR process.

Health Behavior Change

CBPR enables researchers to be sensitive and responsive to the cultural, political, and social context of health behaviors. This includes challenges and opportunities for influencing sensitive health behaviors (e.g., smoking, drug use, sexual practices, domestic violence, obtaining screening tests that involve pelvic or rectal exams) and other health behaviors that can only effectively

be addressed at the community level (e.g., nutrition, physical activity).

Ethical Considerations

CBPR may also present constraints for the conduct of ethical research. In choosing the primary research topic or question, a community-driven process may not yield a result that meets the academic considerations of relevance, novelty, and generalizability. Similarly, community members may object to the publication of study findings or interpretations, even if "objective" research methods were applied. Funding and other rewards for CBPR also may introduce ethical dilemmas. In optimal circumstances, community representatives and organizations should be reimbursed fairly for their participation. If addressed early and forthrightly, many of these ethical concerns may be mitigated. Effective should include gaining insight and assent from all available community leaders, providing appropriate training if appropriate to participants, ensuring financial and nonfinancial recognition for participants, and clarifying rules for ownership and use of study data, analyses, and publications.

Cross-References

- ▶ Cost-Effectiveness Analysis (CEA)
- ▶ Health Behaviors
- ▶ Participatory Research

References and Readings

Higgins, D. L., Maciak, B., & Metzler, M. (2001). CDC Urban Research C. CDC Urban Research Centers: Community-based participatory research to improve the health of urban communities. *Journal of Women's Health & Gender-Based Medicine, 10*(1), 9–15.

Israel, B. A., Schulz, A. J., Parker, E. A., et al. (1998). Review of community-based research: Assessing partnership approaches to improve public health. *Annual Review of Public Health, 19*, 173–202.

Minkler, M., & Wallerstein, N. (2003). *Community based participatory research for health*. San Francisco: Jossey-Bass.

Stratford, D., Chamblee, S., Ellerbrock, T. V., et al. (2003). Integration of a participatory research strategy into a rural health survey. *Journal of General Internal Medicine, 18*(7), 586–588.

Vander Stoep, A., Williams, M., Jones, R., Green, L., & Trupin, E. (1999). Families as full research partners: What's in it for us? *Journal of Behavioral Health Services and Research, 26*(3), 329–344.

Viswanathan, M., Ammerman, A., Eng, E., Gartlehner, G., Lohr, K. N., Griffith, D., Rhodes, S., Samuel-Hodge, C., Maty, S., Lux, L., Webb, L., Sutton, S. F., Swinson, T., Jackman, A., Whitener, L. (2004, July). *Community-based participatory research: Assessing the evidence*. Evidence Report/Technology Assessment No. 99 (Prepared by RTI-University of North Carolina Evidence-based Practice Center under Contract No. 290-02-0016). AHRQ Publication 04-E022-2. Rockville, MD: Agency for Healthcare Research and Quality.

Community-Based Research

▶ Community-Based Participatory Research

Comorbidity

Amy Wachholtz
Department of Psychiatry, University of Massachusetts Medical School, Worcester, MA, USA

Synonyms

Co-occuring

Definition

Comorbidity occurs when an individual experiences two or more disorders at the same time (Eaton, 2006). Comorbidities can occur sequentially, or they can become symptomatic simultaneously. Disorders that are considered comorbidities can be either physical or psychological in nature. It is a common occurrence

that a disorder in one domain (e.g., a physical disorder of spinal cord injury) will trigger or exacerbate a disorder in another domain (e.g., a psychological disorder of depression). Two disorders within the same domain are also considered comorbidities (e.g., depression and anxiety, or chronic obstructive pulmonary disorder and ischemic heart disease). There are some disorders that are such frequent comorbidities that they may eventually be combined under a single label and treated as a single syndrome (e.g., metabolic syndrome which often includes high blood pressure, Type 2 diabetes, obesity, hypercholesterolemia, and dyslipidemia).

Treatment providers will often assess for comorbidities in order to tailor the best treatment approach to that individual. Being aware of a patient's comorbidities allows a treatment provider to educate the patient, consider additional treatment options, and potentially begin treatment for the comorbidity.

Cross-References

- ▶ Anxiety
- ▶ Anxiety and Heart Disease
- ▶ Cancer and Smoking
- ▶ Heart Disease and Cardiovascular Reactivity
- ▶ Heart Disease and Smoking
- ▶ Heart Disease and Stress
- ▶ Insulin Resistance (IR) Syndrome
- ▶ Metabolic Syndrome
- ▶ Obesity: Causes and Consequences
- ▶ Sleep and Health
- ▶ Unipolar Depression

References and Readings

Eaton, W. W. (2006). *Medical and psychiatric comorbidity over the course of life*. Arlington, VA: American Psychiatric Publishing.

Comparative Effectiveness Methodology

- ▶ Comparative Effectiveness Research

Comparative Effectiveness Research

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

CER; Comparative effectiveness methodology

Definition

The definition of Comparative Effectiveness Research (CER) for the Federal Coordinating Council reads as follows (HHS.gov):

Comparative effectiveness research is the conduct and synthesis of systematic research comparing different interventions and strategies to prevent, diagnose, treat, and monitor health conditions. The purpose of this research is to inform patients, providers, and decision-makers, responding to their expressed needs, about which interventions are most effective for which patients under specific circumstances. To provide this information, comparative effectiveness research must assess a comprehensive array of health-related outcomes for diverse patient populations. Defined interventions compared may include medications, procedures, medical and assistive devices and technologies, behavioral change strategies, and delivery system interventions. This research necessitates the development, expansion, and use of a variety of data sources and methods to assess comparative effectiveness.

The inclusion of "behavioral change strategies" makes CER of immediate interest in the field of behavioral medicine.

Description

Sox and Greenfield (2009) discussed various important steps in the development and formalization of CER. A seminal article was published

by Wilensky (2006), and an Institute of Medicine (IOM) report called for a national initiative of research that would support better decision making about interventions in health care (IOM, 2008). A major step occurred when President Obama signed into law the American Recovery and Reinvestment Act of 2009 (ARRA), which allotted US$1.1 billion to CER. The legislation created a Federal Council on CER, and asked the IOM to elicit input from a broad array of stakeholders on which research topics should have the highest priority for funding through the ARRA and to then develop a list of the highest-priority topics for the Secretary of Health and Human Services to consider. The IOM committee formulated a more succinct definition of CER: "CER is the generation and synthesis of evidence that compares the benefits and harms of alternative methods to prevent, diagnose, treat and monitor a clinical condition, or to improve the delivery of care. The purpose of CER is to assist consumers, clinicians, purchasers, and policy makers to make informed decisions that will improve health care at both the individual and population levels."

As Kupersmith and Ommaya (2010) noted, The US Department of Veterans Affairs (VA) has a long history of conducting CER. Along with pharmaceutical interventions, they have had a large focus on behavioral interventions. The success of their CER program has been facilitated by several important aspects of scientific infrastructure related to (1) research question refinement, (2) study design, planning, and coordination, (3) evidence synthesis, and (4) implementation research. In publications that had VA coauthors in two major medical journals, 25% of the published studies were classified as CER. In the future, the CER enterprise will move toward increased input from clinicians in the choice of research topics and enhanced consideration of other methodologies besides the randomized controlled trial. Concato et al. (2010) reviewed and discussed the use of observational studies in CER, focusing on the following: (1) understanding how observational studies can provide accurate results, comparable to those from randomized clinical trials; (2) recognizing strategies used in selected newer methods for conducting observational studies; (3) reviewing selected observational studies from the Veterans Health Administration; and (4) appreciating the importance of fundamental methodological principles when conducting or evaluating individual studies.

Bonham and Solomon (2010) observed that the success of the federal investment in CER will hinge on using the power of science to guide reforms in health-care delivery and improve patient-centered outcomes (as will be true for other sources of investment in this area). They noted that "Translating the results of comparative effectiveness research into practice calls for the rigors of implementation science to ensure the efficient and systematic uptake, dissemination, and endurance of these innovations." Academic medicine is in a strong position to help in various ways: thoroughly integrating its research and training missions with clinical care that is focused on patient-centered outcomes; building multidisciplinary teams that include a wide range of experts such as clinicians, clinical and implementation scientists, systems engineers, behavioral economists, and social scientists; and training future care providers, scientists, and educators to carry innovations forward (Bonham and Solomon, 2010).

An informative discussion was recently provided by Blumenthal (2011) in a paper entitled "New frontiers in cardiovascular behavioral medicine: Comparative effectiveness of exercise and medication in treating depression." As noted, Blumenthal and his colleagues began investigations into cardiac rehabilitation, which they considered to be a "new frontier for behavioral medicine." That field of investigation laid groundwork that has now provided the opportunity to compare exercise therapy, an established component of cardiac rehabilitation, with antidepressant pharmacotherapy as a treatment for depression in cardiac disease patients. Two randomized clinical trials have now been conducted, and, following a detailed discussion of their findings, the author commented as follows: "While these results are preliminary and should be interpreted with caution, it appears that exercise may be comparable with conventional

antidepressant medication in reducing depressive symptoms, at least for patients who are willing to try it, and maintenance of exercise reduces the risk of relapse" (Blumenthal, 2011).

Cross-References

- ▶ Behavioral Medicine
- ▶ Cardiac Rehabilitation
- ▶ Depression: Treatment
- ▶ Institute of Medicine
- ▶ Randomized Clinical Trial

References and Readings

Blumenthal, J. A. (2011). New frontiers in cardiovascular behavioral medicine: comparative effectiveness of exercise and medication in treating depression. *Cleveland Clinical Journal of Medicine, 78*(Suppl. 1), S35–S43.

Bonham, A. C., & Solomon, M. Z. (2010). Moving comparative effectiveness research into practice: Implementation science and the role of academic medicine. *Health Affairs (Millwood)*, *29*, 1901–1905.

Blumenthal, J. A., Califf, R., Williams, R. S., & Hindman, M. (1983). Cardiac rehabilitation: A new frontier for behavioral medicine. *Journal of Cardiac Rehabilitation, 3*, 637–656.

Concato, J., Lawler, E. V., Lew, R. A., Gaziano, J. M., Aslan, M., & Huang, G. D. (2010). Observational methods in comparative effectiveness research. *American Journal of Medicine, 123*(12 Suppl. 1), e16–e23.

Hoffman, B., Babyak, M., Craighead, W. E., Sherwood, A., Doraiswamy, P. M., Coons, M. J., et al. (2010). Exercise and pharmacotherapy in patients with major depression: One-year follow-up of the SMILE study. *Psychosomatic Medicine, 73*, 127–133.

Huang, G. D., Ferguson, R. E., Peduzzi, P. N., & O'Leary, T. J. (2010). Scientific and organizational collaboration in comparative effectiveness research: The VA cooperative studies program model. *American Journal of Medicine, 123*(12 Suppl. 1), e24–e31.

Institute of Medicine (Eden, J., Wheatley, B., McNeil, B., Sox, H., eds.). (2008). *Knowing what works in health care: A roadmap for the nation*. Washington, DC: National Academies Press.

Kupersmith, J., & Ommaya, A. K. (2010). The past, present, and future of comparative effectiveness research in the US Department of Veterans Affairs. *American Journal of Medicine, 123*(12 Suppl 1), e3–e7.

O'Connell, J. M., & Griffin, S. (2011). Overview of methods in economic analyses of behavioral interventions to promote oral health. *Journal of Public Health Dentistry, 71*(Suppl. 1), S101–S118.

Rich, E. C., Bonham, A. C., & Kirch, D. G. (2011). The implications of comparative effectiveness research for academic medicine. *Academic Medicine, 86*, 684–688.

Sox, H. C., & Greenfield, S. (2009). Comparative effectiveness research: A report from the institute of medicine. *Annals of Internal Medicine*, *151*, 203–205.

United States Health and Human Services. HHS.gov. Accessed December 14th, 2011, from http://www.hhs.gov/recovery/programs/cer/draftdefinition.html.

Wilensky, G. R. (2006). Developing a center for comparative effectiveness information. *Health Affairs (Millwood), 25*, w572–w585.

Comparator Group

- ▶ Control Group

Complementary and Alternative Medicine

- ▶ Alternative Medicine
- ▶ Integrative Medicine

Complex Traits

Abanish Singh
Duke University Medical Center, Durham, NC, USA

Definition

Mendelian genetics put forward the concept of dominant and recessive traits, where the phenotypes are controlled by single genes. These traits are known as monogenic or Mendelian traits. Though there are many genes that control Mendelian traits, in contrast, there are features or traits in human genetics which are controlled by multiple genes and whose inheritance does not follow the rules of Mendelian genetics. Such traits are known as complex traits.

Examples of complex traits include disorders such as autism, cardiac disease, cancer,

diabetes, Alzheimer's disease, and asthma. Complex traits are believed to result from gene-gene and gene-environment interactions, genetic heterogeneity, and potentially other yet unknown reasons.

Cross-References

- ▶ Gene-Environment Interaction
- ▶ Gene-Gene Interaction

References and Readings

Frazer, K. A., Murray, S. S., Schork, N. J., & Topol, E. J. (2009). Human genetic variation and its contribution to complex traits. *Nature Reviews Genetics, 10*, 241–251.

Glazier, A. M., Nadeau, J. H., & Aitman, T. J. (2002). Finding genes that underlie complex traits. *Science, 298*, 2345–2350.

Compliance

- ▶ Adherence
- ▶ Medical Utilization

Complications of Atherosclerosis

- ▶ Peripheral Arterial Disease (PAD)/Vascular Disease

Complimentary and Alternative Medicine

- ▶ Alternative Medicine

Composition

- ▶ Family, Structure

Computed Axial Tomography

- ▶ Computerized Axial Tomography (CAT) Scan

Computed Tomography

- ▶ Computerized Axial Tomography (CAT) Scan

Computed Transaxial Tomography

- ▶ CAT Scan

Computer Cartography

- ▶ Geographic Information System (GIS) Technology

Computer-Based Patient Record

- ▶ Electronic Health Record

Computerized Axial Tomography

- ▶ Computerized Axial Tomography (CAT) Scan
- ▶ CAT Scan

Computerized Axial Tomography (CAT) Scan

Siqin Ye
Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Synonyms

CAT scan; Computed axial tomography; Computed tomography; Computerized axial tomography; CT scan; X-ray computed tomography

Definition

Computed axial tomography, or CAT scan, utilizes computer algorithms to combine series of two-dimensional X-ray images (tomographs) to produce three-dimensional representations of the insides of objects. It is used in clinical medicine to noninvasively visualize potential pathologies inside the human body. By eliminating the superimposition of adjacent structures and distinguishing different tissue types based on their densities, CAT scans are able to generate high-resolution images of particular anatomic regions. Intravenous and oral contrast agents can also be used to further enhance image quality, helping to distinguish vasculature and bowel lumen from the surrounding tissue, respectively. Common uses of CAT scan include scanning of the head/brain, to assess strokes, intracranial bleeding, or tumors; of the chest, to assess lung parenchyma, pulmonary embolism, or diseases of the great vessels such as thoracic aortic aneurysm or dissection; of the abdomen and pelvis, to assess pathologies such as kidney stones, appendicitis, pancreatitis, diverticulitis, intra-abdominal abscesses, and various visceral malignancies; and of the bones, to identify osteoporosis and delineate complex fractures (Buzug, 2008). In recent years, there has also been ongoing development of multidetector computed tomography (MDCT) scanners, allowing for further enhanced spatial and temporal resolution. These technological advances have made possible new modalities of CT imaging, such as virtual colonoscopy for colon cancer screening or cardiac CT to visualize coronary arteries as well as other structures of the beating heart (Achenbach & Daniel, 2008).

The popularity of CAT scans has also led to concerns with regard to their potential adverse effects. In addition to the risk of renal injury associated with the use of iodinated intravenous contrast, there has been recognition that the widespread use of CAT scans has led to increased radiation exposure for the general population, with recent estimates showing they may contribute to 1.5–2.0% of all cancers in the United States (Brenner & Hall, 2007). Due to these considerations, it has been recommended that in addition to developing better protocols with lower radiation doses, the decision to obtain a CAT scan for an individual patient should be made judiciously, taking into careful consideration the trade-off between clinical benefit and potential harm, so as to avoid excess testing and radiation exposure.

Cross-References

▶ CAT Scan

References and Readings

Achenbach, S., & Daniel, W. G. (2008). Computed tomography of the heart. In P. Libby, R. O. Bonow, D. L. Mann, D. P. Zipes, & E. Braunwald (Eds.), *Braunwald's heart disease: A textbook of cardiovascular medicine* (pp. 415–438). Philadelphia: Saunders Elsevier.

Brenner, D. J., & Hall, E. J. (2007). Computed tomography-an increasing source of radiation exposure. *The New England Journal of Medicine, 357*, 2277–2284.

Buzug, T. (2008). *Computed tomography: From photon-statistics to modern cone-beam CT*. Berlin, Germany: Springer.

Computerized Tomography (CT)

▶ Brain, Imaging
▶ Neuroimaging

Concentration

▶ Coffee Drinking, Effects of Caffeine
▶ Meditation
▶ Transcendental Meditation

Concordance

Jennifer Wessel
Public Health, School of Medicine, Indiana University, Indianapolis, IN, USA

Definition

Among a pair of twins, the twin pair exhibits an identical phenotype. Within the twin pair, both

individuals share or lack the trait or disease under investigation. Measuring concordance can also be done among siblings or other family members. Greater concordance in MZs versus DZs is suggestive evidence for a genetic contribution to a disease. Concordance is measured as the number of pairs that both exhibit (or not) the trait divided by the total and presented as a percentage.

References and Readings

Nussbaum, R. L., Mc Innes, R. R., & Willard, H. F. (2001). *Genetics in medicine* (6th ed.). Philadelphia: W.B. Saunders.

Spector, T. D., Snieder, H., & MacGregor, A. J. (2000). *Advances in twin and sib-pair analysis* (1st ed.). London: Greenwich Medical Media.

Concurrent Control

▶ Control Group

Concussion

▶ Traumatic Brain Injury

Conditioned Response

▶ Placebo and Placebo Effect

Condom Protected Sex

▶ Condom Use

Condom Use

Rick Crosby
University of Kentucky, Lexington, KY, USA

Synonyms

Barrier method of protection; Condom protected sex; Prophylactic use; Protected sex

Definition

Condom use implies that an FDA-approved latex condom covered the entire head and shaft of the penis from the start of sex (initial penetration) until sex ended (no more penetration). The term also implies that the condom was used properly, thereby avoiding breakage, spillage, leaking, and slipping off the penis. The term applies to penile-vaginal penetration, penile-oral penetration, and penile-anal penetration. In research studies, when a person uses condoms consistently and correctly, he is classified as "having no risk exposure," meaning that he has not engaged in unprotected vaginal sex (UVS), unprotected oral sex (UOS), or unprotected anal sex (UAS). The same classification applies to females who are the recipients of penetrative sex that is 100% condom protected. In addition, condom use can also imply the consistent and correct use of a polyurethane sheath that is closed at one end (intended to cover the cervix) and open at the other end (for penile penetration). Known as the "female condom," this device is also worn by males who will be receptive partners in the act of penile-anal sex.

Description

Condom use is currently the single best method of reducing the global AIDS pandemic and the ever-expanding pandemic of sexually transmitted infections such as Chlamydia, gonorrhea, syphilis, trichomoniasis, genital herpes, and chancroid. The degree of protection conferred by condom use varies as a function of the infection. Evidence is strongest relative to protection against the transmission and the acquisition of the human immunodeficiency virus (HIV). Emerging evidence suggests that condoms can be highly protective against the male insertive partner's acquisition of gonorrhea, Chlamydia, and syphilis. Evidence also supports the protective value of condoms against these same infections for the receptive partner. However, for infections such as human papillomavirus (HPV) and genital herpes (HSV), the protective value of condoms is not

nearly as good simply because these infections spread by skin-to-skin contact of genital areas that condoms do not cover. Nonetheless, it is indeed correct to say that condom use does offer partial protection against HPV and HSV.

Despite the tremendous public health value of condom use, prevailing sociopolitical climates have frequently precluded efforts to educate men and women about condoms and their correct use. This lack of education has proven to be problematic in that a large number of studies show that men and women experience multiple errors and problems when using condoms. The most common error, reported by both men and women, is known as incomplete use. This means that the condom was put on after penetrative sex had begun and/or it was taken off before penetrative sex had ended. Both behaviors have been linked to arousal and erection issues as well as ill-fitting condoms. In addition, condoms that lose their lubrication during sex may be removed prematurely (rather than simply adding lubricant). Breakage is the next most common problem with condom use. Rather than being a problem inherent in the production of condoms, breakage occurs as a consequence of user errors such as applying oil-based lubrication, not leaving an air space in the reservoir tip upon application, using condoms that are too small for the penis, letting condoms contact sharp objects (including teeth and jewelry) and failure to add adequate amounts of water-based lubricants during prolonged sex. The next most common problem is having condoms slip off the penis, either during penetrative sex or during the act of withdrawing the condomized penis after male ejaculation occurs. Loose-fitting condoms, not unrolling condoms all the way to the base of the penis, erection issues, use of erection enhancing drugs, and poorly lubricated condoms have all been associated with slippage during sex. Mistakes that people make when using condoms include putting the unrolled condom on the penis upside down and then "flipping" it over so it will unroll (thereby introducing per-cum [semen] into the outside tip of the condom thus compromising protection). Studies have shown that people will "switch" from one sexual act to another without changing condoms in between acts, thereby creating issues with disease transfer. The sheer volume of condom use errors and problems reported by men and women strongly suggests that all too often condoms fail because the users lacked proper education. These forms of condom failure are also an unfortunate omission in studies of condom effectiveness, thereby creating a bias toward the null hypothesis (i.e., that condoms do not work).

A broad range of behavioral and social issues inextricably surround condom use. For example, a robust finding has been that people are more likely to use condoms with new or "casual" sex partners and far less likely to do so with established partners. Thus, a challenge in behavioral medicine is promoting condom use among at-risk, established, couples. Also, condom use and the use of hormonal contraceptives tend to be inversely correlated, meaning that condom use is reduced or abandoned when a couple begins using highly reliable contraception methods. Here, the challenge in behavioral medicine is to promote the dual use of condoms and contraception. A similar dynamic may exist in relation to vaccines for HPV and microbicidal agents designed to prevent HIV infection – as people perceive less risk as a consequence of the vaccine or microbicide they may reduce or abandon condom use. Condom use will also be problematic in cultures (or among couples) that value reproduction – an inherent downside of condom use for disease prevention is that the behavior precludes desired conception. Low arousability and erection loss are also issues that greatly affect condom use, with several studies indicating incomplete use or lack of use to increase arousability and help maintain erection. The challenge here to behavioral medicine is integrating sex therapy with STI prevention.

References and Readings

Crosby, R. A., Milhausen, R., Yarber, W. L., Sanders, S. A., & Graham, C. A. (2008). Condom "Turn Offs" among adults: An exploratory study. *International Journal of STD and AIDS, 19*, 590–594.

Crosby, R. A., Sanders, S. A., Yarber, W. L., & Graham, C. A. (2003). Condom use errors and problems: A neglected aspect of studies assessing condom effectiveness. *American Journal of Preventive Medicine, 24*, 367–370.
Crosby, R. A., Yarber, W. L., Sanders, S. A., et al. (2007). Men with broken condoms: Who and why? *Sexually Transmitted Infections, 83*, 71–75.
Holmes, K. K., Levine, R., & Weaver, M. (2004). Effectiveness of condoms in preventing sexually transmitted infections. *Bulletin of the World Health Organization, 82*, 454–461.
Misovich, S. J., Fisher, J. D., & Fisher, W. A. (1997). Close relationships and elevated HIV risk behavior: Evidence and possible underlying psychological processes. *Review of General Psychology, 1*(1), 72–107.
Sheeran, P., Abraham, C., & Orbell, S. (1999). Psychosocial correlates of heterosexual condom use: a meta-analysis. *Psychological Bulletin, 125*(1), 90–132.

Confidentiality

Marianne Shaughnessy
School of Nursing, University of Maryland, Baltimore, MD, USA

Synonyms

HIPAA; Patient privacy; Privacy

Definition

Ethical principle that dictates communications are "privileged" and may not be discussed or divulged to third parties.

Description

Confidentiality is a term that commonly applies to conversations between health care providers and patients. Legal protections are available to prevent physicians from revealing certain discussions with patients, even under oath in court. However, the rule only applies to information shared between physician and patient during the course of providing medical care. Traditionally, medical ethics has viewed the duty of confidentiality as a relatively nonnegotiable tenet of medical practice. Issues regarding the confidentiality of health information passed between patients, providers, and insurers led to the evolution of regulatory language to protect patient privacy.

The Health Insurance Portability and Accountability Act of 1996 (HIPAA) includes in its language specific direction for the management of protected health information (PHI) in both clinical and research arenas. The Privacy Rule protects all "individually identifiable health information" held or transmitted by a covered entity or its business associate, in any form or media, whether electronic, paper, or oral. The Privacy Rule calls this information "protected health information (PHI)."

"Individually identifiable health information" is information, including demographic data, that relates to the individual's past, present, or future physical or mental health or condition; the provision of health care to the individual; or the past, present, or future payment for the provision of health care to the individual, and that identifies the individual or for which there is a reasonable basis to believe it can be used to identify the individual. Individually identifiable health information includes many common identifiers (e.g., name, address, birth date, social security number). There are no restrictions on the use or disclosure of de-identified health information. De-identified health information neither identifies nor provides a reasonable basis to identify an individual.

Privacy rules apply only to covered entities. Covered entities are defined as a healthcare provider (physicians, nurse practitioners, psychologists, dentists, clinics, nursing homes, pharmacies, etc.), health plans (insurance companies, Health Maintenance Organization (HMOs), etc.), or a healthcare clearinghouse (entities that process nonstandard health information they receive from another entity into a standard format). Individuals, organizations, and agencies that meet the definition of a covered entity under HIPAA must comply with the rules' requirements to protect the privacy

and security of health information and must provide individuals with information regarding their rights with respect to their health information. If an entity is not a covered entity, it does not have to comply with the Privacy Rule.

Researchers are also bound by the rules regarding confidentiality of protected health information. Generally speaking, researchers are required to safeguard the privacy of all health information obtained in the course of screening or enrollment in a study to the extent permitted by law. In certain types of research, there may be a high risk of identifying information that if disclosed, could have adverse consequences for subjects or damage their financial standing, employability, insurability, or reputation. Certificates of confidentiality are issued by the National Institutes of Health (NIH) to protect identifiable research information from forced disclosure. They allow the investigator and others who have access to research records to refuse to disclose identifying information on research participants in any civil, criminal, administrative, legislative, or other proceeding, whether at the federal, state, or local level. By protecting researchers and institutions from being compelled to disclose information that would identify research subjects, certificates of confidentiality help achieve the research objectives and promote participation in studies by assuring confidentiality and privacy to participants.

Cross-References

- ► Protection of Human Subjects
- ► Research Participation, Risks and Benefits Of

References and Readings

U.S. Department of Health & Human Services Health Information Privacy. Accessed May 9, 2011 from http://www.hhs.gov/ocr/privacy/

U.S. Department of Health & Human Services National Institutes of Health Office of Extramural Research Certificates of Confidentiality Kiosk. Accessed May 12, 2011 from http://grants.nih.gov/grants/policy/coc/

Confounding Influence

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

When investigating the influence of a factor of interest, it is critically important to keep all other potentially relevant influences as constant as possible. That is, the only reasons for differences in how subjects respond to the treatments in a research study should be the nature of the treatments (interventions) themselves. Extraneous influences are called confounding influences: They make it harder to isolate and hence evaluate the degree of influence of the factor of interest.

The list of potential confounding influences for a given study can be extensive and vary from study to study. It is therefore the responsibility of the researcher to design the study and structure the study's research methodology such that confounding influences are controlled to the greatest degree possible.

One example from other entries can be found in the entry titled "► Crossover Design." In these study designs, each subject receives all of the interventions in the study. Because of the potential confounding influence of the order in which the interventions are completed (e.g., subjects may tend to respond better to the first intervention rather than the last, regardless of the nature of the intervention), this factor needs to be controlled for. This potential issue is elegantly solved by counterbalancing the order in which the subjects receive the treatments. In a two-treatment study, for example, half of the subjects would receive Treatment A first and Treatment B second, and the other half would receive the treatments in the reverse order.

Using different but comparable nomenclature, the goal of a research study is to identify one source of systematic influence, the influence that is systematically provided by the factor of interest in the study. It is essential to remove all other

identifiable sources of systematic influence, such as the order in which treatments are administered. Other simple examples include not administering Treatment A only to males and Treatment B only to females, and not administering Treatment A only to relatively young subjects and Treatment B to relatively old subjects.

The process of randomization is a powerful tool used to disperse influences that cannot readily be controlled equally (randomly) across the subjects in a study, thereby removing unwanted systematic influences.

Cross-References

- ▶ Crossover Design
- ▶ Randomization
- ▶ Research Methodology

Congestive Heart Failure

William Whang
Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Synonyms

Heart failure

Description

Congestive heart failure is a condition in which the heart cannot provide enough cardiac output for the metabolic demands of the body. The prevalence of heart failure has been estimated at 2% and is expected to grow due to improved survival of people with cardiac conditions (Mann, 2008). The lifetime risk of developing heart failure has been estimated at 20%. Coronary artery disease is the most frequent cause of heart failure (60–75%) (Lloyd-Jones et al., 2002). Etiologies for heart failure aside from coronary artery disease include viral inflammation of the heart, also known as myocarditis; alcohol toxicity; or genetic mutations.

One way to classify heart failure is according to left ventricular ejection fraction, a measure of contractile function. "Systolic heart failure" is defined by the presence of reduced left ventricular ejection fraction, usually <40%. About half of patients with heart failure may still have preserved left ventricular ejection fraction, so-called heart failure with normal ejection fraction (HFNEF) (Maeder & Kaye, 2009). This is often thought to be due to impaired left ventricular relaxation, or "diastolic dysfunction," but can also occur in the setting of other conditions such as anemia or renal dysfunction.

Symptoms of this condition can include shortness of breath, peripheral edema, and fatigue. Worse symptomatology has been associated with greater mortality risk. New York Heart Association class is one way to indicate the symptom severity of someone with heart failure (Mann, 2008):

Class I – no symptoms and no limitation in ordinary physical activity

Class II – slight limitation during ordinary activity

Class III – marked limitation in activity due to symptoms, even during less-than-ordinary activity

Class IV – symptoms even while at rest, mostly bedbound patients

The prevailing view of pathogenesis of systolic heart failure involves a neurohormonal hypothesis. After an initial insult that results in damage to heart muscle, a number of compensatory systems are activated, mainly involving overactivity of the sympathetic nervous system. Activation of the renin-angiotensin-aldosterone system results in salt and water retention, as well as constriction of peripheral blood vessels. This short-term adaptation leads to detrimental increases in left ventricular size and wall thinning, also referred to as remodeling.

The overall prognosis in patients with heart failure is poor, with 1-year mortality as high as 30–40% without treatment (Mann, 2008). Depression has been estimated by a meta-analysis to occur in about 21% of heart failure patients, and its

presence is associated with worse cardiovascular outcomes and higher overall mortality (Rutledge, Reis, Linke, Greenberg, & Mills, 2006).

The hallmark of pharmacologic therapy for heart failure involves treatment with angiotensin converting-enzyme (ACE) inhibitors and beta blockers, which are known to improve long-term mortality. Of note, there is a relative lack of evidence for therapies for treatment of heart failure with normal ejection fraction, although blood pressure control is thought to play an important role in treatment.

Behavioral interventions for heart failure may include cessation of tobacco/alcohol use, reduction in salt intake, and exercise in selected patients. The *H*eart *F*ailure: *A C*ontrolled *T*rial *I*nvestigating *O*utcomes of Exercise Trai*N*ing (HF-ACTION) trial was performed in 2,331 ambulatory patients with heart failure and reduced left ventricular ejection fraction (average 0.25) (O'Connor et al., 2009). The intervention consisted of a group-based, supervised exercise program for 3 months with transition to home exercise. During a median follow-up duration of 30 months, a nonsignificant reduction in the primary endpoint of all-cause mortality or hospitalization was achieved (HR 0.93, 95% CI 0.84–1.02. $p = 0.13$). Exercise training was also found to be relatively safe in the intervention group.

References and Readings

Lloyd-Jones, D. M., Larson, M. G., Leip, E. P., Beiser, A., D'Agostino, R. B., Kannel, W. B., et al. (2002). Lifetime risk for developing congestive heart failure: The Framingham heart study. *Circulation, 106*(24), 3068–3072.

Maeder, M. T., & Kaye, D. M. (2009). Heart failure with normal left ventricular ejection fraction. *Journal of the American College of Cardiology, 53*(11), 905–918.

Mann, D. L. (2008). Heart failure and cor pulmonale (chap. 227). In A. S. Fauci, E. Braunwald, D. L. Kasper, S. L. Hauser, D. L. Longo, J. L. Jameson, & J. Loscalzo (Eds.), *Harrison's principles of internal medicine, 17e*. New York: McGraw-Hill.

O'Connor, C. M., Whellan, D. J., Lee, K. L., Keteyian, S. J., Cooper, L. S., Ellis, S. J., et al. (2009). Efficacy and safety of exercise training in patients with chronic heart failure: HF-ACTION randomized controlled trial. *Journal of the American Medical Association, 301*(14), 1439–1450.

Rutledge, T., Reis, V. A., Linke, S. E., Greenberg, B. H., & Mills, P. J. (2006). Depression in heart failure a meta-analytic review of prevalence, intervention effects, and associations with clinical outcomes. *Journal of the American College of Cardiology, 48*(8), 1527–1537.

Conjecture

► Theory

Consensus Guideline

► Clinical Practice Guidelines

CONSORT Guidelines

Lisa A. Eaton
Center for Health, Intervention, and Prevention, University of Connecticut, New Haven, CT, USA

Synonyms

Guidelines for reporting randomized controlled trials

Definition

Many studies employ a randomized controlled trial (RCT) design to test the efficacy of products or services. However, inconsistencies in reporting trial information and outcomes of RCTs have stymied the usefulness of these trials in regards to providing readily available data from trial findings. These shortcomings led to the development of Consolidated Standards of Reporting Trials (CONSORT). CONSORT dictates that trial authors answer a series of checklist questions and provide a flowchart representing the trial when reporting outcomes.

The CONSORT Statement seeks to improve reporting information from RCTs, including increasing transparency of trial procedures and outcomes. As of 2010, there are 25 checklist items that cover what information should be included in the title/abstract, introduction, methods, results, discussion, and other (registration, protocol, and funding). In addition, authors adhering to CONSORT Statement guidelines should include a flow chart that depicts, in part, number of participants screened, excluded and why, randomized, received product or service, lost to follow-up and why, assessed, and included in data analysis.

CONSORT was originally developed in the 1990s and has since been amended multiple times. Individuals from varied backgrounds have taken part in forming the specifics of the guidelines. A committee representing the purpose of the CONSORT Statement meets regularly to review, assess, and change as needed statement guidelines. Thus, making this document one that is continually evolving to best represent the reporting of the scientific methods of an RCT. Multiple peer-reviewed, scholarly journals follow the reporting guidelines set forth by the CONSORT Statement which has led to consistency across journals in terms of reporting style. Evaluations have been completed to assess the impact of implementing the CONSORT Statement. Analyses of trial reporting before and after the time period of guideline availability have demonstrated a substantial improvement in transparency of procedures and outcomes as a result of the CONSORT Statement.

Cross-References

► Randomized Clinical Trial

References and Readings

Begg, C., Cho, M., Eastwood, S., Horton, R., Moher, D., et al. (1996). Improving the quality of reporting of randomized controlled trials. The CONSORT statement. *Journal of the American Medical Association, 276*, 637–639.

http://www.consort-statement.org/

Moher, D., Hopewell, S., Schulz, K. F., Montori, V., Gøtzsche, P. C., Devereaux, P. J., et al. (2010). CONSORT 2010 explanation and elaboration: Updated guidelines for reporting parallel group randomised trial. *BMJ, 340*, c869. for the CONSORT Group.

Moher, D., Schulz, K. F., Altman, D. G., & Lepage, L. (2001). The CONSORT statement: Revised recommendations for improving the quality of reports of parallel-group randomised trials. *The Lancet, 357*, 1191–1194.

Construct Validity

Annie T. Ginty
School of Sport and Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

Definition

Construct validity is the extent to which the measurements used, often questionnaires, actually test the hypothesis or theory they are measuring. Construct validity should demonstrate that scores on a particular test do predict the theoretical trait it says it does.

There are two subsets of construct validity: convergent construct validity and discriminant construct validity. Convergent construct validity tests the relationship between the construct and a similar measure; this shows that constructs which are meant to be related are related. Discriminant construct validity tests the relationships between the construct and an unrelated measure; this shows that the constructs are not related to something unexpected. In order to have good construct validity one must have a strong relationship with convergent construct validity and no relationship for discriminant construct validity.

Cross-References

► Psychometric Properties
► Reliability and Validity
► Validity

References and Readings

Bruce, N., Pope, D., & Stanistreet, D. (2008). *Quantitative methods for health research: A practical interactive guide to epidemiology and statistics*. West Sussex: Wiley.

Constructive Coping

▶ Active Coping

Contemplation

▶ Meditation
▶ Transcendental Meditation

Context Effect

▶ Nocebo and Nocebo Effect
▶ Placebo and Placebo Effect

Continuity of Care

Marie Boltz
College of Nursing, New York University, New York, NY, USA

Definition

Continuity of care refers to the seamless provision of health care between settings and over time (Gulliford, Naithani, & Morgan, 2006).

Traditionally, patients have viewed care continuity as a permanent relationship with a dependable, caring health care professional. This view, defined as interpersonal continuity of care, implies that the identified professional is the sole source of care and information for the patient. To health care providers, care continuity has historically implied the exchange of information, e.g., between shifts of nurses, between units of a healthcare facility, and between providers such as acute care and a nursing home. Coleman and colleagues (2006) define the flow of information between different locations or different levels of care within the same location as "transitional care" necessary to ensure the coordination and continuity of health care as patients move through different settings. Organizational approaches that facilitate transitions between settings are the use of transitions coach to educate the patient and family, and coordinate among the health professionals involved in the transition (Coleman, Parry, Chalmers, & Min, 2006) and the transitional care nurse (Naylor et al., 2004) who coordinates the discharge plan and coordinates the plan in the home.

Chronological or longitudinal continuity of care describes health care interactions that occur in the same place, with the same medical record, and with the same professionals, so that there is consistent knowledge of the patient by those providing the care (Saultz, 2003). Interdisciplinary or team-based continuity implies allows previous knowledge of the patient to be present even when the patient requires a wide range of services.

Given that healthcare needs can rarely be met by a single professional or a single provider setting, a multidimensional model of continuity of care is a logical choice, (Gulliford, Naithani, & Morgan, 2006) one that provides a longitudinal and interdisciplinary approach, while providing the dependability and relational aspects of interpersonal continuity. This model relies on integration, coordination, and the sharing of information between different, but stable providers. Evaluation of continuity of care can be conducted from the patient perspective, i.e., the experience of care, or satisfaction with the coordination of care and its interpersonal aspects. It also includes the provider's evaluation of outcomes and care processes (team functioning and case management effectiveness).

References and Readings

Coleman, E. A., Parry, C., Chalmers, S., & Min, S. J. (2006). The care transitions intervention: results of a randomized controlled trial. *Archives of Internal Medicine*, *166*, 1822–1828.

Gulliford, M., Naithani, S., & Morgan, M. (2006). What is "continuity of care?". *Journal of Health Services Research and Policy*, *11*(4), 248–250.

Naylor, M. D., Brooten, D. A., Campell, R. L, Maislin, G., McCauley, K. M., & Schwartz, J.S. (2004). Transitional care of older adults hospitalized with heart

failure: a randomized, controlled trial. *Journal of the American Geriatrics Society*, *52*, 675–684.
Saultz, J. W. (2003). Defining and measuring interpersonal continuity of care. *Annals of Family Medicine*, *1*, 134–143.

Continuous Subcutaneous Insulin Infusion

▶ Insulin Pumps

Contraception

Linda C. Baumann and Alyssa Karel
School of Nursing, University of Wisconsin-Madison, Madison, WI, USA

Abbreviations

STI Sexually transmitted infections

Synonyms

Birth control, family planning

Definition

Contraception, or birth control, is any method, action, device, or medication used to prevent pregnancy. Various methods of contraception include blockage of the sperm from reaching the egg, killing or damaging sperm, preventing the release of an egg from the ovaries, or changing the uterine lining so a fertilized egg will not attach. Many factors can help couples choose the most appropriate contraception based on frequency of sex, plans for pregnancy, age and overall health, side effects, number of sexual partners, protection against sexually transmitted infections (STIs), and contraceptive failure rates. Contraceptive failure rates are most often reported as two numbers, the theoretical failure rate or the rate of contraceptive failure when the method is used correctly during every act of intercourse. The actual failure rate takes into account the actual variation in consistency of contraceptive usage (see Table 1).

Description

The various methods of contraception include barrier methods, natural methods, hormonal methods, intrauterine devices (IUDs), emergency contraception, and surgical sterilization.

The barrier method provides a mechanical or chemical barrier to the sperm from reaching the egg. The most common form of the barrier method is the male condom, which is usually made of thin latex. New materials have been developed and include polyurethane and styrene and styrene ethylene butylenes styrene. These materials have a longer shelf life and can be used with oil-based lubricants without increasing the risk of condom breakage. Male condoms are relatively inexpensive, easily accessible, and carry few health risks. Health risks include hypersensitivity to the latex or lubricant inside the condom. If used correctly every time, male condoms are very effective and carry only a 2% theoretical failure rate; actual failure rates are around 15%.

Female condoms are soft plastic film linings with flexible rings at both ends (see Fig. 1). When used correctly and consistently, this type of contraception carries a slightly higher theoretical and actual failure rate than male condoms at 5% and 21%, respectively. Female condoms are more expensive than male condoms due to the polyurethane material they are made from. Both male and female condoms are a beneficial form of contraception because they not only protect against pregnancy, but also provide protection against sexually transmitted infections (STI).

Spermicides are another barrier method and come in the form of foams, jells, suppositories, creams, films, and tablets. The most widely used types contain nonoxynol-9 and octoxynol-9.

Contraception, Table 1 Overview of contraceptive methods

Method	Failure rate (%) Theoretical/actual		Benefits	Health risks/side effects and disadvantages
Barrier Condom Male condom Female condom	2.0 5.0	15.0 21.0	Provides STI protection Male condoms are relatively cheap and widely available May provide protection against conditions caused by STIs	Occasional hypersensitivity to latex or lubricant inside condom May lead to decreased sensitivity Female condoms are costly and bulky
Spermicide	18.0	29.0	Widely available in many forms and relatively inexpensive	Can cause vaginal or penile irritation Risk of urinary tract infection Frequent use of nonoxynol-9 may increase risk of HIV One of the least effective methods when used alone
Diaphragm Cervical cap Parous women Nuliparous women	6.0 20.0 9.0	16.0 32.0 16.0	May help protect against certain STIs May help protect against cervical cancer	Can cause vaginal or penile irritation May lead to the development of Toxic Shock Syndrome if left in place for too long Risk of bladder irritations that can lead to urinary tract infections
Natural Fertility awareness Coitus interruptus Lactational amenorrhea (LAM)	– 4.0 1.0	25.0 27.0 2.0	Completely natural LAM helps encourage healthy breast-feeding patterns, which benefits both mother and child	Requires motivation and a commitment to learning High failure rates LAM can only be used a maximum of 6 months after delivery
Hormonal methods Combination estrogen and progesterone Pill Injection Patch Vaginal ring	0.3 0.5 0.3 0.3	8.0 3.0 0.8 0.8	Available in many forms Provides many non-contraceptive health benefits including reduced risk of: Ovarian and endometrial cancer, PID, ovarian cysts, osteoporosis, iron-deficiency anemia and dysmenorrhea	Can cause dizziness, headache, nausea, weight changes, mood changes, and breast pain Cary risk of cardiovascular complications such as deep vein thrombosis and pulmonary embolism Risk of heart attack and stroke is present but rare

Progesterone only Pill Injections	0.5 0.3	8.0 3.0	Can be used by women who are nursing and for whom estrogen is contraindicated	May cause irregular bleeding, mood changes, weight gain, dizziness, headaches, and nausea
Intrauterine devices Copper T IUD Levonorgestrel IUD	0.6 0.2	0.8 0.2	May help protect against endometrial cancer After initial insertion requires little maintenance Copper IUDs can remain in uterine and work effectively for up to 10 years Levonorgestrel can be effective for up to 5 years Levonorgestrel IUD has been used to help treat menorrhagia and dysmenorrhea Copper IUD can be used as emergency contraception if placed within 5–8 days of unprotected sex and is the most effective form of emergency contraception	Side effects such as bleeding, pain, perforation, and infection are rare but can be serious May increase the risk of pelvic inflammatory disease if gonorrhea or Chlamydia is present before the insertion If pregnancy does occur, the risk of spontaneous abortion is increased by up to 50% May change bleeding patterns causing increased bleeding and menstrual pain in first few months Levonorgestrel IUD can cause headaches, nausea, dizziness, and weight gain
Surgical sterilization Female sterilization Male sterilization	0.5 0.2	0.5 0.2	Very few side effects Permanent form of contraception and therefore requires no other contraceptive efforts 3 months post operation Helps protect against pelvic inflammatory disease	Complications of surgery and anesthesia are possible Requires a back-up method of contraception for first 3 months post operation

Contraception, Fig. 1 Female condom

Spermicides are inserted deep in the vagina at least 30 min prior to sexual intercourse and create a chemical barrier by killing or inactivating sperm by causing the membrane of the sperm cell to break. Research has shown that frequent use of nonoxynol-9 can damage lower genital tract epithelial surfaces and may increase the risk of HIV infection. New spermicides are being developed to replace nonoxynol-9. When used alone, spermicides are one of the least effective forms of contraception and it is recommended that they be used in conjunction with other forms of contraception. Spermicides do not protect against STIs and carry risks of local tissue irritation.

A diaphragm is a latex rubber cup with a flexible rim that covers the cervix. It is placed in the vagina before intercourse and remains there for 6–8 h after intercourse. It is most often used in conjunction with a spermicide. Diaphragms are relatively effective with a 6% theoretical failure rate and actual failure rate of 16%. However, diaphragms must be prescribed and fitted by a healthcare provider and carry risk of vaginal or penile irritation, urinary tract infection, and in rare cases toxic shock syndrome.

A cervical cap is a small, soft cup that fits snugly over the cervix and is used in conjunction with a spermicide. It is a more effective form of contraception in nulliparous women. Like diaphragms, cervical caps must be fitted by a healthcare provider and carry many of the same health risks.

Natural methods of contraception used to control pregnancy include fertility awareness, coitus interruptus, and lactational amenorrhea. Women using fertility awareness identify ovulation based on body symptoms or the calendar. Symptom-based methods include monitoring cervical mucus changes around the time of ovulation. These changes include the mucus becoming thin and watery. The symptothermal method includes taking regular basal body temperatures to recognize a decrease, which occurs prior to ovulation, and monitoring other cues such as abdominal cramps, breast tenderness, and changes in cervical position to predict ovulation. Calendar methods of fertility awareness include the standard day method and the calendar rhythm method. The standard day method tracks the menstrual cycle counting from the first day of bleeding as day 1; days 9 through 18 are considered fertile days. The calendar rhythm method requires a record of the number of days in a menstrual cycle for 6 months, with estimates of the fertile period calculated by subtracting 18 from the length of the shortest cycle – this day is the estimated first day of the fertile period. To estimate the end of the fertile period, subtract 11 from the length of the longest cycle – this is the estimated last day of the fertile period. These calculations should be updated monthly using the most recent cycles. Fertility awareness is considered one of the least effective forms of contraception, especially in women with irregular menstrual cycles. The theoretical failure rate for fertility awareness is 10%, while the actual failure rate is about 25%.

A second natural method of contraception is coitus interruptus, or withdrawal of the penis from the vagina before ejaculation. The theoretical failure rate is quite low at about 4%, but the actual failure rate is around 27%, which makes this form one of the least effective methods. Pre-ejaculate can be deposited into the vaginal canal prior to ejaculation and contributes to the high failure rate. A male who has recently ejaculated prior to sex should first urinate and clean the tip of the penis to remove any sperm from the previous ejaculation.

The last natural method of contraception is lactational amenorrhea. This method can be used by nursing mothers after delivery because frequent breast-feeding suppresses hormones that cause ovulation. Because the suppression of ovulation is variable, this type of contraception

should not be used longer than 6 months after delivery.

Hormonal methods of birth control suppress ovulation to prevent pregnancy and are the most widely used form of reversible contraception in the United States. Combined estrogen and progesterone and progesterone-only methods are the two available forms of hormonal birth control. Combined birth control methods come in many forms including oral pills, transdermal patches, monthly injections, and vaginal rings. Depending on the form being used, failure rates for combined hormonal contraception vary. Theoretical failure rates for all forms are below 1%; however, some actual rates can be as high as 8%.

The second form of hormonal contraception is progesterone-only contraception. Because this contraception does not contain estrogen, it is advantageous for women who are breastfeeding and for women in whom estrogen is contraindicated. Progesterone-only contraception comes in the form of oral pills and injections that work by thickening the cervical mucus, inhibiting sperm movement, and disrupting the menstrual cycle to prevent ovulation. Generally progesterone-only contraception is not as effective as combination contraception and carries actual failure rates of 8–10%.

Intrauterine devices (IUDs) are the most widely used form of reversible contraception globally. An IUD is a small plastic or metal device that is inserted by a healthcare provider into the uterus. The two most common forms are the copper-bearing IUD and the levonorgestrel IUD. The copper IUD is a plastic frame (or "7") with copper sleeves around it. The levonorgestrel IUD is a plastic T-shaped device that releases small amounts of levonorgestrel, a form of progesterone. The IUD causes a sterile inflammatory response in which sperms are destroyed or immobilized by inflammatory cells. In addition to this inflammatory response, the levonorgestrel further provides contraceptive effect by thickening cervical mucus and causing atrophy of the endometrium. The copper in copper IUDs adds to the contraceptive effect by hampering sperm motility, making it difficult to reach the fallopian tubes. Most IUDs can be left in place for 5–10 years and are therefore a long-term contraceptive plan with little maintenance required after the initial insertion. They are highly effective with theoretical and actual failure rates below 1%.

Emergency contraception is a form of contraception that can be utilized after unprotected sex or after a contraceptive failure. Emergency contraception comes in two forms, pills and an emergency copper IUD insertion, and prevents pregnancy by inhibiting ovulation, fertilization or implantation based on the form used. Emergency contraceptive pills are high doses of either a combined estrogen and progesterone pill or a progesterone-only pill. To be most effective, emergency contraception should be taken as soon as possible after unprotected sex, but can also be effective if taken within 5 days of unprotected intercourse. Emergency insertion of a copper IUD within 5–8 days of unprotected sex is a very effective form of emergency contraception.

Sterilization is a form of permanent contraception and can be done in both men and women. In females, tubal ligation involves a surgical occlusion of both fallopian tubes preventing an egg from entering the uterus. Vasectomy is a male sterilization procedure that involves ligation of the vas deferens. Because these procedures are meant to be permanent, reversal surgery is rare, and when done, rarely successful. Unlike tubal ligation, a vasectomy is not immediately effective and another contraceptive method should be used for the first 3 months post operation. In the past 30 years, the rate of sterilization as a form of contraception has increased dramatically and is currently one of the most widely used forms of contraception. Failure rates are extremely low with both the theoretical and actual rates below 1%.

Globally, many social determinants influence the choice of contraceptive and include gender and the role of women in a culture, age, socioeconomic status, marital status, education level, and religion. For example, in the United States, women aged 22–44 who are less educated are more likely to use sterilization as a contraceptive method while college-educated women of the same age range more often use pills as the preferred method of contraception. Some

religious beliefs sanction natural methods of contraception to space pregnancies as opposed to using hormonal or barrier methods that prevent pregnancy from occurring. Surgical sterilization is most often used by an older population while the pill is the preferred form in the population below the age of 30. The percentages of contraceptive users and the most widely used forms vary by country.

Cross-References

- ▶ Abstinence
- ▶ Family Planning

References and Readings

Callahan, T. L., & Caughey, A. B. (2007). Contraception and sterilization. In N. A. Duffy & K. Horvath (Eds.), *Obstetrics & Gynecology* (pp. 248–266). Baltimore: Lippincott Williams & Wilkins.

Medline Plus. (2010). *Birth control*. Retrieved August 24, 2010, from http://www.nlm.nih.gov/medlineplus/birthcontrol.html.

Mosher, W. D., & Jones, J. (2010). Use of contraception in the United States: 1982–2008. National Center for Health Statistics. *Vital and Health Statistics 23*(29), 1–44.

Rowlands, S. (2009). New Technologies in contraception. *BJOG: An International Journal of Obstetrics & Gynaecology, 116*(2), 230–239.

The American Congress of Obstetricians and Gynecologist. (2007). *Birth control*. Retrieved August 24, 2010, from http://www.acog.org/publications/patient_education/ab020.cfm

Wong, D., Hockenberry, M., Wilson, D., Perry, S., & Lowdermilk, D. (2006). *Maternal child nursing care* (3rd ed.). St. Louis, MO: Mosby Elsevier.

World Health Organization, Department of Reproductive Health and Research (WHO/RHR), & John Hopkins Bloomberg School of Public Health/Center for Communication Programs (CCP). (2008). *Family planning: A global handbook for providers*. Baltimore/Geneva: CCP and WHO.

Control

- ▶ Hyperglycemia
- ▶ Interpersonal Circumplex

Control Group

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Comparator group; Concurrent control (which applies only in some settings)

Definition

A control group is a group of subjects against whose information the information gathered from an investigational group is compared.

To judge the effectiveness of a therapeutic behavioral intervention, or the harm done by engaging in behavioral activities such as smoking, it is necessary to have a reference point. This is provided by data collected from individuals who are deliberately similar to those in the investigational group in as many ways as possible with the single exception of receiving the therapeutic intervention or having engaged in the behavior of concern.

Control groups can be used in experimental studies and nonexperimental (often called observational) studies. Testing the effectiveness of a therapeutic behavioral intervention in a group of individuals who have not previously received it would fall into the category of an experimental study: The researchers administer an experimental treatment. To control for the fact that simply participating in the study may have a sizeable therapeutic benefit (caused by a variety of potential factors, including the extra medical attention given to these subjects), it is necessary to have a control group that experiences all of the circumstances experienced by those in the investigational group with the exception of the intervention of interest. This can be a difficult challenge for those developing the experimental methodology to be used in the study.

Cross-References

- ▶ Case-Control Studies
- ▶ Randomized Clinical Trial

References and Readings

Rothman, K. J., Greenland, S., & Lash, T. L. (Eds.). (2008). *Modern epidemiology* (3rd ed.). Philadelphia: Lippincott Williams & Wilkins.
Turner, J. R. (2012). *Key statistical concepts in clinical trials for pharma*. New York: Springer.

Control Group of a Randomized Trial

- ▶ Usual Care

Co-occuring

- ▶ Comorbidity

Cook-Medley Hostility Scale

Matthew Calamia
Department of Psychology, University of Iowa, Iowa City, IA, USA

Definition

The Cook-Medley Hostility Scale (Ho Scale) (Cook & Medley, 1954) is a 50-item scale derived from the Minnesota Multiphasic Personality Inventory. The creators viewed it as a measure of "chronic hate and anger." Scores on the Ho Scale are related to a variety of health-relevant variables, including alcohol consumption, insulin resistance, and waist-to-hip ratio (Bunde & Suls, 2006). Scores on the Ho are predictive of coronary artery disease and all-cause mortality even after controlling for other health risk factors (Miller, Smith, Turner, Guijarro, & Hallet, 1996). In contrast to the extensive evidence for its predictive validity, the exact construct(s) measured by the scale have been the subject of some debate. A variety of competing measurement models have been proposed, with no clear favorite based on psychometric criteria (Contrada & Jussim, 1992). Although groups of items relating to constructs ranging from hypersensitivity to aggressive responding have been identified, the core factor of the Ho Scale may be best described as reflecting cynicism. A unidimensional index of that primary factor can be derived from the overall scale and was found in at least one study to maintain the predictive ability of the entire scale (Strong, Kahler, Greene, & Schinka, 2005).

Cross-References

- ▶ Cynical Hostility

References and Readings

Bunde, J., & Suls, J. (2006). A quantitative analysis of the relationship between the cook-medley hostility scale and traditional coronary artery disease risk factors. *Health Psychology, 25*, 493–500.
Contrada, R. J., & Jussim, L. (1992). What does the Cook-Medley hostility scale measure? In search of an adequate measurement Model1. *Journal of Applied Social Psychology, 22*, 615–627.
Cook, W. W., & Medley, D. M. (1954). Proposed hostility and pharisaic-virtue scales for the MMPI. *Journal of Applied Psychology, 38*(6), 414–418.
Han, K., Weed, N. C., Calhoun, R. F., & Butcher, J. N. (1995). Psychometric characteristics of the MMPI-2 cook-medley hostility scale. *Journal of Personality Assessment, 65*, 567–585.
Miller, T. Q., Smith, T. W., Turner, C. W., Guijarro, M. L., & Hallet, A. J. (1996). A meta-analytic review of research on hostility and physical health. *Psychological Bulletin, 119*, 322–348.
Strong, D. R., Kahler, C. W., Greene, R. L., & Schinka, J. (2005). Isolating a primary dimension within the cook-medley hostility scale: A rasch analysis. *Personality and Individual Differences, 39*, 21–33.

Coping

Charles Carver
Department of Psychology, University of Miami, Coral Gables, FL, USA

Definition

Coping is efforts to prevent or diminish threat, harm, and loss, or to reduce the distress that is often associated with those experiences.

Description

The concept of coping presumes the existence of a condition of adversity or stress. A person who must deal with adversity is engaged in coping. Thus, coping is inextricably linked to stress. It is often said that stress exists whenever people confront situations that tax or exceed their ability to manage them (Lazarus, 1966; Lazarus & Folkman, 1984). Whenever a person is hard-pressed to deal with an obstacle or impediment or looming threat, the experience is stressful. Adversity takes several forms. *Threat* refers to the impending occurrence of an event that is feared will have bad consequences. *Harm* refers to the perception that bad consequences have already come to pass. *Loss* refers to the perception that something of value has been taken away.

People respond to perceptions of threat, harm, and loss in a wide variety of ways, many of which are labeled coping. Coping is generally defined as efforts to prevent or diminish threat, harm, and loss, or to reduce the distress that is often associated with those experiences. Some theorists prefer to limit the concept of coping to voluntary responses (Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001). Others include automatic and involuntary responses as well (Eisenberg, Fabes, & Guthrie, 1997; Skinner & Zimmer-Gembeck, 2007). It should be noted that it is not easy to distinguish between voluntary and involuntary responses to stress. Furthermore, responses that are intentional and effortful when first used may become automatic with repetition. Some discussions of coping also include unconscious defensive reactions as aspects of coping. This entry is limited, however, to responses that are recognized by the person who is engaging in them.

Distinctions and Groupings Among Coping Responses

Coping is a very broad concept with a long and complex history (Compas et al., 2001; Folkman & Moskowitz, 2004). A great many distinctions have been made within the broad domain (Skinner, Edge, Altman, & Sherwood, 2003). Some of the more important distinctions are described in the sections that follow.

Problem-Focused Versus Emotion-Focused Coping

The first distinction made in modern examination of coping was that made between problem-focused and emotion-focused coping (Lazarus & Folkman, 1984). Problem-focused coping is directed at the stressor itself: taking steps to remove or to evade it, or to somehow diminish its impact if it cannot be evaded. For example, if the arrival of a hurricane is forecast, a homeowner's problem-focused coping might include bringing all potted plants indoors, putting up storm shutters, and buying batteries for use in flashlights. As another example, if layoffs are expected at one's place of employment, problem-focused coping might include saving money, applying for other jobs, obtaining training to enhance hiring prospects, or working harder at the current job to reduce the likelihood of being let go.

Emotion-focused coping, in contrast, is aimed at minimizing the emotional distress that is triggered by stressful events. Because there are many ways to reduce distress, emotion-focused coping includes a very wide range of responses, ranging from self-soothing (e.g., relaxation, seeking emotional support), to expression of negative emotion (e.g., yelling, crying), to a focus on negative

thoughts (e.g., rumination), to attempts to escape cognitively from the stressful situation (e.g., avoidance, denial, wishful thinking).

Problem-focused and emotion-focused coping have different initial or focal goals. The focal goal determines which category a particular response is assigned to. Some behaviors can serve either a problem-focused or an emotion-focused function, depending on the goal behind their use. For example, seeking support is emotion focused if the goal is to obtain emotional support and reassurance; on the other hand, seeking support is problem focused if the goal is to obtain advice or instrumental help.

Although it is easy to distinguish between them in principle, problem-focused coping and emotion-focused coping also tend to facilitate one another. Effective problem-focused coping diminishes the threat or harm, but by doing so, it also diminishes the distress generated by that threat. Effective emotion-focused coping diminishes negative emotions, making it possible to consider the problem more calmly. This often leads to better problem-focused coping. This interwoven relationship between problem- and emotion-focused coping makes it more useful to think of the two as complementary coping functions, rather than as two fully distinct and independent coping categories.

Engagement Versus Disengagement

What turns out to be a particularly important distinction is the distinction between engagement or approach coping and disengagement or avoidance coping (e.g., Skinner et al., 2003). Engagement coping is aimed at actively dealing with the stressor or stress-related emotions. Disengagement coping is aimed at avoiding confrontation with the threat or avoiding the stress-related emotions. Engagement coping includes problem-focused coping and forms of emotion-focused coping such as support seeking, emotion regulation, acceptance, and cognitive restructuring. Disengagement coping includes responses such as avoidance, denial, and wishful thinking. Disengagement coping is often emotion focused, because it typically involves an attempt to escape feelings of distress. Some disengagement coping is almost literally an effort to act as though the threat does not exist, so that no reaction is needed, behaviorally or emotionally. Wishful thinking and fantasy can distance the person from the stressor, at least temporarily, and denial creates a boundary between reality and the person's experience.

Although disengagement coping has the aim of escaping distress, it is generally ineffective in reducing distress over the long term, because it does nothing about the threat's existence and its eventual impact. If you are experiencing a real threat in your life and you respond to it by going to the movies, the threat will generally remain when the movie is over. Eventually, it must be dealt with. Indeed, for many types of stress, the longer a person avoids dealing with the problem, the more difficult or complex it becomes, and the less time is available to deal with it when one does finally turn to it. Finally, some kinds of disengagement coping can create problems of their own. Excessive use of alcohol or drugs can create social and health problems, and shopping or gambling as an escape can create financial problems.

Some have extended the concept of disengagement coping to include giving up on goals that are threatened by the stressor (Carver & Connor-Smith, 2010). This differs from other disengagement responses, in that it addresses both the stressor's existence and its emotional impact by abandoning an investment in something else. Disengaging from the threatened goal may allow the person to avoid negative feelings associated with the threat. Depending on the nature of the goal being abandoned, however, this sort of disengagement can also have adverse secondary consequences.

Accommodative Coping and Meaning-Focused Coping

Most adaptive coping is one or another form of engagement coping. Within engagement coping, distinctions also have been made between attempts to control the stressor itself, called primary control coping, and attempts to adapt or adjust to the stressor, termed accommodative or sometimes secondary control coping (Morling & Evered, 2006; Skinner et al., 2003). The term accommodative is perhaps to be preferred

because it does not carry connotations of exerting control, or of being secondary to other coping efforts.

The concept of accommodative coping is rooted in analyses of the process of successful aging (Brandtstädter & Renner, 1990). It refers to adjustments within the self, which are made in response to constraints inherent in one's life situation. In the realm of coping, accommodation applies to responses such as acceptance, cognitive restructuring, and scaling back of one's goals in the face of insurmountable interference. Another kind of accommodation is self-distraction. Self-distraction is somewhat controversial. Self-distraction is often thought of as disengagement coping. However, there is also evidence suggesting that intentionally engaging in positive activities is a useful means of adapting to uncontrollable events (Skinner et al., 2003).

A concept that is related to accommodation is what has been called meaning-focused coping. In meaning-focused coping, people draw on their beliefs and values to find benefits in stressful experiences or remind themselves of positive aspects of their lives (Tennen & Affleck, 2002). Meaning-focused coping may include reordering one's life priorities and focusing on the positive meaning of ordinary events. The concept of meaning-focused coping has roots in evidence that positive as well as negative emotions are common during stressful experiences, that those positive feelings influence people's outcomes, and particularly the fact that people try to find benefit and meaning in adversity (Helgeson, Reynolds, & Tomich, 2006; Park, Lechner, Antoni, & Stanton, 2009). Although this concept emphasizes the positive changes a stressor brings to a person's life, it is worth pointing out that meaning-focused coping also represents an accommodation to the constraints of one's life situation. Meaning-focused coping involves reappraisal of the situation. It appears to be most likely when stressful experiences are uncontrollable or are going badly.

Stepping Back

This brief review is far from exhaustive. Nonetheless, it should make clear that there are many ways to group and organize coping responses. Further, it should be clear that these distinctions do not form a neat matrix into which all coping reactions can be sorted. A given response typically fits several places. For example, seeking emotional support is engagement, emotion-focused, and accommodative coping. Each distinction that has been introduced can be useful for answering certain questions about responses to stress. No one distinction fully conveys the structure of coping. The distinction that appears to be the most important is that made between engagement and disengagement. Interestingly enough, this is a distinction which also maps well onto goal-based models of personality functioning and social behavior (e.g., Carver & Scheier, 1998).

Relations Between Coping and Well-being

In some respects, the question that everyone wants answered is not "what are the ways in which people cope?" but "how do coping responses affect well-being?" Behind this question lie a number of thorny methodological issues (Carver, 2007). Among them are issues of how often coping should be measured, what time lag should be assumed and thus investigated between coping efforts and eventual outcomes, and whether coping should be viewed as a cluster of responses or a sequence of responses.

In meta-analyses of relations between coping and well-being, effect sizes are typically small to moderate. Coping generally has been linked more strongly to psychological outcomes than to physical health (Clarke, 2006; Penley, Tomaka, & Wiebe, 2002). Nonetheless, most kinds of engagement coping relate to better physical and mental health in samples coping with stressors as diverse as traumatic events, social stress, HIV, and prostate cancer (Clarke, 2006; Littleton, Horsley, John, & Nelson, 2007; Moskowitz, Hult, Bussolari, & Acree, 2009; Penley et al., 2002; Roesch et al., 2005). However, some other less volitional responses that might be seen as reflecting engagement, including rumination, self-blame, and venting, predict poorer

emotional and physical outcomes (Austenfeld & Stanton, 2004; Moskowitz et al., 2009). Higher levels of disengagement coping typically predict poorer outcomes, such as more anxiety, depression, and disruptive behavior, less positive affect, and poorer physical health, across an array of stressors (Littleton et al., 2007; Moskowitz et al., 2009; Roesch et al., 2005). Acceptance coping seems to be a double-edged sword. Acceptance that occurs in the context of other accommodative strategies is helpful, but acceptance that reflects resignation and abandonment predicts distress (Morling & Evered, 2006).

Relations between coping and adjustment also vary with the nature, duration, context, and controllability of the stressor. In meta-analyses of both children and adults, it appears to be important to match one's coping to the stressor's controllability and to the resources that are available. Active attempts to solve problems help when dealing with controllable stressors, but the same responses are potentially harmful when dealing with uncontrollable stressors (Aldridge & Roesch, 2007; Clarke, 2006). Similarly, taking responsibility for uncontrollable stressors predicts distress, but taking responsibility is unrelated to adjustment in the context of controllable stressors (Penley et al., 2002). In contrast, emotional approach coping (e.g., self-regulation and controlled expression of emotion) appears to be most useful in the context of uncontrollable stressors (Austenfeld & Stanton, 2004).

One caveat must be applied to all of these conclusions about the effects of coping. Although coping is almost universally viewed as an ever-changing response to evolving situational demands, most coping research fails to reflect this view. Many studies assess only dispositional coping (overall coping styles), or one-time retrospective reports of overall coping with some stressor. Those studies tell virtually nothing about how timing, order, combination, or duration of coping affects outcomes. In contrast, Tennen, Affleck, Armeli, and Carney (2000) proposed that people typically use emotion-focused coping largely after they have tried problem-focused coping and found it ineffective. This suggests an approach to studying coping in which the question is whether the person changes from one sort of coping to another across successive assessments as a function of lack of effectiveness of the first response used.

The impact of a given coping strategy may be quite brief. For this reason, laboratory and daily report studies are essential to understanding the effects of situational coping strategies (Bolger, Davis, & Rafaeli, 2003). The small number of daily report studies of coping make it clear that the impact of coping changes over time, with responses that are useful one day sometimes having a negative impact on next-day mood or long-term adjustment (DeLongis & Holtzman, 2005). Laboratory research also is useful in disentangling stressor severity from individual differences in stress appraisals by using standardized stressors. Lab studies also make it easier to supplement self-reports with observations of coping and assessment of physiological responses.

Cross-References

▶ Stress

References and Readings

Aldridge, A. A., & Roesch, S. C. (2007). Coping and adjustment in children with cancer: A meta-analytic study. *Journal of Behavioral Medicine, 30*, 115–129.

Austenfeld, J. L., & Stanton, A. L. (2004). Coping through emotional approach: A new look at emotion, coping, and health-related outcomes. *Journal of Personality, 72*, 1335–1363.

Bolger, N., Davis, A., & Rafaeli, E. (2003). Diary methods: Capturing life as it is lived. *Annual Review of Psychology, 54*, 579–616.

Brandtstädter, J., & Renner, G. (1990). Tenacious goal pursuit and flexible goal adjustment: Explication and age-related analysis of assimilative and accommodative strategies of coping. *Psychology and Aging, 5*, 58–67.

Carver, C. S. (2007). Stress, coping, and health. In H. S. Friedman & R. C. Silver (Eds.), *Foundations of health psychology* (pp. 117–144). New York: Oxford University Press.

Carver, C. S., & Connor-Smith, J. (2010). Personality and coping. *Annual Review of Psychology, 61*, 679–704.

Carver, C. S., & Scheier, M. F. (1998). *On the self-regulation of behavior*. New York: Cambridge University Press.

Clarke, A. T. (2006). Coping with interpersonal stress and psychosocial health among children and adolescents: A meta-analysis. *Journal of Youth and Adolescence, 35*, 11–24.
Compas, B. E., Connor-Smith, J. K., Saltzman, H., Thomsen, A. H., & Wadsworth, M. E. (2001). Coping with stress during childhood and adolescence: Problems, progress, and potential in theory and research. *Psychological Bulletin, 127*, 87–127.
DeLongis, A., & Holtzman, S. (2005). Coping in context: The role of stress, social support, and personality in coping. *Journal of Personality, 73*, 1–24.
Duangdao, K. M., & Roesch, S. C. (2008). Coping with diabetes in adulthood: A meta-analysis. *Journal of Behavioral Medicine, 31*, 291–300.
Eisenberg, N., Fabes, R. A., & Guthrie, I. (1997). Coping with stress: The roles of regulation and development. In J. N. Sandler & S. A. Wolchik (Eds.), *Handbook of children's coping with common stressors: Linking theory, research, and intervention* (pp. 41–70). New York: Plenum.
Folkman, S., & Moskowitz, J. T. (2004). Coping: Pitfalls and promise. *Annual Review of Psychology, 55*, 745–774.
Helgeson, V. S., Reynolds, K. A., & Tomich, P. L. (2006). A meta-analytic approach to benefit finding and health. *Journal of Consulting and Clinical Psychology, 74*, 797–816.
Lazarus, R. S. (1966). *Psychological stress and the coping process*. New York: McGraw-Hill.
Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.
Littleton, H., Horsley, S., John, S., & Nelson, D. V. (2007). Trauma coping strategies and psychological distress: A meta-analysis. *Journal of Traumatic Stress, 20*, 977–988.
Morling, B., & Evered, S. (2006). Secondary control reviewed and defined. *Psychological Bulletin, 132*, 269–296.
Moskowitz, J. T., Hult, J. R., Bussolari, C., & Acree, M. (2009). What works in coping with HIV? A meta-analysis with implications for coping with serious illness. *Psychological Bulletin, 135*, 121–141.
Park, C. L., Lechner, S. C., Antoni, M. H., & Stanton, A. L. (Eds.). (2009). *Medical illness and positive life change: Can crisis lead to personal transformation?* Washington, DC: American Psychological Association.
Penley, J. A., Tomaka, J., & Wiebe, J. S. (2002). The association of coping to physical and psychological health outcomes: A meta-analytic review. *Journal of Behavioral Medicine, 25*, 551–603.
Roesch, S. C., Adams, L., Hines, A., Palmores, A., Vyas, P., Tran, C., et al. (2005). Coping with prostate cancer: A meta-analytic review. *Journal of Behavioral Medicine, 28*, 281–293.
Skinner, E. A., Edge, K., Altman, J., & Sherwood, H. (2003). Searching for the structure of coping: A review and critique of category systems for classifying ways of coping. *Psychological Bulletin, 129*, 216–269.
Skinner, E. A., & Zimmer-Gembeck, M. J. (2007). The development of coping. *Annual Review of Psychology, 58*, 119–144.
Tennen, H., & Affleck, G. (2002). Benefit-finding and benefit-reminding. In C. R. Snyder & S. J. Lopez (Eds.), *Handbook of positive psychology* (pp. 584–597). New York: Oxford University Press.
Tennen, H., Affleck, G., Armeli, S., & Carney, M. A. (2000). A daily process approach to coping: Linking theory, research, and practice. *American Psychologist, 55*, 626–636.

Coping Skills Training

▶ Williams LifeSkills Program

Coping Strategies

▶ Coping
▶ Denial

Coping Styles

▶ Coping

Coping with Stress

▶ Stress Management

Copy Number Variant (CNV)

Rany M. Salem[1] and Laura Rodriguez-Murillo[2]
[1]Broad Institute, Cambridge, MA, USA
[2]Department of Psychiatry, Columbia University Medical Center, New York, NY, USA

Synonyms

Structural variant

Definition

A copy number variant (CNV) is a type of genetic variation in which a sequence of nucleotides is repeated in tandem multiple times in an individual's genome. The variability arises from the gain and/or loss of genetic material, causing the number of repeat copies to vary in a population. In contrast to single nucleotide polymorphisms (SNPs), which affect only one nucleotide, CNVs are much larger, ranging from one kilobase to several megabases in size (Conrad et al., 2010). Large CNVs may contain genes, resulting in gene duplication or deletion.

CNVs are inherited, but can also arise de novo (although a rare event) via genomic rearrangements such as deletions, duplications, inversions, translocations, and transposons activity. It is estimated that the human genome contains ~20,000 CNVs (Mills et al., 2011) and covers up 12% of the human genome (Redon et al., 2006). CNVs have been associated with several diseases, including schizophrenia (Xu et al., 2008), autism (Sebat et al., 2007), and others (The Wellcome Trust Case Control Consortium, 2010). The full extent to which they contribute to human disease is not known (Conrad et al., 2010).

Cross-References

- ▶ DNA
- ▶ Human Genome Project
- ▶ Polymorphism
- ▶ Single Nucleotide Polymorphism (SNP)

References and Readings

Conrad, D. F., Pinto, D., Redon, R., Feuk, L., Gokcumen, O., Zhang, Y., et al. (2010). Origins and functional impact of copy number variation in the human genome. *Nature, 464*(7289), 704–712. doi:10.1038/nature08516.

Mills, R. E., Walter, K., Stewart, C., Handsaker, R. E., Chen, K., Alkan, C., et al. (2011). Mapping copy number variation by population-scale genome sequencing. *Nature, 470*(7332), 59–65. doi:10.1038/nature09708.

Redon, R., Ishikawa, S., Fitch, K. R., Feuk, L., Perry, G. H., Andrews, T. D., et al. (2006). Global variation in copy number in the human genome. *Nature, 444*(7118), 444–454. doi:10.1038/nature05329.

Sebat, J., Lakshmi, B., Malhotra, D., Troge, J., Lese-Martin, C., Walsh, T., et al. (2007). Strong Association of De Novo copy number mutations with autism. *Science, 316*(5823), 445–449.

The Wellcome Trust Case Control Consortium. (2010). Genome-wide association study of CNVs in 16,000 cases of eight common diseases and 3,000 shared controls. *Nature, 464*(7289), 713–720. doi:10.1038/nature08979.

Xu, B., Roos, J. L., Levy, S., van Rensburg, E. J., Gogos, J. A., & Karayiorgou, M. (2008). Strong association of de novo copy number mutations with sporadic schizophrenia. *Nature Genetics, 40*(7), 880–885. doi:10.1038/ng.162.

Coronary Artery Bypass Graft (CABG)

Siqin Ye
Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Synonyms

CABG

Definition

Coronary artery bypass graft, or CABG, is a surgical procedure performed to treat advanced coronary atherosclerotic disease.

Description

By using segments of other arteries and veins as conduits to bypass diseased portions of the coronary arteries, CABG can improve cardiac function by restoring blood flow to areas of the heart that were inadequately perfused. The most commonly used grafts include saphenous vein grafts, harvested either openly or endoscopically from the lower extremities; free radial grafts, which are segments of the radial arteries from either wrists; and left or right internal mammary arteries (LIMA or RIMA), which arise from the

subclavian arteries and are anastomosed distally to the target coronary vessels. Currently, the LIMA is most frequently used to bypass the left anterior descending artery (LAD) due its excellent long-term results, while vein grafts, which have much higher rates of graft failure, are used to bypass the other coronary vessels (Morrow & Gersh, 2008).

Despite the advent of percutaneous coronary intervention (PCI), CABG remains one of the most commonly performed surgical procedures in the United States. The main indications for CABG are based on high-risk anatomical features and include significant left main disease or its equivalent (i.e., concomitant proximal LAD and proximal left circumflex artery stenosis), multivessel coronary artery disease that involve the proximal LAD, and triple vessel disease (Eagle et al., 2004). Studies have also demonstrated that patients with left ventricular dysfunction, and especially those with significant amount of viable myocardium on noninvasive imaging, may derive greater benefit from surgical revascularization. In these selected patient populations, CABG has been shown to markedly improve survival compared with medical therapy (Yusuf et al., 1994). However, there are also significant risks associated with CABG. Registries maintained by the Society of Thoracic Surgeons have consistently shown operative mortality of 2–3%. In addition, there are other known perioperative complications, including myocardial infarction, stroke, renal failure, bleeding, and wound infections. Various scoring systems have been derived to predict the risk of these perioperative events and to aid in informed decision making for individual patients. The careful consideration of the benefits and risks of surgery is especially important for those patients with a high-risk profile, such as the frail elderly or those with many comorbidities (Eagle et al., 2004).

There has also been ongoing debate on whether a subset of patients with surgical disease may be treated with PCI rather than CABG. For instance, recent registries have suggested that in patients with uncomplicated left main disease, PCI may yield comparable rates of major adverse cardiovascular events as CABG (Seung et al., 2008). The landmark Synergy between PCI with Taxus and Cardiac Surgery (SYNTAX) trial published in 2009 randomized patients with three-vessel or left main coronary artery disease to PCI or CABG and showed that while patients who underwent PCI had higher rate of repeat revascularization, the rates of death and myocardial infarction were similar between the two arms. In particular, for patients with less complicated lesions, the choice of revascularization strategy did not lead to a significant difference in outcomes (Serruys et al., 2009). On the other hand, new surgical techniques such as off-pump CABG or minimally invasive CABG with hybrid PCI may also significantly alter the risk-benefit balance. With these advances, it is likely that the optimal strategy for revascularization will continue to evolve in the coming years and become increasingly individualized.

Cross-References

- ▶ Bypass Surgery
- ▶ Coronary Artery Disease

References and Readings

Eagle, K. A., Guyton, R. A., Davidoff, R., Edwards, F. H., Ewy, G. A., Gardner, T. J., et al. (2004). ACC/AHA 2004 guideline update for coronary artery bypass graft surgery: Summary article: A report of the American College of Cardiology/American Heart Association task force on practice guidelines (committee to update the 1999 guidelines on coronary artery bypass graft surgery). *Circulation, 110*, 1168–1176.

Morrow, D. A., & Gersh, B. J. (2008). Chronic coronary artery disease. In P. Libby, R. O. Bonow, D. L. Mann, D. P. Zipes, & E. Braunwald (Eds.), *Braunwald's heart disease: A textbook of cardiovascular medicine* (pp. 1353–1417). Philadelphia: Saunders Elsevier.

Serruys, P. W., Morice, M., Kappetein, A. P., Colombo, A., Holmes, D. R., Mack, M. J., et al. (2009). Percutaneous coronary intervention versus coronary-artery bypass grafting for severe coronary artery disease. *The New England Journal of Medicine, 360*, 961–972.

Seung, K. B., Park, D., Kim, Y., Lee, S., Lee, C. W., Hong, M., et al. (2008). Stent versus coronary-artery bypass grafting for left main coronary artery disease. *The New England Journal of Medicine, 358*, 1781–1792.

Yusuf, S., Zucker, D., Passamani, E., Peduzzi, P., Takaro, T., Fisher, L. D., et al. (1994). Effect of coronary artery bypass graft surgery on survival: Overview of 10-year results from randomised trials by the coronary artery bypass graft surgery trialists collaboration. *Lancet, 344*(8922), 563–570.

Coronary Artery Disease

▶ Coronary Heart Disease

Coronary Event

Siqin Ye
Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Synonyms

Cardiac events

Definition

Although no standard definition exists, the term coronary event is used in clinical research to refer to adverse events caused by disease processes affecting the coronary arteries. These may include what are termed "hard" events such as deaths that are attributed to coronary artery disease and nonfatal myocardial infarctions, but also occasionally "soft" events such as angina or revascularizations for worsening coronary artery stenosis. Because coronary event is often such a composite of clinical events of varying significance, there remains considerable debate on what should constitute the most appropriate component endpoints and how to define them, with the recognition that these choices may significantly influence the results and impact of clinical trials and other studies (Kip, Hollabaugh, Marroquin, & Williams, 2008). To address this, guidelines such as the 2001 ACC Clinical Data Standards and the 2007 Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction Expert Consensus Document have been released to standardize the definitions of the most important clinical events such as cardiovascular death and myocardial infarction.

Cross-References

▶ Acute Myocardial Infarction
▶ Angina Pectoris
▶ Cardiac Events
▶ Coronary Artery Bypass Graft (CABG)

References and Readings

ACC Writing Committee for Acute Coronary Syndromes Clinical Data Standards & ACC Task Force on Clinical Data Standards. (2001). American College of Cardiology key data elements and definitions for measuring the clinical management and outcomes of patients with acute coronary syndromes. *Journal of the American College of Cardiology, 38*(7), 2114–2130.

Kip, K. E., Hollabaugh, K., Marroquin, O. C., & Williams, D. O. (2008). The problem with composite end points in cardiovascular studies. *Journal of the American College of Cardiology, 51*(7), 701–707.

Thygesen, K., Alpert J. S., White H. D., on behalf of the Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction. (2007). Universal definition of myocardial infarction. *European Heart Journal*, *28*(20), 2525–2538.

Coronary Heart Disease

William Whang
Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Synonyms

Coronary artery disease

Definition

Coronary heart disease (CHD) is a condition in which the arteries that supply the cardiac muscle,

the coronary arteries, develop reduced luminal size due to the presence of atherosclerosis.

Description

Coronary heart disease (CHD) is a condition in which the arteries that supply the cardiac muscle, the coronary arteries, develop reduced luminal size due to the presence of atherosclerosis (Antman, Selwyn, Braunwald, & Loscalzo, 2008). Atherosclerosis is a progressive condition that starts as fatty streaks and may result in plaque development and ultimately in flow-limiting narrowing or occlusion of coronary arteries. The clinical manifestations of CHD include conditions such as angina, myocardial infarction, and heart failure. Angina, or chest discomfort, develops due to an imbalance between myocardial oxygen demand and the available blood supply. In myocardial infarction, plaque rupture and clot formation at the site of rupture result in occlusion of the artery and loss of blood flow to the heart muscle.

CHD is the major cause of one-third of all deaths in individuals older than 35, and one-half of all middle-aged men and one-third of middle-aged women in the United States will develop CHD (Lloyd-Jones, Larson, Beiser, & Levy, 1999). The Framingham Heart Study, a prospective cohort study of 5,209 individuals that began in 1948 and has continued to collect information and add participants, has defined many of the risk factors for atherosclerotic disease through epidemiologic techniques. Risk factors for coronary heart disease include other medical conditions such as hypertension, diabetes mellitus, and dyslipidemia, as well as behavioral factors such as cigarette smoking (Lloyd-Jones et al., 2010).

Atherosclerotic plaques associated with myocardial infarction are known to have certain features that increase the propensity to develop rupture and clot (Antman et al., 2008). Postmortem studies from cases of sudden death associated with CHD have revealed plaques with thin fibrous caps, relatively large lipid cores, and a high content of a particular type of inflammatory cell, macrophages. It is thought that rupture or erosion of the thin fibrous cap results in activation of the clotting cascade and development of occlusive clot.

Treatment of individuals with coronary heart disease involves primary or secondary prevention of myocardial infarction and heart failure (Antman et al., 2008). Medications such as aspirin and clopidogrel prevent clot formation, beta blockers reduce myocardial workload, and statin medications reduce cholesterol and stabilize coronary plaques. Angiotensin-converting enzyme (ACE) inhibitors reduce blood pressure and resistance in the small arteries and have been shown to prevent cardiac events particularly in patients who have developed left ventricular dysfunction.

Nonpharmacologic treatment of CHD includes percutaneous coronary intervention (PCI), a procedure that involves dilatation of the coronary artery lumen with a balloon and usually followed by implant of a coronary stent (Antman et al., 2008). PCI has been shown to reduce cardiac events in patients who present with myocardial infarction or angina at rest. In some patients with severe CHD involving all three main coronary vessels, particularly in the setting of diabetes or reduced ventricular function, coronary artery bypass graft surgery is a better treatment option.

In terms of behavioral interventions for prevention of CHD, regular aerobic exercise has been associated with improvement in multiple coronary artery disease risk factors, including blood pressure, serum cholesterol, glucose intolerance, and body mass index (Thompson et al., 2007). In addition, healthy dietary patterns have been associated with improved cardiac mortality, as well as lower blood pressure and serum cholesterol (Appel et al. 1997, 2005; Knoops et al., 2004). The 2006 dietary guidelines of the American Heart Association have emphasized maintaining a healthy dietary pattern over a focus on specific nutrients (Lichtenstein et al., 2006). Generally, the recommendations encourage consumption of a variety of fruits, vegetables, and grain products; fat-free dairy products; legumes; poultry; lean meats; and fish, preferably oily fish, at least twice a week. A meta-analysis of 38 randomized controlled trials conducted by the Cochrane Collaboration found that dietary advice reduced low-density lipoprotein cholesterol and blood pressure (Brunner,

Rees, Ward, Burke, & Thorogood, 2007). However, the largest randomized trial to date of a dietary intervention to reduce cardiovascular risk, the Women's Health Initiative Dietary Modification Trial, found no effect on cardiovascular events of group and individual sessions to reduce total fat intake and increase intake of vegetables, fruits, and grains, among 48,835 postmenopausal women (Howard et al., 2006).

A substantial literature has developed documenting the link between psychosocial factors and coronary heart disease, including depression, anger, and anxiety (Albus, 2010). A meta-analysis of 11 prospective cohort studies of healthy individuals estimated a relative risk of 1.64 for adverse cardiac events, including myocardial infarction (MI) and cardiac death, associated with depression (Rugulies, 2002).

Cross-References

▶ Ischemic Heart Disease

References and Readings

Albus, C. (2010). Psychological and social factors in coronary heart disease. *Annals of Medicine, 42*(7), 487–494.

Antman, E. M., Selwyn, A. P., Braunwald, E., & Loscalzo, J. (2008). Chapter 237. Ischemic heart disease. In A. S. Fauci, E. Braunwald, D. L. Kasper, S. L. Hauser, D. L. Longo, J. L. Jameson, & J. Loscalzo (Eds.), *Harrison's principles of internal medicine* (17th ed.). New York: McGraw-Hill.

Appel, L. J., Moore, T. J., Obarzanek, E., Vollmer, W. M., Svetkey, L. P., Sacks, F. M., et al. (1997). A clinical trial of the effects of dietary patterns on blood pressure. *The New England Journal of Medicine, 336*(16), 1117–1124.

Appel, L. J., Sacks, F. M., Carey, V. J., Obarzanek, E., Swain, J. F., Miller, E. R., 3rd, et al. (2005). Effects of protein, monounsaturated fat, and carbohydrate intake on blood pressure and serum lipids: results of the OmniHeart randomized trial. *Journal of the American Medical Association, 294*(19), 2455–2464.

Brunner, E. J., Rees, K., Ward, K., Burke, M., & Thorogood, M. (2007). Dietary advice for reducing cardiovascular risk. *Cochrane Database System Reviews* (4), CD002128.

Howard, B. V., Van Horn, L., Hsia, J., Manson, J. E., Stefanick, M. L., Wassertheil-Smoller, S., et al. (2006). Low-fat dietary pattern and risk of cardiovascular disease: The women's health initiative randomized controlled dietary modification trial. *Journal of the American Medical Association, 295*(6), 655–666.

Knoops, K. T., de Groot, L. C., Kromhout, D., Perrin, A. E., Moreiras-Varela, O., Menotti, A., et al. (2004). Mediterranean diet, lifestyle factors, and 10-year mortality in elderly European men and women: The HALE project. *Journal of the American Medical Association, 292*(12), 1433–1439.

Lichtenstein, A. H., Appel, L. J., Brands, M., Carnethon, M., Daniels, S., Franch, H. A., et al. (2006). Diet and lifestyle recommendations revision 2006: A scientific statement from the American Heart Association Nutrition Committee. *Circulation, 114*(1), 82–96.

Lloyd-Jones, D., Adams, R. J., Brown, T. M., Carnethon, M., Dai, S., De Simone, G., et al. (2010). Executive summary: Heart disease and stroke statistics–2010 update: A report from the American Heart Association. *Circulation, 121*(7), 948–954.

Lloyd-Jones, D. M., Larson, M. G., Beiser, A., & Levy, D. (1999). Lifetime risk of developing coronary heart disease. *The Lancet, 353*(9147), 89–92.

Rugulies, R. (2002). Depression as a predictor for coronary heart disease. A review and meta-analysis. *American Journal of Preventive Medicine, 23*(1), 51–61.

Thompson, P. D., Franklin, B. A., Balady, G. J., Blair, S. N., Corrado, D., Estes, N. A., 3rd, et al. (2007). Exercise and acute cardiovascular events placing the risks into perspective: A scientific statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism and the Council on Clinical Cardiology. *Circulation, 115*(17), 2358–2368.

Coronary Heart Disease (CHD)

▶ Heart Disease and Smoking
▶ Heart Disease and Type A Behavior

Coronary Vasoconstriction

Leah Rosenberg
Department of Medicine, School of Medicine, Duke University, Durham, NC, USA

Definition

Coronary vasoconstriction is the process by which vessels of the heart reduce their overall diameter. This functional capacity is mediated

by a variety of intrinsic and extrinsic stimuli, and may be pathologic and life-threatening.

Description

There are a variety of means by which the arterial and venous flow of the heart are affected by the overall circulating volume in the body. One crucial effector is the sympathetic nervous system, upregulated or suppressed in different situations. The sympathetic nervous system involves the secretion of the hormones epinephrine and norepinephrine from the adrenal medulla. Other important endogenous mediators include endothelin-1, serotonin, thromboxane, and prostaglandins (Rose & Post, 2010).

Coronary Vasoconstriction in Acute Coronary Syndromes and Variant (Prinzmetal's) Angina

Acute coronary syndromes, a term encompassing unstable angina and myocardial infarction, are brought about by a variety of pathophysiological changes that include coronary vasoconstriction. Myocardial infarctions are clinical sequelae of plaque disruption and clot formation over the plaque. Other elements involve platelet activation, dysregulation of the coagulation system, imbalance of myocardial oxygen demand, and finally plaque rupture resulting in vessel occlusion and cell death (Gelfland, Gelfland, & Cannon, 2009). Coronary vasoconstriction, induced by local and circulating levels of vasoconstrictors, also contributes to ischemia or infarction.

Prinzmetal's angina is another clinical syndrome that involves coronary vasoconstriction without plaque disruption. Also referred to as variant angina, Prinzmetal's angina is primarily caused by vasospasm without plaque disruption and thrombus formation. While many Prinzmetal's patients also have atherosclerotic lesions, the clinical presentation and electrocardiogram changes are caused by functional narrowing from coronary vasoconstriction.

Coronary vasoconstriction is the central mechanism of myocardial infarction caused by cocaine. As an analogue of sympathetic outflow, cocaine that is smoked, injected, or inhaled may cause of type of transient vascular obstruction that can lead to myocardial infarction and result in death of the myocardium.

Cross-References

- ▶ Arteries
- ▶ Cardiovascular Disease
- ▶ Vasoconstriction

References and Readings

Gelfland, E. V., Gelfland, E., & Cannon, C. (2009). *Management of acute coronary syndromes*. London: Wiley.

Rose, B., & Post, T. (2010). *Regulation of the effective circulating volume*. UpToDate Online 18.3.

Cortical Activity

▶ Brain Wave

Cortical Dementia

- ▶ Alzheimer's Disease
- ▶ Dementia

Corticosteroids

▶ Glucocorticoids

Corticotropin-Releasing Hormone (CRH)

Jennifer Heaney
School of Sport and Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

Definition

Corticotropin-releasing hormone (CRH) or corticotropin-releasing factor is a hormone that is secreted by the hypothalamus. It is one of the

hypophysiotropic hormones, a group of hormones produced by the hypothalamus that affect the anterior pituitary gland; CRH stimulates the secretion of adrenocorticotropic hormone (ACTH) from the anterior pituitary. CRH plays a key role in the endocrine response to stress as it is involved in one of the initial stages of activation of the hypothalamic-pituitary-adrenal axis (HPA axis) (Martin, Reichlin, & Brown, 1977).

CRH is not only produced in response to stress but exhibits a circadian rhythm of secretion across a 24-h period as a result of input from the central nervous system (Martin et al., 1977). Consequently, hormones that are produced in response to CRH, ACTH and cortisol, also demonstrate a circadian pattern of secretion (Greenspan & Forsham, 1983).

Cross-References

► ACTH

References and Readings

Greenspan, F. S., & Forsham, P. H. (1983). *Basic and clinical endocrinology*. Los Altos, CA: Lange Medical Publications.

Martin, J. B., Reichlin, S., & Brown, G. M. (1997). *Clinical neuroendocrinology*. Philadelphia: F.A. Davis.

Cortisol

Tobias Stalder and Clemens Kirschbaum
Chair of Biopsychology, Technische Universität Dresden, Dresden, Saxony, Germany

Definition

Cortisol (or "hydrocortisone") is a steroid hormone which is essential for life. It is produced in the adrenal cortex and is predominantly regulated by the neuroendocrine hypothalamus-pituitary-adrenal (HPA) axis. Cortisol fulfills vital functions in the regulation of various homeostatic processes and is particularly well known for its role in the body's response to physical and psychological stress. These characteristics combined with its high potency and multitude of physiological effects make cortisol a hormone of prime interest for research in the area of behavioral medicine.

Description

Biosynthesis and Basic Characteristics

Cortisol is mainly synthesized and secreted from the *zona fasciculata* of the adrenal cortex. In addition, it is also produced in smaller amounts in other tissues, including hair follicle cells, the placenta, and the brain (Ito et al., 2005). The main precursor for the production of cortisol is cholesterol from which it is derived via two alternative paths involving several intermediate metabolic steps. As most other hormones, cortisol is secreted in a pulsatile fashion with marked circadian rhythmicity and a mean production ranging from 8 to 25 mg/24 h (mean production: ~13 mg/24 h). Due to its relatively small size (molecular weight: 362.5 Da) and its lipophilic nature, cortisol is able to freely diffuse in and out of target cells.

Cortisol in Blood

Following its synthesis in the adrenal cortex, cortisol is secreted into the blood stream where most of it binds to transport proteins. Approximately 70% of cortisol molecules are bound to cortisol-binding globulin (CBG or transcortin) via high-affinity receptors. A further 15–20% of cortisol is bound to lower-affinity receptors of albumin while an additional 5% is also bound to erythrocytes. Hence, only about 5–10% of cortisol circulates as an unbound or "free" hormone in the blood. Following the free hormone hypothesis (Mendel, 1989), only this unbound fraction of cortisol can enter target cells and is thus biologically active while the larger part of bound cortisol serves as an inactive reservoir. It is assumed that mechanisms influencing the level of circulating transport proteins play an important role in regulating the functional potency of the cortisol signal. Estimates of the biological half-life ($T_{1/2}$)

of unbound cortisol in blood range from 60 to 115 min. The bioavailability of cortisol is thus relatively long compared to other hormones, such as epinephrine or β-endorphin, which show a $T_{1/2}$ in the range of seconds to a few minutes.

Physiological Actions

Mechanisms of Signal Transduction

Cortisol binds to two main types of receptors, the mineralocorticoid (MR) and glucocorticoid receptors (GR). The two receptor types differ with regard to their affinity for cortisol with MRs showing a 6–10 times higher affinity than GRs. As a result, about 90% of MRs are occupied throughout the day, while higher GR occupancy is only reached at times of peak cortisol secretion or during stress responses. Besides their affinity for cortisol, MRs and GRs also differ in terms of their distribution pattern with cortisol-responsive MRs being predominantly located in the kidneys and limbic structures of the brain, while GRs are expressed widely throughout the brain as well as in peripheral tissues (De Kloet, Joëls, & Holsboer, 2005).

The "classical" mechanism of cortisol action comprises its genomic effects. Unbound cortisol is able to freely diffuse into the cells of the body where it binds to high-affinity receptors in the cytoplasm. While unoccupied receptors are guarded by heat-shock-proteins (HSP), cortisol binding releases the HSP which enables the cortisol-receptor complex to enter the cell nucleus. Here it binds to specific sites of the deoxyribonucleic acid (DNA) and acts as a transcription factor to alter the cell's protein biosynthesis. Subsequently, the cortisol-receptor complex is transported back into the cytoplasm. Here it disintegrates and the cortisol molecule, which may have been structurally altered, exits the cell into the extracellular space.

While the time course of genomic effects of cortisol is relatively slow ranging from several minutes to hours, cortisol also affects cell function via faster non-genomic mechanisms. These mechanisms influence a wide range of intracellular processes and are of importance across many peripheral as well as central structures. Targets of non-genomic cortisol action might include lipids and proteins in the cell membrane and cytoplasm as well as membrane MR and GR.

Tissue effects of cortisol are markedly influenced by enzymatic action within target cells. Here, two variants of the enzyme 11β-hydroxysteroid dehydrogenase (11β-HSD) are of particular importance. 11β-HSD type 1, which predominates in adipose and hepatic tissues, converts inactive cortisone to active cortisol and thus has an amplifying effect on local cortisol action. On the other hand, 11β-HSD type 2 is primarily found in the kidneys and placenta where it converts cortisol to its inactive metabolite cortisone.

Effects of Cortisol

Cortisol has a wide range of effects on target tissues throughout the body. Indeed, cortisol is so essential that humans cannot survive removal of the adrenal glands unless glucocorticoid replacement is provided. The effects of cortisol are often permissive rather than direct, which means that it frequently does not initiate an action but provides an environment for the action to take place. Importantly, while being adaptive at normal concentrations of cortisol, many of its actions can have deleterious effects at aberrant concentrations. Both excessive levels and underproduction of cortisol have been implicated in the etiology of various diseases (Chrousos, 2009).

Effects on Carbohydrate and Lipid Metabolism

Cortisol is the primary glucocorticoid in humans, which hints to the fact that one of its pivotal functions lies in facilitating the mobilization of energy resources. It enhances gluconeogenesis in the liver and reduces glucose uptake into muscle and adipose tissue, which increases the amount of glucose available for the body. Cortisol also crucially augments the conversion of protein to glycogen and thus helps to maintain hepatic glycogen stores on which other hormones, like glucagon, can subsequently act to increase glucose levels. Cortisol also increases the breakdown of proteins stored in muscle, bone, and connective tissue and inhibits protein synthesis in non-hepatic tissues which increases the

amount of protein available for gluconeogenesis. Importantly, while this catabolic action is physiologically beneficial at adequate concentrations of cortisol, at excessive levels, it results in the depletion of protein stores which can manifest in symptoms such as thinning of the skin, reduced muscle mass, or osteoporosis.

Besides aiding proteolysis, cortisol is also assumed to facilitate the mobilization of free fatty acids from fat depots which further supports gluconeogenesis. However, cortisol may also have stimulatory effects on appetite and calorie intake and leads to enhanced fat deposition in abdominal and facial areas. Under conditions of chronically elevated cortisol secretion, e.g., in Cushing's syndrome, this leads to a characteristic pattern of central adiposity, as well as fat depositions in the face ("moon face") and at the neck ("buffalo hump").

Effects on Electrolyte Metabolism

The effects of cortisol on sodium and water retention are considerably weaker than those of aldosterone, the primary mineralocorticoid hormone in humans. However, this lack in potency is outweighed by the approximately 200-fold higher concentrations of cortisol compared to aldosterone which indicates that cortisol also plays an important role electrolyte metabolism.

Immunological Effects

Cortisol is the most potent endogenous immunosuppressive substance with strong anti-inflammatory effects. Virtually all steps involved in the local inflammatory response to injury, e.g., dilation of capillaries or tissue swelling, are inhibited by cortisol. It also decreases leukocyte recruitment and effectiveness at the site of inflammation. These effects of cortisol have long been recognized and used in anti-inflammatory drug treatments. Cortisol also profoundly suppresses the immune response to antigens, e.g., by reducing the number and activity of thymus-derived lymphocytes (T-cells). In addition, cortisol inhibits other components of the immune response such as cytokine synthesis, proliferation and differentiation of monocytes as well as activity of macrophages and natural killer cell.

Effects on Brain and Cognition

Cortisol is able to enter the brain where it affects a wide range of neuronal processes and cognitive functions. Cortisol exerts these actions both via the slower genomic pathways as well as via fast non-genomic effects which directly affect the responsivity of neuronal networks. Via these pathways, cortisol interacts with the neurotransmitter systems (including noradrenergic, serotonergic, dopaminergic and cholinergic, and GABAergic neurotransmission) as well as with neuropeptidergic systems, e.g., oxytocin and arginine vasopressin.

One of the best described effects of cortisol on cognitive functions is an enhancing influence on the encoding and consolidation of emotionally relevant information under arousing conditions (de Quervain et al., 2009). However, while cortisol may enhance memory consolidation, acutely elevated cortisol levels are also associated with impaired memory retrieval, particularly of declarative memory, as well as with compromised working memory function. Despite the involvement of other brain regions (e.g., hippocampus and medial prefrontal cortex), close reciprocal interactions between cortisol and noradrenergic neurotransmission in the basolateral nucleus of the amygdala are assumed to be of particular importance for the modulation of these effects on memory. Besides effects on memory, a stimulatory influence of cortisol on psychological arousal has also been reported. Similarly, cortisol can lead to increased amplitude and decreased latency of EEG event-related potentials and heightened EEG frequency.

In addition to these actions under normal functioning, pharmacological administration of high doses of glucocorticoids has been associated with profound psychoactive effects (Lupien, Maheu, Tu, Fiocco, & Schramek, 2007). These include the experience of psychiatric symptoms, such as depression, mania, and psychotic episodes, often collectively referred to as "steroid psychosis." Interestingly, during the initial phase of treatment, elevations of mood and euphoria are often seen while dysphoric mood states and depression predominate with prolonged treatment. This is in line with the fact that Cushing's syndrome,

i.e., chronic endogenous hypercortisolemia, is also frequently associated with depression and/or other psychiatric symptoms which usually subside with successful treatment. Importantly, chronic exposure to excessive amounts of cortisol has also been associated with hippocampal atrophy and cell death as well as with deficits in hippocampus-dependent cognitive functioning. This effect might play a particular role with regard to the cognitive decline often seen with older age.

Regulation

Overview

The synthesis and secretion of cortisol from the adrenal cortex is predominantly controlled by the neuroendocrine HPA axis, a signaling cascade involving the release of corticotropin-releasing hormone (CRH) and adrenocorticotropin hormone (ACTH) as well as numerous other substances, specifically neuropeptides. Both the activity of the HPA axis as well as the secretion of cortisol occur in a pulsatile fashion with approximately 12–18 ultradian pulses per 24-h span. The concentration of circulating cortisol is determined by the frequency and amplitude of individual pulses. The release of cortisol via the HPA axis is under tight negative feedback control, with cortisol inhibiting its own secretion by downregulating levels of CRH and ACTH.

Besides regulation via the HPA axis, there is also considerable evidence that levels of ACTH and cortisol can dissociate under various conditions, suggesting additional extra-pituitary regulatory mechanisms. Here, sympathetic innervations of the adrenal gland via the splanchnic nerve are likely to be of special importance. This pathway is assumed to particularly modulate cortisol secretion by altering adrenal sensitivity to ACTH and is likely to involve both intra-adrenal paracrine interactions as well as direct splanchnic innervation of the adrenal cortex (Bornstein, Engeland, Ehrhart-Bornstein, & Herman, 2008).

Basal Secretion

The secretion of cortisol is subject to considerable circadian variation: Following a circadian nadir during the early night, cortisol levels show a strong increase during the second half of sleep. Upon morning awakening, an additional rise in cortisol levels for approximately 30–40 min post-awakening is seen (the "cortisol awakening response," CAR; Fries, Dettenborn, & Kirschbaum, 2009) which results in circadian peak levels being reached. Subsequently, cortisol levels show a gradual decline over the remainder of the day until they again reach nadir levels during the first half of sleep.

Response to Physiological and Psychological Stress

A prominent feature of cortisol secretion is its marked increase in response to both physiological as well as psychological stress. With regard to the former, cortisol responses have been shown following intense exercise or hard physical work. For a significant cortisol response to occur under these conditions, exercise has to be highly intense or sustained with a maximum oxygen uptake (VO_{2max}) over 70%. The cortisol response to exercise shows no habituation after repeated exposure. In addition to exercise and intense physical work, a range of other physical conditions have been shown to result in cortisol responses, e.g., pain and physical trauma, hypoglycemia, hypoxemia, increased insulin levels, or consumption of a protein-rich meal.

It is now well established that cortisol secretion also responds strongly to psychologically challenging conditions. The magnitude of this response is dependent on both, external factors and characteristics of the individual. Situations containing both uncontrollable and social-evaluative elements tend to result in the largest cortisol responses (Dickerson & Kemeny, 2004). A wide range of psychological factors (e.g., appraisal of the situation, personality traits, coping and attributional style, perceived social support, or adverse early life experiences) as well as physiological predispositions (sex, age, or genetic factors) have been shown to influence the magnitude of the cortisol stress response (Foley & Kirschbaum, 2010). In contrast to the response to physiological challenge, the cortisol response to psychological stress shows

considerable habituation following repeated exposure to the same stress-eliciting situation.

Measurement

Modern laboratory methods allow for rapid and economic cortisol determination in different matrices. Most frequently, cortisol levels are measured in blood, saliva, or urine samples which usually provide information on cortisol production and levels over relatively short time intervals (minutes to days). A recent addition to this methodology is the measurement of cortisol concentrations in hair which is assumed to provide an index of integrated cortisol secretion over prolonged periods of up to several months.

Blood Plasma or Serum

The assessment of cortisol in blood (both plasma and serum) reflects acutely circulating concentrations and thus provides a valuable approach for assessing momentary cortisol levels or dynamic changes in cortisol secretion. Importantly, the assessment of total cortisol in blood comprises both bound and unbound cortisol and their separation can be time consuming and thus expensive. Consequently, blood assessments are not best suited for assessing the bioavailable fraction of cortisol. In addition, blood sampling bears a minor risk of infection and through its invasive nature may itself trigger an acute cortisol stress response.

Saliva

As with cortisol assessments in blood, salivary measurements also reflect acutely circulating cortisol levels. However, as only unbound cortisol can passively diffuse into saliva, salivary cortisol levels only represent the free, biologically active fraction. Importantly, the level of salivary cortisol is unrelated to salivary flow rate and shows only a minimal time lag of 1–2 min to plasma cortisol levels. In addition, saliva sampling is an unintrusive and generally well-accepted method which may easily be carried out under ambulatory conditions. Salivary cortisol assessments are thus increasingly used as the method of choice to determine acute levels of biologically active cortisol in human research (Kirschbaum & Hellhammer, 1994).

A potential limitation relating to both blood and salivary assessments of cortisol is that single spot samples only reflect cortisol secretion during the acute sampling situation. Since many situational variables are known to influence cortisol secretion (see above), drawing conclusions regarding overall functional cortisol status from such "spot data" can be misleading.

Urine

A considerable amount of circulating cortisol is metabolized and excreted into urine. The assessment of urinary cortisol metabolites thus provides a measure of cumulative cortisol secretion over the time period during which urine samples were collected. By using an extended collection period of, e.g., 24 h, the respective results are less influenced by momentary fluctuations in cortisol levels but integrate overall secretory patterns over the sampling period.

Hair

The examination of endogenous cortisol concentrations in human hair has recently been introduced as a new measure of steroid hormone determination. As it is assumed that cortisol is incorporated into the hair shaft during hair growth, the examination of cortisol levels in a specific hair segment is believed to provide a retrospective index of cumulative cortisol secretion over the period during which the respective hair segment has grown. Given a hair growth rate of approximately 1 cm/month, the examination of a 3 cm hair segment should allow the assessment of cumulative cortisol levels over a 3 months period. This largely extended window of examination combined with the possibility of a *retrospective* assessment highlights the potential of hair cortisol analysis as an important future research tool (Stalder & Kirschbaum, in press).

Cross-References

▶ Corticosteroids
▶ Corticotropin-Releasing Hormone (CRH)

- ▶ Glucocorticoids
- ▶ Hypothalamic-Pituitary-Adrenal Axis

References and Readings

Bornstein, S. R., Engeland, W. C., Ehrhart-Bornstein, M., & Herman, J. P. (2008). Dissociation of ACTH and glucocorticoids. *Trends in Endocrinology and Metabolism, 19*, 175–180.

Chrousos, G. P. (2009). Stress and disorders of the stress system. *Nature Review of Endocrinology*, *5*(7), 374–381.

De Kloet, E. R., Joëls, M., & Holsboer, F. (2005). Stress and the brain: From adaptation to disease. *Nature Reviews Neuroscience, 6*, 463–475.

De Quervain, D. J., Aerni, A., Schelling, G., & Roozendaal, B. (2009). Glucocorticoids and the regulation of memory in health and disease. *Frontiers in Neuroendocrinology, 30*, 358–370.

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130*, 355–391.

Foley, P., & Kirschbaum, C. (2010). Human hypothalamus-pituitary-adrenal axis responses to acute psychosocial stress in laboratory settings. *Neuroscience & Biobehavioral Reviews, 35*, 91–96.

Fries, E., Dettenborn, L., & Kirschbaum, C. (2009). The cortisol awakening response (CAR): Facts and future directions. *International Journal of Psychophysiology, 72*, 67–73.

Ito, N., Ito, T., Kromminga, A., Bettermann, A., Takigawa, M., Kees, F., et al. (2005). Human hair follicles display a functional equivalent of the hypothalamic-pituitary-adrenal axis and synthesize cortisol. *The FASEB Journal, 19*, 1332–1334.

Kirschbaum, C., & Hellhammer, D. H. (1994). Salivary cortisol in psychoneuroendocrine research – Recent developments and applications. *Psychoneuroendocrinology, 19*, 313–333.

Lupien, S. J., Maheu, F., Tu, M., Fiocco, A., & Schramek, T. E. (2007). The effects of stress and stress hormones on human cognition: Implications for the field of brain and cognition. *Brain and Cognition, 65*, 209–237.

Mendel, C. M. (1989). The free hormone hypothesis: A physiologically based mathematical model. *Endocrine Reviews, 10*, 232–274.

Stalder, T., Kirschbaum, C. (in press). Analysis of cortisol in hair – State of the art and future directions. *Brain, Behavior and Immunity*. doi:10.1016/j.bbi.2012.02.002.

Cortisone

- ▶ Glucocorticoids

Cost Analysis

- ▶ Cost-Minimization Analysis

Cost Identification

- ▶ Cost-Minimization Analysis

Cost-Benefit Analysis (CBA)

- ▶ Benefit Evaluation in Health Economic Studies

Cost-Comparison Analysis

- ▶ Cost-Minimization Analysis

Cost-Effectiveness

Stephen Birch[1] and Amiram Gafni[2]
[1]Clinical Epidemiology and Biostatistics (CHEPA), McMaster University, Hamilton, ON, Canada
[2]Department of Clinical Epidemiology and Biostatistics, Centre for Health Economics and Policy Analysis, McMaster University, Hamilton, ON, Canada

Definition

Cost-effectiveness is concerned with improving the performance of a health care system by ensuring the resources available to a health care system are used in their most productive way. This can only be achieved through careful consideration of the full consequences and opportunity costs of introducing a new health care program in the context or setting in which it is to be introduced. Appropriate evaluation methods must be

employed to accommodate this information and avoid simplifying assumptions that threaten the evaluation's validity.

Description

Economic evaluation has been defined as "ensuring that the value of what is gained from an activity outweighs the value of what has to be sacrificed" (Wiliams 1983). Hence, economic evaluation reflects the fundamental principles of economics that (1) resources are scarce, (2) choices are made between alternative uses of resources, and (3) a particular deployment of resources involves forgoing the benefits generated from alternative deployments of the same resources. Hence, it requires consideration of both outcome measurement and opportunity cost. Cost-Effectiveness Analysis (CEA) is the most common methodology of economic evaluation in health care, aimed at informing decision-makers faced with constrained resources. For a particular level of health care resources, which need not be the current level, the challenge is to choose from among all possible health care programs the combination of programs that maximizes total health benefits produce.

The theoretical basis for CEA derives from a decision-maker with a fixed budget choosing between many possible programs based on a comparison of the difference in effects between a program under consideration and the current way of serving the same patient population (incremental effects), and the difference in costs between the two programs (incremental costs). Where incremental costs and incremental effects have different signs, the solution is trivial, for example, the new program costs more (i.e., reduces resources available for other unrelated programs) and produces less effects than the current program. Similarly with negative incremental costs and positive incremental effects, a "win-win," no substantial reflection is required. In most cases, however, a new intervention involves incremental effects and incremental costs with the same sign, for example, the intervention is more effective but costs more than the existing intervention. To provide the greater effects of the new treatment, the number of other unrelated treatments must be reduced to release resources to support the additional costs of the new treatment. Here the decision-maker looks to the economist for "inputs" to the decision-making process – in particular decision rules for CEA.

The Decision Rules of CEA

The traditional analytical tool of CEA is the incremental cost-effectiveness ratio (ICER), the incremental cost of the new program divided by the incremental effects of the new program. Maximum health gain from available resources is produced under the following decision rules:

The league table rule: Select programs in ascending order of ICER (i.e., project with lowest ICER first) until available resources are exhausted.

T*he threshold ICER rule*: Select programs with ICER less than or equal to λ, the shadow price of the budget.

Because ICERs have not been estimated for all programs currently delivered in health care systems, comprehensive league tables are not available and the league table rule cannot be followed. The threshold rule has provided the basis for economic evaluation guidelines in many jurisdictions. In each case the use of CEA is linked to addressing the problem of maximizing health improvements from available resources.

This solution is based on assumptions of perfect divisibility and constant returns to scale in all programs. Yet, such conditions do not hold generally in health care decision-making. One cannot divide up an investment to fit whatever budgetary amount is available. A manager must purchase an entire Magnetic Resonance Imaging (MRI) machine, it is not divisible, it is all or nothing. Apart from such physical constraints on divisibility, some programs may not be divisible because of political or ethical constraints. It is ethically problematic to offer vaccination to only 50% of children. Increasing investment in a particular program may not produce

proportionally equal increases in outcomes as program coverage expands from highest need/most severe patients to lesser need/severity groups. So the additional outcomes produced from investing resources in a program may diminish with the scale of the program. Even if the program under evaluation does exhibit constant returns to scale the opportunity, cost of the program is likely to have non-constant returns in the sense that increased resource requirements for the new program mean the decision-maker has to "dig deeper" into his existing budget to fund it. After resources from the least productive current program have been exhausted he must look to other more productive programs meaning that the marginal opportunity cost of the program increases with size.

Because decision-makers are faced with choices between programs of different sizes, and the opportunity costs of programs depend crucially on program size, the different programs are not directly comparable. The ICER is the *average cost per Quality Adjusted Life Year (QALY)* or the inverse of the average rate of return on additional investments required by a program. Comparisons of ICERs across programs ignore problems introduced by the different sizes of programs. They do not compare like with like. Moreover, decision-makers cannot purchase individual units of QALYs. Each program produces a "package" of QALYs, and the average price per QALY may differ by program size. Consequently the ICER threshold decision-rule is not sufficient to maximize health effects from available resources. There is no theoretical justification for asserting that the strategy with the lowest cost-effectiveness ratio is the most desirable one.

To adopt the threshold ICER approach in the absence of the theoretical assumptions requires an unspecified supply of resources with constant marginal opportunity cost. Anything further from the reality of decision-making is hard to imagine.

Even if the assumptions are accepted for the purposes of the theoretical model, the problem of determining a threshold remains. Under the model, the threshold is given by the opportunity cost of the marginal program funded from available resources. This is determined by constructing the ICER league table, but requires information on the incremental costs and effects of *all* possible programs. Hence, the threshold value required to make decisions that produce the maximization of health gains from available resources cannot be determined even if the theoretical assumptions hold.

Extending Economic Evaluation to Identify Efficiency Improvements

For an intervention to represent an efficient use of resources the additional effects it generates must exceed the effects forgone from the most productive alternative use of the same resources. Hence, efficiency cannot be established only by reference to the resources required and outcomes produced by a particular intervention. Information on alternative uses of those resources is also needed and so efficiency is context-specific. Even where incremental costs and effects of an intervention are identical in different settings, it does not mean the efficiency of that intervention is the same in all settings.

If economics is to inform decision-makers about the efficiency of investments, traditional approaches to CEA and the use of ICERs are insufficient. Mathematical approaches to constrained maximization, such as integer programming (IP), solve the decision-maker's problem and are the only universal approach to ranking programs according to efficiency under a resource constraint. The key requirement of the IP approach is that the specification of the problem (i.e., objective function and constraints) must accurately reflect the decision-maker's problem setting.

The substantial data requirements of the IP approach, specifically the incremental costs and effects of all programs together with the resources available for investment, may be difficult to satisfy. However, these requirements reflect the complex nature of the decision-maker's problem.

An alternative practical approach is available (Birch & Gafni 1992; Gafni & Birch 1993) which

satisfies a modified objective of an unambiguous increase in health improvements from available resources (i.e., an objective of improving, as opposed to maximizing, efficiency). This requires that the health improvements of the proposed program be compared with the health improvements produced by that combination of programs that have to be given up to generate sufficient funds for the proposed program. Only where the health improvements of the proposed program exceed the health improvements of the combination of programs to be given up does the new technology represent an improvement in the efficiency of resource utilization. The approach does not rely on an arbitrarily determined threshold value to ascertain the efficiency of the program, nor is it dependent on unrealistic assumptions about perfect divisibility and constant returns to scale. Instead, the source of additional resource requirements is identified and the implications of canceling programs to generate these resources form part of the analysis. Iterative application of this efficiency-improving approach would eventually lead to efficiency maximization as opportunities to further improve efficiency are exhausted.

Concern with maximizing health improvements from available resources may be just one of several objectives that decision-makers face. For example, political considerations associated with providing equal access to services and providing greater priority to health improvements of specific population groups may be important goals. However, the presence of multiple objectives and constraints does not reduce the importance of adopting a constrained maximization model as the basis for analysis. It remains important that whatever goals are identified, these must be pursued efficiently in order to avoid wasting resources. The explicit identification of each objective and constraint enables the full range of policy concerns to be incorporated systematically into the analysis. Hence, the complex objectives faced by decision-makers, far from limiting the role of economic analysis, represent precisely the challenges that the economic model of constrained maximization is intended to accommodate.

Cross-References

▶ Benefit Evaluation in Health Economic Studies

References and Readings

Birch, S., & Gafni, A. (1992). Cost-effectiveness/utility analyses: Do current decision rules lead us to where we want to be? *Journal of Health Economics, 11*, 279–296.

Birch, S., & Gafni, A. (2003). Economics and the evaluation of health care programmes: Generalisability of methods and implications for generalisability of results. *Health Policy, 64*, 207–219.

Birch, S., & Gafni, A. (2006a). Decision rules in economic evaluation. In A. Jones (Ed.), *The Elgar companion to health economics* (pp. 492–502). Cheltenham: Edward Elgar.

Birch, S., & Gafni, A. (2006b). The biggest bang for the buck or bigger bucks for the bang: The fallacy of the cost-effectiveness threshold. *Journal of Health Services Research and Policy, 11*, 46–51.

Drummond, M. (1980). *Principles of economic appraisal in health care*. Oxford: Oxford University Press.

Drummond, M., Sculpher, M., Torrance, G., O'Brien, B., & Stoddart, G. (2005). *Methods for the economic evaluation of health care programmes*. New York: Oxford University Press.

Gafni, A., & Birch, S. (1993). Guidelines for the adoption of new technology: A potential prescription for uncontrolled growth in expenditures and how to avoid it. *Canadian Medical Association Journal*, *148*, 913–917.

Gafni, A., & Birch, S. (2006). Incremental cost-effectiveness ratios (ICERs): The silence of the lambda. *Social Science and Medicine, 62*, 2091–2100.

Weinstein, M., & Zeckhauser, R. (1973). Foundations of cost effectiveness analysis for health and medical practices. *Journal of Public Economics, 2*, 147–157.

Wiliams, A. (1983). The economic role of health indicators. In G. Teeling-Smith (Ed.), *Measuring the social benefits of medicine* (pp. 63–67). London: Office of Health Economics.

Cost-Effectiveness Analysis (CEA)

▶ Benefit Evaluation in Health Economic Studies
▶ Cost-Effectiveness

Cost-Minimization Analysis

Alejandra Duenas
School of Management, IESEG, Paris, France

Synonyms

Cost analysis; Cost identification; Cost-comparison analysis

Definition

This term refers to an economic evaluation tool. Cost-minimization analysis is mostly applied in the health sector and is a method used to measure and compare the costs of different medical interventions. The principal limitations of this cost evaluation method are that it can only be used to compare treatments that provide the same benefits or effectiveness (identical outcomes, e.g., therapeutic effects); moreover, costs need to be determined accurately. In this way, a decision maker can choose the treatment with the lowest total cost. The assessment of costs is performed by identifying the study's perspective, all the resources used, and quantifying them into physical units. The most common perspectives are societal perspective (includes all costs incurred by health care services, social services, patients, and society in general) and third-party payer perspective (includes the costs incurred by an insurance company, a government, etc.). In order to quantify the resources used, a physical unit is defined, such as the number of hospital days, the time that a nurse spends with a patient, number of doctors' visits, etc. Once the units are defined and quantified, they are translated to costs by multiplying the unit costs by the number of units used.

The use of this tool is rather limited as it is difficult to demonstrate that the efficacy of two or more interventions is equivalent. A common application of cost-minimization analysis is the comparison of generic drugs in order to achieve market approval. Some experts consider that cost-minimization analysis is no longer useful (Briggs & O'Brien, 2001) and, furthermore, that other economic evaluation methods such as cost-utility, cost-benefit, and cost-effectiveness analyses are more comprehensive, given that they allow for the comparison of interventions with different effectiveness outcomes and the incorporation of uncertainty.

Cross-References

▶ Cost-Effectiveness Analysis (CEA)

References and Readings

Briggs, A. H., & O'Brien, B. J. (2001). The death of cost-minimization analysis? *Health Economics, 10*(2), 179–184.

Kobelt, G. (2002). *Health economics: An introduction to economic evaluation* (2nd ed.). London: Office of Health Economics.

Cost-Utility Analysis (CUA)

▶ Benefit Evaluation in Health Economic Studies

Couple Therapy

▶ Couple-Focused Therapy
▶ Therapy, Family and Marital

Couple-Focused Therapy

Beate Ditzen[1] and Tanja Zimmermann[2]
[1]Division of Clinical Psychology and Psychotherapy, Department of Psychology, University of Zurich, Binzmuhlestrasse, Zurich, Switzerland
[2]Department of Clinical Psychology, Psychotherapy and Diagnostics, University of Braunschweig, Braunschweig, Germany

Synonyms

Couple therapy; Marital therapy; Marriage counseling

Definition

Couple-focused therapy (CFT) is a psychological therapy with the focus of attention on the relationship between two individuals rather than on one individual. The aim of CFT is to enable a better level of functioning in couples – married or unmarried – who are experiencing distress in their relationship. Couples may seek CFT for a variety of reasons, such as distress in terms of finances, sexuality, communication, infidelity, or individual psychopathology as well as physical health problems with an impact for the couple. Consequently, CFT will differ according to the respective relationship problems. Moreover, couple interventions may also vary based on the phase of the relationship during which they occur: Whereas primary prevention programs or couple education (e.g., the Prevention and Relationship Enhancement Program, PREP, from Howard Markman) might be offered for prevention of future distress relatively early in the relationship, CFT is usually called for when severe problems are present.

In general, the first step of CFT is to identify the areas of dissatisfaction in the relationship, and to implement a treatment plan to which both partners are willing to agree. Based on this treatment plan, therapy sessions will differ according to the chosen model or the philosophy behind the therapy. In the following, some of the best-known approaches will be briefly characterized.

Behavior-Focused Therapy

Traditionally, behavior-focused therapy is based on the idea that both partners (possibly involuntarily) tend to reward and punish specific behaviors during the development of their relationship. Consequently, this behavior exchange is an important treatment focus (e.g., by providing encouragement of positive behavior) in behavioral couple therapy.

Cognitive-Behavioral Therapy

With its roots in behavioral therapy, cognitive-behavioral CFT has enriched the focus on behavior with the perspective on couples' beliefs regarding the relationship. Therapists aim at questioning and modulating presumptions about the positive (or more often negative) motives of each partner and thereby try to prevent negative behavior.

Psychoanalytical Therapy

Psychoanalytical CFT attempts to discover early developmental conflicts in relation to the present interpersonal interactions within the couple. In this approach, couples are thought to be able to improve their relationship through a better understanding of how early parent-child interactions might influence later behavior in adulthood.

Emotion-Focused Therapy

As indicated by the name, the main emphasis in emotion-focused CFT is on the identification and expression of emotional needs in the couple relationship. In particular, the expressions of underlying feelings are supposed to change the perception of the partner and motivate behavior change.

Integrative Therapy

In a number of more recent approaches, researchers have combined a variety of treatment strategies within a consistent theoretical framework, resulting in integrated treatment models (among others the Enhanced Cognitive-Behavioral Couple Therapy by Epstein and Baucom (2003), or the Integrative Behavioral Couple Therapy by Jacobson and Christensen (1998); also see Snyder (1999), Snyder, Castellani, and Whisman (2006)).

CFT programs are broadly evaluated treatment options with effect sizes in the range of $d = 0.72$ for communication and relationship satisfaction, whereas in comparison typically no changes in marital quality in untreated couples are observed (Baucom, Hahlweg, & Kuschel, 2003). However, it should be noted that CFT is no guarantee that the relationship will improve, and there are couples who might benefit more from ending their relationship than from continuing it. This makes the overall evaluation of CFT a challenging topic in behavioral medicine (cf., Christensen, Baucom, Vu, & Stanton, 2005).

Cross-References

- ▶ Cognitive Behavioral Therapy (CBT)
- ▶ Marital Satisfaction

References and Readings

Baucom, D. H., Hahlweg, K., & Kuschel, A. (2003). Are waiting-list control groups needed in future marital therapy outcome research? *Behavior Therapy, 34*, 179–188.

Christensen, A., Baucom, D. H., Vu, C. T., & Stanton, S. (2005). Methodologically sound, cost-effective research on the outcome of couple therapy. *Journal of Family Psychology, 19*(1), 6–17.

Epstein, N. B., & Baucom, D. H. (2003). *Enhanced cognitive-behavioral therapy for couples*. Washington, DC: American Psychological Association.

Jacobson, N. S., & Christensen, A. (1998). *Acceptance and change in couple therapy: A therapist's guide to transforming relationships*. New York: Norton.

Snyder, D. K. (1999). Affective reconstruction in the context of a pluralistic approach to couples therapy. *Clinical Psychology: Science and Practice, 6*(4), 348–365.

Snyder, D. K., Castellani, A. M., & Whisman, M. A. (2006). Current status and future directions in couple therapy. *Annual Review of Psychology, 57*, 317–344.

Covariance Components Model

- ▶ Hierarchical Linear Modeling (HLM)

Co-workers

Karen Jacobs[1], Miranda Hellman[2], Jacqueline Markowitz[2] and Ellen Wuest[2]
[1]Occupational Therapy, College of Health and Rehabilitation Science, Sargent College, Boston University, Boston, MA, USA
[2]Boston University, Boston, MA, USA

Synonyms

Associate; Collaborator; Colleague

Definition

A co-worker is a person who a worker works with, in their role as worker. Co-workers can share their knowledge and expertise when others are faced with problems or novel situations; this can be especially useful when alternative solutions are not readily accessible. The co-worker relationship can also have effects on workplace dynamics, individual stress level, and relationships. Positive relationships between co-workers can be seen as supportive and beneficial in dealing with day-to-day problems and strains arising from employment (Deery, Iverson, & Walsh, 2010), and positive relationships can increase job satisfaction, job involvement, and organizational commitment (Dur & Sol, 2008). This supportive relationship may be more likely to occur in interactionally intense and high stress settings and can help one cope with high job demands. The pace and intensity for the work can be regulated through collaboration between co-workers, and workplace norms are often established through co-worker interaction and collaboration (Deery et al., 2010).

Co-worker relationships can be influenced by a variety of personality traits. Matching co-workers into groups based on these personality traits can lead to strong group cohesion and can create an effective team (Tett & Murphy, 2002). Additionally, supportive and positive co-workers can promote an environment where new ideas are easily and comfortably discussed, which also has positive impacts on the group (Joiner, 2007). Conversely, a mismatch of personality traits can have negative impacts on group dynamics (Tett & Murphy, 2002).

Cross-References

- ▶ Communication, Nonverbal

References and Readings

Deery, S. J., Iverson, R. D., & Walsh, J. T. (2010). Coping strategies in call centres: Work intensity and the role of co-workers and supervisors. *British Journal of Industrial Relations, 48*, 181–200. doi:10.1111/j.1467-8543.2009.00755.x.

Dur, R., & Sol, J. (2008). *Social interaction, co-worker altruism, and incentives*. Amsterdam: Tinbergen Institute.

Joiner, T. (2007). Total quality management and performance: The role of organization support and co-worker support. *International Journal of Quality and Reliability Management, 24*, 617–627. doi:10.1108/02656710710757808.

Tett, R. P., & Murphy, P. J. (2002). Personality and situations in co-worker preference: Similarity and complementarity in worker compatibility. *Journal of Business Psychology, 17*, 223–243.

C-Reactive Protein (CRP)

Jet Veldhuijzen van Zanten
School of Sport and Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

Synonyms

Acute phase proteins; Inflammatory markers

Definition

C-reactive protein (CRP) is an important protein of the acute-phase response, which is a nonspecific physiological and biochemical response to infection, inflammation, and tissue damage. Increases in CRP are found during infection, chronic inflammatory diseases, and following a myocardial infarction. Strenuous exercise and psychological stress can also induce increases in CRP, albeit to a lesser extent compared to the physiologically more traumatic events described above. Therefore, levels of CRP can be reflective of both acute and chronic inflammation (Gabay & Kushner, 1999).

The CRP molecule consists of five calcium-binding nonglycosylated protomers in a pentameric symmetry. CRP is mainly produced by hepatocytes, even though other sources have also been reported. The production is stimulated by cytokines, which are released under the influence of the macrophages and monocytes at the site of the inflammation. Interleukin (IL)-6 has been shown to be most important for CRP production, but other cytokines, such as IL-1, tumor necrosis factor-alpha, interferon gamma, as well as glucocorticoids, can also play a role. Interestingly, specific combinations of these factors can both enhance as well as inhibit CRP production (Gabay & Kushner, 1999; Pepys & Hirschfield, 2003).

The function of CRP is to restore normal structure and function of the tissue that has been affected. CRP recognizes and mediates the elimination of pathogens through activation of the complement system (Gabay & Kushner, 1999; Pepys & Hirschfield, 2003). Even though the aim of the initial increase in CRP is to combat infection and acute inflammation, chronically raised levels have been associated with negative effects for health. Particular attention has been paid to the association between high levels of CRP and increased risk for atherosclerosis and cardiac events; high levels of CRP have been implicated in the pathogenesis, progression, and complications of atherosclerotic plaques (Ridker, 2004).

CRP can be readily assessed in serum using commercially available (high-sensitivity) assays. As the clearance rate of CRP remains stable, the increases in serologically determined CRP are indicative of CRP production. Following the stimulus, it takes on approximately 6 h until an increase is detectable in the serum. The half-life of CRP is less than 24 h.

Cross-References

- ▶ Biomarkers
- ▶ Cardiovascular Risk Factors
- ▶ Inflammation

References and Readings

Gabay, C., & Kushner, I. (1999). Acute-phase proteins and other systemic responses to inflammation. *The New England Journal of Medicine, 340*, 448–454.

Pepys, M. B., & Hirschfield, G. M. (2003). C-reactive protein: a critical update. *Journal of Clinical Investigation, 111*, 1805–1812.
Ridker, P. M. (2004). High-sensitivity C-reactive protein, inflammation, and cardiovascular risk: From concept to clinical practice to clinical benefit. *American Heart Journal, 148*, S19–S26.

Crohn's Disease (CD)

Kyung-Eun Choi and Jost Langhorst
Kliniken Essen Mitte, Klinik für Naturheilkunde und Integrative Medizin, Universität Duisburg-Essen, Am Deimelsberg 34a, Essen, Germany

Synonyms

English: Regional enteritis
Latin: Enteritis regionalis Crohn; Enterocolitis regionalis; Ileitis terminalis; Morbus Crohn (MC)

Definition

Crohn's disease is a chronic inflammatory disorder which can affect the entire gastrointestinal (GI) tract. It is most commonly located in the terminal ileum and the proximal colon but may involve any part of the GI tract. Its incidence in middle Europe is around 1.5–8.5% and its prevalence around 0,036%. The inflammations generally appear in outlined sections, affect all laminae of the intestinal wall, and cause abscesses and fistulae. Key symptoms are persistent diarrhea, abdominal pain, fever, weight loss, and rectal bleeding. The course of the disease can be described with recurrent relapses and symptom-free intervals, both of which vary in length and strength.

Description

In genetically susceptible individuals impaired and inappropriate immune responses to microbial antigens of commensal microorganisms are discussed for pathogenesis. Data on expression suggest that macrophages and epithelial cells could be the locus of the primary pathophysiological defect and that T-cell activation might be a secondary effect inducing chronification of the inflammation, presumably as a backup mechanism to insufficient innate immunity. Genetic predisposition, ethnicity, smoking behavior, nutrition habits, and enhanced drug intake are discussed as further risk or modulating factors. No causal therapy is currently available for Crohn's disease.

Though not curable, glucocorticoids, aminosalicylates, antibiotics, immunomodulatory substances, enteral (specific diets) or parenteral feeding (under avoidance of the digestive tract), and surgical procedures can alleviate symptoms' severity (Akobeng & Thomas, 2007; Butterworth, Thomas, & Akobeng, 2008). Ninety percent of the patients concerned have to undergo surgery at least once in their lifetime, and 20% of operated patients will undergo further surgery within the next 5 years.

Due to a lack of well-designed randomized controlled trials in the field of behavioral medicine, only insufficient evidence is available so far to make firm conclusions about the efficacy of different psychotherapeutic treatment options for induction of remission in Crohn's disease. Neither any specific personality traits nor any family structures in correlation with the occurrence of Crohn's disease have hitherto been clearly identified. Although a causal relationship with critical life events and/or stress has not been established so far, Crohn's disease creates an immense burden on patients and is a critical strain on patients and relatives. Consequently, psychological changes (including higher values of depression, anxiety, and/or emotional instability) are very often observed. From the point of view of behavioral medicine, a behavioral therapy is thus as reasonable as for any other severe chronic disease for which no cure exists. Besides psychotherapy (behavioral, conflict-oriented, psychodynamic, or supportive), relaxation techniques (progressive muscle relaxation, autogenic training) as well as stress management training can be useful in conjunction with standard medical interventions. Such interventions do often not directly affect the course

of the disease, but patients' mental condition and illness-related quality of life improve.

Generally, a positive course of the disease mainly depends on the patients' compliance. Active coping strategies and a problem solution orientation were identified as best predictors for a shorter duration of inflammations and longer relapse-free episodes. The following coping strategies were mostly beneficial in the management of the disease: "accurately adhere to medical advice," "be trustful in medical practitioners," "seek for further information," "actively cope with problems," and "encourage yourself." The probability of actually receiving an adequate medicinal therapy is enhanced with greater compliance. If the relationship between the physician and patient is disturbed or if the patient has no adequate coping strategies, the additional use of psychological interventions should be recommended with higher priority. Especially with phobic reactions after diarrhea, comorbidity with depression and/or anxiety, or other acute psychological conflicts, a supportive psychological therapy should be initiated. Besides solving acute conflicts, general aims of such interventions should lie in an enhancement of the relaxation ability and strengthening of individual coping strategies.

Cross-References

- ▶ Anxiety
- ▶ Compliance
- ▶ Coping Strategies

References and Readings

Akobeng, A. K., & Thomas, A. G. (2007). Enteral nutrition for maintenance of remission in Crohn's disease. *Cochrane Database of Systematic Reviews*(3). doi:10.1002/14651858.CD005984.pub2. Art. No.: CD005984.

Butterworth, A. D., Thomas, A. G., & Akobeng, A. K. (2008). Probiotics for induction of remission in Crohn's disease. *Cochrane Database of Systematic Reviews*(3). doi:10.1002/14651858.CD006634.pub2. Art. No.: CD006634.

Longo, D. L., Fauci, A. S., Kasper, D. L., Hauser, S. L., Jameson, J. L., & Loscalzo, J. (2011). *Harrison's principles of internal medicine (2v.)* (18th ed.). New York: McGraw Hill Professional.

Crossover Design

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Repeated measures design

Definition

Subjects in a crossover design study are assigned to receive two or more treatments in a particular sequence. Imagine a study in which some subjects receive Treatment A on a given day (the first period) and a week later receive Treatment B (the second period). Others subjects (usually close to an equal number) would receive Treatment B first and then, 1 week later, receive Treatment A. Such a study would be described as having a two-period, two-treatment, two-sequence crossover design. Crossover designs can involve various numbers of treatments, sequences, and periods. In these designs, individual subjects are randomized to treatment sequences (as opposed to treatment groups as occurs in parallel groups study designs).

The primary advantage of the crossover design is that each subject serves as his or her own control, providing data in each treatment arm of the study. The design also has some disadvantages, one of which can be difficulty in interpreting the results. Since all subjects receive more than one treatment there can be a carryover effect from one or more early periods to subsequent periods, leading to a biased estimate of the treatment effect(s) of interest.

Cross-References

- ▶ Bias
- ▶ Parallel Group Design
- ▶ Randomization

Cross-Sectional Study

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

A cross-sectional study describes a group of subjects at one particular point in time (Campbell, Machin, & Walters, 2007).

All study designs and methodologies have advantages and disadvantages. The randomized controlled trial, which is placed at the top of the Hierarchy of Evidence by some researchers and therefore is considered a very strong source of evidence, has some disadvantages: They are lengthy, expensive, and may be limited in how well results from them can be generalized to the treatment's effect in the general population in real-world clinical circumstances.

The cross-sectional study is usually comparatively quick and easy to conduct. Examples of its implementation include the use of an interview survey and conducting a mass screening program. Additional advantages are that many risk factors can be studies at the same time, and that they are suitable for studying rare diseases. Disadvantages include the following:

- Only one disease outcome can be studied at once.
- Temporal relationships can be difficult to identify. Since the survey provides a snapshot of information at one time, it is not possible to address the issue of which item of interest that is currently present may have caused (influenced) another item that is also currently present.
- The selection of control subjects can be problematic.
- From a statistical perspective, only relative risk can be obtained.
- Focusing on the subjects, lack of recall and recall bias can be of concern.
- Data derived from these studies cannot meaningfully be used to test the effectiveness of an intervention, i.e., they are not good for answering research questions. Nonetheless, they may be useful for generating hypotheses (asking questions) that can subsequently be further investigated in randomized controlled trials.

Cross-References

- ▶ Absolute Risk
- ▶ Hierarchy of Evidence
- ▶ Randomized Controlled Trial
- ▶ Relative Risk

References and Readings

Campbell, M. J., Machin, D., & Walters, S. J. (2007). *Medical statistics: A textbook for the health sciences* (4th ed.). Chichester, UK: Wiley.

CT Scan

- ▶ CAT Scan
- ▶ Computerized Axial Tomography (CAT) Scan

Cultural and Ethnic Differences

Sana Loue
Department of Epidemiology & Biostatistics, Case Western Reserve University, School of Medicine, Cleveland, OH, USA

Definition

The recognition of culture and its components as a complex and fluid process, rather than a static construct, is critical to attempts to understand the cultural influences on health and health behavior; culture cannot be reduced to a single variable or construct.

Twelve features are essential to an understanding of a culture: history, social status, points

of interaction within and between social groups, value orientations, verbal and nonverbal language and communication processes, family life processes, healing beliefs and practices, religion and religious practices, art and other forms of expression, dietary preferences and practices, recreational forms, and manner and style of dress (Hogan-Garcia, 2003). The subjective components of culture, such as beliefs, values, and explanatory cognitive frameworks, are communicated both verbally and nonverbally; the objective component of culture consists of rules relating to individual and group behavior (Hogan-Garcia, 2003). Culture is constructed by and exists and operates at the levels of the individual and the group and changes over time (Nagel, 1994). Individuals who ascribe to a particular culture share an identity and a framework for understanding the world.

Too often, culture is erroneously assumed to be synonymous with ethnicity. However, ethnicity and culture represent quite different concepts. Ethnicity is a function of both one's self-identification and the identification by others of membership in a specific group based on specific characteristics, such as biological characteristics, nationality, language, and/or religion (Yinger, 1994); it is a function of both cultural history and psychological identity (Melville, 1988). Like culture, one's ethnic identity may change due to changes in one's self-perception and the social context in which one lives. Additionally, individuals may claim membership in various cultures and/or ethnicities simultaneously, and may prioritize these memberships differently depending on any variety of circumstances. As an example, an individual may simultaneously consider herself Polish, Russian, Christian, nondenominational, female, and American.

The concept of ethnicity has also been used confused in the literature with the concept of race. Like ethnicity, the concept of race has been used to explain perceived differences in appearance and behavior across individuals and groups, based on the erroneous assumption that each race is associated with distinct, fixed physical and behavioral characteristics. However, the definition of race, the classification of individuals by race, and the meaning or significance associated with a particular designation have varied over time, place, and purpose of designation, both in the United States and elsewhere (Loue, 2006). Additionally, shifting perceptions of self-identity and self-worth may also influence how an individual self-designates at any particular time and place. Unfortunately, epidemiological literature has frequently confused ethnicity and culture and race by equating ethnicity with country of origin and/or skin color and culture with ethnicity or race, based upon the assumption that any observed differences seen between groups are the result of true and fixed genetic or cultural differences between the groups (Karlsen, 2004).

Additionally, reference to a particular culture, ethnicity, or race assumes and implies homogeneity within the classification being used. However, within every group, differences exist with respect to socioeconomic status, religion, age, understandings of health and illness, educational and employment opportunities, social status and power, and access to services. It is important to recognize that although classifications based on culture and ethnicity may be useful as a shorthand, they are not unitary constructs. A full understanding of the mechanisms that may underlie health disparities requires a more in-depth understanding that is possible only through the examination of the relevant constitutive elements.

The politics of HIV/AIDS illustrates the confusion that often surrounds culture, ethnicity, and race. The human immunodeficiency virus (HIV), the causative agent of AIDS, is transmitted through the exchange of various bodily fluids, such as semen, vaginal fluid, breast milk, and blood. Approximately 1 year after the identification of the first observed cases of the disease, the Centers for Disease Control and Prevention (CDC) labeled Haitians a "risk group," meaning that anyone who was Haitian was believed to be at increased risk of contracting and transmitting the virus by virtue of their group membership, rather than as a function of their individual behaviors (Schiller, Crystal, & Lewellen, 1994). Here, individuals' ethnicity and race were presumed to be congruent with culture and "culture"

was presumed to be a factor in disease transmission. Ironically, this categorization of all Haitians as a risk group reflects US biomedical culture with respect to its understanding at the time of disease process and the meanings of culture, ethnicity, and race.

Description

Culture influences almost every aspect of illness, including how an illness is identified, defined, and made meaningful; the timing and onset of the illness; the symptomatology; the course and outcome of the illness; how individuals, families, providers, and others respond to an experience of the illness; and how individuals seek, utilize, and respond to treatment (Kleinman, 1988). It is beyond the scope of this entry to review the role of culture in each of these aspects across all diseases. Instead, the role of culture as it relates to disease diagnosis, symptomatology, and treatment is examined in the context of several chronic diseases.

As an example, the prevalence of bipolar disorder appears to vary across cultures. Bipolar disorder is a serious, chronic mental illness characterized by manic and depressive episodes (Type I) or hypomanic episodes and major depressive recurrences (Type II). The disorder is associated with impairments in the quality of life, increased rates of suicide, and high financial costs. However, the prevalence of bipolar disorder varies across countries. The consumption of omega-3 fatty acids found in seafood appears to serve a protective effect for individuals who consume large quantities of seafood over their lives, suggesting that nutritional habits play a role in the development of the disease (Noaghiul & Hibbeln, 2003).

Cultural aspects are also implicated in the symptomatology and management of bipolar disorder. The manic phase of bipolar disorder is characterized by an "excessive involvement in activities," that often assumes the form of sexual indiscretions and buying sprees. However, how this excessive involvement manifests may have to be reformulated so as to be consistent with the cultural context in which the individual lives. Clinicians who are unfamiliar with the client's culture may erroneously interpret the client's behavior as symptomatic of bipolar disorder when it is not, or may erroneously ascribe behavior to cultural influences when the behavior actually indicates the presence of bipolar disorder. Similar diagnostic errors have been noted in the context of schizophrenia. The overdiagnosis of schizophrenia among African Americans has been attributed in part to providers' lack of cultural understanding and their consequent misinterpretation of cultural mistrust as paranoia and miscommunications between the provider and the patient. Too, clinicians unfamiliar with the client's culture may be more likely to prescribe or to refrain from prescribing particular pharmacologic treatments based on misunderstandings of the client's behavior.

Culture also plays a role in the prevalence, experience, and course of epilepsy. Epilepsy is a brain disorder that is characterized by a predisposition to generate seizures, with neurobiological, cognitive, psychological, and social consequences. Research findings indicate that the prevalence of the disorder is higher in developing countries compared to more developed countries (Mac et al., 2007). In some cultures and religious groups, such as some Asian Indian and Muslim communities, consanguineous marriages, that is, between blood relatives such as first cousins, is customary. Parental consanguinity had been found to be associated with an increased risk of certain forms of epilepsy. It is important to recognize, however, that not all Asian Indian and Muslims enter into consanguineous marriages and some individuals who are neither Asian Indian nor Muslim may do so.

Individuals may search for an explanation for their seizures, which are often unpredictable and may be uncontrollable. Explanatory models of epilepsy differ across cultures. Individuals from Western developed nations are more likely to ascribe to a biomedical model of the disease, whereas individuals of other cultural backgrounds may attribute the cause of epilepsy to witchcraft, divine punishment, bad luck, or supernatural forces (Allotey & Reidpath, 2007; Mac et al., 2007). The existence of such vast

differences in beliefs regarding the causation of the illness between a patient and a provider may seriously impede communication and adversely affect their ability to work together to control the seizures (Reynolds, 2000). The beliefs that individuals hold regarding their illness also have implications for their willingness to adhere to prescribed treatment, the extent to which they utilize alternative treatments, and their daily functioning. Individuals who believe that their illness lasts only as long as their seizure lasts may refuse to take medication on an ongoing basis, resulting in an inability to control the seizures. Alternative treatments, such as smoke inhalation, herbal preparations, and dietary treatments, may be sought; some of these may be toxic, leading to additional illness. Daily functioning may be limited, not because of the effects of the epilepsy itself, but because individuals and even their health care providers may believe that individuals with epilepsy must restrict their activities, including the avoidance of sun exposure, strenuous exercise, and the obligations demanded by regular employment (Allotey & Reidpath, 2007; Mac et al., 2007). The belief that epilepsy is a contagious disease, common in many countries, may cause people to avoid touching an individual who is experiencing a seizure, even though some forms of help might reduce the likelihood of injury to the individual experiencing the seizure (Mac et al., 2007).

Type 2 diabetes mellitus, which is increasing in prevalence worldwide, results from an interaction between genetic, environmental, and behavioral factors. Numerous studies have reported differences in the prevalence of type 2 diabetes across various ethnic groups. For example, South Asian migrants have been found to have a higher prevalence of type 2 diabetes compared to Westerners; African Americans have been reported to have a higher prevalence compared to Whites (Hussain, Claussen, Ramachandran, & Williams, 2007). These distinctions, which presume a nonexistent homogeneity within the named groups and heterogeneity across groups, can only serve as a foundation for additional study. One must search further for the underlying explanations for these observed differences.

Obesity and physical inactivity have been implicated as factors in the development of type 2 diabetes (Hussain et al., 2007). Accordingly, cultural factors that encourage or promote overeating and a sedentary lifestyle and/or constrain efforts to eat healthily and exercise more may play a role in the development of the disease and its progression. Diet and exercise must both be managed by individuals within the context of their everyday lives and their interactions with others. Standards of modesty in dress may diminish individuals' opportunities to engage in vigorous exercise, attempts to participate in religious fasting rituals may predispose individuals to hypoglycemia, and the consumption of traditional foods, such as those prepared with butter or that are fried, may thwart attempts at weight reduction. Additionally, the standard for what constitutes an ideal body or weight varies across cultures. In some contexts, obesity may signify privilege and affluence, an announcement to the larger world that the individual is able to afford the more costly "status" foods such as meat, butter, and sweets. The ability to refrain from physical exertion, such as that associated with exercise, may also signal higher social and financial status. Individuals' self-identities may be intimately linked to their adherence to specified behavioral norms; their participation in social, religious, and/or other activities; and their relationships with others. Consequently, clinicians' efforts to persuade their patients to modify behaviors may be perceived by the patient not as a necessary change in lifestyle to prevent disease and improve health, but rather as a potential loss of one's identity, status, and membership in a particular group.

In some instances, individuals' interpretations of their symptoms may impede their receipt of potentially helpful treatments. As an example, the term "ataque de nervios," literally an attack of nerves, is utilized by many Puerto Ricans to refer to their response to a specific traumatic event, such as a death in the family or betrayal by one's spouse. (Ataque de nervios is often referred to in the psychiatric literature as a culture-bound syndrome.) That response may include fainting, dizziness, shortness of breath,

weakness, and/or chest pain. The individual may experience feelings of sadness and depression, nervousness and insecurity, or irritability and anger. The experience of an ataque communicates to others in a culturally and socially acceptable manner one's feeling that the world has gone out of control.

An ataque may occur in the absence of any pathology, but in some circumstances may be indicative of an underlying anxiety, affective, or panic disorder. Individuals suffering from ataques may dismiss out of hand the possibility that such experiences suggest an underlying disorder for which they might seek treatment. However, the converse is also true: Clinicians who are unfamiliar with the cultural meaning of ataques may misinterpret the patient's experiences as indicative of pathology when they represent instead a time-limited, culturally sanctioned response to trauma.

As an example of how cultural *change* can impact the diagnosis, prevalence, and treatment of a disease or disorder, consider how understandings of homosexuality have varied over the last 40 years. Once considered a mental illness, individuals who were diagnosed as homosexual were subjected to therapeutic interventions to transform their sexual orientations. Cultural change both within the medical profession and within the larger society in the United States provided the impetus to declassify homosexuality per se as a mental illness requiring treatment. The prevalence of the "illness," the "course of illness," and the "prognosis" were thus dependent on whether the underlying orientation was to be considered an illness at all. Similarly, cultural *change* at a societal level has transformed our understanding of alcohol abuse from that of a moral defect, to an individual medical problem, to a public health issue; our perception of partner violence as a legitimate response to a partner's failure to fulfill role obligations, to a medical diagnosis of the battered partner as a masochist, to a public health and criminal justice issue. Once treated through prayer, alcohol abuse is now seen as amenable to counseling, pharmacologic treatments, and, under some circumstances, legal intervention. Similarly, remedies for partner violence have broadened to include counseling for both the batterer and the battered. Our perception of disease necessarily impacts our prevalence estimates, how the affected individual interprets his or her experience, the course of the individual's illness, and how we as individuals, clinicians, and a society respond to the individual in the context of that experience.

Implications

Much emphasis has been placed on clinicians' need to develop cultural competence in order to better communicate with and counsel their patients. However, all too often, efforts to inculcate cultural competence reduce culture to a laundry list of characteristics attributed to a particular group, characteristics that are presumed to be true of all members of that named group and to exist across time and place. Such efforts fail to recognize the fluid nature of culture at the individual and group levels, the complexity of culture, the heterogeneity that exists within larger groups, and the similarities that exist across groups.

A focus on the development of cultural humility, rather than cultural competence, is more likely to lead to improved communication between providers and their patients, between researchers and their research participants, and across diverse communities. In contrast to cultural competence, which focuses on substantive issues and may lead to both stereotyping and a false sense of security derived from "knowing," cultural humility focuses on process as a key element, requiring that the individual remain open to continual learning, engage in continual self-reflection and self-critique, and attempt to equalize the power imbalances that are inherent in the provider-patient or researcher-participant relationship (Tervalon & Murray-Garcia, 1998). Improved communication and understanding across cultural differences may ultimately lead to improved health for individuals and communities and a reduction in health disparities.

Cross-References

▶ Acculturation
▶ Chronic Disease Management

- ▶ Chronic Disease or Illness
- ▶ Cultural Competence
- ▶ Cultural Factors
- ▶ Ethnic Identities and Health Care
- ▶ Health Behavior Change
- ▶ Health Behaviors
- ▶ Health Beliefs
- ▶ Health Communication
- ▶ Illness Behavior
- ▶ Norms
- ▶ Self-identity
- ▶ Sociocultural Differences

References and Readings

Allotey, P., & Reidpath, D. (2007). Epilepsy, culture, identity, and well-being: A study of the social, cultural, and environmental context of epilepsy in Cameroon. *Journal of Health Psychology, 12*(3), 431–443.

Fadiman, A. (1997). *The spirit catches you and you fall down*. New York: Farrar, Strauss Giroux.

Hogan-Garcia, M. (2003). *The four skills of cultural diversity competence: A process for understanding and practice*. Pacific Grove, CA: Brooks/Cole.

Hussain, A., Claussen, B., Ramachandran, A., & Williams, R. (2007). Prevention of type 2 diabetes: A review. *Diabetes Research and Clinical Practice, 76*, 317–326.

Karlsen, S. (2004). 'Black like Beckham'? Moving beyond definitions of ethnicity based on skin colour and ancestry. *Ethnicity & Health, 9*(2), 107–137.

Kleinman, A. (1988). *Rethinking psychiatry*. New York: The Free Press.

Loue, S. (2006). *Assessing race, ethnicity, and gender in health*. New York: Springer.

Mac, T. L., Tran, D.-S., Quet, F., Odermatt, P., Preux, P.-M., & Tan, C. T. (2007). Epidemiology, aetiology, and clinical management of epilepsy in Asia: A systematic review. *Lancet Neurology, 6*, 533–543.

Melville, M. B. (1988). Hispanics: Race, class, or ethnicity? *Journal of Ethnic Studies, 16*(1), 67–83.

Nagel, J. (1994). Constructing ethnicity: Creating and recreating ethnic identity and culture. *Social Problems, 41*, 152–176.

Noaghiul, S., & Hibbeln, J. R. (2003). Cross-national comparisons of seafood consumption and rates of bipolar disorder. *American Journal of Psychiatry, 160*, 2222–2227.

Reynolds, E. H. (2000). The ILAE/IBE/WHO global campaign against epilepsy: Bringing epilepsy "out of the shadows". *Epilepsy & Behavior, 1*, S3–S8.

Schiller, N. G., Crystal, S., & Lewellen, D. (1994). Risky business: The cultural construction of AIDS risk groups. *Social Science & Medicine, 38*, 1337–1346.

Tervalon, M., & Murray-Garcia, J. (1998). Cultural humility vs. cultural competence: A critical distinction in defining physician training outcomes in multicultural education. *Journal of Health Care for the Poor and Underserved, 9*(2), 117–125.

Yinger, J. M. (1994). *Ethnicity: Source of strength? Source of conflict?* Albany, NY: State University of New York Press.

Cultural Awareness

- ▶ Cultural Competence

Cultural Competence

Elva Arredondo
Division of Health Promotion and Behavioral Sciences, San Diego State University, San Diego, CA, USA

Synonyms

Cultural awareness; Cultural sensitivity

Definition

Cultural competence is defined as a set of congruent behaviors, attitudes, and policies that come together in a system, agency, or among professionals to facilitate effective work in cross-cultural situations (Cross et al., 1989). Linguistic competence is an important component of cultural competency because language is a key aspect of culture.

"Culture" is defined as an integrated pattern of learned human behaviors (e.g., styles of communication, customs) and beliefs (e.g., views on roles and relationships) shared among groups (Robins, Fantone, Hermann, Alexander, & Zweifler, 1998; Donini-Lenhoff & Hendrick, 2000). The word "competence" implies having the capacity to function effectively with a cultural group (Cross, Bazron, Dennis, & Isaacs, 1989).

Description

A key reason for cultural competence in health services administration and public health is to deliver the highest quality of care to all patients, regardless of race or ethnicity, cultural or religious background, or English proficiency (Betancourt, Green, & Carrillo, 2002). Another important reason for delivering culturally competent care is to reduce and eliminate health disparities in health status of diverse people and to enhance the quality of services and health outcomes. Racial and ethnic minorities are more likely to die from many life-threatening diseases compared to members of the majority group. One likely contributor to the disparities in health outcomes and mortality is biased care stemming from conscious or unconscious racial stereotypes (LaVeist, 2002).

Cross et al. (1989) proposed a Cultural Competence Continuum Framework that ranges from "cultural destructiveness" where health services can create harm to "cultural proficiency" where health care services are responsive to the health beliefs, practices, and cultural and linguistic needs of diverse cultural groups. Descriptions of each of the levels in the continuum follow:

- Cultural destructiveness refers to attitudes, practices, and policies within an organization or system that are harmful to a cultural group. This level represents a lack of understanding and unwillingness to learn about other cultures.
- Cultural incapacity involves the lack of capacity to respond to the needs of a cultural group. These practices may consist of disproportionately allocating resources that may ultimately benefit one group at the expense of another.
- Cultural blindness consists of considering all people or groups the same, without acknowledging cultural nuances. This can lead to forced assimilation to institutional attitudes that may blame members of cultural groups for their circumstances.
- Cultural pre-competence involves a commitment to social and civil justice. In this level, it is recognized that continuous expansion of cultural knowledge and resources to address the needs of cultural groups are needed.
- Cultural competence consists of ensuring that the needs of the cultural group are met by the practitioners and health service organizations. It involves being aware of and recognizing group differences and having insight into one's cultural values. In this level, organizations and public health practitioners are able to operate effectively in different cultural contexts.
- Cultural proficiency is a more advanced standard than cultural competence and incorporates all of the concepts of cultural competence, but a higher level of awareness, knowledge, and skills. Culturally proficient practitioners and organizations strive to be innovative and creative in developing and implementing interventions and evaluation tools.

The Cultural Competence Framework involves five essential elements that help health care organizations and public health practitioners change from not understanding the importance of cultural competence to practicing it. These components include: (1) developing a regard for diversity or demonstrating an awareness and commitment to learning about cultural differences; (2) conducting cultural self-assessment or encouraging organizations to take this process into account; (3) understanding the dynamics inherent when cultures interact; (4) accessing cultural knowledge or demonstrating a commitment to integrating lessons learned into the health care delivery skills; and (5) adapting to diversity or developing strategies that translate cultural competency into system change and clinical practice.

Culturally competent care would involve changing from a "one size fits all" model of care to a model in which care is responsive to different cultural communities. Organizations can aim to achieve cultural competence by assuring diversity among board members, staff, and providers, enhancing data collection, providing effective and translation services, and incorporating cultural competence skill development and education. An organization can identify their level of cultural competence through the use of measures that assess cultural attitudes, practices, structures, and policies of programs. Acquiring these data

can help determine areas of weakness to inform the training needed to strengthen cultural and linguistic competency.

Cross-References

- ▶ Cultural and Ethnic Differences
- ▶ Cultural Factors
- ▶ Diversity

References and Reading

Betancourt, J. R., Green, A. R., & Carrillo, E. J. (2002). *Cultural competence in health care: Emerging frameworks and practical approaches*. New York: The Commonwealth Fund.

Cross, T., Bazron, B., Dennis, K., & Isaacs, M. (1989). *Towards a culturally competent system of care* (Vol. 1). Washington, DC: Georgetown University Child Development Center, CASSP Technical Assistance Center.

Donini-Lenhoff, F. G., & Hendrick, H. L. (2000). Increasing awareness and implementation of cultural competence principles in health professions education. *Journal of Allied Health, 29*(4), 241–245.

LaVeist, T. (2002). *Race, ethnicity and health: A public health reader*. San Francisco: John Wiley & Sons.

Robins, L. S., Fantone, J., Hermann, J., Alexander, G., & Zweifler, A. (1998). Improving cultural awareness and sensitivity training in medical school. *Academic Medicine, 73*(Suppl. 10), S31–S34.

Cultural Consonance

- ▶ Cultural and Ethnic Differences

Cultural Factors

Chanita H. Halbert
School of Medicine, University of Pennsylvania, Philadelphia, PA, USA

Synonyms

Folk health beliefs; Myths

Definition

Culture: Culture is a complex system that includes beliefs and values that are socially transmitted within groups who have similar backgrounds and experiences. Culture is often used to refer to individuals from the same racial and ethnic group, but culture is distinct from one's race or ethnicity. Cultural beliefs and values create motivational force, or provide the underlying rationale or impetus to behave, think, and feel in a certain way. Most empirical research has focused on understanding the association between health behaviors and cultural beliefs and values related to religion and spirituality, temporal orientation, and collectivism and individualism (Kagawa-Singer, Dadia, Yu, & Surbone, 2010).

Religion and spirituality: Spirituality and religion are related but distinct factors that have been shown to influence conceptualizations about diseases. Spirituality is defined as having a personal relationship with a higher power and faith, and may be a process used to find meaning in one's life, while religion is defined as a set of practices and beliefs (e.g., dogma, doctrines) that are shared by a community or group. Religion can be thought of as behavioral manifestations of one's spirituality (Taylor, 2001).

Temporal orientation: Temporal orientation is defined as one's cognitive focus of their behaviors, thoughts, and affect in terms of past, present, or future domains. Individuals may think, feel, or act based on perceived consequences that are immediate (present orientation), will happen in the future (future orientation), or has happened in the past (past orientation) (Nuttin, 1985).

Individualism and collectivism: Individualism and collectivism are beliefs and values related to social processes and interactions. Individualism is characterized by placing greater value on personal autonomy, responsibility, and freedom of choice whereas collectivism is characterized by values that include group responsibility and decision making and maintaining harmonious relationships with others (Triandis, McCusker, & Hui, 1990).

Description

Association Between Cultural Factors, Health Behaviors, and Racial and Ethnic Background

By the year 2050, it is estimated that the racial and ethnic composition of the USA will change dramatically and groups that are currently minorities will make up the majority of the US population. In anticipation of this, and the poorer health outcomes that these groups continue to experience, efforts are focusing on developing more effective strategies for health promotion and disease prevention by addressing cultural beliefs and values related to health behaviors. Cultural factors are now being addressed as part of health behavior interventions based on studies which have shown that these factors are associated with health behaviors. Racial and ethnic group differences in cultural beliefs and values also provide support for addressing these factors as part of health promotion and disease prevention efforts. These findings are summarized in the sections below (Smedley, Stith, Nelson, & Institute of Medicine (U.S.), 2003).

Temporal Orientation. As described above, temporal orientation is defined as one's cognitive focus of their behaviors, thoughts, and affect in terms of past, present, or future domains. Studies have shown that future temporal orientation promotes greater psychological well-being, avoidance of risky health behaviors, and adherence to preventive health behaviors and beliefs, whereas present temporal orientation is associated with reduced adherence. There are also racial differences in temporal orientation. For example, Brown and Segal found that African Americans reported greater levels of present temporal orientation related to hypertension management compared to whites. Individuals with higher levels of present temporal orientation reported lower perceptions of susceptibility to adverse effects of uncontrolled disease, perceived fewer benefits of hypertension medication, and reported greater perceptions of burden from the negative aspects of medication. Similar results were reported in a qualitative study of perceptions of cervical cancer screening in Hispanic women. Women in this study reported that reasons for not obtaining screening as recommended included beliefs that less emphasis is placed on screening to prevent future health outcomes because the future cannot be changed or guaranteed. In a community-based sample of African American women, present time orientation was associated with never having a mammogram, but women who had greater levels of future temporal orientation were most likely to participate in genetic counseling for BRCA1 and BRCA2 mutations and receive test results. Greater levels of future temporal orientation were also associated with uptake of genetic counseling for BRCA1 and BRCA2 mutations in samples that consisted mostly of white women (Brown & Segal, 1996; Boyer, Williams, Callister, & Marshall, 2000).

Religion and Spirituality. Religion and spirituality have been examined extensively as predictors of health behaviors and beliefs. For instance, explanatory models for cancer among African American and Hispanic women include the belief that cancer is due to God's will. Other work has shown that religious and spiritual beliefs influence decisions about seeking treatment for breast cancer symptoms and other health behaviors. Lannin and colleagues found that religious and spiritual beliefs, such as prayer about cancer can lead to healing, were associated with a greater delay in seeking treatment for breast cancer symptoms. African American women were significantly more likely than white women to endorse these beliefs. Similar findings have been reported for Hispanics; faith in God was influential in determining the length of time between symptom recognition and seeking care in Hispanic men and women. Studies have also shown that religion and spirituality are important coping resources following breast cancer diagnosis in African American, Hispanic, and white women; however, the importance of these needs may differ depending on one's racial or ethnic background. For example, while 25% of white cancer patients reported five or more spiritual needs following their cancer diagnosis, significantly more African American (41%) and Hispanic (61%) women reported five or more spiritual needs. African American women were significantly more likely than white women to use

God as a source of support following diagnosis. African American prostate cancer survivors also reported significantly greater levels of religiosity compared to white prostate cancer survivors. African American men have also been shown to be significantly more likely than white men to report that faith contributes to good health and faith in God played a role in health-seeking behaviors among Hispanic men. Other work has shown that while religion is very important to the majority of adults diagnosed with disease and one third of healthy adults pray for health conditions, African American men and women were significantly more likely than white men and women to be willing to allocate time with health care providers to discuss spiritual issues rather than health care concerns (Lannin et al., 1998; Moadel et al., 1999; Kub et al., 2003).

Collectivism and Individualism. Individualism and collectivism may also contribute to health behaviors and beliefs, but less empirical data are available on these associations. But studies have examined the relationship between constructs that are similar to individualism and collectivism. Communalism, for example, is defined as having greater recognition of the interdependence of people, particularly family members and familism is defined as having a stronger identification with and attachment to family members. Prior studies have shown that communalism is associated with collectivism and African Americans and Hispanics have been shown to have greater endorsement of collectivism (e.g., interdependence, group responsibility) and familism compared to whites. Other work has shown that greater levels of social integration and the size of one's social network were associated with adherence to breast and cervical cancer screening among Mexican, Cuban, and Central American women. Thompson and colleagues found that African American women who declined to participate in genetic counseling and testing for inherited breast cancer risk reported significantly greater concerns about the impact of testing on family members compared to women who participated in counseling and testing. Further, in a national sample of African American, white, and Hispanic adults, greater levels of individualism were associated with an increased likelihood of eating the recommended number of servings of fruits (Boykin, Jagers, Ellison, & Albury, 1997; Sabogal, Marin, & Perez-Stable, 1987).

Measurement of Cultural Factors

Although cultural beliefs and values are socially transmitted and shared among individuals with similar racial backgrounds and experiences, these factors are most often measured as an individual difference characteristic using self-report measures. Research is now being conducted to develop instruments that measure cultural beliefs and values within specific situational contexts.

Integration of Cultural Factors into Health Promotion and Disease Prevention

Cultural tailoring is an approach that has been used to promote adherence to a wide range of health behaviors that include cancer screening, HIV risk reduction, and informed decision making about genetic testing for inherited disease risk. The premise of cultural tailoring is that information and messages that are customized to one's culturally based beliefs and values will be more effective than generic approaches because they address issues and ways of thinking and coping that are most salient to an individual. Culturally tailored interventions have had mixed results. For instance, Halbert and colleagues developed and evaluated a culturally tailored genetic counseling (CTGC) protocol for African American women as part of a randomized trial. The CTGC protocol included standardized probes to elicit discussion about cultural factors that have been shown to influence decisions about genetic counseling among African American women (e.g., spiritual and religious beliefs, communalism). For example, women were asked what aspects of their spiritual and religious beliefs influence their decision to have genetic testing to facilitate discussion about the role of these factors in decision making about genetic testing for BRCA1/2 mutations. Women who received CTGC reported greater levels of satisfaction compared to those who received standard genetic counseling (SGC), but there were no differences in uptake of BRCA1/2 test results between

women who were randomized to CTGC and SGC. Further, women randomized to CTGC and SGC did not differ in terms of psychological outcomes such as changes in risk perception and cancer worry compared to decliners. In other research, Kreuter and colleagues found that African American women liked culturally relevant health education materials that addressed fruit and vegetable intake and mammography using four cultural constructs (religiosity, racial pride, collectivism, and time orientation) better than materials that were tailored to behavioral constructs. However, women who received both types of education materials (behavioral and culturally relevant materials) were most likely to obtain a mammogram and had greater increases in fruit and vegetable consumption compared to women who received culturally relevant materials, those tailored to behavioral constructs only, and women in the control condition (Kreuter et al., 2005; Kalichman, Kelly, Hunter, Murphy, & Tyler, 1993; Halbert, Kessler, Troxel, Stopfer, & Domchek, 2010).

These findings raise questions about how to develop interventions that are effective in terms of addressing cultural beliefs and values and promoting health behavior change. One issue may be that previous culturally tailored interventions have been based on conceptualizations of cultural factors that are not specific to different health promotion and prevention behaviors. Studies have shown that different cultural values are elicited depending on situational characteristics and context; but existing instruments that measure cultural factors do not relate specifically to different health behaviors (e.g., medication adherence, avoidance of risk factors, early detection) that define the spectrum of health promotion and disease prevention and, therefore, may be less sensitive for interventions studies that aim to address these factors.

Conclusions

Cultural beliefs and values are important to a wide range of health promotion and disease prevention efforts. Empirical evidence is emerging on how to address these factors as part of health behavior interventions.

Cross-References

- ▶ Cultural and Ethnic Differences
- ▶ Ethnicity
- ▶ Religion/Spirituality

References and Readings

Boyer, L. E., Williams, M., Callister, L. C., & Marshall, E. S. (2000). Hispanic women's perceptions regarding cervical cancer screening. *Journal of Obstetric, Gynecologic, and Neonatal Nursing, 30*, 240–245.

Boykin, A. W., Jagers, R. J., Ellison, C. M., & Albury, A. (1997). Communalism: Conceptualization and measurement of an Afrocultural social orientation. *Journal of Black Studies, 27*, 409–418.

Brown, C. M., & Segal, R. (1996). Ethnic differences in temporal orientation and its implications for hypertension management. *Journal of Health and Social Behavior, 37*, 350–361.

Halbert, C. H., Kessler, L., Troxel, A. B., Stopfer, J. E., & Domchek, S. (2010). Effect of genetic counseling and testing for BRCA1 and BRCA2 mutations in African American women: A randomized trial. *Public Health Genomics, 13*(7–8), 440–448.

Kagawa-Singer, M., Dadia, A. V., Yu, M. C., & Surbone, A. (2010). Cancer, culture, and health disparities: Time to chart a new course? *CA: A Cancer Journal for Clinicians, 60*, 12–39.

Kalichman, S. C., Kelly, J. A., Hunter, T. L., Murphy, D. A., & Tyler, R. (1993). Culturally tailored HIV-AIDS risk-reduction messages targeted to African-American urban women: Impact on risk sensitization and risk reduction. *Journal of Consulting and Clinical Psychology, 61*, 291–295.

Kreuter, M. W., Sugg-Skinner, C., Holt, C. L., Clark, E. M., Haire-Joshu, D., Fu, Q., et al. (2005). Cultural tailoring for mammography and fruit and vegetable intake among low-income African-American women in urban public health centers. *Preventive Medicine, 41*, 53–62.

Kub, J. E., Nolan, M. T., Hughes, M. T., Terry, P. B., Sulmasy, D. P., Astrow, A., et al. (2003). Religious importance and practices of patients with a life threatening illness: Implications for screening protocols. *Applied Nursing Research, 16*, 196–200.

Lannin, D. R., Mathews, H. F., Mitchell, J., Swanson, M. S., Swanson, F. H., & Edwards, M. S. (1998). Influence of socioeconomic and cultural factors on racial differences in late-stage presentation of breast cancer. *Journal of the American Medical Association, 279*, 1801–1807.

Moadel, A., Morgan, C., Fatone, A., Grennan, J., Carter, J., Laruffa, G., et al. (1999). Seeking meaning and hope: Self-reported spiritual and existential needs among an ethnically-diverse cancer patient population. *Psycho-oncology, 8*, 378–385.

Nuttin, J. (1985). *Future time perspective and motivation: theory and research method*. Hillsdale, NJ: Erlbaum.
Sabogal, F., Marin, B. V., & Perez-Stable, J. (1987). Hispanic familism and acculturation: what changes and what doesn't? *Hispanic Journal of Behavioral Sciences, 9*, 397–412.
Smedley, B. D., Stith, A. Y., Nelson, A. R., & Institute of Medicine (U.S.). (2003). Committee on understanding and eliminating racial and ethnic disparities in health care. In *Unequal treatment: Confronting racial and ethnic disparities in health care*. Washington, DC: National Academy Press.
Taylor, E. J. (2001). Spirituality, culture, and cancer care. *Seminars in Oncology Nursing, 17*, 197–205.
Triandis, H. C., McCusker, C., & Hui, C. H. (1990). Multimethod probes of individualism and collectivism. *Journal of Personality and Social Psychology, 59*, 1006–1020.

Cultural Sensitivity

▶ Cultural Competence

Custodian

▶ Family, Caregiver

CVD Prevention

▶ Cardiovascular Disease Prevention

Cynical Distrust

▶ Hostility
▶ Hostility, Cynical
▶ Hostility, Measurement of
▶ Hostility, Psychophysiological Responses

Cynical Hostility

▶ Hostility
▶ Hostility, Cynical
▶ Hostility, Measurement of
▶ Hostility, Psychophysiological Responses

Cynicism

▶ Hostility, Cynical
▶ Hostility, Measurement of
▶ Hostility, Psychophysiological Responses

Cystic Fibrosis

Kristen K. Marciel[1] and Judy A. Marciel[2]
[1]Department of Psychology, University of Miami, Coral Gables, FL, USA
[2]Perioperative Services, East Tennessee Children's Hospital, Knoxville, TN, USA

Synonyms

CF

Definition

Cystic fibrosis (CF), a genetic recessive illness, occurs in 1 of 3,500 live births each year in the United States and currently affects approximately 30,000 people, predominantly Caucasians (Cystic Fibrosis Foundation [CFF], 2008). In 1962, average life expectancy was 10 years of age; however, the current life expectancy of a person with CF is approximately 37 (CFF, 2009). More than 46% of people registered with the CF Foundation are adults. Adults with CF now participate in many developmentally-appropriate aspects of life which were previously rare for this population, including completing high school (92%), working part- or full-time (48%), and marrying or living with a partner (38%; CFF, 2008). As people with CF are living longer, there is a need for effective transition from pediatric to adult health care.

CF is caused by a single genetic defect. There are over 1,500 known mutation variations with the most common defect being the Delta F508 mutation (Farrell et al., 2008). In 1989, this defect

was identified on chromosome 7 (Riordan et al., 1989). When working properly, the cystic fibrosis transmembrane conductance regulator gene produces a protein which transports chloride and sodium across cells, particularly in submucosal glands. In CF, this abnormal electrolyte transport results in the production of thick, sticky mucus, affecting the pulmonary, gastrointestinal, pancreatic, and reproductive systems (Welsh & Smith, 1995). Cycles of infection and inflammation result in significant lung damage. Approximately 90% of patients with CF experience pancreatic insufficiency, resulting in difficulty absorbing fats and proteins which leads to undernutrition. Some patients develop CF-related diabetes, liver damage, and bone disease. About 98% of men are infertile due to the absence, malformation, or blockage of the vas deferens. Women have better reproductive capabilities, though conception is often difficult due to excessive cervical mucus.

Diagnostic criteria for CF include both clinical features and laboratory results (Farrell et al., 2008). Laboratory tests include newborn screening (occurring in all 50 states since 2010), quantification of sweat chloride, and genetic testing. Median age of diagnosis is 6 months of age (CFF, 2008), with approximately 70% of children diagnosed before 1 year of age (Walters & Mehta, 2007). A very vigorous treatment regimen is instituted at the time of diagnosis. Some 90% of morbidity and mortality is due to progressive lung disease. Lung function, as measured by percent predicted of forced expiratory volume in one second, slowly declines over time, at a rate of approximately 1–2% each year (Rosenthal, 2007). Given the multiple systems affected by CF, treatments typically include antibiotics (oral, nebulized, and intravenous), enzyme replacement therapy, airway clearance, nebulized bronchodilators, nebulized mucolytic agents, and aggressive nutritional therapies, ranging from increasing caloric intake to enteral nutritional feedings (Eiser, Zoritch, Hiller, Havermans, & Billig, 1995; Orenstein, 1997). The medical regimen is extremely complex and time consuming, which results in significant challenges for adherence.

The Cystic Fibrosis Foundation (CFF) was established in 1955 (CFF, 2008); this organization accredits the more than 115 care centers, manages a patient registry, develops evidence-based practice guidelines, and provides funding for research. The CFF Therapeutics Pipeline includes 33 interventions at various stages of development, including medications to treat symptoms, potentiators and correctors to address the basic defect, and gene therapy to prevent disease.

Cross-References

▶ Pulmonary Function

References and Readings

Cystic Fibrosis Foundation. (2008). *Patient registry 2006 annual report*. Bethesda, MA: Author.

Cystic Fibrosis Foundation. (2009). *2009 annual report*. Bethesda, MA: Author.

Eiser, C., Zoritch, B., Hiller, J., Havermans, T., & Billig, S. (1995). Routine stresses in caring for a child with cystic fibrosis. *Journal of Psychosomatic Research, 39*(5), 641–646.

Farrell, P. M., Rosenstein, B. J., White, T. B., Accurso, F. J., Castellani, C., Cutting, G. R., Durie, P. R., Legrys, V. A., Massie, J., Parad, R. B., Rock, M. J., Campbell, P. W. (2008). Guidelines for diagnosis of cystic fibrosis in newborns through older adults: Cystic Fibrosis Foundation consensus report. *Journal of Pediatrics, 153*(2), S4–S14.

Orenstein, D. M. (1997). *Cystic fibrosis: A guide for patient and family* (2nd ed.). Philadelphia: Lippincott-Raven.

Riordan, J. R., Rommens, J. M., Kerem, B., Alon, N., Rozmahel, R., Grzelczak, Z., Zielenski, J., Lok, S., Plavsic, N., Chou, J. L., Drumm, M. L., Iannuzzi, M. C., Collins, F. S., & Tsui, L. C. (1989). Identification of the cystic fibrosis gene: Cloning and characterization of complementary DNA. *Science, 245*(4922), 1066–1073.

Rosenthal, M. (2007). Physiological monitoring of older children and adults. In M. Hodson, D. Geddes, & A. Bush (Eds.), *Cystic fibrosis* (3rd ed., pp. 345–352). London: Hodder Arnold.

Walters, S., & Mehta, A. (2007). Epidemiology of cystic fibrosis. In M. Hodson, D. Geddes, & A. Bush (Eds.), *Cystic fibrosis* (3rd ed., pp. 345–352). London: Hodder Arnold.

Welsh, M. J., & Smith, A. E. (1995). Cystic fibrosis. *Scientific American, 273*, 52–59.

Cytokine-Induced Depression

► Sickness Behavior

Cytokines

Briain O. Hartaigh
School of Sport and Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

Synonyms

Chemokines; Interleukins; Lymphokines; Monokines

Definition

1. Low molecular weight proteins that stimulate or inhibit the differentiation, proliferation, or function of immune cells.
2. Small cell-signaling protein molecules that are secreted by numerous cells of the body in order to affect the behavior of other cells that bear receptors for them.
3. Extensively involved in intercellular communication.

Description

Cytokines are small cell-signaling protein molecules weighing approximately 25 kDa. Based on their presumed function, cell of secretion, and target of action, cytokines were previously referred to as lymphokines, interleukins (IL), and chemokines. These terms were generally used in an attempt to develop a standardized nomenclature for molecules that were secreted by and which acted on cells of the body. However, due to an ever-increasing number of cytokines with diverse origins, structures, and effects being discovered, these terms are now considered to be obsolete. Although the IL designation is still used, it is anticipated that a nomenclature based on cytokine structures will eventually become established.

Cytokines and their receptors are categorized according to two major structural families:

- *The hematopoietin family*:
  - Includes growth hormones
  - Includes many IL implicated in both innate and adaptive immunity
- *The TNF family*:
  - Also functional in both innate and adaptive immunity
  - Includes many members that are membrane bound

Both major structural families of cytokines are thought to play an active role in local and systemic effects that contribute toward innate and adaptive immunity.

The effect of a particular cytokine on a given cell depends on the cytokine, its extracellular abundance, the presence and quantity of the complementary receptor on the cell surface, and downstream signals activated by receptor binding (the last two factors may vary by cell type). When a cytokine binds to a receptor, a signal is transmitted into the cell which activates particular genes and, in turn, alters the activity of the cell. Once activated, these cells are capable of producing other cytokines. Each individual cytokine can have several different functions depending on which cell(s) it binds to.

Cytokines are strongly involved in regulating immune function. For example, in lymphocyte activation, both T and B cells critically depend on receiving signals delivered by specific cytokines that bind to receptors on their cell membranes. Likewise, cytokines are influential in promoting or inhibiting local and systemic inflammation.

Considering cytokines are multifunctional, it remains complex to simplify these molecules in order to give a precise account. This is due to:

1. The redundant (sharing the same properties) effect of cytokines
2. Multifunctional properties of cytokines (pleiotropy)

3. Several cells may be capable of producing the same cytokine
4. The ability of cytokines to act synergistically or antagonistically with each other

Therefore, it is easier to imagine that these important protein molecules work in a network to promote or inhibit the interaction of the immune system with other physiological systems, by which they remain mutually dependent on each other.

Cross-References

► Interleukins

References and Readings

Janeway, C. A., Travers, P., Walport, M., & Shlomchik, M. J. (2005). *Immunobiology: The immune system in health and disease* (6th ed.). London: Garland Science.

Roitt, I. M., & Delves, P. J. (2001). *Essential immunology* (10th ed.). Oxford: Blackwell Science.

Staines, N., Brostoff, J., & James, K. (1993). *Introducing immunology* (2nd ed.). London: Mosby.

Cytotoxic T Cell Differentiation Factor

► Interleukins, -1 (IL-1), -6 (IL-6), -18 (IL-18)

D

Daily Diary

► Diaries

Daily Hassles

► Daily Stress

Daily Mood Variation

► Diurnal Mood Variation

Daily Stress

C. Renn Upchurch Sweeney
VA Salt Lake City Healthcare System,
Salt Lake City, UT, USA

Synonyms

Daily hassles; Everyday problems

Definition

Daily stress is defined as mundane hassles, strains, or annoyances associated with routine daily activities and transactions of everyday life. Daily stress is relatively minor, but has the potential to disrupt the flow of everyday life and add to overall levels of stress.

Daily stress can be both anticipated and unanticipated. Anticipated daily stressors include, for example, driving in rush hour traffic on the way home from work, paying bills, working long hours, job performance evaluations, or taking children to after-school activities. Unanticipated stressors may include arguments with spouse, car trouble, getting stuck in long lines at the grocery store, getting sick, losing one's keys, or inconveniences due to weather.

Description

Daily stressors are not inherently stressful events, but they are events that people might appraise as stressful. The experience of feeling stressed depends on what events one notices and how one appraises or interprets these events, which is referred to as the "primary appraisal." Events that are stressful for one person may be routine for another. For example, one may see an upcoming job interview as an exciting opportunity. Others may view it as terrifying. Theoretically, the person then engages in a "secondary appraisal" to determine the adequacy of personal and social resources for dealing with the stressor.

Daily stress is different from major life stressors such as getting married, death of a loved one, or

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

divorce. Unlike life events that call for people to make adjustments to their lives, daily hassles are part of everyday life. Daily stress is more frequent and continuous form of stress than less frequent events that constitute major life stressors. Because of its frequency it may be a more important determinant of stress than major life stressors.

Daily stress and minor hassles have been found to be important forms of stress. Research indicates that routine hassles may have significant harmful effects on mental and physical health (i.e., declines in physical health such as headaches or backaches or worsening of symptoms in those already suffering from illness). Minor hassles can produce stress and aggravate physical and psychological health in several ways. First, the effect of minor stressors can be cumulative. Each hassle may be relatively unimportant in itself, but after a day filled with minor hassles, the effects add up. The cumulative impact of small stressors may wear down an individual until the person eventually feels overwhelmed, drained, grumpy, or stressed out. The aggregate effects of everyday hassles have the potential to compromise well-being or predispose an individual to become ill. Second, daily stress can contribute to the stress produced by major life stressors and influence the relationship between major life events and illness. That is, daily stress can contribute to the stress produced by major life events. If a major life event is experienced at a time when minor life events are also high in number, the stress may be greater than it would otherwise be. Alternatively, major life events, either positive or negative, can also affect distress by increasing the number of daily hassles they create.

References and Readings

Cooper, C. L., & Derre, P. (2007). Stress: A brief history from the 1950s to Richard Lazarus. In A. Monat, R. S. Lazarus, & G. Reevy (Eds.), *The Praeger handbook on stress and coping* (2007th ed., Vol. 1, pp. 7–31). Westport, CT: Greenwood Publishing.

Kohn, P. M. (1996). On coping adaptively with daily hassles. In M. Zeidner & N. S. Endler (Eds.), *Handbook of coping: Theory research, & applications* (pp. 181–201). Oxford, England: Wiley.

Taylor, S. (2006). *Health psychology* (6th ed.). New York: McGraw-Hill.

Weiten, W. (1995). *Themes and variations* (3rd ed.). Pacific Groove, CA: Brooks/Cole.

Dangerous Drinking

▶ Binge Drinking

Data

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Numerical information; Numerical representation of (biological, psychological, behavioral) information

Definition

Data is a plural construct indicating more than one piece of numerical information. The singular form of the term is datum. Statistical analysis (certainly of the type useful in the discipline of behavioral medicine) almost always uses more than one piece of numerical information, and the term datum does not occur again in any other methodology entry in this encyclopedia.

Description

Accordingly, plural words are used in conjunction with the word data: "the data are, the data were, these data, the data show, etc." If you are uncertain as to how to construct a phrase including the word data, replace the word data in your mind with the word results. While the terms data and results are not truly synonymous, the word results is also a plural construct. This strategy will

therefore likely help you express a phrase including the word data correctly.

Data can generally be classified into one of the following scales of measurement: nominal, ordinal, and ratio. Nominal scales involve names of characteristics. Common examples from behavioral medicine include sex (male and female subjects in a research study) and race or ethnicity. An ordinal scale is defined as one in which an ordering of values can be assigned. Age of study subjects categorized as less than 25 years of age, 25–30 years of age, and 31 years of age and older is one example. Data measured on a ratio scale can be manipulated in certain ways not possible with the previous scales. For example, someone weighing 220 pounds (lbs) can be said to weigh twice as much as another subject weighing 110 lbs. The same applies for height and age. The feature of the ratio scale that makes such comparisons possible is that the value of zero on the scale represents a true zero – a weight of zero and a height of zero (no matter what the unit of measurement) means that there is no weight or height, respectively.

You may have noticed what appears to be an initial contradiction in the previous paragraph: Age is discussed in both the ordinal scale and the ratio scale discussions. The reason for this apparent paradox is that data can be measured (recorded) on one scale but reported on another. Imagine that 100 subjects participate in a research study, and each of their ages is recorded in years and months. Then, for various reasons, the subjects are each placed into one of three ordinal categories: those aged less than 25 years of age, 25–30 years of age, and 31 years of age and older. This is perfectly acceptable, but any statistical analysis performed would have to take into account the scale on which the data are reported: Different analyses are appropriate for different kinds of data.

It is also of interest to note that, while it may be convenient to report the subjects' ages in this ordinal fashion, a certain degree of precision in the information is lost. For example, two subjects aged 26 and 29 years, respectively, would both be placed in the middle category. Therefore, although they provide different raw data (their age in years and months) they contribute equally to the total number of subjects in that category.

Cross-References

- ▶ Efficacy
- ▶ Sample Size Estimation

Database Development and Management

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

The goal of experimental methodology and operational execution in behavioral medicine research, like all research, is to provide optimum quality data for subsequent statistical analysis and interpretation. These data need to be stored and managed. Databases facilitate such storage and management. Data management is therefore an important intermediary between data acquisition and data analysis.

Description

Analysis of data collected in behavioral medicine clinical trials is typically conducted using files of data contained in a database. It is of critical importance that the data collected from all sources are accurately captured in the database. A brief list of such data includes: subject identifiers (rather than their names); age, sex, height, and weight; questionnaire data concerning a multitude of topics; and physiological measurements made before, during, and possibly after the treatment period(s).

A data management plan for a clinical trial is written along with the study protocol and possibly a statistical analysis plan before the study

commences (statistical details can also be included in the study protocol). The data management plan identifies the documentation that will be produced as a result of all of the data collected during the conduct of the trial. This plan covers items such as:

- The form(s) on which raw (source) data will be recorded.
- Entering data.
- Cleaning the data.
- Creating data reports.
- Transferring data.
- Quality assurance processes.

The quality assurance (QA) component is vital. While differing definitions of quality activities can be found, Prokscha (2007) defined quality assurance (QA) as a process involving the prevention, detection, and correction of errors or problems, and quality control (QC) as a check of the process. The data stored in the database need to be complete and accurate. Processes that check data and correct them where necessary (i.e., make a change to the database) need to be formalized, and all corrections documented in an audit trail such that a later audit can reveal exactly how the final database was created. That is, following initial data entry, the audit trail will record "who, what, when, why" information for all changes subsequently made.

Having collected optimal quality data, first-rate data management is also critical. Many data that are collected can now be fed directly from the measuring instrument to computer databases, thereby avoiding the potential of human data entry error. However, this is not universally true. Therefore, careful strategies have been developed to scrutinize data as they are entered. The double-entry method requires that each data set is entered twice (usually by different operators) and that these entries are compared by a computer for any discrepancies. This method operates on the premise that two identical errors are probabilistically very unlikely, and that every time the two entries match the data are correct. In contrast, dissimilar entries are identified, the source data located, and the correct data point entry confirmed.

To facilitate the eventual statistical analysis of the enormous amount of data acquired during a clinical trial, recording and maintaining them is extremely important. Database development, implementation, and maintenance therefore require attention. The goals of a database are to store data in a manner that facilitates prompt retrieval while not diminishing their security or integrity.

There are several types of database models. Clinical research typically utilizes one of two types, the flat file database or the relational database. Each has its advantages and disadvantages, and these will be considered by data managers before they decide which type to employ. The flat file database model is simple but restrictive, and it becomes less easy to use as the amount of data stored increases. This model can also lead to data redundancy (the same information, e.g., a subject's birth date, being entered multiple times) and consequently to potential errors. This model works well for relatively small databases.

Relational databases are more flexible, but they can be complex, and careful initial work is needed. This work involves initial logical modeling of the database. The defining feature of a relational database is that data are stored in tables, and these tables can be related to each other. This reduces data redundancy. Subject identifiers in one table, for example, can be related to their heights in another table, their baseline blood pressure in another table, and so on, thereby eliminating the need to store identifiers with each individual set of measurements. Since these databases can contain huge amounts of tables, use of one of several commercially available relational database management systems is typical.

Cross-References

► Study Protocol

References and Readings

Prokscha, S. (2007). *Practical guide to clinical data management* (2nd ed.). Boca Raton, FL: Taylor & Francis.

Dean Ornish

▶ Ornish Program and Dean Ornish
▶ Preventive Medicine Research Institute (Ornish)

Death

▶ Mortality

Death Anxiety

Chad Barrett
Department of Psychology, University of Colorado Denver, Denver, CO, USA

Synonyms

Fear of Death; Thanatophobia

Definition

Death anxiety refers the fear of and anxiety related to the anticipation, and awareness, of dying, death, and nonexistence. It typically includes emotional, cognitive, and motivational components that vary according to a person's stage of development and sociocultural life experiences (Lehto & Stein, 2009). Death anxiety is associated with fundamental brain structures that regulate fight-or-flight responses and record emotionally charged explicit and implicit memories (Panksepp, 2004). Cognitive dimensions of death anxiety can include an awareness of the salience of death and a variety of beliefs, attitudes, images, and thoughts concerning death, dying, and what happens after death (Lehto & Stein, 2009). Death anxiety can be experienced consciously or unconsciously; it can motivate individuals to ameliorate their death anxiety through distraction (Greenberg, Pyszczynski, Solomon, Simon, & Breus, 1994), attempts to enhance self-esteem (Bassett, 2007), or by pursuing positive life changes (Tedeschi & Calhoun, 2004). Individuals experiences of death anxiety can be influenced by their developmental stage. Young adults are mostly concerned about dying too soon, and adult parents are mostly concerned about the effect of their possible death on other family members. Elderly adults are often more concerned with becoming a burden on others, dying alone, or dying among strangers (Kastenbaum, 2000). Sociocultural influences can also shape the cognitive, experiential, and emotional components of death anxiety (Kübler-Ross, 2002; Lehto & Stein, 2009).

Description

Most people report some fear of death, but only a few people report high levels of death anxiety (Kastenbaum, 2000). According to Noyes et al. (2000), only 3.8% of respondents indicated that they were much more nervous than most people about death or dying, and 9.8% indicated they were somewhat more nervous than most people. Stressful experiences can often increase a person's level of death anxiety, (e.g., life-threatening encounters, tragedies, disasters, health problems, illness, or death of a friend or family member, etc.) (Kastenbaum). A meta-analysis of research on death attitudes among older adults indicated that health problems were associated with elevated levels of death anxiety (Fortner & Neimeyer, 1999). In a later review of the literature on death attitudes, Neimeyer, Wittkowski, and Moser (2004) noted that the relationship between death anxiety and health problems is sometimes equivocal. While many studies found positive associations between health problems and death anxiety, others found no significant relationship. Neimeyer et al. discussed more sophisticated studies and that such conflicting findings may be the result of moderator variables such as social support, coping styles, and religious beliefs. Increased social support, approach- and acceptance-based coping strategies, intrinsic religiosity, and beliefs in a positive afterlife are typically associated with less death anxiety.

Cross-References

- ▶ End-of-Life Care
- ▶ Palliative Care

References and Readings

Bassett, J. A. (2007). Psychological defenses against death anxiety: Integrating terror management theory and Firestone's separation theory. *Death Studies, 31*, 727–750.

Fortner, B. V., & Neimeyer, R. A. (1999). Death anxiety in older adults: A quantitative review. *Death Studies, 23*, 387–411.

Greenberg, J., Pyszczynski, T., Solomon, S., Simon, L., & Breus, M. (1994). Role of consciousness and accessibility of death-related thoughts in mortality salience effects. *Journal of Personality and Social Psychology, 67*, 627–637.

Kastenbaum, R. (2000). *The psychology of death* (3rd ed.). New York: Springer.

Kübler-Ross, E. (2002). *On death and dying: Questions and answers on death and dying; on life after death*. New York: Quality Paper Book Club.

Lehto, R. H., & Stein, K. F. (2009). Death anxiety: An analysis of evolving concept. *Research and Theory for Nursing Practice: An International Journal, 23*, 23–41.

Neimeyer, R. A., Wittkowski, J., & Moser, R. P. (2004). Psychological research on death attitudes: An overview and evaluation. *Death Studies, 28*, 309–340.

Noyes, R., Jr., Hartz, A. J., Doebbeling, C. C., Malis, R. W., Happel, R. L., Werner, L. A., et al. (2000). Illness fears in the general population. *Psychosomatic Medicine, 62*, 318–325.

Panksepp, J. (2004). *The foundations of human and animal emotions*. New York: Oxford University Press.

Tedeschi, R. G., & Calhoun, L. G. (2004). Posttraumatic growth: Conceptual foundations and empirical evidence. *Psychological Inquiry, 15*, 1–18.

Death Rate

- ▶ Mortality Rates

Death, Assisted

- ▶ Euthanasia

Death, Sudden

Ana Victoria Soto[1] and William Whang[2]
[1]Medicine – Residency Program, Columbia University Medical Center, New York, NY, USA
[2]Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Synonyms

Sudden cardiac death

Definition

Death within 1 h of the onset of acute symptoms.

Description

Sudden cardiac death (SCD) is an important public health problem, with an annual incidence estimated between 180,000 and 250,000 cases in the United States. The working definition of SCD is death within 1 h of the onset of symptoms, in the absence of preceding evidence of severe pump failure. In prior decades, the majority of SCD cases have been estimated to occur due to rapid cardiac arrhythmia, specifically ventricular tachycardia (VT) and ventricular fibrillation (VF). More recent data indicate that VT/VF is the presenting rhythm in SCD about 30–40% of the time. SCD may also occur due to life-threatening slow heart rhythms (bradycardia) or due to other causes such as massive pulmonary embolism or intracranial hemorrhage (Hinkle & Thaler, 1982; Lloyd-Jones et al., 2010).

In prospective cohort studies, women have a lower incidence of sudden death than men. Coronary artery disease (CAD) is the most common finding in SCD and is discovered in as many as 80% of SCD cases. However, among the large population of patients with coronary artery disease, the absolute risk of SCD is still low. In addition, a substantial proportion of SCDs occur in the absence of known prior heart disease.

Clinical risk factors for SCD have been developed, and the most reliable of which is reduced left ventricular ejection fraction by cardiac imaging such as echocardiogram. However, the prevailing clinical indicators of risk are still limited in their specificity, and identification of individuals at high risk remains a major challenge (Chugh et al., 2008).

Two major mechanisms have been implicated in SCD in the setting of CAD. First, acute plaque rupture may lead to coronary artery occlusion, inadequate blood flow to cardiac muscle (ischemia), and subsequent VT and VF. Another potential mechanism related to CAD results from the presence of myocardial scar from a prior myocardial infarction. With this myocardial substrate, heterogeneity in depolarization and conduction can allow for the development of reentry, in which a tachycardia circuit develops and which manifests as VT that can eventually degenerate to VF. Other underlying cardiac abnormalities can also predispose to SCD. For instance, cardiomyopathies due to causes other than CAD (e.g., alcohol, long-standing hypertension, sarcoidosis) are also associated with SCD. In addition, primary electrical abnormalities, such as inherited ion channel disorders, are relatively rare but potent causes of sustained ventricular arrhythmia in the absence of structural heart disease (Virmani, Burke, & Farb, 2001).

The major treatment against SCD is a preventive therapy, the implantable cardioverter-defibrillator. Randomized controlled trials of primary prevention ICD therapy have demonstrated survival benefit in patients with left ventricular ejection fraction <0.36 and with symptoms of congestive heart failure (Bardy et al., 2005; Moss et al., 2002).

A number of studies have noted an association between psychosocial factors, in particular depression, and SCD. For instance, Empana et al. examined data from enrollees of a health maintenance organization in Washington state, in a case control study involving 2,228 out-of-hospital cardiac arrests. Cases of out-of-hospital cardiac arrest ($n = 2,228$) among patients aged 40–79 years were identified from emergency medical service incident reports, and their ambulatory medical records were examined for the existence of depression. Compared with nondepressed subjects, the adjusted odds ratio of cardiac arrest was increased in less severely depressed subjects (OR 1.30, 95% CI 1.04–1.63) and further increased in severely depressed (OR 1.77, 95% CI 1.28–2.45) (Empana et al., 2006). In a cohort analysis involving 915 individuals aged 70 years or older in northern Finland, Luukinen et al. found that depression was associated with increased risk of sudden death (HR 2.74, 95% CI 1.37–5.50), whereas the risk of non-sudden death was not significantly increased. (Luukinen, Laippala, & Huikuri, 2003) In the Nurses' Health Study of 63,000 female nurses without known cardiovascular disease at study outset, cohort analyses indicated a significant association between depression and SCD in multivariable models that included hypertension, diabetes, and hypercholesterolemia (HR 2.33, 95% CI 1.47–3.70). The relationship of depression at study outset to subsequent SCD appeared to be related to a specific association with antidepressant use (Whang et al., 2009). A separate cohort analysis of the Nurses' Health Study included 72,359 women with no history of cardiovascular disease or cancer in 1988 and used the Crown-Crisp Index to assess phobic anxiety. During 12 years of follow-up, women who scored 4 or greater on the CCI were at higher risk of SCD (HR 1.59, 95% CI 0.97–2.60). After adjustment for possible intermediaries (hypertension, diabetes, and elevated cholesterol), a trend toward increased risk persisted for SCD ($P = 0.06$) (Albert, Chae, Rexrode, Manson, & Kawachi, 2005).

Cross-References

▶ Sudden Cardiac Death

References and Readings

Albert, C. M., Chae, C. U., Rexrode, K. M., Manson, J. E., & Kawachi, I. (2005). Phobic anxiety and risk of coronary heart disease and sudden cardiac death among women. *Circulation, 111*(4), 480–487.

Bardy, G. H., Lee, K. L., Mark, D. B., Poole, J. E., Packer, D. L., Boineau, R., et al. (2005). Amiodarone or an implantable cardioverter-defibrillator for congestive heart failure. *The New England Journal of Medicine, 352*(3), 225–237.
Chugh, S. S., Reinier, K., Teodorescu, C., Evanado, A., Kehr, E., Al Samara, M., et al. (2008). Epidemiology of sudden cardiac death: Clinical and research implications. *Progress in Cardiovascular Diseases, 51*(3), 213–228.
Empana, J. P., Jouven, X., Lemaitre, R. N., Sotoodehnia, N., Rea, T., Raghunathan, T. E., et al. (2006). Clinical depression and risk of out-of-hospital cardiac arrest. *Archives of Internal Medicine, 166*(2), 195–200.
Hinkle, L. E., Jr., & Thaler, J. T. (1982). Clinical classification of cardiac deaths. *Circulation, 65*, 457–464.
Lloyd-Jones, D., Adams, R. J., Brown, T. M., Carnethon, M., Dai, S., De Simone, G., et al. (2010). Executive summary: Heart disease and stroke statistics–2010 update: A report from the American Heart Association. *Circulation, 121*(7), 948–954.
Luukinen, H., Laippala, P., & Huikuri, H. V. (2003). Depressive symptoms and the risk of sudden cardiac death among the elderly. *European Heart Journal, 24*(22), 2021–2026.
Moss, A. J., Zareba, W., Hall, W. J., Klein, H., Wilber, D. J., Cannom, D. S., et al. (2002). Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. *The New England Journal of Medicine, 346*(12), 877–883.
Virmani, R., Burke, A. P., & Farb, A. (2001). Sudden cardiac death. *Cardiovascular Pathology, 10*, 211–218.
Whang, W., Kubzansky, L. D., Kawachi, I., Rexrode, K. M., Kroenke, C. H., Glynn, R. J., et al. (2009). Depression and risk of sudden cardiac death and coronary heart disease in women: Results from the Nurses' Health Study. *Journal of the American College of Cardiology, 53*(11), 950–958.

Decision Analysis

► Clinical Decision-Making

Decision Authority

► Job Demand/Control/Strain

Decision Latitude

► Job Demand/Control/Strain

Decision Making

► Clinical Decision-Making

Deep Sleep

► Slow-Wave Sleep

Defense Mechanism

► Denial

Defensive Coping

► Defensiveness

Defensive Denial

► Defensiveness

Defensiveness

Carolyn Korbel[1] and Sonia Matwin[2]
[1]The Neurobehavioral Clinic and Counseling Center, Lake Forest, CA, USA
[2]Department of Psychiatry, Harvard Medical School, Boston, MA, USA

Synonyms

Avoidant coping; Defensive coping; Defensive denial; Repression; Repressive coping

Definition

Defensiveness is defined as a coping strategy that is characterized by a general orientation away from threatening self-relevant information and a denial or minimization of negative affects such as distress, anxiety, or anger.

Description

Defensiveness is characterized by a general orientation away from threatening self-relevant information and a denial or minimization of negative affects such as distress, anxiety, or anger (Weinberger, Schwartz, & Davidson, 1979). Self-relevant information that is perceived as being inconsistent with personal goals and beliefs is likely to trigger defensive coping reactions (Croyle, Sun, & Hart, 1997). Defensiveness appears to occur normatively in response to self-relevant health risk information, but also to vary across individuals as a more enduring orientation to coping with distress. There is much conceptual overlap between defensiveness, repressive coping, avoidant coping, and denial in the literature, in that each share a core coping process of minimizing, denying, or repressing distress, negative affect, or distressing information to serve emotion regulation goals (Myers, 2010).

Defensiveness has been most frequently assessed through the use of measures of self-reported defensiveness (Weinberger et al., 1979), such as the Marlowe-Crowne Social Desirability Scale (MCSD; Crowne & Marlowe, 1960). Those who score high on social desirability are thought to minimize, deny, or repress negative emotions such as anxiety and anger, reflecting a defensive or self-deceptive orientation to the self that involves avoidance of distress-arousing thoughts. Measures of self-reported trait anxiety are also frequently used in conjunction with the MCSD to identify those who minimize or deny negative affects and who score high on defensiveness to capture a true defensive or, interchangeably, repressive coping group (Myers, 2010; Weinberger et al., 1979). Those who have a defensive or repressive coping style are less likely to report negative affect, distress, somatic symptoms, and chronic stress across a variety of tasks, experimental conditions, and self-report measures. Although defensive/repressive copers deny distress in response to stressful experimental conditions, physiological indicators of distress are often observed.

Defensiveness occurs rather frequently in the population. It has been estimated that 10–20% of the general population, 30–50% of those with particular chronic illnesses, and up to 50% of the elderly use defensive or repressive coping strategies (see Myers, 2010 for a review). In the context of behavioral medicine, defensiveness appears to prompt cognitive, behavioral, and physiological variations, which may have important implications for health. Specifically, the current literature suggests that:

1. Defensiveness is associated with information processing variations that occur normatively in response to self-relevant health threat information.
2. Defensiveness may have direct effects on physiological functioning.
3. Defensiveness is associated with greater morbidity and mortality in a number of chronic illnesses and disease states.

Defensive Cognitive Processing

Defensiveness influences the way that information is processed when threatening self-relevant information is perceived. Defensive cognitive processing variations appear to occur normatively in response to perceiving personally threatening health risk information. Defensive denial processes tend to appear early in the health-threat appraisal process and tend to diminish over time. They are less extreme when individuals are aware of direct actions to eliminate the threat, and are less common when positive states and experiences (e.g., positive mood, optimism, self-affirmation) are bolstered prior to threat perception, or when active coping alternatives are available and reasonable to execute. Defensive cognitive processing variations such as (a) minimization of the seriousness of health threats, (b) self-serving prevalence estimates,

(c) tendencies to denigrate the accuracy or validity of an undesirable test result, and (d) biased processing of health risk information occur frequently in response to perceived health-threat information. These normative defensive processes may play an important role in regulating emotional distress in the short term so that rational health-protective actions can be identified, enacted, and maintained (Croyle et al., 1997; Wiebe & Korbel, 2003).

Physiological Effects of Defensiveness

An emerging literature has identified links between a generalized defensive or repressive coping style and physiological variations in responding which may have direct impacts on health (see Myers, 2010 for a review). It has been hypothesized that the effort required to repress, minimize, or deny negative thoughts and emotions characteristic of defensive coping may result in heightened autonomic reactivity and may impact cardiovascular arousal. Defensiveness has been associated with increased cardiovascular reactivity to stress via increased sympathetic demand when defensive processes are initiated and maintained. Homeostatic changes in baseline cardiovascular functioning are thought to occur over time in response to increased sympathetic reactivity. Cardiovascular disease risk may be increased in defensives through a physiological mechanism of increased stress reactivity, possibly triggering changes in vascular functions or structure that may alter resting blood pressure levels.

Increased Prevalence of Morbidity and Mortality among Defensive Copers

There is an extensive body of literature that links trait-like defensive and repressive coping with poor physical health (see Myers et al., 2007 for a comprehensive review). Repressive/defensive coping appears to both contribute to poor health and disease progression, and to also be used more frequently among those with chronic illnesses. There is a fairly extensive literature linking repressive coping with increased risk for mortality in coronary heart disease (CHD) and myocardial infarction (MI). Repressive coping is associated with a twofold increased risk of death, MI, and other cardiac events. In addition, heightened levels of defensiveness are associated with hypertension, high blood pressure, as well as high lipid and glucose levels. For example, high scores on the MCSD have been associated with elevated blood pressure and heart rate reactivity. Additional support for the association between defensiveness and elevated blood pressure in the general population was found in Jorgensen, Johnson, Kolodziej, and Schreer (1996) meta-analysis. Further, a meta-analysis by Mund and Mitte (2011) suggested that repressive copers are at greater risk of developing cancer and coronary heart disease.

Cross-References

- Cancer Risk Perceptions
- Cognitive Distortions
- Coping
- Defensive Coping
- Denial
- Health Behaviors
- Repressive Coping

References and Readings

Crowne, D. P., & Marlowe, D. (1960). A new scale of social desirability independent of psychopathology. *Journal of Consulting Psychology, 24*(4), 349–354.

Croyle, R. T., Sun, Y., & Hart, M. (1997). Processing risk factor information: Defensive biases in health-related judgments and memory. In K. Petrie & J. Weinman (Eds.), *Perceptions of health and illness: Current research and applications* (pp. 267–290). London: Harwood Academic.

Jorgensen, R. S., Johnson, B. T., Kolodziej, M. E., & Schreer, G. E. (1996). Elevated blood pressure and personality: A meta-analytic review. *Psychological Bulletin, 120*(2), 293–320.

Mund, M., & Mitte, K. (2011). The costs of repression: A meta-analysis on the relation between repressive coping and somatic diseases. *Health Psychology*. doi:10.1037/a0026257. Nov 14, 2011 (No pagination specified).

Myers, L. (2010). The importance of the repressive coping style: Findings from 30 years of research. *Anxiety, Stress, and Coping, 23*(1), 3–17.

Myers, L., Burns, J. W., Derakshan, N., Elfant, E., Eysenck, M. W., & Phipps, S. (2007). Current issues in repressive coping and health. In J. Denollet, I. Nyklicek, & A. Vingerhoets (Eds.), *Emotion regulation: Conceptual and clinical issues* (pp. 69–86). New York: Springer.

Weinberger, D. A., Schwartz, G. E., & Davidson, R. J. (1979). Low-anxious, high-anxious and repressive coping styles: Psychometric patterns and behavioral responses to stress. *Journal of Abnormal Psychology, 88*, 369–380.

Wiebe, D. J., & Korbel, C. (2003). Defensive denial, affect, and the self-regulation of health threats. In L. Cameron & H. Leventhal (Eds.), *The self-regulation of health and illness behavior* (pp. 184–203). New York: Harwood Academic.

Degenerative Diseases: Disc or Spine

Daniel Gorrin
Department of Physical Therapy, University of Delaware, Newark, DE, USA

Definition

The intervertebral disc is a structure located between adjacent vertebral bodies that functions primarily as a shock absorber. The disc is comprised of a fibrocartilaginous outer layer called the annulus fibrosus and a gelatinous inner layer called the nucleus pulposus (made up of collagen fibrils embedded within a water/mucopolysaccharide mix). The disc is connected to the cartilaginous end plates located on the cranial and caudal aspects of the vertebral bodies. The end plates assist in providing the disc with nutrients.

Degenerative disc disease is a potential cause of back pain marked by an atraumatic, gradual onset of symptoms. Due to its primary function as a shock absorber, the disc is subject to significant "wear and tear" during the course of a lifetime. As the patient increases in age, the disc may undergo a degenerative process in which water is lost from the nucleus pulposus and replaced with fibrocartilage. Systemic, cellular, and biochemical changes related to aging may also contribute to degeneration of the disc. Pain resulting from degenerative disc disease is thought to be caused by a combination of irritation of the disc's nociceptive fibers and inflammatory products found within the damaged disc.

The degenerative process can also affect the outer layer of the disc, the annulus fibrosis, which could increase the risk of a herniation of the nucleus pulposus. Tears in the annulus or degeneration of the annulus can limit the structure's ability to contain the gel-like nucleus. Release of the nucleus can cause impingement and irritation of the surrounding spinal nerve roots or even the spinal cord itself. This condition (commonly referred to as a "bulging" or "herniated" disc) can result in localized pain at the site of the herniation or pain in the areas supplied by the nerve (radiculopathy).

Patients who experience degenerative disc disease or a disc herniation are likely to regain full function with non-operative treatment. Physical therapy interventions including mobilization, manipulation, traction, core stabilization exercises, electrical stimulation, and biofeedback are used in treatment of these conditions. Appropriate pharmacological intervention, nerve root injections, and epidural injections can also help provide pain relief. Operative treatment may be indicated if the patient presents with severe neurological deficits or receives no benefit from conservative treatment.

References and Readings

Boyling, J. D., & Palastanga, N. (1994). *Grieve's modern manual therapy* (2nd ed.). New York: Churchill Livingstone.

Drake, R. L., Wayne Vogl, A., & Mitchell, A. W. M. (2010). *Gray's anatomy for students* (2nd ed.). Philadelphia: Churchill Livingstone Elsevier.

Magee, D. J., Zachazewski, J. E., & Quillen, W. S. (2009). *Pathology and intervention in musculoskeletal rehabilitation* (1st ed.). St. Louis, MO: Saunders Elsevier.

McGill, S. (2002). *Low back disorders: Evidence based prevention and rehabilitation* (1st ed.). Champaign, IL: Human Kinetics.

Yue, J. J., Guyer, R. D., Johnson, J. P., Khoo, L. T., & Hochschuler, S. H. (2011). *The comprehensive treatment of the aging spine: Minimally invasive and advanced techniques* (1st ed.). Philadelphia: Elsevier Saunders.

Degenerative Diseases: Joint

Beth Schroeder
University of Delaware, Newark, DE, USA

Synonyms

Chronic inflammatory polyarthritis; RA

Definition

Rheumatoid arthritis (RA) is a chronic inflammatory condition that affects the synovium of joints in the body. While the exact cause of RA is unknown, it is considered to be an autoimmune condition, in which the body attacks its own healthy cells. In a normal, healthy joint, the synovial membrane provides synovial fluid that functions to reduce friction and lubricate the joint surfaces and to provide nutrients to the cartilage. With RA, the synovial membrane becomes inflamed and thickens, forming a pannus. A pannus is an accumulation of tissue that ultimately causes damage to structures in the joint capsule, such as the cartilage, subchondral bone, and even ligaments.

RA presents with periods of both exacerbation and remission. The typical signs and symptoms of RA include pain, morning stiffness, swelling, and decreased mobility of the affected joints. Normally, multiple joints are affected in a symmetric pattern, with both sides of the body being affected. The small joints of the hands and feet are the most common joints to be affected, as well as the cervical spine. As the condition progresses, laxity in the ligaments supporting the joint may develop, causing joint deformities or dislocations.

RA's symptoms are not only musculoskeletal, but the condition can also affect the cardiovascular, renal, and pulmonary systems that may also present in this condition, including fatigue, loss of appetite, and decreased endurance. In some patients, firm skin nodules may form in areas such as the elbows or fingers. It usually presents between the ages of 20–50 years old, and females are more affected.

Although there is no cure for RA, there are pharmacotherapy treatment options that target the pain and inflammation with hopes of slowing the progressive of the disease. The use of medications includes analgesics, nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids, disease-modifying antirheumatic drugs (DMARDS), and biologic response modifiers (BMRs). Physical therapy is also a critical component in the management of RA. During periods of an active flare, joint protection and rest are the most important. Exercise, though, should still be a component of treatment, organized into short bouts with frequent rest periods to limit fatigue. In order to prevent deformations and maintain adequate range of motion, it is very important that patients consistently change positions of the affected joints. If swelling is not present, it is also recommended that light stretching be done.

Because the manifestations of RA are irreversible, management and treatment of RA is a lifelong process. It is essential for patients to work closely with their health care providers in order to establish an individualized treatment plan in order to maintain as much functional independence as possible.

References and Readings

A.D.A.M. Medical Encyclopedia [Internet]. *Rheumatoid arthritis*. Atlanta, GA: A.D.A.M.; ©2010. [Last reviewed February 07, 2010; cited April 18, 2011]; [about 6 p.]. Retrieved from http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0001467/

Goodman, C. C., & Fuller, K. S. (2009). *Pathology: Implications for the physical therapist* (3rd ed.). St. Louis, MO: Saunders Elsevier.

Kisner, C., & Colby, L. A. (2002). *Therapeutic exercise*. Philadelphia: F.A. Davis Company.

Degenerative Parkinsonism

▶ Parkinson's Disease: Psychosocial Aspects

Degrees of Freedom

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

Degrees of freedom may be defined as the number of squares of deviations from the mean minus the number of independent linear restrictions placed upon the quantities involves. For n numbers there are n squares of deviations from the mean, of which only $(n - 1)$ are independent. That is, when $(n - 1)$ are specified, the nth is also specified.

Description

This is the most esoteric definition in the Methodology category of entries, and please do not be concerned if it sounds a little hard to digest. The following scenario will make it much clearer.

Suppose you are asked a question: "Choose any five numbers that add up to 100." How much choice do you actually have? A few moments' thought will reveal that you only have four choices. You can choose any four numbers you wish, but having done so, you have no choice about the fifth. Whatever the sum of the four numbers you have chosen, only one number will take you from there to 100, and hence you have no choice.

Now consider this issue in a slightly different way, but one that is precisely equivalent: "Choose any five numbers that have a mean of 20." This is an equivalent task since any five numbers that have a mean of 20 will add up to 100. Therefore, once again, there are only four choices: These four choices precisely determine the value that must be chosen as the fifth. Hence, in a group of scores with a fixed mean, there is one less degree of freedom than the total number of scores. This explanation ties in to the information provided in the entry titled "▶ Variance," in which the calculation of variance from a term called "the sum of squares" is described: Variance is obtained by dividing the sum of squares by the degrees of freedom.

A word of further explanation is in order here. While the term $(n - 1)$ is used by the majority of statisticians, it is possible that you might on occasion see the term n used as the denominator instead of $(n - 1)$ when calculating variance. The mathematical reasoning behind the statement that $(n - 1)$ is the better choice is beyond the scope of this encyclopedia's discussions. Nonetheless, a simple explanation makes the point.

Whenever a single research study is conducted, a sample of study participants is chosen from the population of all individuals who could theoretically have participated as subjects. The data collected from the subjects who did participate facilitate the precise calculation of the variance in the characteristic of interest to the researchers (reduction in blood pressure, reduction in an assessment of depression, etc.). However, this precisely calculated value is not of primary interest. What is of most interest is an estimate of the variance that would be seen in the general population of individuals who may be exposed to the intervention of interest if the study reveals that the intervention is both safe and also of therapeutic benefit. That is, the goal is to use statistical methodology to estimate in an optimum manner how well the results of a single experimental study will generalize to the general population of patients should the intervention become widely used. The best estimate of the population variance is generated by the use of $(n - 1)$ as the degrees of freedom, that is, as the denominator when the sum of squares is divided by the degrees of freedom.

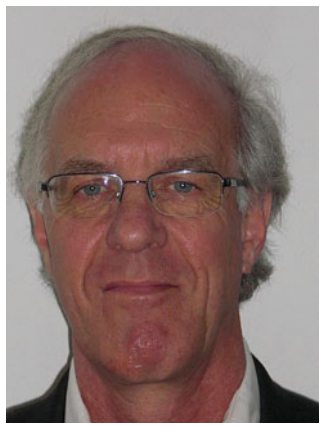
Cross-References

▶ Standard deviation
▶ Variance

Dekker, Joost

Joost Dekker
Department of Psychiatry and Department of Rehabilitation Medicine, VU University Medical Centre, Amsterdam, The Netherlands

Biographical Information



Joost Dekker was born in Doetinchem, the Netherlands, on June 23, 1951. He received a BSc in chemistry (Utrecht, 1973, cum laude) and MSc in psychology (Utrecht, 1980). He obtained his PhD in 1988 (Utrecht, cum laude). He is a licensed health psychologist. He was senior researcher at the WHO Collaborating Center on Quality of Life in Relation to Health Care, Amsterdam (1987–1988), and senior researcher and subsequently head of the research department at the Netherlands Institute of Primary Health Care, Utrecht (1988–2001). He was also director of the Institute of Health and Welfare Studies, Amsterdam (2001–2007).

Dekker is currently Professor of Allied Health Care at the Department of Psychiatry and the Department of Rehabilitation Medicine, VU University Medical Center, Amsterdam, the Netherlands (1997–present). He is chair of the research track Soma & Psyche in the Mental Health research program, EMGO Institute for Health and Care Research, and leader of the Quality of Life research program, Cancer Center Amsterdam. He is "dosent" of Psychological Aspects of Rehabilitation at the Faculty of Sports and Health Sciences, University of Jyväskylä, Finland.

Dekker's research concerns behavioral factors in somatic disease, in the clinical epidemiological tradition. He focuses on musculoskeletal disorders, neurological disorders, and – recently – cancer. He has obtained grants from numerous agencies, including the Ministry of Health, Netherlands Organization for Health Research and Development, and NGOs. He is (co)author of more than 225 international peer-reviewed scientific publications, more than 80 national peer-reviewed scientific publications, and more than 90 scientific publications in books, reports, and other journals. He served as editor in chief (2007–2011) and associate editor (1993–2006) of the International Journal of Behavioral Medicine. He performs editorial services for a wide range of scientific journals and has supervised 21 successfully defended PhD theses.

Dekker is president elect of the International Society of Behavioral Medicine (2010–2012) and will serve as president from 2012 to 2014. Other positions in ISBM include member of the Board (2007–present), member of the Governing Council (1994–2006), chair of the Strategic Planning Committee (2004–2006), chair of the Nominations Committee (2006), and co-chair (1996–1998) and chair (1998–2002) of the Education and Training Committee. He is involved in other international and national boards and committees. Examples include the Society of Behavioral Medicine, the Cochrane Collaboration, Netherlands Health Council, Netherlands Health Research Council, Netherlands Behavioral Medicine Federation, and Royal Netherlands Society of Physiotherapy. He has contributed to the organization of numerous international and national conferences.

Major Accomplishments

Dekker developed the theory on behavioral and neuromuscular factors in activity limitations in

osteoarthritis. The theory provides an integrated model of how behavioral factors (negative affect and avoidance of activities) and neuromuscular factors (muscle weakness, poor proprioception, and laxity of joints) interact, resulting in activity limitations in this clinical condition. Empirical support for the theory has been obtained in cross-sectional and longitudinal research (Dekker, Tola, Aufdemkampe, & Winckers, 1993; van der Esch, Steultjens, Knol, Dinant, & Dekker, 2006; van der Esch et al., 2007; Steultjens, Dekker, & Bijlsma, 2002).This theory was used to develop therapeutic approaches aimed at improved performance of activities in osteoarthritis. Examples include "behavioral graded activity" and "stability training." These therapeutic approaches have been and are being evaluated in randomized clinical trials, which are providing evidence in support of these approaches (Pisters, Veenhof, Schellevis, de Bakker, & Dekker, 2010; Veenhof et al., 2006).

He contributes to the integration of psychology and rehabilitation (including rehabilitation medicine, physiotherapy, and occupational therapy). This integrated approach results in novel theories and innovative treatments. The previously mentioned research on activity limitations in osteoarthritis illustrates the integration of psychology and rehabilitation. Other examples include the development of therapeutic approaches for neurological patients, specifically stroke patients with apraxia (Donkervoort, Dekker, Stehman-Saris, & Deelman, 2001; Donkervoort, Dekker, & Deelman, 2006; van Heugten et al., 1998) and patients with dementia (Graff et al., 2006).

Dekker also contributes to the scientific foundation of rehabilitation medicine, physiotherapy, and occupational therapy. This work concerns the application of the International Classification of Functioning in these disciplines (Dekker, 1995), summarizing the evidence in support of exercise therapy in a wide range of disorders (Baar, Assendelft, Dekker, Oostendorp, & Bijlsma, 1999; Smidt et al., 2005), assessing prognostic factors for quality of life (Braamse et al., 2011; van der Waal, Terwee, van der Windt, Bouter, & Dekker, 2005), summarizing the evidence in support of occupational therapy in numerous disorders (Steultjens, Dekker, Bouter, Leemrijse, & van den Ende, 2005), assessing the impact of comorbidity on pain and activity limitations (van Dijk et al., 2010), and contributing to the development of measurement instruments and clinimetrics (Dekker, Dallmeijer, & Lankhorst, 2005).

He strongly supports the implementation of his research into clinical practice. This has resulted in the foundation of an outpatient clinic for advanced rehabilitation in osteoarthritis, the development and implementation of a national consensus on the treatment of osteoarthritis (van den Ende et al., 2010), and the implementation of screening and treatment for psychological distress in patients with multiple sclerosis and cancer.

In the role of editor in chief, Dekker contributed to the definition of the profile of the International Journal of Behavioral Medicine (Dekker, 2007). *IJBM* has been defined as an interdisciplinary journal, publishing research on factors relevant to health and illness. The scope of *IJBM* extends from biobehavioral mechanisms, clinical studies on diagnosis, treatment, and rehabilitation to research on public health, including health promotion and prevention. *IJBM* is an international journal: manuscripts originate from all over the world, addressing issues related to both local and global health.

Cross-References

- ▶ Arthritis
- ▶ Cancer Survivorship
- ▶ Chronic Pain
- ▶ Evidence-Based Behavioral Medicine (EBBM)
- ▶ Exercise
- ▶ International Society of Behavioral Medicine
- ▶ Neurological
- ▶ Occupational Therapy
- ▶ Physical Therapy
- ▶ Psychometrics
- ▶ Quality of Life: Measurement
- ▶ Rehabilitation

References and Readings

Baar, M. E. V., Assendelft, W. J. J., Dekker, J., Oostendorp, R. A. B., & Bijlsma, J. W. J. (1999). Effectivenss of exercise therapy in patients with osteoarthritis of the hip or knee: A systematic review of randomized clinical trials. *Arthritis & Rheumatism, 42*, 1361–1369.

Braamse, A. M., Gerrits, M. M., van Meijel, B., Visser, O., van Oppen, P., Boenink, A. D. et al (2011). Predictors of health-related quality of life in patients treated with auto- and allo-SCT for hematological malignancies. *Bone Marrow Transplant*. doi:10.1038/bmt.2011.130.

Dekker, J. (1995). Application of the ICIDH in survey research on rehabilitation: The emergence of the functional diagnosis. *Disability and Rehabilitation, 17* (3–4), 195–201.

Dekker, J. (2007). Defining the profile. *International Journal of Behavioral Medicine, 14*, 1–2.

Dekker, J., Dallmeijer, A. J., & Lankhorst, G. J. (2005). Clinimetrics in rehabilitation medicine: Current issues in developing and applying measurement instruments 1. *Journal of Rehabilitation Medicine, 37*(4), 193–201.

Dekker, J., Tola, P., Aufdemkampe, G., & Winckers, M. (1993). Negative affect, pain and disability in osteoarthritis patients: The mediating role of muscle weakness. *Behavior Research and Therapy, 31*, 203–206.

Donkervoort, M., Dekker, J., & Deelman, B. (2006). The course of apraxia and ADL functioning in left hemisphere stroke patients treated in rehabilitation centres and nursing homes. *Clinical Rehabilitation, 20*(12), 1085–1093.

Donkervoort, M., Dekker, J., Stehman-Saris, F. C., & Deelman, B. G. (2001). Efficacy of strategy training in left hemisphere stroke patients with apraxia: A randomised clinical trial. *Neuropsychological Rehabilitation, 11*, 549–566.

Graff, M. J., Vernooij-Dassen, M. J., Thijssen, M., Dekker, J., Hoefnagels, W. H., & Rikkert, M. G. (2006). Community based occupational therapy for patients with dementia and their care givers: Randomised controlled trial. *British Medical Journal, 333*(7580), 1196.

Pisters, M. F., Veenhof, C., Schellevis, F. G., de Bakker, D. H., & Dekker, J. (2010). Long-term effectiveness of exercise therapy in patients with osteoarthritis of the hip or knee: A randomized controlled trial comparing two different physical therapy interventions. *Osteoarthritis and Cartilage, 18*(8), 1019–1026.

Smidt, N., de Vet, H. C., Bouter, L. M., Dekker, J., Arendzen, J. H., de Bie, R. A., et al. (2005). Effectiveness of exercise therapy: A best-evidence summary of systematic reviews. *The Australian Journal of Physiotherapy, 51*(2), 71–85.

Steultjens, M. P., Dekker, J., & Bijlsma, J. W. (2002). Avoidance of activity and disability in patients with osteoarthritis of the knee: The mediating role of muscle strength. *Arthritis and Rheumatism, 46*(7), 1784–1788.

Steultjens, E. M., Dekker, J., Bouter, L. M., Leemrijse, C. J., & van den Ende, C. H. (2005). Evidence of the efficacy of occupational therapy in different conditions: An overview of systematic reviews. *Clinical Rehabilitation, 19*(3), 247–254.

van den Ende, C. M., Bierma-Zeinstra, S. M., Vlieland, T. P., Swierstra, B. A., Voorn, T. B., & Dekker, J. (2010). Conservative treatment of hip and knee osteoarthritis: A systematic, step-by-step treatment strategy. *Nederlands Tijdschrift voor Geneeskunde, 154*, A1574.

Van der Esch, M., Steultjens, M., Harlaar, J., Knol, D., Lems, W., & Dekker, J. (2007). Joint proprioception, muscle strength, and functional ability in patients with osteoarthritis of the knee. *Arthritis and Rheumatism, 57*(5), 787–793.

van der Esch, M., Steultjens, M., Knol, D. L., Dinant, H., & Dekker, J. (2006). Joint laxity and the relationship between muscle strength and functional ability in patients with osteoarthritis of the knee. *Arthritis and Rheumatism, 55*(6), 953–959.

van der Waal, J. M., Terwee, C. B., van der Windt, D. A., Bouter, L. M., & Dekker, J. (2005). The impact of non-traumatic hip and knee disorders on health-related quality of life as measured with the SF-36 or SF-12. A systematic review. *Quality of Life Research, 14*(4), 1141–1155.

van Dijk, G. M., Veenhof, C., Spreeuwenberg, P., Coene, N., Burger, B. J., van Schaardenburg, D., van den Ende, C. H., Lankhorst, G. J., Dekker, J. (2010). CARPA Study Group. Prognosis of limitations in activities in osteoarthritis of the hip or knee: a 3-year cohort study. *Archives of Physical Medicine and Rehabilitation*, *91*:58–66.

van Heugten, C. M., Dekker, J., Deelman, B. G., van Dijk, A. J., Stehmann-Saris, J. C., & Kinebanian, A. (1998). Outcome of strategy training in stroke patients with apraxia: A phase II study. *Clinical Rehabilitation, 12*(4), 294–303.

Veenhof, C., Koke, A. J., Dekker, J., Oostendorp, R. A., Bijlsma, J. W., van Tulder, M. W., et al. (2006). Effectiveness of behavioral graded activity in patients with osteoarthritis of the hip and/or knee: A randomized clinical trial. *Arthritis and Rheumatism, 55*(6), 925–934.

Delay Discounting

► Impulsivity

Deliberate Self-Harm

► Suicide

Delta Sleep

► Slow-Wave Sleep

Dementia

Bonnie Levin
Department of Neurology, University of Miami Medical Center, Miami, FL, USA

Synonyms

Cognitive impairment; Cortical dementia; Dementing illness

Definition

Dementia is a disorder characterized by a progressive decline in intellectual function or behavior severe enough to cause impairment in social and occupational functioning.

Description

The term dementia is derived from the Latin words de ("without") and mens ("the mind"). The most widely used criterion for diagnosing dementia is the DSM-IV, which defines dementia as a disorder characterized by progressive decline in intellectual function or behavior severe enough to cause impairment in social and occupational functioning. Memory loss is the hallmark feature as well as impairment in one or more cognitive abilities, including language, reasoning, executive function, praxis, and visuospatial skills.

The DSM-V, which is expected to be published in 2012, has adopted the term "Neurocognitive Disorders" and further subdivided it into "Major" and "Minor" to replace the DSM-IV classification of "Delirium, Dementia, and Amnestic and Other Cognitive Disorders."

There are four dementia syndromes that account for approximately 90% of cases. They are Alzheimer's disease, vascular dementia, dementia with Lewy bodies, and frontotemporal dementia.

Alzheimer's disease: Alzheimer's disease (AD) is the most common dementia accounting for 50% of all cases. The major pathology is an abnormal extracellular accumulation of beta-amyloid peptide and intracellular accumulation of tau protein. Beta-amyloid is believed to be the main component of senile plaques (SPs) and tau is involved in the development of neurofibrillary tangles (NFT). Neuropathological examination of AD brains reveals that most cases of AD have a combination of NFT and SP. The NFTs initially appear in the hippocampus and entorhinal cortex and then extend to the neocortex. SPs tend to be seen more in the association cortex. Memory changes have been correlated with hippocampal and entorhinal pathology whereas more global cognitive decline is seen with neocortical involvement. AD onset is typically insidious, often taking years before the correct diagnosis is made. The first clinical criteria based on consensus were published in l983, referred to as the NINCDS-ADRDA. The advancement in MR imaging, PET imaging, CSF assays, and other biomarkers have shown that the older criteria are no longer well suited to diagnose AD and newer guidelines for all-cause dementia and AD dementia, which is further subdivided into amnestic and non-amnestic presentations, have recently been published (Dubois et al., 2007).

Cardinal features of the disease are progressive decline in mental status functions, including memory loss, and one or more cognitive impairments involving language, executive, visuospatial/perceptual dysfunction, apraxia, and agnosia. The cognitive deficits seen in AD are progressive and interfere with activities of daily living (ADL). The average time course for AD is between 8 and 12 years after diagnosis, but it can last as long as 20. It is now accepted that there is a prodromal phase in which individuals exhibit mild cognitive impairment, also referred to as MCI, before reaching the threshold for early dementia (Peterson, 2000).

There are three stages of AD. In mild AD, individuals typically present with problems recalling recent events with relative sparing of older memories. Other frequent cognitive problems include difficulty in solving problems and carrying out complex multi-step tasks, making sound decisions, planning difficulties, and problems in holding information in mind. Changes in personality are also common, with irritability and apathy among the most frequent complaints voiced by caregivers. Individuals in the early stages of AD may also exhibit empty speech, problems finding words, and have difficulty expressing their ideas. In the midstage or moderate AD, individuals become more confused and their memory loss is more pervasive. They may have difficulty retrieving older memories such as their address, school they attended, or names of relatives. Assistance with basic ADLs such as grooming, toileting, and other self-care activities may be necessary. Personality changes are more pervasive and it is not unusual for caregivers to report aggression and paranoia. In the late or severe stage of AD, afflicted individuals have lost the ability to communicate beyond occasional words or phrases and require full time assistance for all self-care activities. At this stage, motor symptoms are common as well as loss of bowel, bladder, and swallowing abilities. Most AD patients die of complications of chronic illness (pneumonia).

Vascular dementia: It is estimated that nearly two thirds of individuals who experience a stroke will have some degree of cognitive impairment, with roughly a third exhibiting frank dementia (Selnes & Vinters, 2006). Cognitive impairment resulting from vascular factors has been termed, "vascular cognitive impairment" or VCI. Various components of the "metabolic syndrome," a term that refers to a cluster of cardiovascular risk factors, including diabetes, hypertension, hyperlipidemia, hypertriglycemia, and impaired glucose tolerance, have been linked to age-related cognitive decline. Postmortem studies have revealed that VCI can also coexist with AD pathology, and those with both pathologies show a greater degree of cognitive impairment (REF). Since many of the vascular risk factors can be modified following changes in one's lifestyle (diet, exercise, not smoking, etc.) and medication, it may be possible to improve or even decrease the incidence of VCI with the appropriate intervention (Gorelick et al., 2011).

Dementia with Lewy bodies: Dementia with Lewy bodies (DLB), also known as Lewy body dementia, Lewy body disease, and cortical Lewy body disease, is the second most common dementia after Alzheimer's disease. DLB can present as a movement disorder resembling Parkinson's disease with cognitive changes or with memory and dysexecutive changes suggestive of Alzheimer's disease with visual hallucinations and/or delusions. Other presenting features of DLB include fluctuating levels of attention, characterized by drowsiness, starring off, lethargy, a history of falling, sleep-related disturbances, and autonomic dysregulation involving body temperature, blood pressure, urinary difficulties, constipation, and swallowing difficulties. Risk factors are age (>60 years), gender (male), and family history.

Frontal temporal dementia: Frontal temporal dementia (FTD) is a category of conditions involving atrophy and neuronal loss of the frontal and temporal lobes, resulting in prominent language impairment and behavioral decline. It is the most prevalent dementia among younger individuals. It is estimated that between 20% and 50% of individuals with dementia under 65 years of age have FTD (REF). Three FTD syndromes have been proposed: behavioral variant, semantic dementia, and progressive nonfluent aphasia. In the behavioral variant, neuropsychiatric features, characterized by emotional dysregulation, are prominent early in the disease. Social inappropriateness, lack of insight, apathy, disinhibition, and diminished activity are frequent as well as more extreme behaviors including poor hygiene, hyperorality, shoplifting, and other impulse control problems. This variant is often misdiagnosed as depression due to the apathetic behavioral style. Frank psychosis is unusual but seen most often among individuals with Alzheimer's disease (Cardarelli, Kertesz, & Knebl, 2010; Neary et al., 1998). In semantic dementia, patients present with fluent speech that is devoid of meaning and may contain

semantic paraphasias. The central feature is language output characterized by the use of words that approximate the intended word, such as "thing to eat with" for knife or "clothes" for skirt. In addition, this variant is also associated with associative agnosia, or the inability to recognize and assign meaning to objects and facial recognition deficits, including well-known figures such as celebrities (Cardarelli et al., 2010; Neary et al., 1998). The third variant, progressive nonfluent aphasia, is characterized by speech that is agrammatical, nonfluent, stuttering or halting, and effortful. Word retrieval difficulties or frank anomia are common with phonemic paraphasias such as saying "dat" for cat or "drother" for mother. Other impairments include difficulties with comprehension, reading, and repetition. Median survival for FTD is comparable to AD, approximately 9 years. Since there is no treatment for FTD, intervention is at the level of establishing behavioral management strategies for issues related to behavioral conduct and psychological counseling for caregivers and family members (Cardarelli et al., 2010; Neary et al., 1998).

Other Dementias

Treatable Dementia

There are a number of treatable dementias. The most common are those resulting from metabolic disorders such as a vitamin B-12 deficiency, normal pressure hydrocephalus, chronic substance abuse, subdural hematoma following trauma and hypothyroidism. For this reason, it is important to first rule out the treatable dementias with the help of a careful medical work-up, blood tests, and neuroimaging.

Rapidly Progressive Dementia (RPD)

There is a group of dementing conditions that develop subacutely and involve rapid decline of cognitive, behavioral, and motor function. A variety of etiologies can lead to RPD including neurodegenerative, toxic-metabolic, neoplastic, infectious, and inflammatory conditions (Geschwind, Shu, Harman, Sejvar, & Miller, 2008; Rosenbloom & Alireza, 2011).

The most widely studied RPD subgroup is the prion disease Creutzfeldt-Jakob disease or CJD. The sporadic form of CJD (sCJD) typically presents with mental status alterations characterized by dementia and/or psychiatric changes accompanied by cerebellar and extrapyramidal symptoms. sCJD onset is usually between 50 and 70 years and is equally prevalent in males and females, with a short median survival of 5 months. Of note, psychiatric complaints and behavioral symptoms such as depression, malaise and marked anxiety can precede the dementia and movement disorder. The EEG in the later stages of the disease has a distinctive diagnostic pattern of periodic sharp waves. The other form, referred to as variant CJD (vCJD), is rarer and can affect either young or older adults. Mean age of onset is 29 years and typically presents as a psychiatric disturbance lasting 6 or more months before other symptoms begin. Although the classic EEG pattern described above for sCJD is not typically present, the diagnostic feature of vCJD is the pulvinar sign on MRI (Geschwind et al., 2008).

Assessment of Dementia

The diagnosis of dementia should be ascertained through a combination of careful history taking, an interview with the patient and an informant, and neuropsychological testing performed by a qualified professional. The type of cognitive battery used to assess dementia will depend on several factors, including the time allotted for assessment, the willingness of the patient to participate in the testing process, and clinician availability.

Assessment of dementia requires an understanding of the normative aging process, brain anatomy and neural circuitry, and neuropathology. The mental status evaluation should focus on three components: cognition, personality/mood, and behavioral function. The type of examination can vary from bedside screening to a comprehensive evaluation.

The most widely known measure is the Mini-Mental State Examination (MMSE), a short screening instrument developed in the l970s to assess cognition in the elderly. It is untimed,

consists of 11 questions and a total possible score of 30 points. Originally designed for psychogeriatric patients (Rosenbloom & Alireza, 2011), this measure has been used to assess mental status in a wide variety of neurologic and general medical disorders as a dementia screen, not as a diagnostic tool. Its purpose is threefold: to screen for cognitive impairment, to assess severity of impairment, and to monitor change over time with repeated assessments.

There are many advantages of the MMSE, some of which include the ease of administration, the availability of international translations, the use of cutoff scores that make inter-study comparisons easier, and the availability of norms for age and education. However, there are several drawbacks to the MMSE (Nieuwenhuis-Mark, 2010). One criticism of this screening instrument is that it relies heavily on intact verbal skills, a problem for those with limited language ability or with a low educational level. Also there is a lack of consensus as to which cutoff score is best to use and whether the norms have been collected on representative samples. Although the MMSE has wide international use and has been translated into many different languages, there are questions as to whether the translations are really comparable to the original test due to the fact some of the items may not be relevant in other cultures (e.g., reciting "no, ifs, ands, and buts"). The most serious criticism is that the MMSE was developed as a cognitive screen, but it is widely used as a diagnostic tool and has been shown to be insensitive in discriminating between age-related cognitive change, mild cognitive impairment, and early dementia (Mitchell, 2009).

Ideally, bedside examinations and screening measures should be supplemented with more comprehensive testing using standardized neuropsychological measures with known reliability and sensitivity for detecting cognitive impairment. A qualified neuropsychologist will select measures designed to assess specific cognitive abilities including general intelligence, language abilities (e.g., verbal fluency, word retrieval, comprehension), conceptual reasoning and abstraction, perception, spatial cognition (visuoconstructive graphomotor and assembling abilities), attention and memory (working memory, verbal and nonverbal immediate and delayed recall), motor speed and the executive functions (e.g., skills involved in planning, organization, set shifting, goal setting, and problem solving). Additional questionnaires are frequently included to assess a patient's emotional status, in particular symptoms of anxiety and depression. A major goal of the neuropsychological evaluation is to identify a specific pattern of cognitive change associated with a particular dementia. Another goal is to provide a baseline from which to compare future evaluations. A third goal is to identify cognitive strengths and weaknesses that can be used to address treatment and management issues.

Prevention of Dementia

There is compelling evidence from basic and clinical research that aerobic exercise may be neuroprotective. Transgenic mouse models, epidemiologic, biomarker, and prospective clinical studies have linked exercise with improved cognition and provided evidence that physical activity directly modulates known risk factors associated with dementia, including obesity, vascular disease, hypertension, diabetes, inflammation, and cardiovascular disease. The mechanism by which increased physical activity improves cognition is not known. It has been proposed that physical exercise may reduce beta-amyloid deposits in the brain, increase synaptogenesis and plasticity, boost endorphins and growth factors, or increase brain perfusion (Savica & Peterson, 2011).

Education and intellectual stimulation have also been credited with having a neuroprotective role against dementia and cognitive decline (Stern, 2002). Numerous observational studies have shown a strong and positive association between higher intellectual activities and reduced cognitive decline. A lower educational level is associated with a 30% increased risk of having AD. It is possible that higher education is a proxy for higher SES which confers an advantage in terms of overall health, lifestyle, and reduced stress.

More recent studies have provided preliminary evidence showing that cognitive training, i.e., mental exercises to improve memory, reasoning, and processing, may improve cognition and instrumental activities of daily living. Preliminary evidence has shown in animals that increased mental activity is linked to increased brain volume (Willis et al., 2006).

Diet: Although it has long been held that diet is linked to cognition, the evidence remains controversial. Most attention has centered on the Mediterranean diet, one which is high in vegetables and non-saturated fat, and linked to a lower risk of cognitive decline. It is argued the diet is high in antioxidants and its relationship to reducing oxidative stress is the key factor responsible for improved cognition (Savica & Peterson, 2011).

Social Networks: There are several studies suggesting that social connectivity, as defined by participation in social activities and maintaining interpersonal relationships, may be protective against cognitive decline. Conversely, social disengagement, loneliness, and not living with a partner have been found to be linked to AD, although this association did not apply to those who were divorced or widowed (Seeman, Lusignolo, Albert, & Berkman, 2001). A valid criticism of these studies is that it is difficult to know whether reduced social connectivity is a risk factor or an actual symptom of AD, given that social withdrawal is a feature of early dementia.

Cross-References

- ▶ Aging
- ▶ Alzheimer's Disease
- ▶ Cognitive Function
- ▶ Cognitive Impairment
- ▶ Geriatrics

References and Readings

Cardarelli, R., Kertesz, A., & Knebl, J. A. (2010). Frontotemporal dementia: A review for primary care physicians. *American Family Physician, 82*, 1372–1377.

Dubois, B., Feldman, H. H., Jacova, C., Dekosky, S. T., Barberger-Gateau, P., Cummings, J., et al. (2007). Research criteria for the diagnosis of Alzheimer's disease: Revising the NINDS-ADRDA criteria. *Lancet Neurology, 8*, 734–746.

Geschwind, M. D., Shu, H., Harman, A., Sejvar, J. J., & Miller, B. (2008). Rapidly progressive dementia. *Annals of Neurology, 64*(1), 97–108.

Gorelick et al. (2011). Inclusion of stroke in cardiovascular risk prediction instruments: A statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke, 42*, 2672–2713.

Mitchell, A. J. (2009). A meta-analysis of the accuracy of the mini-mental state examination in the detection of dementia and mild cognitive impairment. *Journal of Psychiatry Research, 43*(4), 411–431.

Neary, D., Snowden, J. S., Gustafson, L., Passant, U., Stuss, D., Black, S., et al. (1998). Frontotemporal lobar degeneration: A consensus on clinical diagnostic criteria. *Neurology, 51*(6), 1546–1554.

Nieuwenhuis-Mark, R. E. (2010). The death knoll for the MMSE: Has it outlived its purpose? *Journal of Geriatric Psychiatry and Neurology, 23*(3), 151–157.

Peterson, R. C. (2000). Mild cognitive impairment: Transition between aging and Alzheimer's disease. *Neurologia, 15*, 93–101.

Rosenbloom, M. H., & Alireza, A. (2011). The evaluation of rapidly progressive dementia. *Neurologist, 7*(2), 67–74.

Savica, R., & Peterson, R. C. (2011). Prevention of dementia. *Psychiatric Clinics of North America, 34*(1), 127–145.

Seeman, T. E., Lusignolo, T. M., Albert, M., & Berkman, L. (2001). Social relationships, social support, and patterns of cognitive aging in healthy, high-functioning older adults: MacArthur studies of successful aging. *Health Psychology, 20*(4), 243–255.

Selnes, O. A., & Vinters, H. V. (2006). Vascular cognitive impairment. *Nature Clinical Practice Neurology, 2*, 538–547.

Stern, Y. (2002). What is cognitive reserve? Theory and research application of the reserve concept. *Journal of International Neuropsychology Society, 8*, 448–460.

Willis, S. L., Tennstedt, S. L., Marsiske, M., Ball, K., Elias, J., Koepke, K. M., et al. (2006). Long-term effects of cognitive training on everyday functional outcomes in older adults. *Journal of the American Medical Association, 296*(23), 2805–2814.

Dementia Screening Tests

- ▶ Screening, Cognitive

Dementing Illness

► Dementia

Demographics

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Subject characteristics

Definition

Demography can be defined as the statistical science focusing upon the distribution, density, vital statistics, and various other defining characteristics of human populations. Demographics therefore include characteristics such as sex, age, race/ethnicity, height, weight, and socioeconomic class.

When reporting a research study, it is necessary to provide a summary of the relevant demographic characteristics of the subjects who participated in the study. Ultimately, the goal of a clinical study is not to provide precise information for that particular subject sample but to collect information that generalizes to the population from whom that particular sample was chosen. Therefore, a given subject sample needs to reflect that population adequately for such generalization to be meaningful.

Not all demographic information is always of relevance. In some studies, perhaps a clinical trial of a new drug, it may not be necessary to report the socioeconomic status of the study participating in the study. If there is no biologically plausible reason to think that individuals from different socioeconomic strata would respond differently to the drug, it is not necessary to report this information. In contrast, sex and age may be considered to be of considerable relevance if there are biologically plausible reasons to think that these factors may influence drug responses.

Cross-References

► Generalizability

Demyelinating Disease

► Multiple Sclerosis: Psychosocial Factors

Denial

Alefiyah Z. Pishori
Department of Psychology, University of Connecticut, Storrs, CT, USA

Synonyms

Coping strategies; Defense mechanism

Definition

The psychological concept of denial refers to a cognitive and emotional coping strategy involving the negation of a fact or reality. In the context of health psychology, denial refers to the negation of a health problem, effecting either oneself or someone else. Individuals experiencing denial refuse to believe facts that are difficult to face, such as being diagnosed with a terminal or chronic illness. Denial is a common and normal process; it can be either protective or harmful in managing a health problem, depending on the extent of the denial and how it impacts individuals' decision making. For instance, denial has been identified as a useful and necessary first step in the process of coping with a terminal or life-threatening illness to allow individuals to adjust

to the situation. However, denial can become a problem when it persists and prevents individuals from actively coping with the truth and/or receiving necessary care and treatment for their illnesses. Family members or friends of an individual diagnosed with an illness may also engage in denial; they may cope with the experience of learning their loved one is ill by rejecting the idea. If their denial persists as well, they may advise their loved one to not seek treatment or reject the truth regarding what is happening to their loved one.

Description

Denial as a psychological process has a long history, originating with Sigmund Freud's theory of defense mechanisms. Freud conceptualized defense mechanisms as strategizes utilized by individuals to protect themselves from difficult memories by distorting them or making them inaccessible to consciousness; denial was one such strategy. Over time, theorists have identified different types of denial that an individual may experience or engage.

Defensive Denial

Defensive (or pathogenic) denial is a specific form of denial in which individuals use defensive processes to manage their emotional responses and allow for appropriate decisions regarding health threats. This is a dynamic process that accommodates new information and can be distinguished from other forms of denial that ignore reality. Defensive denial minimizes threats but is responsive to reality and information regarding the situation. It may lead to the minimization of health threats, protective social comparisons that minimize the individuals' risks, or questioning the validity of health threat information. It is unclear how defensive denial impacts behavior, although it has been suggested that it may decrease negative affect and thus allow individuals to engage in protective health behaviors (Wiebe & Korbel, 2003). However, if a person experiencing pathogenic denial is not exposed to information regarding the seriousness of the situation, he/she may not recognize the truth of his/her situation (Janis, 1983).

Malignant Denial

Malignant (or pathological) denial is a maladaptive form of denial that prevents individuals from receiving necessary treatment for their conditions. What makes this form of denial malignant is the impact it has on health-care decisions: individuals will ignore doctors and refuse treatment. For instance, individuals suffering from psychological conditions, such as substance abuse and eating disorders, often deny they have a problem and refuse help or treatment. Individuals with infectious diseases who are experiencing malignant denial may engage in behaviors that put others at risk of becoming infected as well (Kalichman, 2009). Thus, this form of denial is a serious concern as it can negatively impact both an individual's own and others' health.

Denial versus Avoidance

Denial must be distinguished from avoidance. Avoidance refers to individuals' refraining from reminders of the truth, although they are cognitively aware of the facts, whereas denial suggests that individuals have refused to accept the facts. Thus, an avoidant individual may recognize he/she is ill but refrain from going to the doctor for treatment, while an individual in denial would refuse to see the doctor because he/she would not acknowledge he/she had an illness. Although these concepts are often confused and the terms used interchangeably, they are conceptually distinct.

Cross-References

- ▶ Avoidance
- ▶ Defensiveness

References and Readings

Breznitz, S. (1983). *The denial of stress*. New York: International Universities.

Cohen, S. (2001). *States of denial: Knowing about atrocities and suffering*. Malden, MA: Blackwell.
Janis, I. L. (1983). Preventing pathogenic denial by means of stress inoculation. In S. Breznitz (Ed.), *The denial of stress* (pp. 35–76). New York: International Universities Press.
Kalichman, S. (2009). *Denying AIDS: Conspiracy theories, pseudoscience, and human tragedy*. New York: Copernicus Books.
Wiebe, D., & Korbel, C. (2003). Defensive denial, affect, and the self-regulation of health threats. In L. D. Cameron & H. Leventhal (Eds.), *The self-regulation of health and illness behavior* (pp. 184–203). New York: Routledge.

Dependence, Drug

John Grabowski
Department of Psychiatry, Medical School, University of Minnesota, Minneapolis, MN, USA

Synonyms

Drug abuse; Substance abuse; Substance use disorders

Definition

Problematic use of drugs altering behavior and psychological function is categorized in terms of patterns and consequences of use. The common worldwide codification of these disorders is found in International Statistical Classification of Diseases and Related Health Problems 10 (ICD-10), which refers to these disorders as *Mental and behavioral disorders due to psychoactive substance use*. The generic ICD-10 definition is: "*a wide variety of disorders that differ in severity and clinical form but that are all attributable to the use of one or more psychoactive substances, which may or may not have been medically prescribed*." The American Psychiatric Association codification of these disorders is found in the Diagnostic and Statistical Manual – Fourth Edition (DSM-IV), which uses the diagnostic label of *Substance-Related Disorders*. The generic DSM-IV definition is: "*The Substance-Related Disorders include disorders related to the taking of a drug of abuse (including alcohol), to the side effects of a medication, and to toxin exposure*." While "dependence" historically has had a precise scientific definition, common use is often confused with "abuse," "addiction," and other terms.

Description

Determinants of Dependence

Problematic use of drugs altering behavior and psychological function ("psychoactive drugs") is determined by circumstances of use, route of administration, dose, and drug or medication. Direct biological and behavioral effects of a chemical or drug determine the likelihood of drug taking. The "abuse liability" or "abuse potential" is determined with standardized preclinical/animal laboratory procedures. In these experiments, the test drug is made available through an intravenous line, as a liquid for oral consumption, or on occasion as vapor or smoke. The animal has the opportunity to press a lever or engage in some other response producing drug delivery. The rate of responding and frequency of drug delivery are compared to the vehicle, or solution without drug (placebo). If the drug is "self-administered" at higher rates than vehicle, it is deemed to have "rewarding" or reinforcing effects that will sustain drug seeking and drug taking.

Within a series of similar drugs (e.g., stimulants, anxiolytics) the relative reinforcing effect can be established as a hierarchy from least to most reinforcing. In turn, this is characterized as relative abuse liability. Generally, though not always, the animal self-administration patterns predict the likelihood of human self-administration. In these experiments, food and water may be concurrently or sequentially available for comparison to drug intake or to determine the effect of drug on other behaviors. Numerous comparisons can be made, and other paradigms implemented, to further characterize the properties and behavioral consequences of drug self-administration. In animals, the core

biological effects of the agents are examined and in humans a variety of self-report descriptive measures, such as "liking," "willingness to take again," and "unpleasant effects" are also determined. Arguably, it is the balance of *immediate* pleasurable to unpleasant effects that determine possible persistent use. Untoward effects that follow excessive use, for example, "hangovers" are not necessarily deterrents to resumption of drinking alcohol. Ultimately, when use persists and dependence emerges, a variety of untoward outcomes occur. Continued use in patterns that produce hazardous and debilitating outcomes (biological, behavioral, social) are key features in determination of dependence.

Within drug self-administration studies, whether with nonhuman animals or humans, dose-ranging studies are conducted with, for example, "low," "medium," and "high" doses, again with comparison to placebo. The result is often, though not always an inverted U-shaped curve with lower doses consumed less than intermediate doses, while very high unit doses may generate less drug taking (due to increasing adverse effects or satiating doses achieved with less output). In some instances, for example, sedatives, the medication itself may impair ability to continue self-administration. Other experimental strategies determine whether changes in intake are due to incapacitating effects of the drug, titration to seek optimal effect, adverse effects at a particular dose, or other factors. While the interactions may be complex, dose is an important factor in self-administration and establishing drug dependence.

The route of administration (intravenous, inhalation, insulfflation, oral) may alter the likelihood of drug dependence. More rapid onset is typically observed with intravenous and inhalation routes and it is generally thought that this may increase the probability of persistent use. However, individual preferences or dislikes may intervene; for example, many people are unwilling to use injection paraphernalia. Still, while IV heroin use produces a singular and pronounced effect, orally ingested opioids for nontherapeutic purposes can also produce profound dependence. Most agents with moderate to high abuse potential can be expected to be associated with dependence in some people, regardless of route, when used outside of therapeutic regimens.

Other factors important to use and ultimately dependence may include drug availability and social circumstances. Social factors are commonly important in initial use even though later use may be solitary. The relative ease of obtaining a drug makes initial exposure and frequent use more likely for those individuals who are responsive to the effects. Still, most individuals exposed to drugs do not proceed to a level of use that can be categorized as dependence. Knowing who will, or will not proceed to dependence, that is, who is vulnerable to effects of a particular drug and likely to engage in persistent use is a matter of considerable interest.

Behavioral/Psychological and Physical Dependence

The various diagnostic and scientific schemata may differentiate or emphasize two aspects of dependence that are commonly inseparable: behavioral or psychological dependence and physical dependence. As drug action and determinants of persistent use have been more effectively delineated, these distinctions may be less useful but are separable in some circumstances.

Drug dependence typically refers to persistent use despite problems across the spectrum of personal and social activities as well as biological/medical and psychological harm. In current terminology, dependence refers to patterns of behavior that precede, are concurrent with, and follow use. Drug seeking (soliciting/purchasing drugs from others) can be elaborate and time consuming. The behavior of drug taking, legal or illegal, is typically characterized by ritualized events.

These behaviors may be socially accepted as well as being behaviorally and psychologically relatively benign, for example, persistent coffee/caffeinated beverage consumption at moderate doses. Caffeine, most commonly through coffee or carbonated beverage consumption, is thought to be the most widely used drug in

the world. In this example, two prominent ritualized patterns exist. One entails the legal purchase of beans or ground coffee, special home apparatus for grinding and preparing coffee along with the spectrum of containers from which it is consumed. A second pattern that has evolved in recent decades stems from the long-standing practice of coffee with meals or in coffee shops. Now, the elaborate rituals are well represented by Starbucks, Caribou, and other chains as well as myriad local purveyors. The user determines size (volume), dose (singles, doubles, triples), dairy product additions ranging from skimmed milk to heavy cream, additional additives be they spices or liqueur flavorings. The use, dose, drug-taking style, may differ from person to person and time to time for the particular person. Persistent caffeine use has clear biological and psychological effects and cessation of use leads to an array of symptoms.

Heroin use likewise entails procedures and rituals: mixing, drawing drug into a syringe, tying off an extremity to gain access to a vein and injection. Use by smoking or insufflation is also accompanied by a systematized approach to self-administration. These events may be solitary or in groups. The consequences of drug administration are then experienced. Heroin or other persistent opioid use for nontherapeutic purposes often follows a course of increasing dosing as tolerance emerges to the euphoriant effects, increase in associated illegal contacts during drug seeking, increasing cost, and deterioration of social circumstances. Tolerance is not consistent across all effects. Thus, diminished euphoriant effect leading to higher dosing with opioids is not matched by tolerance to respiratory depressant effects and death may ensue. In the case of potent stimulants, cardiovascular excitation/dysfunction or seizures occur as doses increase despite reduction in perceived euphoriant effects. These patterns of use in the face of untoward consequences are emblematic of dependence.

The behaviors immediately following drug use are dependent on the characteristics of the agent. Sedating drugs produce feelings of euphoria or pleasure, varying levels of lethargy and somnolence to virtual unconsciousness, and at the extreme, death, usually from respiratory depression. Stimulant-type drugs generate patterns of energized behavior ranging from active euphoria and self-confidence to highly stereotypic behaviors, hallucinations, and psychosis, and at the extreme, death due to cardiovascular accident or collapse.

Dependence may also refer to a biological state, historically referred to in pharmacology as "addiction" or more recently as "physical dependence," in which a distinctive profile and sequence of symptoms emerges when use is discontinued. The constellation of symptoms observed on abrupt discontinuation in the presence of physical dependence is referred to as a "withdrawal syndrome" (composed of withdrawal signs and symptoms). The consistency of such patterns is most evident for drugs with sedative-like properties such as opiates (e.g., heroin), benzodiazepines (e.g., diazepam), barbiturates (e.g., pentobarbital), and alcohol. These symptoms are typically the reverse of those associated with high-dose drug use; for example, with opioids, behavioral activation, increases in respiration, and increased gastrointestinal activity over baseline emerge. Direct physical symptoms dissipate over days but behavioral and biological symptoms that have been conditioned by repeated pairings of drug self-administration and previously neutral environmental stimuli may persist for months. They may be elicited by environmental circumstances in which drug use or withdrawal symptoms previously occurred. In the case of stimulants, behavioral malaise, impairment in performance, diminution of blood pressure, and other symptoms are common. For the licit drug caffeine, the most pronounced withdrawal symptoms are headache, inattention and associated performance deficits, and fatigue. The constellation of symptoms is often a determinant of reemergent drug seeking and drug taking, or in treatment jargon, "relapse."

As noted, in special circumstances, the components of dependence may be separable. This is informative with respect to the contribution of environmental circumstances, behavioral

consequences, and biological underpinnings. These separable determinants can be examined in the laboratory, for example, by comparing the behavior of animals that have learned to self-administer drugs to those that have passively received the drugs. Comparable instances exist in humans. Remarkable resilience and persistence of behavior emerges for individuals using psychoactive drugs to excess, be the drug stimulant or sedative like. However, patients receiving high doses of intravenous opioids in hospital settings for pain, invariably have a pattern of biological adaptations characteristic of dependence that dictate physiological sequelae to abrupt cessation of dosing will occur. Thus, for example, the constipation associated with high-dose opioid use is followed by increased gastrointestinal motility. Still, it is extraordinarily uncommon on recovery and discharge from the hospital for these patients to engage in the patterns of drug seeking and drug taking that are evident in individuals whose use was established through illicit acquisition and seeking euphoriant effects. Similarly, patients treated with methadone or buprenorphine for previous heroin dependence, are, on one hand physically dependent and tolerant to many opioid effects at a stable dose, but unlikely to seek and use illicit opioids. In other circumstances, for example, treatment of attention deficit-hyperactivity disorder, drugs with some dependence liability are used effectively for treatment and diminish the likelihood of illicit drug use and abuse; thus treating one disorder diminishes a range of problems, including propensity to illicit drug use. These examples point to the importance of a range of environmental and behavioral factors contributing to substance use disorders.

Acquisition, Maintenance, and Elimination of Drug Dependence

In the laboratory, nonhuman subjects (e.g., rodents, primates) generally self-administer, or will work to obtain, the same agents that are taken by humans in both laboratory and natural settings. Conversely, discovery of a drug used for euphoriant purposes in human populations, can generally be translated into animal paradigms with evidence of self-administration. There are some exceptions and special cases; for example, nonhuman organisms rarely self-administer hallucinogens, but for a variety of pharmacological and social reasons, these are not self-administered by humans in the same way as, for example, heroin. Persistent harmful heroin, cocaine, alcohol, or other drug use is clearly distinctively different from either therapeutic drug use or even "controlled" licit or illicit drug use.

Acquisition of drug taking is essentially a social and cultural phenomenon. For legal psychoactive drugs (e.g., alcohol), society sets relatively clear rules and norms generally with a view to harm reduction and control. Most people conform to the accepted social and legal constraints while some go on to dependence. For illegal drug use, the social group sets the norms. Both entail socially driven exposure.

Stable use of legal drugs may differ considerably within and across drugs. Caffeinated beverages may be consumed consistently throughout the day with late day cessation to permit undisrupted sleep. Historically, nicotine/cigarettes were consumed in virtually all social and nonsocial environments during the "maintenance phase." Here the change in social norms over many decades has resulted in limited venues for use, dramatic increases in prices, and remarkable reductions in use across the population in some countries, while unfettered use is permitted and maintained in other countries, thus pointing to the importance of social determinants in use and maintenance. Illegality of a drug sets maximum constraints on some aspects of use that must be circumvented but also establishes a separate market beyond regulatory control. Even with the alternative social constraints, not all individuals who use, be it stimulants (e.g., cocaine), opioids (e.g., heroin), or marijuana, go on to dependence.

Some of the factors that contribute to increased reinforcing effectiveness when a drug is self-administered (immediacy of effect, genetic makeup) have been considered. Yet the factors leading to transition from casual, stable, or infrequent use of a drug, legal or illegal, to

persistent, compulsive-like use by any given individual is not well understood.

Elimination of drug use and termination of self-administration and dependence can be studied in the animal laboratory and some findings are translatable to the human case. In the human natural environment, elimination or cessation may result from a variety of self-imposed regimens or externally applied circumstances. Severe dependence typically requires a course of specific treatment for the substance use disorder(s). For drugs that produce profound withdrawal syndromes, for example, alcohol, the first phase may require intensive medical management. However, this is distinct from the typically required course of treatment involving cognitive behavior therapy, substantial social and behavioral adjustments, and possibly maintenance medications.

Cross-References

- ▶ Addictive Behaviors
- ▶ Cognitive Behavior Therapy
- ▶ Substance Abuse: Treatment

References and Readings

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Carroll, M. E., & Overmier, J. B. (Eds.). (2001). *Animal research and human health: Advancing human welfare through behavioral science [electronic resource]*. Washington, DC: American Psychological Association.

Fischman, M. W., & Mello, N. K. (Eds.). (1989). *Testing for abuse liability of drugs in humans*. National Institute on Drug Abuse. Rockville, MD: U.S. Dept. of Health and Human Services, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, National Institute on Drug Abuse; [Washington, DC: Supt. of Docs., U.S. G.P.O., distributor].

Higgins, S. T. (1997). Applying learning and conditioning theory to the treatmentof alcohol and cocaine abuse. In B. Johnson & J. Roache (Eds.), *Drug addiction and its treatment* (pp. 367–386). Philadelphia: Lippincott-Raven.

Johanson, C. E., Schuster, C. R., Hatsukami, D., & Vocci, F. (2003). Abuse liability assessment of CNS drugs. *Drug and Alcohol Dependence, 70*(3), S1–S114.

McKim, W. A. (2007). *Drugs and behavior: An introduction to behavioral pharmacology* (6th ed.). Upper Saddle River, N.J: Pearson Prentice Hall.

Thompson, T., & Unna, K. R. (Eds.). (1977). *Predicting dependence liability of stimulant and depressant drugs*. Baltimore: University Park Press.

World Health Organization. (1992). *ICD-10 classifications of mental and behavioural disorder: Clinical descriptions and diagnostic guidelines*. Geneva: Author.

Depression

- ▶ Beck Depression Inventory (BDI)
- ▶ Negative Thoughts
- ▶ Pregnancy Outcomes: Psychosocial Aspect

Depression Assessment

- ▶ Depression: Measurement

Depression Diagnosis

- ▶ Depression: Measurement

Depression: Measurement

Samantha Yard and Kimberly Nelson
Department of Psychology,
University of Washington, Seattle, WA, USA

Synonyms

Depression assessment; Depression diagnosis

Definition

The measurement of depression is a process conducted by clinicians and researchers for the purpose of (1) identifying people who may require treatment for depression, (2) identifying people who meet specific diagnostic criteria for

depression, and (3) quantifying the severity of depressive symptomatology.

Description

Depression is defined by a cluster of behaviors and symptoms that have both mental and physical manifestations and affect a wide range of functionality. Specific criteria for depression include experiencing persistent depressed mood or loss of interest or pleasure in most things along with at least four out of the following additional markers: sleep disturbance, feelings of worthlessness or guilt, appetite or weight changes, concentration problems, decreased energy, psychomotor retardation or agitation, and suicidality. Depression is ideally diagnosed through a face-to-face interview with a trained mental health professional based on the current versions of the Diagnostic and Statistical Manual of Mental Disorders (DSM) or the International Classification of Disease (ICD) criteria. It is also important to note that symptoms that do not meet criteria can be impairing and thus may be important to measure as well. As such, depression can be measured both categorically (i.e., meeting diagnostic criteria or not) and continuously (i.e., symptom severity). Depression measurement can be broken down into three distinct functions: screening, diagnosis, and quantification of symptoms. Instrument choice should be made according to these functions, in addition to what population is being assessed, what specific hypotheses are being tested, and if there is a desire to compare the results to other research findings.

Screening instruments are designed to capture anyone who could potentially meet diagnostic criteria for depression and should be referred for further evaluation. They can additionally be used to estimate the prevalence of possible depression in a given setting. They typically do not require a lot of time, have empirically supported cutoff criteria, and can be self-administered or administered by a nonclinician. As screening instruments are unable to diagnosis depression on their own, further assessment by a clinician is needed to establish a diagnosis.

Diagnosis of depression is typically done through a face-to-face interview with a trained clinician. Sometimes, as is typical in medical settings, this occurs only with those patients who have been screened as possibly depressed with the final diagnosis confirmed by a clinician. In research settings, the gold standard for depression diagnosis is a standardized diagnostic interview, such as the Structured Clinical Interview (SCID) or the Composite International Diagnostic Interview (CIDI). Structured diagnostic interviews can take over an hour depending on the patient and are typically administered by a clinician who has also been shown to reliably distinguish cases from among multiple participants. It is important to note that diagnostic interviews are meant to categorize individuals as diagnosed or not but will not capture people who fall just below the criteria, yet are still impaired.

Symptom rating scales offer a continuous assessment of depression severity. They are typically brief, can be self-administered or administered by a nonclinician, and may include a cutoff for screening purposes. They are particularly useful in monitoring changes in depression symptoms over time, and thus can be used in both clinical and research settings to evaluate treatment effects. Although many symptom rating scales have established clinical significance or severity cutoff values, they are unable to diagnose depression on their own; thus, assessment by a clinician or a structured diagnostic interview is needed for diagnosis.

There are multiple things to consider when making a depression measurement choice. First, depression and depressive symptoms can co-occur with or result from a variety of physical conditions such as hypothyroidism, cancer, diabetes, HIV/AIDS, heart disease, medication side effects, substance withdrawal, and other illnesses. Therefore, evaluating for additional medical issues can be important in the assessment of depression. Second, practical considerations should be taken into account, including the length, whether it assesses specific aspects of depression (i.e., measurement of suicidality and somatic symptoms separately), reading level, response formats, need for training to administer,

Depression: Measurement, Table 1 Twenty-two most commonly used and validated measures of depression

Measure	No. of items	Assessment type	Administration	Clinical cutoff?	Depression specific?	Time frame assessed
BDI; BDI-PC	7, 13, 21	Screening; symptom rating	Self	Yes	Yes	Today
CES-D	10, 20	Screening; symptom rating	Self	Yes	Yes	Past week
CGI	3	Symptom rating	Clinician	No	No	Varies
CIDI	Variable	Diagnostic	Interviewer	No	Yes	Past year
CIS-R	Variable	Diagnostic	Interviewer	No	No	Varies
DADS/DUKE-AD	7	Screening	Self	Yes	No	Past week
DEPS	10	Symptom rating	Self	Yes	Yes	Past month
GHQ-12	12	Symptom rating	Self	No	No	Past few weeks
HADS	14	Diagnostic	Self	Yes	No	Currently
HAM-D/HDRS	17–29	Symptom rating	Clinician	No	Yes	Varies
HSCL	13, 25	Screening; symptom rating	Self	Yes	No	Past week
QIDS/IDS	16, 30	Diagnostic; symptom rating	Clinician; self	No	Yes	Past week
K6/K10	6, 10	Symptom rating	Self; interviewer	Yes	No	Past month
MADRS	10	Symptom rating	Clinician	No	Yes	Varies
MDI	10	Diagnostic; symptom rating	Self	Yes	Yes	Past 2 weeks
MINI	Variable	Diagnostic	Clinician	No	No	Lifetime
PHQ/PRIME-MD	2, 9	Screening; diagnostic	Self; clinician	Yes	Yes	Past month/ 2 weeks
SCID	Variable	Diagnostic	Clinician	No	No	Lifetime
SCL-90-R; BSI	53, 90	Symptom rating	Self	Yes	No	Past week
SDDS-PC	5	Screening; diagnostic	Self; clinician		No	Past month
SQ	1	Screening	Self	No	Yes	Past year
ZSDS	20	Screening; symptom rating	Self	Yes	Yes	Recently

CES-D Center for Epidemiologic Studies Depression Scale, CGI Clinical Global Impression, CIDI Composite International Diagnostic Interview, CIS-R Revised Clinical Interview Schedule, DADS/DUKE-AD Duke Anxiety Depression Scale, DEPS The Depression Scale, GHQ-12 General Health Questionnaire, HADS Hospital Anxiety and Depression Scale, HAM-D/HDRS Hamilton Depression Rating Scale, HSCL Hopkins Symptom Checklist, QIDS (IDS) [Quick] Inventory of Depressive Symptoms, K6/K10 Kessler Psychological Distress Scale, MADRS Montgomery-Asberg Depression Rating Scale, MDI Major Depression Inventory, MINI Mini-International Neuropsychiatric Interview, PHQ Patient Health Questionnaire, PRIME-MD Primary Care Evaluation of Mental Disorders, SCID Structured Clinical Interview for DSM Disorders, research version, SCL-90-R Symptom Check List Revised, BSI Brief Symptom Inventory, SDDS-PC Symptom Driven Diagnostic System-Primary Care, SQ Single Question, ZSDS Zung Self-Rating Depression Scale

Note: Some measures come in more than one version with varying lengths and differences in administration

and whether it can be used to assess treatment response and severity of depression along with being able to diagnose. Finally, the Depression Task Force for the DSM-V is considering changing the criteria for depression to include a specification of the severity of depression, using the Patient Health Questionnaire (PHQ-9) or the Clinical Global Impressions (CGI) scale. This forthcoming change in criteria may influence the choice to include the PHQ-9 or CGI in the measurement of depression before measures are adapted to the new criteria.

Below we have provided Table 1 with the twenty-two most commonly used and validated measures of depression. For each measure, we indicate the number of items, type of measure (i.e., screening, diagnostic, and/or symptom rating scale), who can administer the measure (i.e., clinician, interviewer, self), whether it has any established cutoffs for severity or clinical significance, whether the scale or one of the subscales assesses depression specifically – as opposed to general psychological distress or mental illness, and the timeframe the assessment covers.

Cross-References

► Depression: Symptoms

References and Readings

Sharp, L., & Lipsky, M. (2002). Screening for depression across the lifespan: a review of measures for use in primary care settings. *American Family Physician, 66*(6), 1001–1008.

Simoni, J. M., Safren, S. A., Manhart, L. E., Lyda, K., Grossman, C. I., Rao, D., et al. (2010). Challenges in addressing depression in HIV research: Assessment, cultural context, and methods. *AIDS and Behavior*. doi:10.1007/s10461-010-9836-3. Advanced online publication.

Williams, J., Noël, P., Cordes, J., Ramirez, G., & Pignone, M. (2002). Is this patient clinically depressed? *Journal of the American Medical Association, 287*(9), 1160–1170.

Depression: Symptoms

Ellen-ge Denton
Department of Medicine Center for Behavioral Cardiovascular Health, Columbia University Medical Center, New York, NY, USA

Synonyms

Diagnostic features of depression

Definition

Major depressive disorder (MDD) criteria requires five or more diagnostic features, with either (1) depressed mood or (2) anhedonia being present during the same 2 week period and a change in previous daily functioning.

Diagnostic features include (1) depressed mood (feeling sad or empty); (2) markedly diminished interest or pleasure in almost all activities (anhedonia); (3) weight loss or weight gain (at least 5% body weight change in a month), increased or decreased appetite; (4) insomnia or hypersomnia; (5) feelings of restlessness or feeling slowed down (psychomotor agitation or retardation); (6) fatigue, loss of energy; (7) feelings of worthlessness, excessive or inappropriate guilt; (8) diminished ability to think or concentrate, indecisiveness; and (9) recurring thoughts of death and/or suicide.

Dysthymic disorder or a chronic depressive disorder requires depressed mood, for more days than not, over a period of 2 years, and two or more of the diagnostic features previously mentioned. During the 2-year period, symptoms of depressed mood and the minimum of two diagnostic features are to have never remitted for more than 2 months at a time.

Diagnostic features include (1) poor appetite or overeating, (2) insomnia or hypersomnia, (3) low energy or fatigue, (4) low self-esteem, (5) poor concentration or difficulty making decisions, and (6) feelings of hopelessness.

- Depression symptoms must cause clinically significant distress or interfere with daily functioning (i.e., social, occupational) or daily tasks.
- Depression symptoms are not due to a general medical condition or due to the direct physiological effects of a substance.
- Depression symptoms are not better accounted for by bereavement.

Description

Symptoms of depression can be categorized by a marked change in a person's (1) physical well-being, evidenced by changes in sleep and eating

behaviors; (2) emotional well-being, such as feelings of sadness and/or hopelessness; and (3) thoughts. For example, negative thought patterns. Hallmark symptoms of depression are loss of interest in activities (anhedonia) and a depressed mood (melancholia), as at least one of these symptoms are necessary for MDD diagnostic criteria. Depression symptoms can have catatonic features, melancholic features, atypical features, and postpartum onset.

Depression symptoms may present differently among children and adolescents. Some children may present with mood irritability or a failure to make expected weight gains. Diagnostic duration of symptoms for children and adolescents is typically at least 1 year.

Depression diagnosis is two times more likely among women than men. Women's increased likelihood for depression is found in the general population, across cultural groups and across demographic groups (Bromet et al., 2011). Although studies have not concluded that depression symptoms differ by gender (Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993), some studies suggest men exhibit more externalizing symptoms of depression (angry outbursts, irritability, withdrawal, blunted affect, etc.) while comparatively, women have more melancholic symptoms of depression (sadness, guilt, etc.) (Hatzenbuehler, Hilt, & Nolen-Hoeksema, 2010).

Depressive symptoms are quite common in several biomedical health conditions and have been shown to predict worse prognosis in heart disease (Bush, 2002; Frasure-Smith & Lesperance, 2006), even if at low levels. Because depression is a heterogeneous construct with multidimensional characteristics, the cardiovascular literature has begun to identify depression symptom clusters that are associated with worse coronary heart disease outcome. For example, some authors have found somatic depressive symptoms to be associated with cardiac disease severity (de Jonge et al., 2006; Watkins et al., 2003). A recent study comparing cognitive affective symptoms to somatic affective depressive symptoms found that somatic affective symptoms predicted worse cardiovascular outcome while cognitive affective symptoms did not (Martens, Hoen, Mittelhaeuser, de Jonge, & Denollet, 2009). Somatic affective symptoms include sadness, dissatisfaction, pessimism, suicidal ideas, crying, work difficulty, insomnia, fatigability, loss of appetite, somatic preoccupation, and loss of libido.

References and Readings

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Bromet, E., Andrade, L., Hwang, I., Sampson, N., Alonso, J., de Girolamo, G., et al. (2011). Cross-national epidemiology of DSM-IV major depressive episode. *BMC Medicine, 9*(1), 90.

Bush, D. E. (2002). Cardiac disease and depression in the elderly. *Cardiology in Review, 19*(11), 10–15.

de Jonge, P., Ormel, J., van den Brink, R. H. S., van Melle, J. P., Spijkerman, T. A., Kuijper, A., et al. (2006). Symptom dimensions of depression following myocardial infarction and their relationship with somatic health status and cardiovascular prognosis. *The American Journal of Psychiatry, 163*(1), 138–144.

Frasure-Smith, N., & Lesperance, F. (2006). Depression and coronary artery disease. *Herz, 31*(Suppl. 3), 64–68.

Goldberg, D., Kendler, K. S., Sirovatka, P. J., & Regier, D. A. (2010). *Diagnostic issues in depression and generalized anxiety disorder: Refining the research agenda for DSM-5*. Arlington, VA: American Psychiatric Association.

Hales, R. E., Yudofsky, S. C., & Gabbard, G. O. (2008). *The American psychiatric publishing textbook of psychiatry* (5th ed.). Arlington, VA: American Psychiatric.

Hatzenbuehler, M. L., Hilt, L. M., & Nolen-Hoeksema, S. (2010). Gender, sexual orientation, and vulnerability to depression. In J. C. Chrisler & D. R. McCreary (Eds.), *Handbook of gender research in psychology* (pp. 133–151). New York: Springer.

Kessler, R. C., McGonagle, K. A., Swartz, M., Blazer, D. G., & Nelson, C. B. (1993). Sex and depression in the National Comorbidity Survey I: Lifetime prevalence, chronicity and recurrence. *Journal of Affective Disorders, 29*(2–3), 85–96.

Martens, E. J., Hoen, P. W., Mittelhaeuser, M., de Jonge, P., & Denollet, J. (2009). Symptom dimensions of post-myocardial infarction depression, disease severity and cardiac prognosis. *Psychological Medicine, 40*(05), 807.

Watkins, L. L., Schneiderman, N., Blumenthal, J. A., Sheps, D. S., Catellier, D., Taylor, C. B., et al. (2003). Cognitive and somatic symptoms of depression are associated with medical comorbidity in patients after acute myocardial infarction. *American Heart Journal, 146*(1), 48–54.

Depression: Treatment

Tatsuo Akechi
Department of Psychiatry and Cognitive-Behavioral Medicine, Graduate School of Medical Sciences, Nagoya City University, Nagoya, Aichi, Japan

Synonyms

Management of depression; Pharmacotherapy for depression; Psychotherapy for depression

Definition

Effective treatment methods of patients with depressive disorders

Description

There are several types of treatment for depression, and these are mainly somatotherapy and psychotherapy. Somatotherapy for depression usually includes pharmacotherapy and electroconvulsive therapy (ECT). In addition, other types of somatotherapy including transcranial magnetic stimulation (TMS) can be available now in several countries such as USA (Gelenberg, 2010).

With regard to pharmacotherapy, there are several different types of drugs, so-called antidepressants. Antidepressants include selective serotonin reuptake inhibitors (SSRI), serotonin noradrenaline reuptake inhibitors (SNRI), tricyclic antidepressants (TCA), tetracyclic antidepressants, monoamine oxidase inhibitors (MAOIs), and other types of antidepressant drugs (Gelenberg, 2010; Hales & Yudofsky, 2003; Sadock & Sadock, 2003). The effectiveness of these antidepressants is generally comparable between classes and within classes of medications. On the other hand, side effect profiles clearly differ among the different classes of antidepressants. Pharmacotherapy is most widely used for treatment of depression. Especially pharmacotherapy is recommended as an initial treatment choice for patients with mild to moderate major depressive disorder as defined by DSM-IV-TR. The choice of each antidepressant is usually determined by anticipated side effects and safety for the individual patient. In general, the SSRIs and other newer antidepressants are better tolerated and safer than either TCAs or the MAOIs, although many patients still benefit from older drugs including TCAs. During pharmacotherapy, patients should be carefully and regularly monitored to evaluate side effects. Overall, approximately two-thirds of the patients with major depression respond to an adequate trial of antidepressant medication. However, far few achieve full remission of symptoms.

ECT is recommended as a treatment of choice for patients with severe major depressive disorder and those with psychotic features. Other cases such as a suicidal patient with an urgent need for response can also be appropriate for ECT treatment. ECT has the highest response and remission rates among any antidepressant treatment. ECT is generally provided 2–3 times per week and total of 6–12 treatments. ECT is a safe treatment, and it is suggested that risks of morbidity and mortality do not exceed those associated with anesthesia alone. Side effects of ECT include short-time confusion, memory impairment, headache, muscle aches, and so on. ECT is the use of electrically induced repetitive firings of the neurons in the CNS. The mechanisms of action of ECT are complex and not completely understood.

TMS was approved for use in patients with major depressive disorder in USA. TMS uses a magnetic field to stimulate or inhibit cortical neurons. Because the area of cortex stimulated is related to placement of the coil on the skull, the coil is most often placed over the left dorsolateral prefrontal cortex for treatment of depression. There are few findings regarding long-term follow-up data of TMS treatment effect. So more longer-term data and further refinement of TMS are needed.

Regarding psychotherapy, cognitive-behavioral therapy (CBT) and interpersonal psychotherapy (IPT) are most well-known and proven psychotherapeutic approaches for patients with depressive

disorders (Gelenberg, 2010). CBT combines cognitive psychotherapy with behavioral therapy, including behavioral activation, and its goal is to reduce depressive symptoms by challenging and reversing irrational beliefs and distorted attitudes and encouraging patients to change their maladaptive preconceptions and behaviors in real life. On the other hand, IPT focus on interpersonal factors that may interact with the development of depressive disorders. The goal of IPT is to intervene by identifying the trigger of depression, facilitating mourning in the case of bereavement, promoting recognition of related affects, resolving role disputes and role transitions, and building social skills to improve relationships and to acquire needed social supports. Although these psychotherapies are recommended as an initial treatment choice for patients with mild to moderate major depressive disorder, these should be used in combination with pharmacotherapy for severe major depressive disorder.

Treatments of depression generally include several different steps, and these are acute phase treatment, continuous phase treatment, and maintenance phase treatment. Primary aims of the acute phase treatment are to improve symptoms of depression and achieve a full return to the patient's functioning. Continuous phase treatments are mainly provided to reduce the risk of relapse for a patient who has been successfully treated. Regarding patients who have had multiple major depressive episodes or who have chronic features, maintenance phase treatment should be considered in order to reduce the risk of a recurrent depressive episode.

Cross-References

- ▶ Antidepressant Medications
- ▶ Cognitive Behavioral Therapy (CBT)
- ▶ Psychoeducation
- ▶ Social Support

References and Readings

Gelenberg, A. J. (2010). *Practice guideline for the treatment of patients with major depressive disorder* (American journal of psychiatry, 3rd ed., Suppl. 167, pp. 1–118). Washington, DC: American Psychiatric Association.

Hales, R. E., & Yudofsky, S. C. (2003). *Textbook of clinical psychiatry* (4th ed.). Washington, DC: The American Psychiatric Publishing.

Sadock, B. J., & Sadock, V. A. (2003). *Kaplan & Sadock's synopsis of psychiatry* (9th ed.). Philadelphia: Lippincott Williams & Wilkins.

Descriptive Data

- ▶ Aggregate Data

Developmental Disabilities

Monica Dowling
Miller School of Medicine, University of Miami, Miami, FL, USA

Synonyms

Autism spectrum disorders; Intellectual disability

Definition

Developmental disabilities (DD) is an umbrella term for a group of interrelated, chronic, nonprogressive, neurological, or brain-based disorders which are defined as "severe, life-long disabilities attributable to mental and/or physical impairments, manifested before age 22, that result in substantial limitations in three or more areas of major life activities: capacity for independent living, economic self-sufficiency, learning, mobility, receptive and expressive language, self-care, self-direction" (Administration on Developmental Disabilities, 2007). In the case of young children, DD is likely to result in significant limitations. Low IQ scores are typically associated with DD, and individuals with intellectual disabilities comprise the largest group considered to have DD (Larson et al., 2000), but other conditions may impose functional limitations on individuals whose intelligence is at or

above average. Sensory impairments are included only as occurring in combination with impairment in intellectual and adaptive functioning. The current conceptualization includes diagnostic classifications of intellectual disability (formerly mental retardation), autism spectrum disorders (ASD), cerebral palsy (CP), and specific syndromes whose behavioral phenotype includes limitations in intellectual and adaptive functioning (e.g., fragile X, trisomies, Prader-Willi, Smith-Magenis) but does not specify an etiology or medical diagnosis.

Description

Epidemiology

Researchers from the CDC estimate the prevalence of DD by tracking five conditions: intellectual disability (ID), autism spectrum disorders (ASD), cerebral palsy (CP), hearing loss, and vision impairment. The essential features of ASD are impaired reciprocal social interactions, delayed or unusual communication styles, and restricted or repetitive behavior patterns. ID is defined as a condition marked by an IQ < 70 with concurrent limitations in adaptive functioning, previously referred to as mental retardation. Cerebral palsy is defined as a group of nonprogressive, but often changing, motor impairment syndromes secondary to brain lesions/anomalies arising at any time during brain development or as a result of neonatal insult.

Developmental disabilities affect approximately 17% of children younger than 18 years of age in the USA (Bhasin, Brocksen, Avchen, & Van Naarden, 2006). In 2006–2008 nearly 10 million children aged 3–17 had a developmental disability on the basis of parent report (Boyle et al., 2011). The most recent CDC prevalence estimates of ASD are 1 in 88 children (Baio, 2012). Population-based estimates of functional limitations and health services utilization among children with DD were 4–32 times higher than for children without DD (Boulet, Boyle, & Schieve, 2009), while the cost to society of ASD alone is estimated to be $35–$90 billion annually (Ganz, 2007). The CDC estimates that the average lifetime cost associated with intellectual disability (IQ < 70) is approximately $1,014,000 per person (in 2003 dollars).

Etiology

The likelihood of identifying an underlying etiology increases with the degree of disability. Prenatal causes include *genetic abnormalities* including chromosomal abnormalities (e.g., trisomies, X-linked, microdeletions, and subtelomeric rearrangements), single gene disorders (e.g., X-linked recessive conditions), and multifactorial/polygenic conditions (e.g., spina bifida); *congenital infections* (e.g., rubella, syphilis); alcohol and other drug or teratogen exposure; and maternal disorders. Perinatal causes include *placental complications, preeclampsia/eclampsia, birth trauma/anoxia, complications of prematurity* (e.g., IVH), *infections* (e.g., bacterial meningitis), and *metabolic abnormalities*. Postnatal causes include *infections, trauma, environmental pollutants/neurotoxins, malnutrition*, and *inborn errors of metabolism* (e.g., PKU).

Genetic disorders now account for approximately 55% of moderate to severe ID (IQ < 50) and 10–15% of mild ID (IQ 50–70), and these percentages continue to increase with the use of new molecular techniques. More than 1,000 genetic disorders leading to developmental disabilities have been identified, many with active research programs (Tartaglia, Hansen, & Hagerman, 2007). Fragile X syndrome (FXS), the most common form of inherited ID, is caused by a mutation in a single gene (FMRP1) on the X chromosome, resulting from expansions of cytosine-guanine-guanine (CGG) repeats, which interferes with the normal transcription of a single protein (FMRP). Other disorders, such as Smith-Magenis or velocardiofacial syndrome (also known as 22q11.2 deletion syndrome), are microdeletion syndromes. Prader-Willi and Angelman syndromes are both the result of deletions on the same chromosome (15), but the expression is related to inheritance from either the father (Prader-Willi) or mother (Angelman). Still other disorders are characterized by the addition or absence of an entire chromosome (e.g., Down syndrome or trisomy 21; Klinefelter or

47, XXY; Turner or 45, X), leading to overexpression or imbalance of many genes and subsequent abnormalities.

Diagnosis

The American Academy of Pediatrics has recommended that developmental surveillance be incorporated into every well-child visit and that any concerns should be promptly addressed with standardized developmental screening tests (AAP, 2006). In addition, screening tests should be administered regularly at the 9-, 18-, and 24-month visits, including ASD specific measures. There is no universally accepted screening tool appropriate for all populations and all ages. However, accurate, cost-effective, and parent-friendly questionnaires are available for ages 1 month to 5 1/2 years in multiple languages (e.g., Ages and Stages Questionnaires, third Ed., Brookes Publishing). In addition, tools such as the M-CHAT and a follow-up interview used to screen for ASD are available, at no cost (www.firstsigns.com), covering a range of ages (e.g., 16–48 months) and in many languages. Once identified as being at risk, diagnostic developmental and medical evaluations should be pursued, typically involving pediatric subspecialists and using valid and reliable measures of cognition, adaptive behavior, communication, social, and neuropsychological functioning.

Intervention/Current Best Practices

Prevention has focused on *educational initiatives* to eliminate or minimize risk factors such as smoking and alcohol use during pregnancy or lead and mercury exposure as well as *medical initiatives* such prenatal screening and treatment for infectious disease (e.g., syphilis, CMV), genetic screening and counseling for carriers of genetic disorders, and the use of vaccines to prevent maternal or child infections (e.g., rubella, meningitis) (Brosco, Mattingly, & Sanders, 2006). In addition, early identification and treatment (e.g., newborn screening for genetic and metabolic disorders and fetal alcohol syndrome) have been successful in limiting the impact of severe developmental disabilities (Powell et al., 2010; Bertrand, 2009) and pilot programs using a variety of assays using urine or blood are under way (e.g., cytomegalovirus). Even late treatment has been successful in partially reversing the severe cognitive impact associated with metabolic disorders such as untreated PKU (Grosse, 2010).

Emerging areas of knowledge that influence practice include targeted pharmacological and evidence-based treatments for specific disorders such as fragile X and ASD, as well as a growing body of clinical guidelines specific to conditions in the pediatric age range. Evidence-based comprehensive treatment programs for young children with ASD emphasize behavioral- and/or development-based models. For example, the UCLA Young Autism Project, the Princeton Child Development Institute, and the Douglass Developmental Disabilities Center utilize traditional behavioral interventions(e.g., discrete trial training). The Learning Experiences and Alternative Program for Preschoolers and their Parents (LEAP) and the Walden Early Childhood Program utilize behavioral interventions in naturalistic settings and incidental teaching. Division TEACCH incorporates both behavioral and developmental approaches, while the Denver Early Start Model has a developmental orientation. In addition, care consideration guidelines are currently being developed for fragile X and other disorders having unique behavioral phenotypes (e.g., velocardiofacial, Smith-Magenis), and as understanding of the underlying mechanisms advances, targeted treatment studies are under way that may eventually reverse the neurodevelopmental abnormalities (e.g., medications that regulate the activity of the mGluR5 pathway in fragile X). At this time, best practice includes the need for intensive, multidisciplinary treatment programs for individuals with developmental disabilities and their families that focus on strengths and include medical, behavioral, educational, and therapeutic interventions.

References and Readings

Accardo, P. J. (Ed.). (2008). *Capute and Accardo's neurodevelopmental disabilities in infancy and childhood* (3rd ed.). Baltimore: Brookes.

Administration on Developmental Disabilities. (2007). *What are developmental disabilities?* Washington, DC: Administration on Developmental Disabilities. Retrieved December 28, 2010, from www.acf.hhs.gov/opa/fact_sheets/add_factsheet.html

American Academy of Pediatrics, Council on Children with Disabilities. (2006). Identifying infants and young children with developmental disorders in the medical home: An algorithm for developmental surveillance and screening. *Pediatrics, 118*(1), 405–420.

Bertrand, J. (2009). Interventions for children with fetal alcohol spectrum disorders (FASDs): Overview of findings for five innovative research projects. *Research in Developmental Disabilities, 30*(5), 986–1006.

Bhasin, T. K., Brocksen, S., Avchen, R. N., & Van Naarden, B. K. (2006, January). Prevalence of four developmental disabilities among children aged 8 years: Metropolitan Atlanta Developmental Disabilities Surveillance Program, 1996 and 2000. *MMWR Surveillance Summaries, 55*(SS01), 1.

Boulet, S., Boyle, C. A., & Schieve, L. A. (2009). Health care use and health and functional impact of developmental disabilities among US children, 1997–2005. *Archives of Pediatrics & Adolescent Medicine, 163*(1), 19–26.

Boyle, C. A., Boulet, S., Schieve, L. A., Cohen, R. H., Blumberg, S. J., Yeargin-Allsopp, M., Visser, S., & Kogan, M. D. (2011). Trends in the prevalence of deveoplmental disabilities in US children, 1997–2008. *Pediatrics. 127*(6), 1034–1042.

Brosco, J. P., Mattingly, M., & Sanders, L. M. (2006). Impact of specific medical interventions on reducing the prevalence of mental retardation. *Archives of Pediatrics & Adolescent Medicine, 160*(3), 302–309.

Ganz, M. L. (2007). The lifetime distribution of the incremental societal costs of autism. *Archives of Pediatrics & Adolescent Medicine, 161*(4), 343–349.

Gross, S. D. (2010). Late-treated phenylketonuria and partial reversibility of intellectual impairment. *Child Development, 81*(1), 200–211.

Larson, S., Lakin, C., Anderson, L., Kwak, N., Lee, J. H., & Anderson, D. (2000). Prevalence of mental retardation and/or developmental disabilities: Analysis of the 1994/1995 NHIS-D. *MR/DD Data Brief, 2*(1), 1–11.

Powell, K., Van Naarden Braun, K., Singh, R., Shapira, S. K., Olney, R. S., & Yeargin-Allsopp, M. (2010). Prevalence of developmental disabilities and receipt of special education services among children with an inborn error of metabolism. *Journal of Pediatrics, 156*(3), 420–426.

Baio, J. (2012, March). Prevalence of autism spectrum disorders-Autism and Developmental Disabilities Monitoring Network, United States, 2008. *MMWR Surveillance Summaries, 61*(SS03), 1–19.

Tartaglia, N. R., Hansen, R. L., & Hagerman, R. J. (2007). Advances in Genetics. In S. L. Odom, R. H. Horner, M. E. Snell, & J. Blacher (Eds.), *Handbook of developmental disabilities* (pp. 98–128). New York: Guilford Press.

Developmental Psychology

▶ Child Development

Deviance

▶ Stigma

Dex Suppression Test

▶ Dexamethasone Suppression Test

Dex Test

▶ Dexamethasone Suppression Test

Dexamethasone Suppression Test

Brigitte M. Kudielka
Department of Medical Psychology & Psychological Diagnostics, University of Regensburg, Regensburg, Germany

Synonyms

Dex suppression test; Dex test; DST; HPA axis negative feedback testing

Definition

Dexamethasone is a synthetic glucocorticoid and acts as a ligand of glucocorticoid receptors. The principle of the dexamethasone suppression test (DST) is based on this binding capability. Via receptor binding, dexamethasone exerts a negative feedback function on the hypothalamus-pituitary-adrenal (HPA) axis (de Kloet, Vreugdenhil, Oitzl, & Joels, 1998). The HPA axis is a hierarchical hormonal system encompassing the hypothalamus, the pituitary gland, and the adrenal cortex with their respective hormones CRH (corticotropin releasing hormone), ACTH (adrenocorticotropin hormone), and cortisol. Beside its role in stress regulation, the HPA axis is vital for supporting normal physiological functioning. Its functioning is controlled by several negative feedback loops. Generally, the DST is applied as a standard diagnostic tool to assess feedback sensitivity of the HPA axis in clinical settings (e.g., in major depression, posttraumatic stress disorder, etc.) as well as in psychoneuroendocrinological research (e.g., in stress research) (Bellingrath, Weigl, & Kudielka, 2008; Yehuda et al., 1993).

The dexamethasone suppression test normally consists of the oral intake of a single dose of dexamethasone (see below) which then leads to the suppression of ACTH at the level of the pituitary and subsequently to a reduced cortisol secretion from the adrenal cortex. Of course, dexamethasone can also be applied intravenously. In contrast to endogenous glucocorticoids like cortisol, dexamethasone primarily acts at the level of the pituitary (due to the blood-brain barrier). Selected doses of dexamethasone vary depending on the aim of the diagnostic test, the tested population, or the given research question. A typical dose to identify individuals with increased cortisol suppression after dexamethasone intake (indicating increased negative feedback sensitivity) is the application of 0.5 mg of dexamethasone. Oral intake normally takes place at 11 pm and cortisol measurements are repeatedly performed during the following morning or day to account for the normal circadian rhythm of cortisol (and ACTH) with highest levels in the morning and decreasing levels over the remainder of the day (except for stress-related superimposed hormone surges). After ingestion of a standard dose of 1 mg of dexamethasone, an almost complete suppression of the cortisol secretion in healthy individuals can be expected. That means, cortisol levels are typically suppressed to concentrations less than 5 μg/dl until the following afternoon after the intake of 1 mg dexamethasone the night before. However, in the normal population up to 10% non-suppressors can be identified. Fortunately, there are virtually no side effects reported for a single dexamethasone intake of 1–2 mg of dexamethasone. In different patient groups, either no cortisol suppression (e.g., in Cushing's disease) or cortisol super-suppression (e.g., in some patients suffering from posttraumatic stress disorder) can be observed. For the diagnosis of Cushing's disease, dexamethasone doses of up to 8 mg are applied. However, it is of note here that other tests need to complement a definite diagnosis of Cushing's disease.

In recent years, a much lower dexamethasone dosage of 0.25 mg was suggested (mainly for research purposes) to increase the sensitivity of the DST, called low-dose DST (Cole, Kim, Kalman, & Spencer, 2000; Yehuda, et al., 1993). This version of the test is advantageous if subtle differences are to be detected in HPA axis negative feedback sensitivity in apparently healthy individuals (Bellingrath et al., 2008). After intake of 0.25 mg of dexamethasone, endogenous cortisol concentrations of about 5 μg/dl can normally be expected.

One should be aware of the fact that an unequivocal interpretation of DST results should additionally account for the following two issues: Since the exact amount of circulating dexamethasone is dependent on metabolic functioning, bioavailability of dexamethasone should be controlled for, especially in certain patient groups with known metabolic dysfunctions. Furthermore, in order to rule out altered reactivity of the adrenal cortex to ACTH signals, one should want to additionally check the extent of ACTH suppression.

Finally, the DST can also be combined with other pharmacological provocation tests like the CRH stimulation tests (Heuser, Yassouridis, & Holsboer, 1994). Over the last decades, the combined Dex-CRH test applying a premedication of 1.5 mg dexamethasone the night before followed by a CRH application (e.g., 100 μg or 1 μg/kg body weight) the following afternoon proved its usefulness especially for the assessment of HPA axis feedback regulation in psychiatric disorders.

Cross-References

- ▶ ACTH
- ▶ Adrenal Glands
- ▶ Adrenocorticotropin
- ▶ Corticotropin-Releasing Hormone (CRH)
- ▶ Cortisol
- ▶ Depression
- ▶ Endocrinology
- ▶ Glucocorticoids
- ▶ Hypothalamic-Pituitary-Adrenal Axis
- ▶ Pituitary-Adrenal Axis
- ▶ Stress

References and Readings

Bellingrath, S., Weigl, T., & Kudielka, B. M. (2008). Cortisol dysregulation in school teachers in relation to burnout, vital exhaustion, and effort-reward-imbalance. *Biological Psychology, 78*(1), 104–113.

Cole, M. A., Kim, P. J., Kalman, B. A., & Spencer, R. L. (2000). Dexamethasone suppression of corticosteroid secretion: evaluation of the site of action by receptor measures and functional studies. *Psychoneuroendocrinology, 25*(2), 151–167.

de Kloet, E. R., Vreugdenhil, E., Oitzl, M. S., & Joels, M. (1998). Brain corticosteroid receptor balance in health and disease. *Endocrine Reviews, 19*(3), 269–301.

Heuser, I., Yassouridis, A., & Holsboer, F. (1994). The combined dexamethasone/CRH test: A refined laboratory test for psychiatric disorders. *Journal of Psychiatric Research, 28*(4), 341–356.

Yehuda, R., Southwick, S. M., Krystal, J. H., Bremner, D., Charney, D. S., & Mason, J. W. (1993). Enhanced suppression of cortisol following dexamethasone administration in posttraumatic stress disorder. *The American Journal of Psychiatry, 150*(1), 83–86.

DHA

- ▶ Omega-3 Fatty Acids

Diabesity in Children

Francine Kaufman
Medtronic, Northridge, CA, USA

Synonyms

Childhood obesity; Obesity

Definition

The association of obesity and diabetes has been recently referred to as diabesity.

Description

In 2000, then Surgeon General David Satcher announced that the epidemic of obesity in the United States was increasingly affecting children and adolescents (Satcher, 2001). At the time of the surgeon general's announcement, approximately 16% of youth were categorized as being obese, defined as a body mass index (BMI) greater than the 95th percentile for age and gender. Overall, that same childhood obesity rate persists in the USA today; however, minority children have significantly higher rates. Data from the HEALTHY study in middle school–aged children showed that approximately 50% of sixth grade students in middle schools with a predominate minority population were overweight or obese (BMI $\geq$ 85th percentile for age and gender) (HEALTHY, HEALTHY Study Group, 2010).

Concomitant with the rise in childhood obesity is a corresponding increase in the incidence of the metabolic syndrome and type 2 diabetes in pediatric subjects. Before the 1990s, it was rare for most pediatric centers to have patients with

type 2 diabetes. By 1994, type 2 diabetes patients represented up to 16% of new cases of diabetes in children in urban areas, and by 1999, depending on geographic location, the range of percentage of new cases of type 2 was between 8% and 45% (HEALTHY, HEALTHY Study Group, 2010). The SEARCH study showed that after age 10 years, of all American Indian children who have diabetes, two thirds have type 2; of all Hispanic and African American children with diabetes, approximately a third have type 2; and only 8% of non-Hispanic White children affected with the disease have type 2 (SEARCH, 2006). Therefore, type 2 diabetes occurs mainly in ethnic minorities in the United States, as has been described in children in a number of countries throughout the world.

A period of prediabetes, defined as either elevated fasting glucose levels, impaired glucose tolerance, and/or elevated A1C (5.7– $<$ 6.4%), occurs before the development of frank type 2 diabetes. Type 2 diabetes in children and youth, as in adults, is caused by the combination of insulin resistance and relative B cell secretary failure. Plasma insulin concentrations appear normal or elevated, but there is a loss of first-phase insulin secretion that cannot compensate for underlying insulin resistance. There are a number of genetic and environmental risk factors for insulin resistance and limited B cell reserve, including ethnicity, obesity, sedentary behavior, family history of type 2 diabetes, puberty, high and low birth weight, and female gender. Family education level, SES, maternal diabetes or excessive weight gain, failure to breast feed, and exposure to an obesogenic environment are additional risk factors.

Type 2 diabetes in pediatric subjects has a variable presentation, although many children present with symptoms caused by elevated glucose. There is an associated increase in A1C $\geq$6.4% which can be used to make the diagnosis. Few pediatric subjects with type 2 diabetes can be treated with diet and exercise alone; therefore, pharmacologic therapy is most often required. Depending on initial glucose levels and the degree of symptoms caused by hyperglycemia, practitioners usually prescribe metformin for those subjects who are mildly affected, or subjects begin insulin therapy if they have more significant hyperglycemia. Relatively few pediatric subjects use other or combination therapy. Unfortunately, while they are undergoing the present pharmacologic regimens, many patients appear unable to achieve glycemic targets over the long term. Specific treatment algorithms for pediatric patients with type 2 diabetes that are aimed at achieving glycemic targets have not been investigated in youth. The ongoing Treatment Options for type 2 Diabetes in Adolescents and Youth (TODAY) trial, sponsored by the National Institutes of Health, has investigated best treatments for type 2 diabetes in pediatric subjects and will provide evidence for improving the outcome of pediatric type 2 diabetes.

The term diabesity was coined to raise awareness about the adverse health effects of obesity. Today, obesity and diabetes has become a healthcare crisis of epidemic proportions. It is a given that individuals and families must change their behavior if we are to reverse the present trends. But they cannot do it on their own. Reversing the trends will require the coordinated efforts from local, state, and national governments; public and private industries; community and religious organizations; schools; and the health-care system. Information must be provided, social norms must change, and, most importantly, an environment that supports healthy lifestyles must be created. Only then will the childhood diabesity epidemic be reversed.

Cross-References

▶ Obesity in Children
▶ Type 2 Diabetes Mellitus

References and Readings

HEALTHY, HEALTHY Study Group. (2010). A school-based intervention for diabetes risk reduction. *The New England Journal of Medicine, 363*, 443–453.

Kaufman, F. R. (2005). Type 2 diabetes in children and youth. *Endocrinology and Metabolism Clinics of North America, 34*, 659–676.

Satcher, D. (2001). *The Surgeon General's call to action to prevent and decrease overweight and obesity*. Rockville, MD: Public Health Service, Office of the Surgeon General, United States Department of Health and Human Services. Available at http://www.surgeongeneral.gov/topics/obesity/calltoaction/CalltoAction.pdf

SEARCH Study Group: The Burden of Diabetes Mellitus Among U.S. Youth. (2006). Prevalence estimates from the SEARCH for Diabetes in Youth Study. *Pediatrics, 118*, 1510–1518.

Diabetes

Luigi Meneghini
Diabetes Research Institute, University of Miami, Miami, FL, USA

Synonyms

Hyperglycemia

Definition

Diabetes (mellitus) is defined as elevated blood glucose levels (hyperglycemia), which over time can lead to chronic microvascular complications such as diabetic retinopathy, nephropathy, and neuropathy. Diabetes is caused by deficiency in insulin production (type 1 diabetes), which in many cases can be accompanied by increased insulin demand, also known as insulin resistance (type 2 diabetes). Type 2 is the most common form of diabetes accounting for 85–90% of all cases. Diabetes can be diagnosed by way of a fasting plasma glucose ($\geq$126 mg/dl), an elevated HbA1c ($\geq$6.5%) or an oral glucose tolerance test (2-h postchallenge plasma glucose $\geq$200 mg/dl). Alternatively, someone presenting with symptoms of hyperglycemia, such as excessive thirst or urination, blurring of vision, or weight loss, combined with a random plasma glucose of $\geq$200 mg/dl, can also be diagnosed with diabetes.

Control of hyperglycemia is essential to reduce the risk of chronic microvascular complications associated with diabetes. This can be done through adopting healthy lifestyle and dietary habits, the use of oral medications to lower blood glucose, and/or the use of insulin replacement therapy. Diabetes is often a familial disease with genetic and environmental predisposing factors. First-degree relatives of individuals with diabetes have an approximate fivefold to tenfold increase in the risk of developing diabetes compared to the general population.

While diabetes mellitus ("sweet siphon") refers to the more common form of diabetes, characterized by hyperglycemia, diabetes insipidus ("bland or tasteless siphon") refers to an inability to retain free water due to deficiencies in the production or action of antidiuretic hormone (vasopressin).

References and Readings

Joslin, E. P., & Kahn, C. R. (2005). *Joslin's diabetes mellitus* (14th ed.). Philadelphia: Lippincott Williams & Willkins.

Diabetes Education

Luigi Meneghini
Diabetes Research Institute, University of Miami, Miami, FL, USA

Synonyms

Patient education; Self-management education

Definition

Diabetes education is the process of enabling patients with diabetes to gain knowledge, problem-solving skills, empowerment, and ability to manage their condition through the application of appropriate treatments and lifestyle intervention.

Description

Diabetes is a chronic condition whose management and control is highly dependent on both appropriate treatment prescriptions and patient implementation of those recommendations. Proper diabetes management requires a high degree of involvement on the part of the patient if it is to be successful. Education for patients with diabetes requires transfer of knowledge, as well as problem-solving skills, that incorporates the needs, goals, and experiences of patients with diabetes, with the ultimate objective of supporting "informed decision making, self-care behaviors, problem solving, and active collaboration with the health-care team." The implementation of appropriate treatment prescriptions and self-management behaviors needs to ultimately lead to improved clinical outcomes, health, and quality of life.

Diabetes self-management standards have been modified over the years to incorporate evidence-based recommendations. Guiding principles for these standards, which need to be adhered to by any entity seeking accreditation for their educational activities, dictate that diabetes education should improve clinical outcomes and quality of life, incorporate empowerment models, and individualize the educational approach to include behavioral and psychological strategies that are culturally and age appropriate. Group education, ongoing reassessment and education, and behavioral goal setting are critical elements to successful patient education programs. The American Diabetes Association has published specific standards for diabetes self-management education (DSME) that address several areas. DSME programs need a clear organizational structure, mission statement, and goals that are overseen by an advisory group, which include representatives from health professionals, patients, community, and other stakeholders. The DSME programs need to determine the specific educational needs of their target populations and identify appropriate resources to meet those needs; they must designate a professional coordinator to manage the program. Professional and experienced educators (nurse, dietitian, pharmacist, etc.) develop and implement educational interventions based on a curriculum that relies on current evidence and practice guidelines. Patients need to receive a documented individual assessment and education plan, with a personalized follow-up plan for ongoing education and support. Measurement of patient-defined goals forms the basis for patient reassessment and ongoing support and education. The DSME also needs to measure and document the effectiveness of the education process and use this opportunity for continuous quality improvement.

Cross-References

▶ Patient Education
▶ Self-management Education

References and Readings

Funnell, M. M., Brown, T. L., Childs, B. P., Haas, L. B., Hosey, G. M., Jensen, B., et al. (2011). National standards for diabetes self-management education. *Diabetes Care, 34*(Suppl 1), S89–S96.

Diabetes Foot Care

Kathleen Michael
School of Nursing, University of Maryland, Baltimore, MD, USA

Synonyms

Diabetic foot care

Definition

Routine care of the feet for individuals who have diabetes, including inspection, skin and nail care, and prevention of injury.

Description

Diabetes may cause nerve damage that affects feeling in the feet. Diabetes may also reduce blood flow to the feet, making it harder to heal injuries or to resist infection.

Most people with diabetes can prevent serious foot problems by taking some simple actions. Routine foot care should include an annual foot exam by a healthcare provider, or more often if foot problems are present. The exam includes evaluation for injuries or breaks in the skin, nail problems, pain, sensitivity, or changes in foot shape or skin color. In some cases, healthcare providers may recommend specially fitted shoes.

Individuals with diabetes should inspect their feet every day, looking for red spots, cuts, swelling, or blisters. They should wash feet daily and apply moisturizing lotion to tops and bottoms of feet, but not between toes. Toenails should be trimmed straight across and filed if they can be easily seen and reached, otherwise a foot care specialist should trim nails. Shoes and socks should be worn at all times to prevent injury to the feet. Other self-care measures to prevent foot problems include keeping blood glucose levels controlled, not smoking, and avoiding sitting and crossing legs for prolonged periods. Increased activity may promote foot health along with other overall benefits to the cardiovascular system.

Cross-References

► Preventive Care

Diabetes Prevention Program

David G. Marrero
Diabetes Translational Research Center, Indiana University School of Medicine, Indianapolis, IN, USA

Synonyms

Type 2 diabetes prevention

Definition

There are several factors that increase a person's risk for developing type 2 diabetes mellitus. These include increased age, a family history of diabetes, race (persons of color having greater risk), obesity, body fat distribution, physical inactivity, and evidence of a metabolic defect as measured by either elevated fasting glucose, impaired glucose tolerance, or elevated glycosylated hemoglobin A1c. Because many of these risk factors are modifiable, notably obesity and activity patterns, it should be possible to reduce risk by interventions designed to help high risk persons reduce weight and increase their levels of physical activity. There is increasing evidence that this is indeed the case. In 1997, the Chinese first reported that lifestyle intervention in persons with impaired glucose tolerance (IGT) resulted in a significant reduction in the incidence of diabetes, with a 40% reduction occurring over a 6-year period (Pan et al., 1997). In 2001, the Finns reported that lifestyle intervention in persons with IGT resulted in a 58% reduction in 3-year diabetes incidence (Tuomilehto et al., 2001), and in 2002, the American Diabetes Prevention Program (DPP) study reported an identical 3-year reduction in diabetes incidence (Knowler et al., 2002). This entry focuses on the DPP.

Description

The DPP was a three-group randomized clinical trial that was conducted in 27 centers across the United States. The 3,234 subjects were all 25 years of age or older, had IGT, and a body mass index (BMI) of at least 24 kg/m^2. All ethnic groups were represented with 45% of the cohort being African American, Hispanic American, American Indian, or Asian/Pacific Islander. In addition, 68% of the cohort was women, 31% between the ages of 25–44, 49% between 45 and 59, and 20% 60 and above. Subjects were randomly assigned to a medication condition (using metformin), a medication placebo control condition, or a lifestyle intervention. The lifestyle

intervention was an intensive program with very specific goals: a minimum of 7% loss of body weight and maintenance of this weight loss through the course of the trial and a minimum of 150 min per week of physical activity with brisk walking being the standard.

The lifestyle intervention was a 16-session core curriculum implemented over 24 weeks to account for holidays and regionally defined special events. Each session was taught by a lifestyle coach who worked with the subject one on one. In addition, subjects had access to a dietitian, a behaviorist, and exercise physiologist if they so elected. Frequent contact with the lifestyle coach and support staff was the norm with most subjects following a weekly meeting schedule (The Diabetes Prevention Program (DPP) Research Group, 2002).

The intervention provided education and training in diet and exercise methods and behavior modification skills. Emphasis was placed on the use of self-monitoring techniques to assess dietary intake and diet composition, active problem solving to reduce the impact of personal and social cues to eat in ways counterproductive to achieving weight goals, and building self esteem, empowerment, and social support to reinforce lifestyle modifications. The intervention was individualized to address social and cultural factors that impact eating behavior, and a long-term maintenance program was introduced following the core curriculum.

The intervention was successful in reducing the risk for developing type 2 diabetes by 58%. Subjects in the lifestyle condition lost an average of 7 kg following the core curriculum and maintained a negative weight loss with an average loss of approximately 4 kg and 36 months postcore. In addition, 74% of the subjects in the lifestyle condition achieved the minimum study goal of 150 min of physical activity per week with the mean activity level at the end of the core curriculum being 224 min per week. Importantly, the intervention was effective for all participants, regardless of race, age, or gender.

As noted above, this is the same percentage of risk reduction obtained by the Finns. It is exceedingly rare in the annals of human clinical trials that two independent studies conducted on separate continents would report identical findings. A reasonable assumption is that the Finnish and American trials used identical lifestyle interventions (both were delivered to individual participants rather than in group sessions). However, they were quite different with the American trial being substantially more intensive. In the Finnish trial, each participant in the lifestyle intervention group had seven sessions with a nutritionist during the first year of the study and one session every 3 months thereafter (Tuomilehto et al., 2001).

It is tempting to conclude that the American approach to lifestyle intervention was less efficient than that used by the Finns, but there are differences between the Finnish and American participants worth noting. The mean body mass index (BMI; kg/m^2) in the Finnish sample was about 31, and in the American sample it was about 34, suggesting that the Americans were 9–10 kg heavier, on average, than the Finns. The DPP cohort was heterogeneous in terms of age and race/ethnicity whereas the Finns studied a fairly homogenous population. In addition, because of local environmental and cultural differences between Finland and the USA, it is likely there were fewer opportunities for physical activity for American participants than for Finns.

The prevention of type 2 diabetes is clearly a behavioral issue that involves implementing interventions designed to modify eating and physical activity behaviors. Future efforts need to consider how to translate efficacy studies such as those reviewed here into the broader public health. Such efforts need to involve behavioral scientists in the design of these interventions.

Cross-References

▶ Diabetes Education
▶ Type 2 Diabetes

References and Readings

Knowler, W. C., Barrett-Connor, E., Fowler, S. E., Hamman, R. F., Lachin, J. M., Walker, E. A., et al. (2002). Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *The New England Journal of Medicine, 346*, 393–403.

Pan, X. R., Li, G. W., Hu, Y. H., Wang, J. X., Yang, W. Y., An, Z. X., et al. (1997). Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and Diabetes Study. *Diabetes Care, 20*, 537–544.

The Diabetes Prevention Program (DPP) Research Group. (2002). The Diabetes Prevention Program (DPP): Description of lifestyle intervention. *Diabetes Care, 25*, 2165–2171.

Tuomilehto, J., Lindström, J., Eriksson, J. G., Valle, T. T., Ämalainen, H., Lanne-Parikka, P., et al. (2001). Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *The New England Journal of Medicine, 344*, 1343–1350.

Diabetes: Psychosocial Factors

Maartje de Wit
Medical Psychology, VU University Medical Center, Amsterdam, North Holland, The Netherlands

Definition

Diabetes psychosocial factors are those factors associated with the psychological and social well-being of people with diabetes, as well as how those factors are related to diabetes-related self-management behaviors and glycemic control.

Description

The daily self-care of patients with diabetes mellitus type 1 or 2 is crucial for achieving blood glucose targets. Self-management is the foundation of diabetes treatment. A good understanding of the changes and challenges faced by people with diabetes is therefore essential in guiding these patients. We should remember that despite medication and improvements in administration systems, over one third of patients have long-term poorly controlled diabetes and thus a greatly increased risk of micro- and macro-vascular complications (Harris, 2000; Writing Team for the Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications Research Group, 2002). Psychological and social factors play an important role in the self-management of diabetes. This involves more than just knowledge of the patient. Research on self-care of diabetes patients shows that especially perceptions, attitudes, emotions, and social support are important in the process of behavior change.

Adaptation and Self-management

Diabetes is a chronic disease that puts specific demands on the daily life of patients. The most important task is keeping blood glucose values within normal limits in different situations. This requires the patient to be always aware of the effects of diet, physical activity, and glucose-lowering medication. Patients using insulin are advised to monitor their blood glucose levels frequently, to anticipate changing circumstances, and if necessary, to correct the glucose concentration in a timely manner. Fluctuations in blood glucose levels are often unavoidable. Low blood glucose (hypoglycemia) may seriously disrupt daily functioning and thus lead to frustration and anxiety in patients as well as in their family members.

Many patients with type 2 diabetes have, in addition to impaired glucose regulation, metabolic problems requiring a change of lifestyle and drug treatment. An increasing number of patients with type 2 diabetes need to take several oral medications each day, and many of them also need daily insulin injections. For many, this treatment appears a difficult task, which translates into poor treatment adherence. Diabetes is truly regarded as one of the most psychologically damaging chronic diseases with a high risk of "burnout."

After Diagnosis

The adjustment process starts with the diagnosis. In type 1 diabetes, the majority of cases are diagnosed early in childhood and impact the entire family. Understandably, the diagnosis causes strong feelings of fear and uncertainty. Most children and their parents appear to adjust quite well to the new situation after some time (Anderson,

2003). Successful adaptation depends on the family situation and the quality of care provided. Generally, during adolescence, a worsening of diabetes is seen. Increasing insulin resistance plays a role, but also the tendency of adolescents to diminish their attention to their diabetes and to take more risks. Conflicts may arise in the family, which in turn contribute to poorer adjustment of blood glucose of youths. However, young people with diabetes tend to rate their psychosocial well-being equal to that of their healthy peers (de Wit et al., 2007).

Diabetes mellitus type 2 is, in most cases, diagnosed in adulthood although in recent years the mean age at diagnosis has decreased. Research shows that when type 2 diabetes is diagnosed at an early stage, this causes little or no emotional reaction (Adriaanse & Snoek, 2006). This is presumably because a medical treatment is usually not an issue and initially "only" lifestyle changes of patients are requested. Longitudinal research has shown that the significance of diabetes and the psychological impact of this disease changes over time (Thoolen, de Ridder, Bensing, Gorter, & Rutten, 2006). It is therefore important not only to pay attention to adaptation problems soon after diagnosis, but also in the subsequent treatment process. In patients with type 1 or type 2 diabetes, possible health complications may occur that seriously complicate daily functioning and adversely affect quality of life.

Social Support

Social support is a complex construct, but generally is found to have positive effects on diabetes management. Research has demonstrated positive effects on adherence and control for both structural support (e.g., family, friends, co-workers, density of support networks) and functional support (e.g., diabetes-specific help, communication style, cohesiveness). Especially in adolescents, the importance of a supportive family has been shown. Open, empathic communication within families and continued parental involvement in diabetes care is important for achieving good adherence and glycemic outcomes (Anderson, 2003). Evaluation of interventions designed to enhance support, such as social skills training, and improved understanding about diabetes for families, has revealed positive effects on adherence and control. Several trials have demonstrated that group instruction to impart diabetes knowledge and coping skills produces better results than individualized instruction. Support provided through self-help groups or through a mentor (a well-adjusted patient) has been promoted but not researched in the context of diabetes.

Quality of Life

Diabetes, with daily requirements for self-monitoring and management in order to avoid the short-term consequences of hypoglycemia and the long-term complications associated with hyperglycemia, has a substantial impact on daily life. The demands of daily self-care can easily interfere with normal routines and friendships, thereby compromising emotional and social well-being. Attaining strict glycemic control as well as good quality of life (QoL) is a challenge for people with diabetes, their families, and health-care providers. This has led to considerable interest in diabetes-specific quality of life, assessed through a wide range of concerns including morale, well-being, depression, and role functioning. Studies looking into the relationship between diabetes control and QoL find low correlations, if any, although there is evidence to suggest that patients suffering from diabetes-related complications (neuropathy, retinopathy, nephropathy) on average report lower levels of QoL compared to patients without secondary complications (Snoek, 2000). In children, the relationship between glycemic control and QoL is complex and inconsistent across studies as well (Bryden et al., 2001; Hoey et al., 2001; de Wit et al., 2007).

Psychiatric Comorbidity

Diabetes has long been associated with the psychological constitution of patients. Their mental state was considered to be the cause of the disease or as a factor in diabetes regulation.

Indeed, a meta-analysis does show that depression increases the risk of developing type 2 diabetes by 30%, taking known risk factors into account (Knol et al., 2006). There is evidence that patients with poorly controlled type 1 diabetes, as measured by levels of glycosylated hemoglobin (HbA1c), can be distinguished psychologically as a group from patients with well-controlled diabetes on measures of depression and eating disorders. In patients with type 2 diabetes, there is also evidence of a relationship between depression and poorer glycemic control. There is increasing evidence that psychiatric comorbidity is more frequent in adults as well as adolescents with diabetes than in the general population (Anderson, Freedland, Clouse, & Lustman, 2001; Bryden et al., 2001), with adverse consequences for diabetes control. Below three major mental disorders that can complicate the treatment of diabetes are discussed.

Eating Disorders

Food and postponement of food are inextricably linked to a disturbance of blood glucose control in people with diabetes. Time to think about what you eat and when, can result in feelings of frustration and "binge eating," especially if the diet is restrictive. This may explain the increased prevalence of "binge eating disorder" in female patients with type 2 diabetes (Kenardy et al., 2001). In girls with type 1 diabetes, the prevalence of bulimia nervosa is elevated (Colton, Olmsted, Daneman, Rydall, & Rodin, 2004). Eating disorders almost always go along with an elevated HbA1c, frequent fluctuations in blood glucoses, and a greatly increased risk of early development of microvascular complications. Underdosing of insulin as a way to lose weight is not uncommon, particularly among adolescent girls. Among girls with type 1 diabetes, 10% admit to skipping some insulin injections, and 7.5% report injecting less insulin than is required in order to lose weight (Neumark-Sztainer et al., 2002). The treatment of severe eating disorders in diabetes is complex and requires a close collaboration between diabetes clinicians and professionals of clinics specialized in eating disorders.

Anxiety

Extreme anxiety may affect diabetes control, primarily by the disturbing effect of stress hormones, but also by avoidance behavior. One must be careful in giving alarming information like risk of diabetes-related health complications ("fear appeals") if one wants to encourage patients to improve self-management. Most diabetes patients are already concerned about the potential complications of their illness, and further increasing this fear probably does more harm than good. Two fears specific to patients with diabetes require special attention, namely, fear of injections and self-monitoring of blood glucose and fear of hypoglycemia. Although the prevalence of extreme anxiety for the injection of insulin and for self-monitoring of blood glucose is low among diabetic patients using insulin (0.3–1.0%), this fear may be accompanied by great distress and poor diabetes regulation. Moreover, 40% of patients with a phobic fear of injections also have a phobia of pricking the finger to obtain a blood sample. Data on the effects of psychological treatment for self-testing or injection fear are scarce. Both phobias are often associated with other psychiatric disorders, which makes these patients particularly vulnerable (Mollema, Snoek, Ader, Heine, & van der Ploeg, 2001).

Hypoglycemia remains the major side effect of intensive insulin therapy. Exact data are lacking, but a large proportion of patients using insulin have frequent worries about hypoglycemia. More uncommon is a phobic fear of hypoglycemia which can arise once a patient experienced a severe hypoglycemia with loss of consciousness. Patients with a compulsive or panic disorder can be extremely afraid of hypoglycemia without ever having had a real risk. A complicating factor is that anxious patients often do not know whether the symptoms of sweating, dizziness, and heart palpitations they are experiencing are due to dropping blood glucose levels or a panic attack. It is understandable that phobic patients may pursue "safe" blood glucose levels, which translates into a higher HbA1c. Patients with milder forms of fear benefit from hypoglycemia prevention training, which

aims to improve their symptom perception and better recognition of risk factors for hypoglycemia (Cox et al., 2001). Phobic patients and partners can benefit from cognitive behavioral therapy where they can learn to examine how realistic their views on hypoglycemia are and replace irrational thoughts with more adaptive cognitions.

Depression

Mood disorders are twice as common in patients with diabetes compared to the general population. The prevalence of moderate to severe depression among both type 1 and type 2 diabetes patients is estimated at 10–20% (Anderson et al., 2001). For adolescents with type 1 diabetes, the risk of depression is 2–3 times higher compared to their healthy peers (Hood et al., 2006). The relationship between diabetes and depression is not entirely clear. Probably biochemical and psychosocial factors play a role. Patients with depressive symptoms have poorer glycemic control and more complications and are more often hospitalized. Early recognition and treatment of depression in people with diabetes will probably result in major health benefits. Both psychological and pharmacological treatments of depression in diabetes patients are proven to be effective (Katon et al., 2004).

Sexual Problems

It is estimated that approximately 50% of men with a diabetes duration greater than 5 years have some degree of erectile dysfunction, with adverse effects on their perceived quality of life. It seems that these sexual problems are not often discussed with health-care professionals (De Berardis et al., 2002). Neuropathy and metabolic disorders are considered as the main causes of erectile dysfunction, but acute fluctuations in blood glucose and psychological factors may play a role as well. Drug treatment of erectile dysfunction, sometimes in combination with psychotherapy or marriage counseling, may be effective.

Less is known about sexual dysfunction in women with diabetes, but recent research among women with type 1 diabetes showed that they have more problems with sexual arousal and lubrication compared to healthy women. Sexual problems in female patients are often associated with depressive symptoms, making it difficult to determine cause and effect.

Conclusion

Successful management of diabetes requires considerable motivation and adaptability of the patient. Because people with diabetes are at increased risk for psychological problems that may complicate self-management behaviors, attention to the psychosocial functioning of patients is important in all phases of treatment. The fact that depression and other psychosocial problems are often not recognized and discussed calls for systematic monitoring of psychological well-being of diabetic patients as part of the regular appointments. Research into the effects in youth and adults with diabetes has shown that such an approach is feasible and that the well-being of patients and their satisfaction with care increase (Pouwer, Snoek, van der Ploeg, Ader, & Heine, 2001; de Wit et al., 2008). Nurses can play an important role in such approach. Additional psychological assessment and intervention can be provided as needed. Diabetes is a largely self-managed disease. Consequently, if the patient is unwilling or unable to self-manage his or her diabetes on a day-to-day basis, outcomes will be poor, regardless of how advanced the treatment technology is. Cognitive, emotional, behavioral, and social factors have a vital role in diabetes management, particularly because research has shown depression and other psychological problems are prevalent and negatively impact on well-being and metabolic outcomes. There is more to diabetes than glucose control; a biopsychosocial approach is required for optimal results. Motivational counseling and behavior change programs in type 2 diabetes have been shown to be effective in improving adherence and warrant further dissemination in primary and secondary care. In type 1 diabetes, adolescents are at increased risk of coping difficulties and poor diabetes outcomes, and warrant special attention. For all age groups, monitoring of

patients' emotional well-being as an integral part of routine diabetes care is recommended. Discussion of quality-of-life issues in the context of clinical diabetes care in itself promotes increased adherence and patient satisfaction, and has proven to increase recognition of signs of emotional problems and "diabetes burnout." Integrating psychology in diabetes management can help to effectively tailor care to the patient's individual needs and improve outcomes.

Cross-References

- ▶ Quality of Life
- ▶ Self-management
- ▶ Self-monitoring
- ▶ Self-regulation Model

References and Readings

Adriaanse, M. C., & Snoek, F. J. (2006). The psychological impact of screening for type 2 diabetes. *Diabetes/Metabolism Research and Reviews, 22*(1), 20–25.

Anderson, B. J. (2003). Diabetes self-care: Lessons from research on the family and broader contexts. *Current Diabetes Reports, 3*(2), 134–140.

Anderson, R. J., Freedland, K. E., Clouse, R. E., & Lustman, P. J. (2001). The prevalence of comorbid depression in adults with diabetes: A meta-analysis. *Diabetes Care, 24*(6), 1069–1078.

Bryden, K. S., Peveler, R. C., Stein, A., Neil, A., Mayou, R. A., & Dunger, D. B. (2001). Clinical and psychological course of diabetes from adolescence to young adulthood: A longitudinal cohort study. *Diabetes Care, 24*(9), 1536–1540.

Colton, P., Olmsted, M., Daneman, D., Rydall, A., & Rodin, G. (2004). Disturbed eating behavior and eating disorders in preteen and early teenage girls with type 1 diabetes: A case-controlled study. *Diabetes Care, 27*(7), 1654–1659.

Cox, D. J., Gonder-Frederick, L., Polonsky, W., Schlundt, D., Kovatchev, B., & Clarke, W. (2001). Blood glucose awareness training (BGAT-2): Long-term benefits. *Diabetes Care, 24*(4), 637–642.

de Wit, M., Delemarre-van de Waal, H. A., Bokma, J. A., Haasnoot, K., Houdijk, M. C., Gemke, R. J., et al. (2007). Self-report and parent-report of physical and psychosocial well-being in Dutch adolescents with type 1 diabetes in relation to glycemic control. *Health and Quality of Life Outcomes, 5*, 10.

de Wit, M., Delemarre-van de Waal, H. A., Bokma, J. A., Haasnoot, K., Houdijk, M. C., Gemke, R. J., et al. (2008). Monitoring and discussing health-related quality of life in adolescents with type 1 diabetes improve psychosocial well-being: A randomized controlled trial. *Diabetes Care, 31*(8), 1521–1526.

De Berardis, G., Franciosi, M., Belfiglio, M., Di Nardo, B., Greenfield, S., Kaplan, S. H., et al. (2002). Erectile dysfunction and quality of life in type 2 diabetic patients: A serious problem too often overlooked. *Diabetes Care, 25*(2), 284–291.

Harris, M. I. (2000). Health care and health status and outcomes for patients with type 2 diabetes. *Diabetes Care, 23*(6), 754–758.

Hoey, H., Aanstoot, H. J., Chiarelli, F., Daneman, D., Danne, T., Dorchy, H., et al. (2001). Good metabolic control is associated with better quality of life in 2,101 adolescents with type 1 diabetes. *Diabetes Care, 24*(11), 1923–1928.

Hood, K. K., Huestis, S., Maher, A., Butler, D., Volkening, L., & Laffel, L. M. B. (2006). Depressive symptoms in children and adolescents with Type 1 diabetes: Association with diabetes-specific characteristics. *Diabetes Care, 29*(6), 1389.

Katon, W. J., Von Korff, M., Lin, E. H., Simon, G., Ludman, E., Russo, J., et al. (2004). The pathways study: A randomized trial of collaborative care in patients with diabetes and depression. *Archives of General Psychiatry, 61*(10), 1042–1049.

Kenardy, J., Mensch, M., Bowen, K., Green, B., Walton, J., & Dalton, M. (2001). Disordered eating behaviours in women with Type 2 diabetes mellitus. *Eating Behaviors, 2*(2), 183–192.

Knol, M. J., Twisk, J. W., Beekman, A. T., Heine, R. J., Snoek, F. J., & Pouwer, F. (2006). Depression as a risk factor for the onset of type 2 diabetes mellitus. A meta-analysis. *Diabetologia, 49*(5), 837–845.

Mollema, E. D., Snoek, F. J., Ader, H. J., Heine, R. J., & van der Ploeg, H. M. (2001). Insulin-treated diabetes patients with fear of self-injecting or fear of self-testing: Psychological comorbidity and general well-being. *Journal of Psychosomatic Research, 51*(5), 665–672.

Neumark-Sztainer, D., Patterson, J., Mellin, A., Ackard, D. M., Utter, J., Story, M., et al. (2002). Weight control practices and disordered eating behaviors among adolescent females and males with type 1 diabetes: Associations with sociodemographics, weight concerns, familial factors, and metabolic outcomes. *Diabetes Care, 25*(8), 1289–1296.

Pouwer, F., Snoek, F. J., van der Ploeg, H. M., Ader, H. J., & Heine, R. J. (2001). Monitoring of psychological well-being in outpatients with diabetes: Effects on mood, HbA(1c), and the patient's evaluation of the quality of diabetes care: A randomized controlled trial. *Diabetes Care, 24*(11), 1929–1935.

Snoek, F. J. (2000). Quality of life: A closer look at measuring patients' well-being. *Diabetes Spectrum, 13*, 24.

Thoolen, B. J., de Ridder, D. T., Bensing, J. M., Gorter, K. J., & Rutten, G. E. (2006). Psychological outcomes

of patients with screen-detected type 2 diabetes: The influence of time since diagnosis and treatment intensity. *Diabetes Care, 29*(10), 2257–2262.
Writing Team for the Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications Research Group. (2002). Effect of intensive therapy on the microvascular complications of type 1 diabetes mellitus. *JAMA: The Journal of the American Medical Association, 287*(19), 2563–2569.

Diabetic Foot Care

► Diabetes Foot Care

Diabetic Neuropathy

Jenny T. Wang[1] and Jason S. Yeh[2]
[1]Department of Medical Psychology, Duke University, Durham, NC, USA
[2]Obstetrics and Gynecology, Division of Reproductive Endocrinology and Fertility, Duke University Medical Center, Durham, NC, USA

Synonyms

Nerve damage

Definition

Diabetic neuropathy is nerve damage resulting from high blood sugar levels (hyperglycemia) and poor metabolic health in individuals with diabetes mellitus. Diabetic neuropathy can affect any number of organs or organ systems. Although it can develop after the initial diagnosis is made, it is commonly used as a symptom to diagnose diabetes in a patient. A significant percentage of patients have clinical evidence of nerve damage at the time of diagnosis, which suggests that even prediabetes can cause early diabetic neuropathy. In general, the more poorly controlled the diabetes, the more severe the diabetic neuropathy. Studies have shown that nerve conduction through the body slows significantly with each percent rise in glycosylated hemoglobin (HbA1c) values. The most commonly encountered forms of diabetic neuropathy include distal symmetric polyneuropathy, autonomic neuropathy, polyradiculopathy, and mononeuropathy.

Distal symmetric polyneuropathy is the most common type and is often synonymous with diabetic neuropathy. It is characterized by the symmetrical damage of sensory nerves that initially affects the lower extremities. The natural history of symmetric polyneuropathy illustrates the principle that the longest axons are affected first. Consequently, patients initially report symptoms in their toes and feet, which eventually progress to the classic bilateral "stocking and glove" numbness. Individuals with peripheral neuropathy can experience debilitating pain, tingling, and numbness in their hands and feet. Because many patients ultimately lose all sensation in their feet, they must be fitted with nonabrasive shoes and are taught to check their hands and feet daily for abrasions and injuries that can progress into limb and life-threatening ulcers.

Autonomic neuropathy includes a wide spectrum of symptoms that can affect multiple organ systems such as the cardiovascular, gastrointestinal, genitourinary, and even the neuroendocrine system. Its diagnosis can be difficult because of multiple organ involvement and insidious onset. Symptoms of cardiac neuropathy include exercise intolerance, resting tachycardia, and silent myocardial infarction. Neuropathic disease of the upper gastrointestinal tract can cause dysphagia, retrosternal pain, and "heartburn." More concerning is delayed stomach emptying which can cause nausea, vomiting, early satiety, prolonged fullness after eating and anorexia. When autonomic disease affects the lower gastrointestinal tract, patients present with severe constipation, diarrhea, and even bowel

incontinence. Neuropathy affecting the genitourinary system can cause bladder dysfunction, erectile dysfunction, and painful intercourse due to decreased vaginal lubrication. Less commonly, neuropathy can even cause hypoglycemia unawareness where patients become unable to perceive dangerously low blood sugar levels.

Diabetic polyradiculopathies refer to several types of asymmetric proximal nerve disease in the diabetic patient, the most common being diabetic amyotrophy and diabetic thoracic polyradiculopathy. Diabetic amyotrophy is the more common of the two and involves an acute onset of pain followed by weakness involving one proximal leg, with concurrent autonomic failure and weight loss. If the disease affects the contralateral leg, symptoms can occur immediately or much later after the initial episode. No treatments have been shown to be effective for diabetic amyotrophy. Thoracic polyradiculopathy, another type of diabetic polyradiculopathy, describes an injury of the high lumbar or thoracic-level nerve roots. These patients present with severe abdominal pain and have frequently undergone multiple studies to identify the cause of their symptoms.

Lastly, there are two types of diabetic mononeuropathy: cranial and peripheral. Cranial lesions commonly affect nerves surrounding the eye and typically result in unilateral eye symptoms including pain, drooping eyelid, and double vision. The most common peripheral lesions in diabetic patients are median nerve mononeuropathy at the wrist and common peroneal mononeuropathy near the ankle, both of which can result in pain, drooping, weakness, and decreased range of motion.

Improving the symptoms of diabetic neuropathy can be difficult; most efforts are made to prevent the onset and worsening of existing diabetic neuropathy. Treatment of diabetic neuropathy emphasizes tight blood sugar control, managing pain symptoms through pharmacotherapy (i.e., analgesics, certain antidepressants, steroids) and/or psychosocial interventions (e.g., meditation, relaxation training), and practicing diligent foot care (i.e., washing feet, inspecting for cuts, bruises, or blisters).

Successful diabetes management is associated with several behavioral and lifestyle factors, which have been shown to improve with psychosocial interventions such as motivational interviewing, health coaching, and cognitive behavioral therapy. Well-controlled diabetes is often the result of adherence to a healthy diet and exercise regimen, keeping track of carbohydrate intake, frequent and routine checks of blood sugar levels, taking required amounts of insulin, and discontinuing negative behaviors such as smoking or excessive drinking. Modification of these behaviors in children and adults has resulted in improvements in diabetes management, which can prevent or slow the development of diabetic neuropathy.

Cross-References

- ▶ Blood Glucose
- ▶ Chronic Disease Management
- ▶ Diabetes
- ▶ Diabetes Education
- ▶ Diabetes Foot Care
- ▶ Hyperglycemia

References and Readings

Kronenberg, H., & Williams, R. H. (2008). *Williams textbook of endocrinology* (11th ed.). Philadelphia: Saunders Elsevier.

Diabetologist (Diabetes Specialist)

Janine Sanchez
Department of Pediatrics, University of Miami, Miami, FL, USA

Synonyms

Endocrinologist

Definition

A diabetologist is a physician with expertise in diabetes care. The physician is often board certified in pediatric or adult endocrinology with special interest or extra training in diabetes care or research. However, diabetology is not a recognized medical specialty and has no formal training programs. Thus, any physician whose practice and/or research efforts are concentrated mainly in diabetes care may be considered a diabetologist/diabetes specialist.

Cross-References

- ▶ Diabetes
- ▶ Endocrinology

References and Readings

Menon, R. (2003). *Pediatric diabetes* (1st ed.). Norwell, MA: Springer.
Sperling, M. A. (2009). *Pediatric endocrinology* (3rd ed.). Philadelphia: W.B. Saunders.

Diagnostic Criteria

- ▶ Psychiatric Diagnosis

Diagnostic Features of Depression

- ▶ Depression: Symptoms

Diagnostic Interview

- ▶ Interview

Diagnostic Interview Schedule

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

DIS

Definition

The National Institute of Mental Health Diagnostic Interview Schedule was discussed in the Archives of General Psychiatry by Robbins, Helzer, Croughan, and Ratcliff (1981). The interview schedule allowed lay interviewers or clinicians to make psychiatric diagnoses according to *DSM-III* criteria, Feighner criteria, and Research Diagnostic Criteria. It was being used in a set of epidemiological studies sponsored by the National Institute of Mental Health Center for Epidemiological Studies. Its accuracy has been evaluated in a test-retest design comparing independent administrations by psychiatrists and lay interviewers to 216 subjects (inpatients, outpatients, ex-patients, and nonpatients).

The National Institute of Mental Health Diagnostic Interview Schedule for Children, Version 4 (NIMH DISC IV or "DISC") is a highly structured diagnostic interview used to assess psychiatric diagnoses of children and adolescents. The DISC was designed to be administered by interviewers with no formal clinical training following the rules and conventions outlined in the DISC training manual. The DISC questions elicit the diagnostic criteria specified in the *Diagnostic and Statistical Manual of Mental Disorders – Fourth Edition (DSM-IV)* and the WHO International Classification of Diseases, Version 10 (ICD-10).

The Generic or "12 month" DISC was used in NHANES. Seven of the 34 diagnostic assessments were included in NHANES over the 6-year

period that the DISC was administered: generalized anxiety disorder, panic disorder, eating disorder, elimination disorders, major depression/dysthymic disorder, attention deficit disorder/hyperactivity (ADD/ADHD), and conduct disorder. In each module, questions are asked about specific symptoms during the past year, and then follow-up questions in cases of positive endorsement. Two of the DISC modules in NHANES, eating disorder and major depression/dysthymic disorder, were comprised of two parallel interviews. A youth-informant interview (DISC-Y) administered in-person to children asked questions about themselves, and a parent-informant interview (DISC-P) administered by telephone to a parent or caretaker asked questions about their child. Only the DISC-Y was administered for generalized anxiety disorder and panic disorder, and only the DISC-P was administered for elimination disorder, ADD/ADHD, and conduct disorder. Depending on the module, responses and diagnostic scores derived from the interviews can be combined or examined separately.

Cross-References

- ▶ Anxiety Disorder
- ▶ Depression: Measurement
- ▶ National Health and Nutrition Examination Survey (NHANES)
- ▶ Panic Disorder

References and Readings

Everson-Rose, S. A., & Clark, C. J. (2010). Assessment of psychosocial factors in population studies. In A. Steptoe (Ed.), *Handbook of behavioral medicine: Methods and applications* (pp. 291–306). New York: Springer.

Robbins, L. N., Helzer, J. E., Croughan, J., & Ratcliff, K. S. (1981). National Institute of Mental Health diagnostic interview schedule: Its history, characteristics, and validity. *Archives of General Psychiatry, 38*, 381–389.

Snowling, M. J., & Hulme, C. (2011). Annual research review: The nature and classification of reading disorders: a commentary on proposals for DSM-5. *Journal of Child Psychology and Psychiatry*, Dec, 5th [Epub ahead of print].

Diaries

C. Renn Upchurch Sweeney
VA Salt Lake City Healthcare System,
Salt Lake City, UT, USA

Synonyms

Daily diary; Event sampling

Definition

Diaries are self-report instruments often used in behavioral medicine research to examine psychological processes (i.e., affect, social interaction, marital and family interactions, stress, physical symptoms, mental health, well being) within the natural context of everyday life. Diaries require study participants to keep track of cognitions, emotions, or behaviors in a log for a particular period of time and are designed to "capture life as it is lived" (Bolger, Davis, & Rafaeli, 2003).

Examples of diaries include paper and pencil diaries, augmented paper diaries (ancillary devices are programmed to prompt participants to respond at a particular time), and electronic diaries (i.e., palm pilots, PDAs). Diaries can be collected repeatedly over a number of days, once daily (daily diary), or even sampled several times during the day.

Advantages

One advantage of diary methods is that information is gathered in the context of the participant's everyday life, which may illicit behavior that is more representative than that observed in the laboratory setting. Secondly, diary methods reduce the likelihood of retrospection. That is, the time between an experience and the recounting of the experience by the participant is minimized. By asking the participants to record information when an event occurs or shortly thereafter, diaries also reduce biases related to recall (the greater the time between events and its recollection, the greater the potential for

distortion), recency effects (more recent events are more likely to influence judgments) and salience (moments of peak intensity or personal relevance influence judgments more than less salient experiences). Finally, diary studies eliminate the difficulty of summarizing multiple events.

Disadvantages

Despite the many advantages of diary methods, several disadvantages also exist. For example, diaries require experimenters to conduct training sessions to ensure that participants understand the diary protocol, which can be time consuming for the experimenter. Secondly, diaries can be onerous for participants. The burden of repeated queries and responses places substantial demands on the participant and requires a greater level of participant commitment compared to other types of research studies. Thirdly, the act of completing the diary may affect participants' responses or alter participants' understanding of a particular construct. For example, a more complex understanding of the surveyed topic may develop or the experience of the diary study may change participants' conceptualization of the topic to fit with those measured in the diary. Finally, participants may develop a habitual response style when making repeated diary entries, which may have negative consequences. For example, participants may skim over sections of a diary questionnaire that rarely apply to them, but inadvertently omit responses to these questions at relevant times.

Cross-References

► Ecological Momentary Assessment

References and Readings

Bolger, N., Davis, A., & Rafaeli, E. (2003). Diary methods: Capturing life as it is lived. *Annual Review of Psychology, 54*, 579–616.

Fiske, S. T., Gilbert, D. T., & Lindzey, G. (Eds.). (2009). *Handbook of social psychology* (Vol. 1). Hoboken, NJ: Wiley.

Green, A. S., Rafaeli, E., Bolger, N., Shrout, P. E., & Reis, H. T. (2006). Paper or plastic? Data equivalence in paper and electronic diaries. *Psychological Methods, 11*, 87–105.

Laurenceau, J., & Bolger, N. (2005). Using diary methods to study marital and family process. *Journal of Family Psychology, 19*, 86–97.

Tennen, H., Affleck, G., & Armeli, S. (2003). Daily processes in health and illness. In J. Suls & K. Wallston (Eds.), *The social psychological foundations of health and illness* (pp. 495–529). Oxford, England: Blackwell.

Diastolic Blood Pressure (DBP)

Annie T. Ginty
School of Sport and Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

Synonyms

Blood pressure

Definition

Diastolic blood pressure is the force exerted by the artery walls during ventricular relaxation. It is the lowest pressure measured and normal range is considered to be <80 mmHg (Tortora & Grabowski, 1996).

Cross-References

► Blood Pressure, Elevated
► Blood Pressure, Measurement of
► Systolic Blood Pressure (SBP)

References and Readings

Tortora, G. J., & Grabowski, S. R. (1996). *Principles of anatomy and physiology* (8th ed.). New York: Harper Collins College.

Diathesis-Stress Model

Kristen Salomon and Alvin Jin
Department of Psychology, University of South Florida College of Arts & Sciences, Tampa, FL, USA

Synonyms

Risk factors

Definition

Diathesis refers to a predisposition or vulnerability for the development of a pathological state. Diathesis-stress models argue that certain pathological states or diseases emerge from the combination of a predisposition with stressful events (Zuckerman, 1999). Most models specify that neither the diathesis nor stress alone is sufficient to produce the disorder. Instead, stress activates the diathesis, which then leads to the disorder. More broadly, diathesis-stress models are similar to the idea of risk-factors for stress-related diseases.

Description

History

Early diathesis-stress models primarily focused on psychiatric disorders such as schizophrenia, depression, and anxiety disorders, born out of the observation that these disorders tend to be inherited and yet also show a significant relationship to life stress (Zuckerman, 1999). These early diathesis-stress models identified fixed biological and/or hereditary factors as predispositions, and often argued for singular directionality, i.e., that the stress acted upon the diathesis. Later, the idea of diathesis was expanded to include physiological, behavioural and psychological diatheses, some of which may be acquired (Zuckerman). Broadening the scope of diatheses to include "non-biological" factors also resulted in a change in the presumed directionality, such that diatheses may influence the experience of stress.

Diatheses

Zuckerman (1999) has argued that diatheses are traits, and as such they not only should be present before the onset of the disorder but also should not change as the result of the disorder. Diatheses may be conceptualized as dichotomous, i.e., present or not. However, many diathesis-stress models suggest that the degree of diathesis present sets a threshold of vulnerability to stress. The greater the level of diathesis present, the less stress required to activate it and create the pathological state (Zuckerman). Some diathesis-stress theories, such as Fowles (1992) theory of schizophrenia, argue that stress may not be necessary for the disorder to develop. If the level of diathesis is high enough, the threshold is met and, even in the absence of stress, the disorder will develop (Zuckerman, 1999).

The Role of Stress

One important issue for diathesis-stress models is the potential confounding of stress with the diathesis. For example, if a personality trait, such as neuroticism, is identified as a diathesis for a disorder, such as anxiety, then the issue becomes whether the diathesis is reacting to stress or is the cause of stress. Therefore, many diathesis-stress models define stress in terms of external events rather than defining stress as subjectively reported reactions to events (Monroe & Simons, 1991). Further, identifying genetic, biological, and/or physiological diatheses, rather than psychological ones, also serves to avoid the problem of confounding (Zuckerman, 1999). Also, diathesis-stress models often assume that the stress must occur in close temporal proximity to the onset of the disorder (Zuckerman, 1999). Some stress-diathesis models suggest that not only do diatheses differ by disorder but also by the type of stress necessary to activate a specific diathesis. For example, for major depressive disorder, stressors that involve loss of one's social structure (e.g., job loss, divorce, death of a loved

one) have been identified as those that combine with diatheses to produce the disorder (Monroe & Simons, 1991).

Specifying Diathesis-Stress Models

Conceptualizations of diatheses and stressors that are binary (present or not) lead to relatively simple models. If both the diathesis and stress are present, the disorder will occur, but if one or both are absent, the disorder should not occur. However, most research on diathesis-stress models suggests that neither diatheses nor stress are dichotomous. Some models have suggested that diatheses are categorical, such as evidence suggesting that allelic variation in the 5-HTT-linked polymorphic region (5-HTTLPR) of the serotonin-transporter gene serves as a diathesis for anxiety-related disorders (Lesch et al., 1996). However, these models do not consider the polygenic nature of most disorders and they are likely artificially categorizing dimensional variability in gene expression (Zuckerman, 1999). Further, stress is often scaled in terms of the severity of individual stressors (i.e., traumatic stress producing posttraumatic stress disorder; PTSD) or in the total number of stressors (i.e., more instances of loss associated with higher rates of depression). Continuous diatheses and stressors lead to more complex models. Models may specify additive effects, such that more stress is required to bring about the disorder in someone with less of the diathesis than in someone with a greater degree of the diathesis. Interactive models may suggest that if the diathesis is absent, no amount of stress may bring about the disorder, but once present, the diathesis can vary in its loading, thus requiring different amounts of stress to bring about the disorder. Thus, important questions to consider when developing diathesis stress models involve the nature of the diathesis (categorical, continuous, continuous only if present), the diathesis threshold necessary for the disorder to emerge, the type (e.g., loss, fear) and nature (categorical or continuous) of the stress necessary to activate the diathesis, the nature of the effects of each (additive, interactive), and whether the diathesis and stress are independent of one another or correlated.

Current State of Stress-Diathesis Models

Recently, the basic diathesis-stress model has been expanded to include predispositions that protect individuals from developing stress-related disorders, or resilience (Belsky & Pluess, 2009). Instead of focusing on why some people fall victim to disorders in the face of stress, resilience research focuses on why some people seem resistant to a disorder, even in the face of extreme stress. However, resilience is not the opposite of diathesis, but instead, individuals may differ in their overall plasticity to both negative (i.e., stress) and positive (i.e., supportive) environmental influences (Belsky & Pluess, 2009).

Cross-References

- ▶ Resilience
- ▶ Risk Factors
- ▶ Stress

References and Readings

Belsky, J., & Pluess, M. (2009). Beyond diathesis stress: Differential susceptibility to environmental influences. *Psychological Bulletin, 135*(6), 885–908.

Fowles, D. C. (1992). Schizophrenia – diathesis stress revisited. *Annual Review of Psychology, 43*, 303–336.

Lesch, K. P., Bengel, D., Heils, A., Sabol, S. Z., Greenberg, B. D., Petri, S., et al. (1996). Association of anxiety-related traits with a polymorphism in the serotonin transporter gene regulatory region. *Science, 274*, 1527–1531.

Monroe, S. M., & Simons, A. D. (1991). Diathesis-stress theories in the context of life stress research: Implications for the depressive disorders. *Psychological Bulletin, 110*, 406–425.

Zuckerman, M. (1999). *Vulnerability to psychopathology: A biosocial model*. Washington, DC: American Psychological Association.

Diet and Cancer

- ▶ Cancer and Diet

Dietary Fatty Acids

► Essential Fatty Acids

Dietary Lipids Absorption

► Fat Absorption

Dietary Requirements

► Nutrition

Dietary Supplement

► Nutritional Supplements

Differential Psychology

► Individual Differences

Diffuse Optical Imaging (DOI)

► Brain, Imaging
► Neuroimaging

Diffusion

► Dissemination

Dimeric Glycoprotein

► Fibrinogen

Dimsdale, Joel E.

Joel E. Dimsdale
Department of Psychiatry, University of California San Diego, La Jolla, CA, USA

Biographical Information

Joel Dimsdale was born in Sioux City, Iowa, in 1947, and obtained his BA degree in biology from Carleton College. He then attended Stanford University, where he obtained an MA degree in Sociology and an MD degree. He obtained his psychiatric training at Massachusetts General Hospital and then completed a fellowship in psychobiology at the New England Regional Primate Center. He was on the faculty of Harvard Medical School from 1976 until 1985, when he moved to University of California, San Diego (UCSD).

Dimsdale is distinguished professor emeritus and Research Professor in the department of psychiatry at UCSD. His clinical subspecialty is consultation psychiatry. He is an active investigator, a former career awardee of the American Heart Association, and is past-president of the Academy of Behavioral Medicine Research, the American Psychosomatic Society, and the Society of Behavioral Medicine. He is on numerous editorial boards, is editor-in-chief emeritus of

Psychosomatic Medicine, and is a previous guest editor of *Circulation*. He has been a consultant to the President's Commission on Mental Health, the Institute of Medicine, and is a long-time reviewer for NIH. He consults to the National Academy of Sciences regarding behavioral issues in space. He is a member of the DSM V taskforce and chairs the workgroup studying somatic symptom disorders. Dimsdale is the former chair of the UCSD Academic Senate and currently chairs the Systemwide University of California Faculty Welfare Committee.

Dimsdale is an active teacher who supervises CL psychiatry. He mentors trainees and junior faculty members from psychiatry, psychology, pulmonary medicine, nephrology, anesthesiology, and surgery. Dimsdale directs UCSD's K12 training grant for fostering the careers of outstanding young clinical faculty.

Dimsdale's major research interests include sympathetic nervous system physiology as it relates to stress, blood pressure, and sleep; cultural factors in illness; and quality of life. He is the author of more than 500 publications as well as the editor of four books.

Major Accomplishments

Dimsdale has been an active investigator who has mentored generations of medical students, residents, psychology students, and post docs. He has been repeatedly tapped for leadership positions in national organizations, on medical journals, and in university governance.

References and Readings

Bardwell, W., Moore, P., Ancoli-Israel, S., & Dimsdale, J. (2003). Fatigue in obstructive sleep apnea is driven by depressive symptoms and not apnea severity. *The American Journal of Psychiatry, 160*, 350–355.

Bardwell, W., Natarajan, L., Dimsdale, J., Rock, C., Mortimer, J., Hollenbach, K., & Pierce, J. (2006). Objective cancer-related variables are not associated with depressive symptoms in women treated for early-stage breast cancer. *Journal of Clinical Oncology, 24*, 2420–2427.

Dimsdale, J. E. (1974). Coping behavior of Nazi concentration camp survivors. *The American Journal of Psychiatry, 131*, 792–797.

Dimsdale, J. (1988). A perspective on type A behavior and coronary disease. *The New England Journal of Medicine, 318*, 110–112.

Dimsdale, J. (2000). Stalked by the past: The impact of ethnicity on health. *Psychosomatic Medicine, 62*, 161–170.

Dimsdale, J. (2008). Psychological stress and cardiovascular disease. *Journal of the American College of Cardiology, 51*, 1237–1246.

Dimsdale, J., & Creed, F. (2009). The proposed diagnosis of somatic symptom disorders in DSM-V to replace somatoform disorders in DSM-IV – A preliminary report. *Journal of Psychosomatic Research, 66*(6), 473–476.

Dimsdale, J., Graham, R., Ziegler, M., Zusman, R., & Berry, C. (1987). Age, race, diagnosis, and sodium effects on the pressor response to infused norepinephrine. *Hypertension, 10*, 564–569.

Dimsdale, J. E., Hackett, T. P., Hutter, A., Block, P., & Catanzano, D. (1978). Type A personality and the extent of coronary atherosclerosis. *The American Journal of Cardiology, 42*, 583–586.

Dimsdale, J. E., Hartley, L. H., Guiney, T., Ruskin, J., & Greenblatt, D. (1984). Post-exercise peril: Plasma catecholamines and exercise. *Journal of the American Medical Association, 251*, 630–632.

Dimsdale, J. E., & Moss, J. (1980). Plasma catecholamines in stress and exercise. *Journal of the American Medical Association, 243*, 340–342.

Dimsdale, J., Newton, R., & Joist, T. (1989). Neuropsychological side effects of beta blockers. *Archives of Internal Medicine, 149*, 514–525.

Golomb, B. A., Criqui, M. H., White, H. L., & Dimsdale, J. E. (2004). Conceptual foundations of the UCSD statin study: A randomized controlled trial assessing the impact of statins on cognition, behavior, and biochemistry. *Archives of Internal Medicine, 164*, 153–162.

Mills, P., Dimsdale, J., Coy, T., Ancoli-Israel, S., Clausen, J., & Nelesen, R. (1995). Beta-two adrenergic receptor characteristics in sleep apnea patients. *Sleep, 18*, 39–42.

Ng, B., Dimsdale, J., Rollnik, J., & Shapiro, H. (1996). The effect of ethnicity on prescriptions for patient controlled analgesia for post-operative pain. *Pain, 66*, 9–12.

Profant, J., & Dimsdale, J. (1999). Race and diurnal blood pressure patterns: A review and meta-analysis. *Hypertension, 33*, 1099–1104.

Thomas, K., Bardwell, W., Ancoli-Israel, S., & Dimsdale, J. (2006). The toll of ethnic discrimination on sleep architecture and fatigue. *Health Psychology, 25*(5), 635–642.

von Kanel, R., Loredo, J., Ancoli-Israel, S., Mills, P., Natarajan, L., & Dimsdale, J. (2007). Association between polysomnographic measures of disrupted sleep and prothrombotic factors. *Chest, 131*, 733–739.

Ziegler, M., Nelesen, R., Mills, P., Ancoli-Israel, S., Clausen, J., Watkins, L., & Dimsdale, J. (1995). The effect of hypoxia on baroreflexes and pressor sensitivity in sleep apnea and hypertension. *Sleep, 18*, 859–865.

DIS

► Diagnostic Interview Schedule

Disability

Diane Dixon
Department of Psychology, University of Strathclyde, Glasgow, Scotland, UK

Synonyms

Activity limitations; Impairment; Participation restrictions

Definition

The World Health Organization views disability not as a property of an individual person but as an interaction between features of a person's body and their social and physical environment. Disability can exist at the level of impairments (to body structures and functions), activity limitations, and/or participation restrictions. Impairments are defined as a significant deviation or loss in body functions or structures. Activity limitations are difficulties a person has in performing activities; an activity is the execution of a task or action. Participation restrictions are problems a person experiences in involvement in life situations; participation is involvement in life situations.

Governments also define disability within antidiscrimination legislation and to provide access to government support and services. For example, in the United Kingdom, the Equality Act (2010) considers a person to have a disability if they have a physical or mental impairment that has a substantial and long-term adverse effect on their ability to perform normal day-to-day activities.

Description

The World Health Organization estimates that, worldwide, 650 million people live with disabilities of various types. It is expected that this figure will continue to rise as the world's population ages and the prevalence of chronic illness increases. The management of disability is complex and typically involves multidisciplinary teams and input from multiple services. As a consequence, the management of disability will benefit from the use of theoretical frameworks that are able to accommodate such multidisciplinary ways of working.

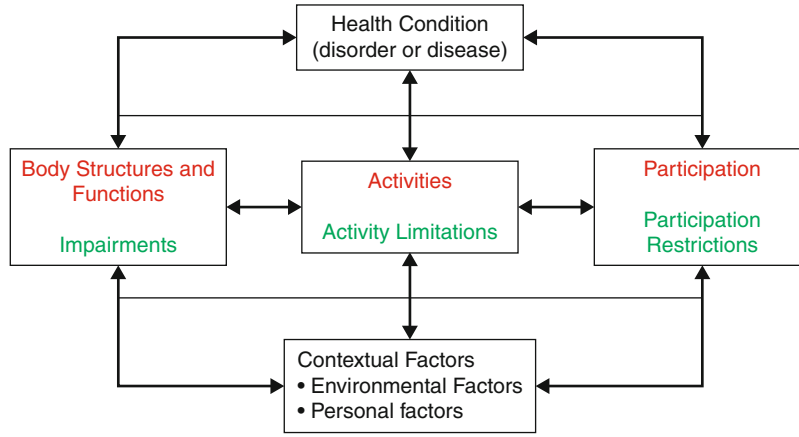
Conceptualizing Disability

The World Health Organization's International Classification of Functioning, Disability and Health (ICF) provides such an integrative framework (WHO, 2001). A summary schematic of the ICF is shown in Fig. 1.

The WHO designed the ICF as a classification system for health and health-related states. However, the ICF can also operate as a complex model of health and disability. The ICF identifies three health components, namely, body structures and functions, activities and participation, and their corollaries of impairment, activity limitations, and participation restrictions (see the "Definition" tab for a description of each component).

The ICF has several features of particular relevance to behavioral medicine (Dixon & Johnston, 2010).

First, the relationships between the components are reciprocal. This means that impairments can cause activity limitations but also that activity limitations can cause impairments. For example, osteoarthritis is a health condition in which the structure of the hip joint is impaired; this impairment is experienced as joint stiffness and pain (impairments). A person with osteoarthritis of the hip might, as a result of such impairments,



Disability, Fig. 1 Summary schematic of the ICF

experience difficulties getting up and down stairs and walking (activity limitations), and these activity limitations might restrict their ability to use buses or trains, which might reduce their ability to visit the cinema in town (participation restrictions). However, reduced walking might also cause further impairments in the structure and function of the hip joint, as muscle strength weakens with reduced use. Thus, within the ICF, reductions in impairment can be achieved through interventions that target activity limitations and vice versa. This makes the ICF suitable for use by multidisciplinary teams typically required for the effective management of the consequences of chronic illness. For example, medical doctors can intervene surgically or pharmacologically; allied health professionals can intervene with a range of therapies, for example, physiotherapy and speech and language therapy; social services can intervene with adjustments to the home environment, for example, provision of ramp access to the home, an electric wheelchair, and other assistive devices.

Second, the role of the environment and personal factors in disability is recognized by the contextual factors component of the ICF. These contextual factors enable other disciplines to contribute to our understanding of disability. The ICF provides a detailed description of the environmental factors, which include assistive products and technologies, the natural and man-made environment, social services, systems, and policies. These environmental factors enable diverse disciplines, such as architecture and town planning, to contribute to achieving reductions in disability. The personal factors component is less well described by the ICF; however, personal factors have been operationalized in the form of individual cognitions and emotions. Inclusion of the personal factors component and the observation that activity limitations and participation restrictions are behavior(s) enables psychology to inform our understanding of disability. Psychology can be defined as the scientific study of behavior, and as such, models of behavior and behavior change can be used to understand the factors that influence disability. Further, the inclusion of behavioral models of disability delivers the evidence base on how to intervene to change behavior (Bandura, 1969; Michie et al., 2009), which enables reductions in disability to be achieved, again without the need to reduce chronic impairments.

A behavioral approach to disability conceptualizes disability as behavior, which is influenced by the same psychological processes that affect any other type of behavior. As a consequence, an individual with a health condition will be motivated to engage in an activity or participate in a social situation because it achieves the things they like, because they believe other people would like them to do so, and because they believe they are able to do so. The behavioral approach can be used to explain, in part, the so-called disability paradox. The disability paradox is the observation that two people, living in

identical social and environmental situations, experience different levels of disability, i.e., people with severe impairments might report lower than expected levels of disability, whereas an individual with mild impairment might experience higher than expected levels of disability. This observed discordance between impairments and activity limitations and participation restrictions may, in part, be explained by differences in cognitions, emotions, or coping strategies. The behavioral approach, in particular, should not be used to "blame" people with disabilities for those disabilities. The behavioral approach does not support the idea that disability arises because an individual lacks the motivation to overcome their impairments and limitations. Rather, the behavioral model emphasizes that every person is influenced by biological, personal, social, and environmental factors, and those influences are unique to each individual. Indeed, using behavioral models to conceptualize the personal factors component of the ICF supports the aim of the WHO to account for activity and activity limitations in the same terms for all individuals. Within this integrative framework, it is only the relative importance of each factor that differs between people, not the nature of the factors per se. For example, compared to the significant role of impairment, the role of motivational factors is likely to be a much weaker determinant of whether or not a person who has just had a stroke leaves their home to walk into town to visit the cinema. However, over the course of their recovery, the role of impairment factors might reduce and the role of motivational factors might increase, so that 6 months after their stroke, the individual might not walk into town to visit the cinema simply because there are no movies they want (are motivated) to see.

Measuring Disability

Clinical practice and research requires methods of measurement of disability so that the severity of a health condition can be assessed and the effectiveness of interventions evaluated. The ICF provides detailed descriptions of the body structures and functions and activities that should be assessed for any given health condition. However, the ICF does not indicate how those structures, functions, or activities should be assessed. In general, disability is measured by assessing the ability of an individual to perform particular activities relevant to their health conditions. For example, a person who has experienced a stroke might be assessed for their ability to perform activities of daily living, such as the ability to dress, to use the stairs, and to transfer from bed to chair, whereas a person with a diagnosis of dementia might be asked to complete measures of cognitive function.

In general, two methods of measurement are available: self-report and observation. Self-report requires the individual to describe the limitations and difficulties they experience. Self-report measures typically use standard questionnaires, for example, activities of daily living can be measured by a wide variety of instruments, including the Barthel Index, the Sickness Impact Profile (and its UK equivalent the Functional Limitations Profile), and the Katz ADL scale. Self-report measures have the advantage of being suitable for use in a variety of settings, including the person's own home, they are inexpensive, and can assess a wide range of activities over a long time course. In addition, proxy reports are sometimes used; proxy reporters are usually the primary caregiver. However, both self- and proxy reports have the disadvantage of being open to reporting errors.

Observational measures require a trained observer to record whether an individual is able (or not) to successfully perform relevant and defined activities. Observational measures are regarded as being more accurate than self-report measures but have several disadvantages. They are restrictive, in that they typically assess only those activities performed in the limited setting of the hospital or in the limited period available for a home visit, and as such, they too might under or over estimate disability.

Self-report and observational measures can be supplemented by objective electronic measures, for example, pedometers provide step counts, and accelerometers measure activity in general. However, such devices might have restrictive utility for particular groups, for example, elderly people

might walk with a gait that fails to register accurately on pedometers. In addition, with the exception of a step count, these devices do not discriminate between particular behaviors, for example, they are not able to distinguish between the wide variety of activities of daily living measured by self-report instruments, and at best they can discriminate between walking, standing, sitting, and lying.

Information about the WHO-ICF can be found at: http://www.who.int/classifications/icf/en/ this site provides a detailed description of the ICF and contains a great beginner's guide to the ICF http://www.who.int/classifications/icf/training/icfbeginnersguide.pdf

The importance of regarding behavior as a primary health outcome is made very effectively by Professor Robert Kaplan (Kaplan 1990).

Cross-References

- ▶ Activities of Daily Living (ADL)
- ▶ Aging
- ▶ Chronic Disease Management
- ▶ Chronic Disease or Illness
- ▶ Efficacy Cognitions
- ▶ Functional Capacity, Disability, and Status
- ▶ Geriatric Medicine
- ▶ Gerontology
- ▶ Health Psychology
- ▶ Illness Cognitions and Perceptions
- ▶ Independent Living
- ▶ Measures of Quality of Life
- ▶ National Institute on Aging
- ▶ Quality of Life
- ▶ Self-care
- ▶ Self-management

References and Readings

Bandura, A. (1969). *Principles of behavior modification*. New York: Holt, Reinhart and Winston.

Dixon, D., & Johnston, M. (2010). Disability. In D. French, K. Vedhara, A. A. Kaptein, & J. Weinman (Eds.), *Health psychology* (pp. 317–328). Chichester: Blackwell.

Kaplan, R. M. (1990). Behavior as the central outcome in health-care. *American Psychologist, 45*, 1211–1220.

Michie, S., Abraham, C., et al. (2009). Effective techniques in healthy eating and physical activity interventions: A meta-regression. *Health Psychology, 28*(6), 690–701.

WHO. (2001). *International classification of functioning, disability and health: ICF*. Geneva: Author.

Disability Assessment

- ▶ Health Assessment Questionnaire

Disability-Adjusted Life Years (DALYs)

Marijke De Couck
Free University of Brussels (VUB),
Jette, Belgium

Definition

The disability-adjusted life year (DALY) has emerged in the international health policy lexicon as a measure of overall "disease burden." It is an expansion on a previous measure, namely, years of life lost (YLL), which did not take into account the impact of disability. DALYs for a disease or health condition are calculated as the sum of the years of life lost (YLL) due to premature mortality in the population and the years lost due to disability (YLD) for incident cases of the health condition: DALY = YLL + YLD (World Health Organization [WHO], 2010). The YLL correspond to the number of deaths multiplied by the standard life expectancy at the age at which death occurs. The basic formula for YLL is the following for a given cause, age, and sex:

$$YLL = N \times L$$

where:

- N = number of deaths
- L = standard life expectancy at age of death in years

L reflects the difference between the standard life expectancy at that age and age of death. Because YLL measure the incident stream of "lost years of life" due to deaths, an incidence perspective is also taken for the calculation of YLD. To estimate YLD for a particular cause in a particular time period, the number of incident cases in that period is multiplied by the average duration of the disease and a weight factor that reflects the severity of the disease on a scale from 0 (perfect health) to 1 (dead). The basic formula for YLD is the following:

$$YLD = I \times DW \times L$$

where:

- I = number of incident cases
- DW = disability weight
- L = average duration of the disease until remission or death (years)

One DALY can be thought of as one lost year of "healthy" life. The sum of these DALYs across the population, or the burden of disease, can be thought of as a measurement of the gap between current health status and an ideal health situation where the entire population lives to an advanced age, free of disease and disability (WHO, 2010).

Several countries and organizations are using DALYs to identify health priorities and cost-effective interventions and to allocate resources for health (The World Bank, 1993). Several treatments or medications can be compared by this measure, which has been done in a few studies (Renaud, Basenya, de Borman, Greindl, & Meyer-Rath, 2009). DALY also measures psychological factors (e.g., emotional, behavioral, cognitive, and social functions), which are considered in the weighted disability. DALY can be used to compare several kinds of interventions, like psychological versus pharmacological, as has been done by several studies (Heuzenroeder et al., 2004).

However, there are a few disadvantages of the DALY. It is a metric which is used to provide a single number to capture all of the health costs caused by a disease. One DALY could represent 1 year of life lost (due to early death), 1.67 years spent with blindness, 5.24 years with significant malaria episodes, 41.67 years spent with intestinal obstruction due to ascariasis (a parasite), or many possible combinations of these and other symptoms (Lopez, Mathers, Ezzati, Jamison, & Murray, 2006). Thus, the same amount of DALYs, though numerically identical, could represent very different disabilities over time, which may not be comparable.

Cross-References

- ▶ Disability
- ▶ Longevity
- ▶ Quality of Life
- ▶ Quality of Life: Measurement

References and Readings

Heuzenroeder, L., Donnelly, M., Haby, M. M., Mihalopoulos, M., Rossell, R., Carter, R., et al. (2004). Cost-effectiveness of psychological and pharmacological interventions for generalized anxiety disorder and panic disorder. *The Australian and New Zealand Journal of Psychiatry, 38*, 602–612.

Lopez, A. D., Mathers, C. D., Ezzati, M., Jamison, D. T., & Murray, C. J. L. (2006). *Global burden of disease and risk factors*. Washington, DC: World Bank. Chapter 1.

Renaud, A., Basenya, O., de Borman, N., Greindl, I., & Meyer-Rath, G. (2009). The cost effectiveness of integrated care for people living with HIV including antiretroviral treatment in a primary health care centre in Bujumbura, Burundi. *AIDS Care, 21*, 1388–1394.

The World Bank. (1993). *The World development report 1993. Investing in health*. Washington, DC: Author.

World Health Organization. (2010). *Global burden of disease (GBD)*. Accessed April 15, 2010, from http://www.who.int/healthinfo/global_burden_disease/en/index.html

Disasters and Health: Natural Disasters and Stress/Health

- ▶ Psychosocial Factors and Traumatic Events

Disclosure

Pamela S. King
Pediatric Prevention Research Center, Department of Pediatrics, Wayne State University School of Medicine, Detroit, MI, USA

Synonyms

Emotional disclosure

Definition

Disclosure has been defined as sharing of personal information with others through verbal communication or written expression of what individuals verbally reveal about themselves to others (including thoughts, feelings, and experiences).

Description

Disclosure can be a complex process. It is often difficult to know what, how much, when, and to whom to disclose. Many factors influence the decision to disclose information, including personality traits such as anxiety, impulsivity, and extraversion (see Costa & McCrae, 1992), as well as social norms, one's affect, goals, and previous experiences with disclosure, the behavior of the confidant, and one's relationship with the confidant. People often choose not to disclose for fear of negative consequences (e.g., embarrassing or hurting one's confidant, punishment, reduction in autonomy, rejection, harassment, discrimination). Although disclosure can result in negative outcomes, research suggests that disclosure can be beneficial in a variety of ways. Disclosure provides people with an opportunity to express their thoughts and feelings, to elicit social support, gain new coping strategies, and build intimacy in their personal relationships. Disclosure may also allow people to find meaning in traumatic experiences, and may promote personal growth and self-acceptance.

Disclosure has been investigated in a variety of contexts. Researchers have investigated disclosure of a "concealable stigmatized identity," such as mental illness, experiences of abuse or assault, epilepsy, or an HIV-positive diagnosis. In each of these cases, people have personal information that is socially devalued but is not apparent to others. Disclosure has been examined among people who have a chronic illness that is not readily apparent, such as cancer or diabetes. Researchers have also studied disclosure of health information to family, friends, and health care providers (e.g., parents disclosing HIV status to children, adolescents sharing diabetes management information with parents). Research within each of these domains has examined how people make decisions to disclose, how confidants react to disclosure, and how people are affected by their disclosure decisions.

Much of the disclosure literature has focused on the effects of written disclosure on health outcomes. These studies use a laboratory writing technique which typically involves random assignment to one of two (or more) groups: an experimental group that discloses emotional material, and a control group that does not. Both groups are asked to write about assignments for 3–5 days, for 15–30 min each day. Those assigned to the control conditions are asked to write about superficial topics. Those in the experimental group are asked to write about their thoughts and feelings about an important emotional issue that has affected them and their life. Writing for both groups is usually done in the laboratory, with no feedback given. This body of research suggests that disclosure of emotional events can have immediate effects on skin conductance, heart rate, and blood pressure. In addition, written disclosure about emotional issues can have long-term effects on both immune functioning and health outcomes. Disclosure may influence health in several ways. First, research suggests that nondisclosure is a form of inhibition that requires physiological work, reflected in autonomic and central nervous system arousal. Disclosing may reduce inhibition, thus reducing

autonomic and central nervous system arousal and facilitating better health outcomes. Disclosure may also lead to changes in cognitive processes, such as decreases in rumination or increases in mastery, self-acceptance, and self-concept, which in turn have benefits for health. Finally, disclosure may lead to better health by increasing social support. When individuals disclose, they may gain information from others about effective coping strategies or they may obtain emotional support, both of which could contribute to better health outcomes.

Recent research has examined a different form of disclosure – the link between adolescent disclosure to parents and adolescent health outcomes. Prior research identified parental monitoring as an important predictor of a variety of adolescent behaviors including risky sexual behavior, substance abuse, and poor adherence. Additional investigation, however, revealed that measures of parental monitoring were assessing how much knowledge parents had, rather than the way in which they obtained that knowledge. Adolescent disclosure may be the primary source of parents' knowledge about adolescents' activities. Research indicates that among adolescent disclosure, parent solicitation, and parent behavioral control, disclosure is the best predictor of delinquent behavior. Research is now exploring the role that adolescent disclosure to parents plays in explaining adolescent health outcomes, such as adherence to the type 1 diabetes regimen.

Cross-References

- ▶ Emotional Expression
- ▶ Expressive Writing and Health

References and Readings

Chaudoir, S. R., & Fisher, J. D. (2010). The disclosure process model: Understanding disclosure decision making and postdisclosure outcomes among people living with a concealable stigmatized identity. *Psychological Bulletin, 136*(2), 236–256.

Frattaroli, J. (2006). Experimental disclosure and its moderators: A meta-analysis. *Psychological Bulletin, 132*(6), 823–865.

Graybeal, A., Sexton, J. D., & Penneaker, J. W. (2002). The role of story-making in disclosure writing: The psychometrics of narrative. *Psychology and Health, 17*(5), 571–581.

Pachankis, J. E. (2007). The psychological implications of concealing a stigma: A cognitive-affective-behavioral model. *Psychological Bulletin, 133*(2), 328–345.

Pennebaker, J. W. (1995). *Emotion, disclosure, & health*. Washington, DC: American Psychological Association.

Smetana, J. G., Metzger, A., Gettman, D. C., & Campione-Barr, N. (2006). Disclosure and secrecy in adolescent-parent relationships. *Child Development, 77*(1), 201–217.

Stattin, H., & Kerr, M. (2000). Parental monitoring: A reinterpretation. *Child Development, 71*, 1072–1085.

Discrimination

▶ Stigma

Discrimination and Health

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

This term refers to deliberate inequalities in access to and in care for health conditions or to the unequal exposure to health risks as a function of people's ethnic or demographic background (age, gender, beliefs, skin color, etc.). This problem exists in both developed and developing countries. Furthermore, discrimination and health can even be seen on a more global level – the inequality in health care between rich and poor countries and the consequent disease burden carried by poor countries, where global economic and political forces sustain this. Numerous studies show differences between low and high socioeconomic status (SES) groups on health

conditions, and there is accumulating evidence linking actual and perceived discrimination with poor health conditions (Ahmed, Mohammed, & Williams, 2007; Williams & Mohammed, 2009). Importantly, physicians may even provide less medical advice to people who are less similar to them, i.e., of lower SES (Tschann, Adamson, Coates, & Gullion, 1988). This is doubly harmful since low SES people already have poorer health risks, and receiving less medical advice may add to their health risks. Both biological and psychosocial pathways may link discrimination to poor health (Ahmed et al., 2007). Biologically, low SES people may be exposed to poorer environments (poor sanitation, pollution), whose adverse health effects are clear – infectious diseases, pulmonary problems, and possibly cancers.

However, the mere exposure to environmental stressors (e.g., crowding, social violence) may add onto these biological agents, in contributing to either unhealthy behaviors (e.g., heavy alcohol consumption), or to psychophysiological processes such as stress-induced inflammation (Maes et al., 1998) or stress-induced DNA damage (Gidron, Russ, Tissarchondou, & Warner, 2006), eventually resulting in poorer health. Clear discrimination leading to health problems could be seen when urban or even country institutions marginalize certain parts of society (based on ethnicity or SES) by pressing them to reside in poorer and more health-risky areas of cities (Ahmed et al., 2007). One major challenge to research and of course to curb this severe problem is the assessment of discrimination. Its assessment can be done by analysis of legislative records (institutional discrimination), employment uptake as a function of ethnicity (controlling for education), and at an individual level. The latter includes various scales such as the perceived discrimination scale (Williams, Yu, Jackson, & Anderson, 1997), which assesses 10 daily aspects of discrimination (e.g., being treated as less intelligent or in a less courteous manner than others). This domain is a very important example where behavioral and social sciences interact with biomedical sciences for understanding and beginning to ameliorate such severe health problems at a macro level.

Cross-References

- ▶ Health Disparities
- ▶ Socioeconomic Status (SES)

References and Readings

Ahmed, A. T., Mohammed, S. A., & Williams, D. R. (2007). Racial discrimination & health: Pathways & evidence. *Indian Journal of Medical Research, 126*, 318–327.

Gidron, Y., Russ, K., Tissarchondou, H., & Warner, J. (2006). The relation between psychological factors and DNA-damage: A critical review. *Biological Psychology, 72*, 291–304.

Maes, M., Song, C., Lin, A., De Jongh, R., Van Gastel, A., Kenis, G., et al. (1998). The effects of psychological stress on humans: Increased production of pro-inflammatory cytokines and a Th1-like response in stress-induced anxiety. *Cytokine, 10*, 313–318.

Tschann, J. M., Adamson, T. E., Coates, T. J., & Gullion, D. S. (1988). Behaviors of treated hypertensive patients and patient demographic characteristics. *Journal of Community Health, 13*, 19–32.

Williams, D. R., & Mohammed, S. A. (2009). Discrimination and racial disparities in health: Evidence and needed research. *Journal of Behavioral Medicine, 32*, 20–47.

Williams, D., Yu, Y., Jackson, J. S., & Anderson, N. B. (1997). Racial differences in physical and mental health: Socioeconomic status, stress and discrimination. *Journal of Health Psychology, 2*, 335–351.

Disease Acuity

- ▶ Disease Severity

Disease Burden

Yori Gidron
Faculty of Medicine and Pharmacy
Free University of Brussels (VUB),
Jette, Belgium

Definition

The term “disease burden” is a term of major importance in medicine and behavioral medicine.

It refers to the intensity or severity of a disease and to its possible impact on daily life. Thus, it could be at times referring to both illness severity and impact, resulting in confusion between concepts. Concerning disease severity, this reflects the amount of pathology in a given illness. For example, disease burden in cancer may be indexed by level of a tumor marker – e.g., CA125 in ovarian cancer, or CEA in colon cancer. Additionally, tumor stage could reflect disease burden as well. In coronary artery disease (CAD), this can be indexed by the number of arteries occluded over 50%, or the percentage of occlusion in a given artery (e.g., 40%, 99%). In infectious diseases, disease burden could reflect the amount of viral load in HIV patients. Concerning a disease's impact, disease burden can be measured by its effects on longevity or on quality of life (QOL). An illness with a high disease burden would thus impair one's psychological, physical, or social functioning aspects of QOL. For example, a patient with severe CAD could have debilitating chest pain which reduces his or her mobility, thus impairs work, elicits anxiety and depressive reactions, and reduces his or her social contacts with friends, family, and work colleagues.

In behavior medicine, researchers often take into account disease burden as a covariate, when testing the relation between a psychological factor and recovery or prognosis, independent of disease burden. For example, Denollet et al. (1996) showed that the Type-D personality (high distress and social inhibition) predicted mortality in coronary heart disease, independent of disease burden indexed by number of diseased vessels and left ventricular functioning. In a study on patients' recovery from coronary bypass surgery, Scheier et al. (1989) found that patients' trait optimism predicted recovery, independent of disease burden indexed by extent of surgery and number of occluded vessels. Hundreds of studies examine disease burden by assessing its impact on QOL and general well-being. A few standardized disease burden indices exist including the Charlson Index and the index of coexistent disease (Greenfield, Aronow, Elashoff, & Watanabe, 1988). The former only considers the number and severity of comorbid diseases, while the latter additionally considers patients' functional status in 12 categories (e.g., feeding, mental status, vision, respiration). Another unique disease burden index is the Smith index, which considers in a formula with weights a patient's emergency room visits during 6 months, blood urea nitrogen value, arterial pO_2, total white blood cell count, and presence of anemia. These indices can enable researchers in behavior medicine to assess disease burden in a standardized manner across patient conditions, and then examine the role of psychosocial factors in prognosis, independent of disease burden estimates. Finally, one may also estimate the impact of such disease burden indices on psychological functioning.

References and Readings

Denollet, J., Sys, S. U., Stroobant, N., Rombouts, H., Gillebert, T. C., & Brutsaert, D. L. (1996). Personality as independent predictor of long-term mortality in patients with coronary heart disease. *Lancet, 347*, 417–421.

Greenfield, S., Aronow, H., Elashoff, R., & Watanabe, D. (1988). Flaws in mortality data: The hazards of ignoring comorbid disease. *Journal of the American Medical Association, 260*, 2253–2255.

Scheier, M. F., Matthews, K. A., Owens, J. F., Magovern, G. J., Sr., Lefebvre, R. C., Abbott, R. A., et al. (1989). Dispositional optimism and recovery from coronary artery bypass surgery: The beneficial effects on physical and psychological well-being. *Journal of Personality and Social Psychology, 57*, 1024–1040.

Disease Management

Harry Prapavessis
University of Western Ontario, London, ON, Canada

Synonyms

Chronic care; Integrated health care; Managed care

Definition

Disease management is a patient centered, integrative health care intervention approach for managing the signs and symptoms of chronic diseases in defined populations of individuals. The main aims of disease management interventions are to optimize care, improve quality of life, and reduce costs associated with treating chronic conditions by coordinating patient care using a multidisciplinary approach.

Description

Disease management came about as a response to a changing health landscape wherein the prevalence and complexity of chronic conditions is on the rise requiring treatment regimens to adjust. With traditional approaches, patient information is spread across practitioners and specialists who are responsible for disparate aspects of patient care and respond in reactive ways to acute conditions. In contrast, disease management approaches plan for the long-term management of chronic conditions by reducing fragmentation and improving the continuity and coordination of the patient care process. This strategy is predicated on the understanding that improvements in medical therapy and self management can reduce the signs and symptoms of disease and improve treatment outcomes.

Disease management interventions are based in both primary care and private sector operations. While each of these shares the common goal of improving the health of the patient (e.g., increase functional ability, improve health status, reduce hospitalization time, and increase care compliance with treatment plans) and decreasing associated costs, different approaches are taken. Primary care interventions are integrated into the health care delivery system and focus mainly on communication with patients and between professionals (e.g., physicians, pharmacists, nurses, nurse practitioners, physical therapists). Private sector interventions are adjunct to the health care system (e.g., employers and insurers) and mainly focus on cost containment.

Three common key agents of change emerge in disease management literature: the patient, the health care team, and the environment. The patient is viewed as an active agent in his or her own care. It is up to the individual to seek information and be motivated to carry out the treatment plan. As such, self-management (► Self-management) interventions are hypothesized to affect outcomes (e.g., health status and health care use) by producing behavior change. There are many modifiable lifestyle factors that can be adopted by the patient to prevent or minimize the effects of disease (e.g., adherence to treatment regimens, physical activity, proper nutrition, medical screening, limited sun exposure, and substance avoidance). For example, epidemiological studies reveal that people who are more physically active have a lower risk of certain cancers than those who are sedentary. Evidence of self management interventions consistently demonstrates self-efficacy, and knowledge and skills are important mediators to target behavior change interventions.

The role of the health care team in disease management is to monitor conditions and to create a treatment plan, either alone or with the patient and health care team. A key challenge for the health care professional is to keep abreast of advancing knowledge and expertise necessary to care for patients. Education, decision-making support, time, and resources are targets for disease management intervention that should result in changed attitudes or behavioral intention, which is hypothesized to lead to professional behavior change. Ultimately, professionals who have increased expertise, knowledge, and support should provide higher quality treatment thus improving health outcomes.

Interventions that target the health care environment often focus on organizational design and relational coordination. Effective relational coordination interventions often involve the assembly of a coordinated (coordination within, across, and between care teams and community resources) multidisciplinary patient care team. Effective organizational design interventions often address task allocation, information transfer, appointment scheduling, and case management. These strategies are based on the belief that seamless care delivery (e.g., structural, financial, and

functional) and collaboration among diverse care providers (e.g., nurses, nurse practitioners, dieticians, social workers, physicians, physician assistants, and public health workers) will improve health and fiscal outcomes.

Although there is some evidence to support various disease management strategies, definitive conclusions about the overall effectiveness on patient outcomes and cost reduction cannot yet be made and will require further exploration.

Cross-References

- ▶ Adherence
- ▶ Aerobic Exercise
- ▶ Behavior Change
- ▶ Behavior Modification
- ▶ Self-management

References and Readings

http://www.carecontinuum.org/

Singer, S., Burgers, J., Friedberg, M., Rosenthal, M., Leape, L., & Schneider, E. (2010). Defining and measuring integrated patient care: Promoting the next frontier in health care delivery. *Medical Care Research and Review, 68*(1), 112–127.

Wagner, E. H. (2000). The role of patient care teams in chronic disease management. *British Medical Journal, 320*(7234), 569–572.

Disease Manifestation

- ▶ Disease Onset

Disease Onset

Steven Gambert
Department of Medicine, School of Medicine, University of Maryland, Baltimore, MD, USA

Synonyms

Disease manifestation

Definition

A disease is a medical condition that is considered to be abnormal, impairs bodily functions, and is associated with specific signs and symptoms. The disease onset is the first time that there has been noted to be a "change" in one's usual health status with the identified signs and/or symptoms being able to be directly attributable to a specific disease process.

Description

Diseases may be identified as having an acute, subacute, or chronic onset with symptoms developing over a wide variety of time from minutes to months. Certain diseases will be easier to identify as to their exact time of origin, or disease onset. An example would be the onset of nausea and vomiting from a food-borne toxin that has been ingested. Other diseases are much harder to characterize as to their onset. Alzheimer's disease, for example, is rarely identified until it has reached clinical significance at which time most individuals and their close contacts on more careful analysis can identify early warning signs that appeared years earlier and likely marked the onset of the disease. While symptoms may present in a classic manner, such as chest pain accompanying the onset of a myocardial infarction, at times, the presentation is atypical/nonspecific, making the diagnosis as to disease onset challenging. In the elderly, for example, it is not uncommon for a myocardial infarction to present with no chest pain but rather shortness of breath being the initial symptom at disease onset.

There are many examples of diseases that have variable disease onset. One such example is coronary artery disease. Individuals who present with coronary artery disease prior to age 50 are more likely to have a genetic predisposition; individuals affected later in life may have additional risk factors including dietary influences and coexisting diseases such as hypertension. Another example of a disease with a variable disease onset is Alzheimer's disease. Individuals affected prior to age 65 are

said to have a presenile dementia of the Alzheimer's type and are more likely to have a genetic predisposition for this earlier onset. Individuals affected after age 65 are said to have senile dementia of the Alzheimer's type (SDAT) (Bertram & Tanzi, 2008).

Cross-References

- ▶ Acute Disease
- ▶ Alzheimer's Disease

References and Readings

Bertram, L., & Tanzi, R. E. (2008). Thirty years of Alzheimer's disease genetics: The implications of systematic meta-analyses. Nature reviews. *Neuroscience, 9*(10), 768–778.

Disease Severity

Steven Gambert
Department of Medicine, School of Medicine, University of Maryland, Baltimore, MD, USA

Synonyms

Disease acuity; Disease severity index

Definition

Disease severity is a term used to characterize the impact that a disease process has on the utilization of resources, comorbidities, and mortality. It is often used by funding agencies to determine what is an appropriate payment for hospitalization or nursing home payments based on Diagnosis-Related Groups or RUGS (Resource Utilization Groups). There are several "severity indexes" that may be used to quantify the severity of illness such as the Glascow Coma Scale for assessing the severity of cognitive dysfunction or the Mini-Mental Examination that quantifies the degree of dementia.

Disease Severity Index

- ▶ Disease Severity

Disinhibition

- ▶ Behavioral Inhibition
- ▶ Impulsivity

Disparities

- ▶ Health Disparities

Dispersion

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Scatter; Variability

Definition

The spread, or dispersion, of a group of numbers around a central value is an important characteristic of a data set. It can be calculated in various ways. The range, the simplest measure of dispersion, is the arithmetic difference between the largest (maximum) value and the smallest (minimum) value. However, while it can be useful in initial "visual inspections" of a data set, this measure of dispersion is only a rough guide to the amount of variation present. Because it only takes into account two values from a data set, it utilizes a (potentially very) small part of the information available.

Imagine a data set containing 100 numbers. When calculating the range, only two of these numbers would be used. In other words, 98% of the available information would not be used in deriving this measure of dispersion. Consider a hypothetical data set of 100 numbers where the minimum value is 20, the maximum value is 80, and all of the 98 other numbers lie between 55 and 75. Now consider a second hypothetical data set of 100 numbers where the minimum value is again 20, the maximum value is again 80, but the other 98 numbers are spread out between 25 and 55. While the range would be identical in both cases (i.e., 60), it is intuitive that the overall natures of the two sets of numbers are quite different.

Two more sophisticated measures of dispersion for a data set are its variance and its standard deviation. These measures are intimately related to each other and take account of all values in the data set.

Cross-References

- ▶ Standard Deviation
- ▶ Variance

Disposition

- ▶ Personality

Dispositional Optimism

Lauren Zagorski
Department of Psychology, The University of Iowa, Iowa City, IA, USA

Synonyms

Optimism

Definition

Dispositional optimism is a stable personality trait characterized by general positive expectations that influence motivated action. When confronted with obstacles in achieving a desired future state, those who are optimistic anticipate positive outcomes from their actions. According to this self-regulatory model, optimism plays a role in negative feedback loops that guide goal-directed behavior. Consequently, optimistic individuals display a cross-situational tendency to enhance efforts toward their goals instead of disengaging and withdrawing efforts. It is also theorized that optimism is implicated in the propensity to attribute the cause of negative events as external and unstable. This explanatory style then influences future expectancies and behavior.

Various investigations of dispositional optimism have revealed a positive relationship with problem-focused and engagement coping strategies. This is the proposed pathway for optimism's potential benefits for well-being and adjustment to stressors. Dispositional optimism has been linked to positive psychological and physical outcomes among patients with chronic illnesses including cardiovascular disease, AIDS, and cancer.

Cross-References

- ▶ Life Orientation Test (LOT)
- ▶ Optimism and Pessimism: Measurement
- ▶ Self-regulation Model

References and Readings

Carver, C. S., & Scheier, M. F. (2002). Optimism. In C. R. Snyder & S. J. Lopez (Eds.), *Handbook of positive psychology* (pp. 231–243). New York: Oxford University Press.

Carver, C. S., Scheier, M. F., & Segerstrom, S. C. (2010). Optimism. *Clinical Psychology Review, 30*(7), 879–889. doi:10.1016/j.cpr.2010.01.006.

Scheier, M. F., & Carver, C. S. (1992). Effects of optimism on psychological and physical well-being: Theoretical overview and empirical update. *Cognitive Therapy and Research, 16*(2), 201–228. doi:10.1007/BF01173489.

Dispositional Pessimism

▶ Optimism, Pessimism, and Health
▶ Pessimism

Dissemination

Wynne E. Norton
Department of Health Behavior, School of Public Health, University of Alabama at Birmingham, Birmingham, AL, USA

Synonyms

Diffusion; Implementation

Definition

Dissemination refers to "the targeted distribution of information and intervention materials to a specific public health or clinical practice audience," whereas *implementation* refers to "the use of strategies to adopt and integrate evidence-based health interventions and change practice patterns within specific settings" (National Institutes of Health [NIH], 2010).

Broadly speaking, dissemination and implementation science (D&I) is focused on bridging the research-to-practice gap in health care and public health. The overall objectives of D&I research are to understand barriers toward the effective use of evidence-based health interventions, programs, practices, and treatments in health care and public health and, importantly, to create and test strategies to move such health innovations into everyday settings more quickly, effectively, and broadly.

D&I science is highly interdisciplinary, drawing on expertise from systems science, psychology, sociology, health services research, organizational behavior, and clinical research, among other fields. The field has witnessed considerable growth, expansion, and interest among researchers, policymakers, and practitioners in the United States and international settings in the past decade. This has included the emergence of speciality journals (e.g., Implementation Science), conferences (e.g., National Institutes of Health Conference on the Science of Dissemination and Implementation), review panels, funding announcements, and training programs (e.g., Implementation Research Institute).

References and Readings

Dearing, J. W. (2008). Evolution of diffusion and dissemination theory. *Journal of Public Health Management and Practice, 14*(2), 99–108.
Green, L. W., Ottoson, J. M., Garcia, C., & Hiatt, R. A. (2009). Diffusion theory and knowledge dissemination, utilization, and integration in public health. *Annual Review of Public Health, 30*, 151–174.
Greenhalgh, T., Robert, G., Macfarlane, F., Bate, P., & Kyriakidou, O. (2004). Diffusion of innovations in service organizations: Systematic review and recommendations. *The Milbank Quarterly, 82*(4), 581–629.
Implementation Science. (2011). www.implementationscience.com
Implementation Research Institute. (2011). http://cmhsr.wustl.edu/Training/IRI/Pages/ImplementationResearchTraining.aspx
National Institutes of Health. (2010). *Program announcement: Dissemination and implementation research in health (R01)*. Retrieved from http://grants.nih.gov/grants/guide/pa-files/PAR-10-038.html
NIH Conference on the Science of Dissemination and Implementation. (2011). http://conferences.thehillgroup.com/obssr/DI2011/about.html
Proctor, E. K., Landsverk, J., Aarons, G., Chambers, D., Glisson, C., & Mittman, B. (2009). Implementation research in mental health services: An emerging science with conceptual, methodological, and training challenges. *Administration and Policy in Mental Health, 36*(1), 24–34.
Rabin, B. A., Brownson, R. C., Haire-Joshu, D., Kreuter, M. W., & Weaver, N. L. (2008). A glossary for dissemination and implementation research in health. *Journal of Public Health Management and Practice, 14*(2), 117–123.

Dissemination and Implementation

▶ Research to Practice Translation

Distant Intercessory Prayer

Kevin S. Masters
Department of Psychology, University of Colorado, Denver, CO, USA

Definition

Distant intercessory prayer is simply defined as prayer said on behalf of someone else when that person is not present. This is different from intercessory prayer in which prayer is also said on behalf of someone else but the person being prayed for is present during the prayer.

Description

Intercessory prayer is the age-old practice of praying for someone else. The first empirical study on this topic was conducted by Sir Francis Galton and published in 1872. Galton demonstrated that individuals who were often prayed for, in this case, members of the royal family, did not live longer than others. In the context of modern behavioral medicine, intercessory prayer still usually consists of prayer for improvement in health status or healing. One could serve as one's own intercessor (as when praying for oneself) or could be the recipient of prayers from others, i.e., intercessors. Prayers said by oneself or by others in the presence of the recipient of the prayer could theoretically be effective in improving health through a number of mechanisms including not only the actual prayer itself (i.e., divine intervention or some type of beneficial energy) but also via social support or other naturalistic psychological mechanisms. Distant intercessory prayer, however, occurs when the intercessor is not physically present with the recipient of the prayer. In this way, a more stringent test of the effects of prayer per se can be tested. Further, some studies in this area used blinding strategies wherein patients did not know whether they were in the prayer or no-prayer group, and other studies went even further and included patients who did not even know they were included in a prayer research project.

Empirical research on the effects of distant intercessory prayer has been carried out with patients experiencing many different disorders including various forms of cardiac disease, leukemia, mental health problems, renal failure, rheumatoid arthritis, infertility, sepsis, and alcohol abuse. Reviews and meta-analyses were conducted by Masters and colleagues (Masters & Spielmans, 2007; Masters, Spielmans, & Goodson, 2006) and the more recently published Cochrane Collaboration Review (Roberts, Ahmed, Hall, & Davison, 2011). Each of these analyses found no credible evidence that distant intercessory prayer was associated with a beneficial overall effect. The authors of the Cochrane report carefully pointed out that their review of the empirical data in no way addresses metaphysical questions regarding the existence of God or any deity. Similarly, Masters (2005) argued that distant intercessory prayer studies lack a strong theological basis and also produce noninterpretable findings because, critically, they are not able to control the amount of prayer delivered to or for the no-prayer control group. That is, when a group of researchers assigns individuals to a no-prayer control group, it only means that the researchers or their chosen intercessors will not pray for those individuals. This, however, does nothing to stop family, close friends, health-care professionals, or others from praying for them. There has never been a reason postulated that suggests the prayers of intercessors in a prayer study would be more effective than those of others in the patients' experiential world. Many other questions remain unaddressed in these studies including qualifications of intercessors, theoretical understanding of both significant and nonsignificant findings, within group variations in outcomes, and any firm theory or even coherent hypothesis to offer an explanation for the expected effects. One study (Benson et al., 2006) even suggested that individuals undergoing coronary artery bypass grafting who knew they were receiving intercessory prayer had a higher incidence of complications.

Finally, in some studies, when significant findings were found, they (a) were not on the variables that were the object of prayer and (b) occurred in the absence of controls for multiple comparisons.

It bears repeating, however, that the argument here is not against the conduct of intercessory prayer, distant or present, by those who believe in such practices. Rather, it is an argument that empirical studies have not demonstrated an overall superiority for groups in prayed for versus not prayed for groups and that, because of the inherent methodological limitations of such research, these studies are actually unable to test their central idea/hypothesis and thus will never render a definitive database upon which to ascertain the health effects of intercessory prayer.

Cross-References

► Prayer

References and Readings

Benson, H., Dusek, J. A., Sherwood, J. B., Lam, P., Bethea, C. F., Carpenter, W., et al. (2006). Study of the therapeutic effects of intercessory prayer (STEP) in cardiac bypass patients: A multicenter randomized trial of uncertainty and certainty of receiving intercessory prayer. *American Heart Journal, 151*, 934–942.

Galton, F. (1872). Statistical inquiries into the efficacy of prayer. *Fortnightly Review, 12*, 125–135.

Masters, K. S. (2005). Research on the healing power of distant intercessory prayer: Disconnect between science and faith. *Journal of Psychology and Theology, 33*, 268–277.

Masters, K. S., & Spielmans, G. I. (2007). Prayer and health: Review, meta-analysis, and research agenda. *Journal of Behavioral Medicine, 30*, 329–338.

Masters, K. S., Spielmans, G. I., & Goodson, J. T. (2006). Are there demonstrable effects of distant intercessory prayer? A meta-analytic review. *Annals of Behavioral Medicine, 32*, 337–342.

Roberts, L., Ahmed, I., Hall, S., & Davison, A. (2011). *Intercessory prayer for the alleviation of ill health (Review). The Cochrane Collaboration*. New York: John Wiley and Sons.

Distraction (Coping Strategy)

Lara Traeger
Behavioral Medicine Service, Massachusetts General Hospital/Harvard Medical School, Boston, MA, USA

Synonyms

Behavioral disengagement; Diversion; Mental disengagement

Definition

Distraction refers to a classification of coping strategies that are employed to divert attention away from a stressor and toward other thoughts or behaviors that are unrelated to the stressor. In both adult and pediatric populations, distraction (for example, focusing on an external object or imagining a peaceful place) may be used to deal with pain and discomfort during medical procedures. Other examples of distraction include daydreaming or engaging in substitute activities to keep one's mind from ongoing stressors related to a chronic illness. There are many ways to group coping strategies together. For instance, distraction has been considered a type of emotion-focused coping (Lazarus and Folkman, 1984), which involves minimizing the emotional distress related to a stressor. Distraction has also been categorized as passive coping, a type of coping that has been associated with helplessness, avoidance, and poorer psychological outcomes in the long term. Examples of distraction that reflect a sense of helplessness or avoidance are statements such as "I try to think about anything else but my illness because I've quit trying to deal with it" and "I can't think about my illness so I've been watching television or movies to escape."

Cross-References

- ▶ Coping
- ▶ Passive Coping Strategies

References and Readings

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.

Distress

- ▶ Mental Stress
- ▶ Psychological Stress
- ▶ Stress

Distressed Personality Type

- ▶ Type D Personality

Disuse Atrophy

- ▶ Sarcopenia

Diuretic

Nicole Brandt[1] and Rachel Flurie[2]
[1]School of Pharmacy, University of Maryland, Baltimore, MD, USA
[2]University of Maryland, Baltimore, MD, USA

Synonyms

Fluid pill; Water pill

Definition

A diuretic is a drug that increases the rate of urine flow. As a drug class, the major aim of diuretics is to decrease extracellular fluid volume, and their major site of action is the kidneys. Secondarily, diuretics increase extracellular sodium excretion. Different classes of diuretics work by different mechanisms in the kidneys, and they alter cation and anion levels in the body as well as renal hemodynamics.

The main classes of diuretics include carbonic anhydrase inhibitors, loop diuretics, thiazide diuretics, potassium-sparing diuretics, and osmotic diuretics. Carbonic anhydrase inhibitors (e.g., acetazolamide, dichlorphenamide, and methazolamide) work at the proximal convoluted tubule of the nephron, where carbonic anhydrase plays a role in sodium bicarbonate reabsorption. By inhibiting sodium bicarbonate reabsorption, these drugs cause sodium bicarbonate and water excretion. Carbonic anhydrase inhibitors are used clinically to treat severe acute glaucoma because their effect of decreasing bicarbonate reabsorption leads to decreased bicarbonate secretion in the eye and lowers intraocular pressure.

Loop diuretics (e.g., furosemide, bumetanide, ethacrynic acid, and torsemide) work at the thick ascending loop of henle and inhibit the sodium-potassium-chloride symport, thereby increasing excretion of ions and water. Clinically they are used to quickly get rid of edema in heart failure, ascites, and pulmonary edema.

Thiazides (e.g., hydrochlorothiazide, thiazide, chlorthalidone, indapamide, and metolazone) work at the distal convoluted tubule and inhibit the sodium-chloride symport. They are used mainly in hypertension because they lower blood pressure and to a lesser extent to reduce edema.

Potassium-sparing diuretics act by two different mechanisms. Spironolactone and eplerenone antagonize aldosterone in the collecting tubules while amiloride and triamterene block the epithelial sodium channels in the collecting tubules.

They all increase sodium excretion and decrease potassium and hydrogen excretion. These drugs may be used in combination with loop or thiazide diuretics to counterbalance the potassium wasting of those drug classes. The ones that affect aldosterone are also used in heart failure.

Osmotic diuretics (glycerine, isosorbide, mannitol, and urea) cause excess water excretion from intracellular compartments thereby increasing urine volume and renal blood flow. They are used to maintain high urine flow in certain clinical situations (e.g., severe hemolysis or rhabdomyolysis) and can be used to reduce intraocular or intracranial pressure.

There are multiple types of diuretics whose clinical indications can vary depending on the patient's comorbidities. It is imperative with this class of medications to weigh the impact not just on efficacy but also how these medications can impact their quality of life due to issues with urinary frequency and possibly incontinence.

References and Readings

Brunton, L. L., Chabner, B. A., & Knollmann, B. C. (2010). *Goodman & Gilman's the pharmacological basis of therapeutics* (12th ed.). New York: McGraw-Hill Professional.

Trevor, A. J., Katzung, B. G., & Susan, B. (2010). *Masters: Pharmacology: Examination & board review* (9th ed.). New York: McGraw-Hill Medical.

Diurnal Mood Variation

Brant P. Hasler
Psychiatry, Western Psychiatric Institute and Clinic University of Pittsburgh School of Medicine, Pittsburgh, PA, USA

Synonyms

Daily mood variation; Diurnal rhythms in mood; Mood variability

Definition

The term *diurnal mood variation* is most commonly used in the context of the symptomatology of mood disorders, referring to noticeable diurnal (daily) changes in overall mood state experienced by some individuals suffering from depression. Historically, the specific patterns of these diurnal mood changes were thought to characterize various subtypes of depression, although empirical evidence for this has been mixed. Diurnal mood variation has been most closely linked to melancholic depression (also known as endogenous or somatic depression), which was thought to be characterized by a pattern of feeling worst (most depressed) upon awakening in the morning, than feeling progressively better as the day continues into the afternoon and evening. The opposite (atypical) pattern – feeling best in the morning, then worsening over the course of the day – was considered to be less common, and thought to characterize atypical (or nonendogenous) depression. Diurnal patterns in mood have also been noted in healthy individuals, and a circadian rhythm component to mood is now well established, although the specific term "diurnal mood variation" is less commonly used in this context.

Description

In the context of depression, diurnal mood variation is typically assessed by asking the patient to retrospectively describe the pattern, either during the course of a clinical interview or as an item on a questionnaire (e.g., Hamilton Rating Scale for Depression). Efforts to assess diurnal mood variation prospectively, both within and across days, suggest that the presence and direction (morning worse vs. evening worse) of diurnal mood variation are highly unstable over time, and vary independently of overall depressive symptoms (Gordijn, Beersma, Bouhus, Reinink, & Van den Hoofdakker, 1994). The instability of diurnal mood variation, as well as poor agreement between assessment approaches, suggests that caution is warranted when interpreting

diurnal mood variation as an indicator of depression subtype (e.g., melancholic vs. atypical). This caution was underscored by a study of 37 patients with major depression that found no relationship between pattern of diurnal mood variation and either typical (i.e., weight loss and insomnia) or atypical (i.e., weight gain and hypersomnia) depressive symptoms (Leibenluft, Noonan, & Wehr, 1992). Likewise, a much larger dataset of 3,744 outpatients with major depression from the STAR*D study also suggested a need to revise the conventional wisdom regarding links between specific patterns of diurnal mood variation and depression subtypes (Morris et al., 2007). Specifically, nearly a quarter of patients reported diurnal mood variation, but the majority of these patients (48.6%) reported evening worsening in mood, with only 31.9% or 19.5% reporting morning or afternoon worsening in mood, respectively. Morris and colleagues also reported that any diurnal mood variation, rather than morning worsening, per se, increased the likelihood of having melancholic symptoms of depression.

Diurnal mood variation is thought to have clinical relevance as a predictor of treatment response, although the empirical evidence for this is mixed. Early studies have suggested that morning worse morning mood predicted favorable treatment responses to total sleep deprivation and tricyclic antidepressants. In contrast, more recent studies indicated that greater mood variability, rather than any specific pattern of diurnal mood variation, is associated with improved response to total sleep deprivation treatment (Gordijn et al., 1994), and that diurnal mood variation does not predict treatment response to selective serotonin reuptake inhibitors (SSRIs) (Morris et al., 2007).

Although the classic conception of diurnal mood variation has thus far proven to have arguable utility, more sophisticated investigations of diurnal patterns in mood may still provide important insights into the pathophysiology of depression. These studies are distinguished from past research by using prospective designs along with more frequent within-day assessments to provide greater temporal resolution of mood variability. A number of studies have been influenced by advances in affective science, and thus used separate scales to assess positive and negative affect rather than using a unidimensional mood measure (Watson, 2000). These studies have generally reported that positive affect shows a 24-h rhythmic pattern, while negative affect lacks systematic daily variation. Positive affect is lowest in the early morning hours, rises throughout the day, to peak in the late afternoon and early evening, and declines during the night. Accumulating evidence indicates that the 24-h patterns in positive affect are due in part to endogenous circadian rhythms and not simply a reflection of affective responses to sociocultural rhythms in the environment (Boivin et al., 1997; Murray, Allen, & Trinder, 2002).

Diurnal rhythms in mood may be altered in depression, most commonly manifesting as a blunted peak in positive affect. In a study by Peeters, Berkhof, Delespaul, Routtenberg, and Nicolson (2006) depressed individuals reported lower peaks of positive affect, and these peaks occured later in the day, compared to healthy non-depressed individuals. The depressed group also reported decreased negative affect throughout the day, with greater moment-to-moment variability. (Negative affect in the healthy group did not follow a systematic pattern, consistent with previous reports.) In interpreting these results, the authors hearkened back to the classic definition of diurnal mood variation, noting that retrospective mood evaluation, via a unidimensional mood construct, likely hinged on an internal calculus of the varying combinations of positive and negative affect occurring throughout the day. A more recent study by Murray and colleagues (Murray, 2007) suggested that changes in the diurnal rhythms of positive affect are not only apparent in categorical comparisons of depressed and healthy individuals, but can also serve to characterize the severity of depression. Compared to a group with mild depression, the group with more severe depression had a less discernable rhythm in positive affect with a notably blunted peak, along greater overall negative affect throughout the day. Cumulative evidence indicates that altered circadian function may be present in depression.

Cross-References

- ▶ Circadian Rhythm
- ▶ Depression: Measurement
- ▶ Depression: Symptoms
- ▶ Depression: Treatment
- ▶ Hamilton Rating Scale for Depression (HAM-D)
- ▶ Mood

References and Readings

Boivin, D. B., Czeisler, C. A., Dijk, D. J., Duffy, J. F., Folkard, S., Minors, D. S., et al. (1997). Complex interaction of the sleep-wake cycle and circadian phase modulates mood in healthy subjects. *Archives of General Psychiatry, 54*(2), 145–152.

Gordijn, M. C., Beersma, D. G., Bouhus, A. L., Reinink, E., & Van den Hoofdakker, R. H. (1994). A longitudinal study of diurnal mood variation in depression; characteristics and significance. *Journal of Affective Disorders, 31*(4), 261–273.

Leibenluft, E., Noonan, B. M., & Wehr, T. A. (1992). Diurnal variation: Reliability of measurement and relationship to typical and atypical symptoms of depression. *Journal of Affective Disorders, 26*(3), 199–204.

Morris, D. W., Rush, A. J., Jain, S., Fava, M., Wisniewski, S. R., Balasubramani, G. K., et al. (2007). Diurnal mood variation in outpatients with major depressive disorder: Implications for DSM-V from an analysis of the sequenced treatment alternatives to relieve depression study data. *The Journal of Clinical Psychiatry, 68*(9), 1339–1347.

Murray, G. (2007). Diurnal mood variation in depression: A signal of disturbed circadian function? *Journal of Affective Disorders, 102*, 47–53.

Murray, G., Allen, N. B., & Trinder, J. (2002). Mood and the circadian system: Investigation of a circadian component in positive affect. *Chronobiology International, 19*(6), 1151–1169.

Peeters, F., Berkhof, J., Delespaul, P., Routtenberg, J., & Nicolson, N. A. (2006). Diurnal mood variation in major depressive disorder. *Emotion, 6*(3), 383–391.

Watson, D. (2000). *Mood and temperament*. New York: Guilford Press.

Wirz-Justice, A. (2008). Diurnal variation of depressive symptoms. *Dialogues in Clinical Neuroscience, 10*(3), 337–343.

Diurnal Rhythms in Mood

- ▶ Diurnal Mood Variation

Diversion

- ▶ Distraction (Coping Strategy)

Diversity

C. Andres Bedoya
Behavioral Medicine Service Department of Psychiatry, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA

Synonyms

Cultural competence; Heterogeneity; Multiculturalism

Definition

Diversity involves a difference between an individual or group in comparison to an established "norm" (Kato & Mann, 1996). This may be influenced by context and based on a number of factors including, but not limited to, ethnicity, race, culture, gender, age, sexual orientation, religion/spirituality, health status, disability, veteran status, or socioeconomic status (Jackson, 2006). Such factors are not mutually exclusive and may occur in any myriad of combinations.

Description

The field of behavioral medicine has made significant advances over the past three decades, providing a strong body of interdisciplinary evidence and theory to support the efficacy of applying research and practice for the promotion of health and prevention of illness (Belar & Deardorff, 2009; Smith & Suls, 2004). Similar to the medical field as a whole, however, these advances have historically focused on the experience of middle-class Euro-American white males and been based on a traditional Western

view of health (Kazarian & Evans, 2001; Smith, Kendall & Keefe, 2002).

Yet demographic diversity within the United States continues to increase in a number of areas (Jackson, 2006). For example, the Census Bureau noted that in 2008, ethnic and racial minorities consisted of approximately one-third of the population. Census projections indicate that by mid-century, these minority groups will continue to increase in number and represent slightly more than a majority of the population as a whole. Latinos, in particular, are projected to account for one in three of all Americans. Over this period, the average age is also expected to increase, resulting in a greater proportion of Americans who are older. Similarly, other diverse groups are projected to continue to grow, including the number of people who identify as members of sexual minority groups.

Diversity is a salient issue as it has a number of implications regarding the prevalence of illness and health-related disparities (Agency for Healthcare Research and Quality, 2009; Smith & Shuls, 2004). For example, compared to whites, African Americans experience significant health disparities in areas such as number of new cases of AIDS, diabetes-related lower extremity amputations, and lack of prenatal care within the first trimester of pregnancy. African Americans, Asian Americans, and Hispanics over the age 50 are significantly less likely to receive preventative screenings such as a colonoscopy or proctoscopy. Similarly, Hispanic and African Americans with depression are less likely than whites to receive mental health care.

An increasingly diverse demographic landscape will require that behavioral medicine adapt in order to appropriately address the needs of diverse groups (Belar & Deardorff, 2009). Through development of cultural competence skills, the field can better understand patients' sociocultural contexts, as well as recognize and appropriately respond to key cultural features. To this end, cultural competence involves development within three domains: self-awareness of one's own attitudes and beliefs; knowledge of the population of interest; and tools that can be applied with diverse groups (Jackson, 2006).

Similarly, cultural competence can be extended to involve a context competence (Smith & Suls, 2004). From this point of view, diversity must be addressed within all aspects of behavioral medicine – clinical practice, research, education, and policy.

This provides the opportunity to explore the ways that sociodemographic characteristics are linked to health, illness, and related behaviors (Smith, Kendall & Keefe, 2002). The call to address diversity within behavioral medicine provides the opportunity to establish generalization of prior findings, as well as explore within-group differences that are related to health-related behavior and health outcomes. Such competence will require added interdisciplinary collaboration, for example, with multicultural psychology, and involve other levels of systems such as members of a community or organization.

Cross-References

- ▶ Cultural and Ethnic Differences
- ▶ Cultural Competence
- ▶ Disability
- ▶ Discrimination and Health
- ▶ Ethnic Differences
- ▶ Gender Differences
- ▶ Health Disparities
- ▶ Income Inequality and Health
- ▶ Minority Health
- ▶ Religion/Spirituality
- ▶ Sexual Orientation
- ▶ Sociocultural Differences
- ▶ Socioeconomic Status (SES)

References and Readings

Agency for Healthcare Research and Quality. (2009, March). *National healthcare disparities report, 2008*. Retrieved March 1, 2011, from www.ahrq.gov/qual/qrdr08.htm

Belar, C. D., & Deardorff, W. W. (2009). *Clinical health psychology in medical settings: A practitioner's guidebook* (2nd ed.). Washington, DC: American Psychological Association.

Jackson, Y. (2006). *Encyclopedia of multicultural psychology*. Thousand Oaks, CA: Sage.

Kato, P. M., & Mann, T. (1996). *Handbook of diversity issues in health psychology*. New York: Plenum Press.
Kazarian, S. S., & Evans, D. R. (2001). *Handbook of cultural health psychology*. New York: Academic Press.
Office of Behavioral and Social Sciences Research, National Institutes of Health. http://obssr.od.nih.gov/scientific_areas/social_culture_factors_ in_health/index.aspx
Office of Minority Health, U.S. Department of Health and Human Services. http://minorityhealth.hhs.gov/
Smith, T. W., Kendall, P. C., & Keefe, F. J. (2002). Behavioral medicine and clinical health psychology: Introduction to the special issue, a view from the decade of behavior [Special issue]. *Journal of Consulting and Clinical Psychology, 70*(3), 459–462.
Smith, T. W., & Shuls, J. (2004). Introduction to the special section on the future of health psychology [Special issue]. *Health Psychology, 23*(2), 115–118.

Divorce and Health

Tamara Goldman Sher[1] and Kathryn Noth[2]
[1]The Family Institute at Northwestern University, Evanston, IL, USA
[2]Illinois Institute of Technology, College of Psychology, Chicago, IL, USA

Synonyms

Marital dissolution; Separation

Definition

Divorce is the legal dissolution of a marriage. It is simultaneously a legal and psychological process (Margulies & Luchow, 1992). Not all people are equally distressed by divorce, and a variety factors contribute to level of distress endured. The degree of distress that stems from the divorce process and from divorced marital status predicts mental and physical health in both the short and long term. Remarriage can reduce the health risks of divorce. Possible mechanisms underlying the relationship between divorce and health (e.g., psychophysiological mechanisms, immune functioning) have been explored.

Description

It has long been understood that marriage, or a stable, long-term intimate relationship, increases life expectancy, decreases morbidity, and enhances quality of life (Burman & Margolin, 1992). In fact, it has been noted that the evidence linking social relationships to health and mortality is as strong as that linking cigarette smoking, blood pressure, and obesity to health (Umberson, Williams, Powers, Liu, & Needham, 2006). However, it is also understood that not all marriages protect equally, and a poor relationship may be more health damaging than no relationship at all.

A number of studies have also found that being divorced is more health-damaging than being married (e.g. Hughes & Waite, 2009; Kiecolt-Glaser, Kennedy, Malkoff, & Fisher, 1988). In an early study on the effects of marriage versus divorce on immunity status among a sample of 32 married versus 32 divorced men, it was found that separated/divorced men were more distressed and lonelier and reported significantly more recent illness than did married men; the former also had significantly poorer values on two functional indices of immunity (antibody titers to two herpes viruses) while not differing significantly on quantitative indices (percentages of helper and suppressor cells and their ratio).

In understanding the complexity of the marital status/marital quality and health relationship, it is important to understand the differences between those who are divorced, those who remain married and those who remarry in terms of morbidity and mortality. In the same study of immunological functioning, it was found that among married men, poorer marital quality was associated with greater distress and poorer immunological functioning. Among separated/divorced subjects, those who had separated within the past year and who had initiated the separation were less distressed, reported better health, and had better immunological functioning than did noninitiators (Kiecolt-Glaser et al., 1988). Taken together, the results suggest that while being married can be advantageous in staying healthy (having

increased immunity against disease), the effects of marriage on health depend on the quality of that marriage. And, given a poor marriage, it is better to be the one initiating divorce than the one who is not the initiator when it comes to immune functioning. Other researchers have attempted clarify the psychophysiological mechanisms underlying the relationship between divorce and health. One study looked at divorce-related psychological adjustment and blood pressure in a sample of recently separated or divorced adults ($n = 70$). Individuals who reported higher degrees of divorce-related emotional distress demonstrated elevated resting blood pressure at study entry. The same study found that men who reported greater emotional difficulty associated with the divorce experience showed greater cardiovascular reactivity when thinking about the divorce (Sbarra et al. 2009).

A lot of the early research on health and marriage compared those who were married with those who were unmarried on health outcomes. Fewer studies have examined the effect of *changes* in marital status on either emotional or physical well-being. Two large sample studies, one of 8,809 people in the United States (Hughes & Waite, 2009) and one of over 5,000 people in Great Britain (Bennett, 2006), looked at transitions into and out of marriage. Results from both studies found a clear positive effect of marriage. It was also found that a change in marital status predicted health worsening (Bennett, 2006; Hughes & Waite, 2009) with a difference between those who were divorced and those who were widowed. Interestingly, for the newly divorced, health problems increased at the point of the divorce, while for the newly widowed, problems increased later in time, perhaps because of a cumulative stress effect (Bennett, 2006). Finally, results revealed strong and consistent effects of marriage on later health in that among those who have ever been divorced or widowed, the remarried generally show better health than those who have not remarried (Hughes & Waite, 2009).

Others have assessed whether findings that divorce is deleterious to health would hold-up to longer-term analyses. In a study focused on the immediate and the more long-term (10 years later) effects of divorce on women's health ($n = 244$), it was found that in the years immediately following their divorce, women reported significantly higher levels of psychological distress than married women; no differences in physical illness were found between groups (Lorenz, Wickrama, Conger, & Elder, 2006). A decade later, the divorced women reported significantly higher levels of illness, even after controlling for age, remarriage, education, income, and prior health. The authors concluded that as a stressor, divorce has a more acute effect on psychological health, while physical illness risk accumulates incrementally in response to the relatively stable dimensions of chronic stress over time. That is, the divorce *process* has more of an acute effect on the psychological health of women while *being divorced* is a chronic stressor, and illness can be understood as a cumulative response to the concomitant chronically stressful conditions (e.g., financial hardship, lack of social support).

In looking at the effects of marriage, marriage transition, and divorce, there are a number of confounding issues including gender effects, age effects, and time married effects that are beyond the scope of this entry. It is clear that divorce affects men and women differently and the young versus the old differently. However, the issue of the relative impact of marriage, divorce, and marriage transitions on the health of men versus women is not a clear one. While some studies have found differences between men and women (e.g., Dupre & Meadows, 2007), others have found no such differences (e.g., Williams, 2003) in health impact. In trying to reconcile these discrepant findings, some have looked at the possibility of different mechanisms underlying the effects for men versus women. A number of studies have associated the mortality disadvantage of divorce on women to the financial losses resulting from divorce, as opposed to the divorce itself (Lillard & Waite, 1995; Prigerson, Maciejewski, & Rosenheck, 1999; Wickrama, Conger, & Lorenz, 1995). In contrast, men seem to benefit from the sense of a "settled life" that marriage provides; they are less likely to engage

in risky health behaviors and experience gain from the household tasks taken over by women in marriage (Lillard & Waite, 1995).

In further trying to elucidate the underlying mechanisms in the relationship between divorce and health, some researchers have looked at divorce and illness prevention behaviors. However, rather than finding that it is a lack of illness prevention among the unmarried that leads to worse health outcomes, the opposite appears to be the case. A number of studies have in fact found that marriage *decreases* healthy behaviors such as weight control (Lee et al., 2005) and fitness levels (Ortega et al., 2011). These findings are consistent among the married versus the nonmarried and across the divorced versus remarried, with remarriage showing the same negative health trends as the continuously married men and women.

Thus, it remains to be determined why marriage is protective and divorce is harmful in terms of health outcomes overall. Other than general health effects, a number of studies have investigated more discrete health outcomes that result from marriage, divorce, and transitions. For example, there have been consistent findings that distressed marriages lead to more specific poor health outcomes such as coronary disease (Eaker, Sullivan, Kelly-Hayes, D'Agostino, & Benjamin, 2007; Zhang & Hayward, 2006), slow wound healing (Kiecolt-Glaser et al., 2005), and cardiac events (Orth-Gomer et al., 2000) compared to happier marriages. It has also been found that marital status determines specific health outcomes. For example, remarriage after divorce significantly reduces risk of COPD incidence, even after adjusting for smoking habit (Noda et al., 2009), and coronary heart disease mortality among divorced, widowed, and never married men and women is greater than among the married (Lindgarde, Furu, & Ljung, 1987; Weiss, 1973; Zhang & Hayward, 2006).

It remains a truism that being married or in another long-term intimate relationship is better for one's health than being single, widowed, or divorced for both general health and a number of specific health conditions. However, the reasons for these findings are not well understood. It is clearly not that married people take better care of themselves physically; in fact, the opposite seems to be the case. It is possible that the stress of divorce accounts for these differences but then again so does the stress of remaining in an unhappy relationship. For women, this stress seems to be primarily financial, while for men, this stress appears more task-oriented. It is also better to be remarried than to remain divorced or widowed, although the transitions into and out of marriage themselves seem to be harmful. Clearly, more information is needed to understand the complicated relationships between marital status and health, marital history and health, and marital quality and health.

Cross-References

- ▶ Immune Responses to Stress
- ▶ Marital Satisfaction
- ▶ Marriage and Health
- ▶ Psychophysiological

References and Readings

Bennett, K. M. (2006). Does marital status and marital status change predict physical health in older adults? *Psychological Medicine: A Journal of Research in Psychiatry and the Allied Sciences, 36*(9), 1313–1320.

Burman, B., & Margolin, G. (1992). Analysis of the association between marital relationships and health problems: An interactional perspective. *Psychological Bulletin, 112*(1), 39–63.

Dupre, M. E., & Meadows, S. O. (2007). Disaggregating the effects of marital trajectories on health. *Journal of Family Issues, 28*(5), 623–652.

Eaker, E. D., Sullivan, L. M., Kelly-Hayes, M., D'Agostino, R. B., & Benjamin, E. J. (2007). Marital status, marital strain, and risk of coronary heart disease or total mortality: The Framingham offspring study. *Psychosomatic Medicine, 69*(6), 509–513.

Hughes, M. E., & Waite, L. J. (2009). Marital biography and health at mid-life. *Journal of Health and Social Behavior, 50*(3), 344–358.

Kiecolt-Glaser, J. K., Kennedy, S., Malkoff, S., & Fisher, L. (1988). Marital discord and immunity in males. *Psychosomatic Medicine, 50*(3), 213–229.

Kiecolt-Glaser, J. K., Loving, T. J., Stowell, J. R., Malarkey, W. B., Lemeshow, S., Dickinson, S. L., et al. (2005). Hostile marital interactions, proinflammatory

cytokine production, and wound healing. *Archives of General Psychiatry, 62*(12), 1377–1384.
Lee, S., Cho, E., Grodstein, F., Kawachi, I., Hu, F. B., & Colditz, G. A. (2005). Effects of marital transitions on change in dietary and other health behaviors in US women. *International Journal of Epidemiology, 34*(1), 69–78.
Lillard, L. A., & Waite, L. J. (1995). Til death do us part: Marital disruption and mortality. *American Journal of Sociology, 100*(5), 1131–1156.
Lindgarde, F., Furu, M., & Ljung, B.-O. (1987). A longitudinal study on the significance of environmental and individual factors associated with the development of essential hypertension. *Journal of Epidemiology and Community Health, 41*(3), 220–226.
Lorenz, F. O., Wickrama, K. A. S., Conger, R. D., & Elder, G. H., Jr. (2006). The short-term and decade-long effects of divorce on women's midlife health. *Journal of Health and Social Behavior, 47*(2), 111–125.
Margulies, S., & Luchow, A. (1992). Litigation, mediation, and the psychology of divorce. *Psychiatry & Law, 20*(4), 483–504.
Noda, T., Ojima, T., Hayasaka, S., Hagihara, A., Takayanagi, R., & Nobutomo, K. (2009). The health impact of remarriage behavior on chronic obstructive pulmonary disease: Findings from the US longitudinal survey. *BMC Public Health, 9*, 412.
Ortega, F. B., Brown, W. J., Lee, D. C., Baruth, M., Sui, X., & Blair, S. N. (2011). In fitness and in health? A prospective study of changes in marital status and fitness in men and women. *American Journal of Epidemiology, 73*(3), 337–344.
Orth-Gomer, K., Wamala, S. P., Horsten, M., Schenck-Gustafsson, K., Schneiderman, N., & Mittleman, M. A. (2000). Marital stress worsens prognosis in women with coronary heart disease: The Stockholm Female Coronary Risk Study. *Journal of the American Medical Association, 284*(23), 3008–3014.
Prigerson, H. G., Maciejewski, P. K., & Rosenheck, R. A. (1999). The effects of marital dissolution and marital quality on health and health service use among women. *Medical Care, 37*(9), 858–873.
Sbarra, D. A., Law, R. W., et al. (2009). Marital dissolution and blood pressure reactivity: Evidence for the specificity of emotional intrusion-hyperarousal and task-related emotional difficulty. *Psychosomatic Medicine, 71*(5), 532–540.
Umberson, D., Williams, K., Powers, D. A., Liu, H., & Needham, B. (2006). You make me sick: Marital quality and health over the life course. *Journal of Health and Social Behavior, 47*(1), 1–16.
Weiss, N. S. (1973). Marital status and risk factors for coronary heart disease: The United States health examination survey of adults. *British Journal of Preventive & Social Medicine, 27*, 41–43.
Wickrama, K., Conger, R. D., & Lorenz, F. O. (1995). Work, marriage, lifestyle, and changes in men's physical health. *J Behavioral Medicine, 18*(2), 97–111.
Williams, K. (2003). Has the future of marriage arrived? A contemporary examination of gender, marriage, and psychological well-being. *Journal of Health & Social Behavior, 44*(4), 470–487.
Zhang, Z. M., & Hayward, M. D. (2006). Gender, the marital life course, and cardiovascular disease in late midlife. *Journal of Marriage and the Family, 68*(3), 639–657.

Dizygotic Twins

Jennifer Wessel
Public Health, School of Medicine, Indiana University, Indianapolis, IN, USA

Synonyms

Fraternal twins; Nonidentical twins

Definition

Dizygotic (DZ) twins are pairs of siblings resulting from the same pregnancy. They develop from two separate eggs that have each been fertilized by a different sperm. These siblings share, on average, 50% of their genes, as do ordinary full siblings. In contrast to monozygotic (MZ) twins, who are always same-sex pairs, DZ twins can be same-sex pairs or opposite-sex pairs.

The employment of opposite-sex pairs in twin studies allows assessments of whether genetic and shared environmental familial influences on behavior are different for males and females. If there are sex differences, the correlation for opposite-sex pairs will typically be lower than that for same-sex pairs.

Cross-References

▶ Monozygotic Twins
▶ Twin Studies

References and Readings

Elston, R. C., Olson, J. M., & Palmer, L. (2002). *Biostatistical genetics and genetic epidemiology* (1st ed.). Chichester: John Wiley and Sons.

Nussbaum, R. L., Mc Innes, R. R., & Willard, H. F. (2001). *Genetics in medicine* (6th ed.). Philadelphia, PA: W.B. Saunders.

Spector, T. D., Snieder, H., & MacGregor, A. J. (2000). *Advances in Twin and Sib-pair analysis* (1st ed.). London: Greenwich Medical Media.

DNA

Edward L. Perkins
Biomedical Sciences, Mercer University School of Medicine, Savannah, GA, USA

Synonyms

Genetic material

Definition

The acronym DNA is now so well known in popular as well as scientific literature that it often appears with no accompanying definition: The words deoxyribonucleic acid are rarely heard, but they are what the acronym represents. DNA is a very large molecule or macromolecule, with each word of its full name being descriptive of its nature. Ribose is one form of sugar (along with glucose, fructose, sucrose, and others). The prefix "deoxy-" specifies a ribose that has lost one of its oxygen atoms at a specific site in the molecule. Nucleic acids are a group of complex compounds derived from carbohydrates, purines and pyrimidines, and phosphoric acid.

Nucleic acids are found in all living cells, and also in viruses, which themselves are not actually "alive" until they hijack another cell's genetic material and make it work to their advantage. Nobel Laureate Sir Peter Medawar has captured this occurrence very well by calling a virus "a piece of nucleic acid surrounded by bad news" (cited by Bryson, 2004).

DNA molecules contain many copies of four bases: adenine (A), guanine (G), thymine (T), and cytosine (C). Each of these bases can be regarded as a molecule in its own right, being comprised of carbon, hydrogen, oxygen, and nitrogen atoms. Adenine and guanine are purines, chemical structures composed of two carbon rings, and thymine and cytosine are pyrimidines, which are composed of one ring of carbon atoms.

Purines and pyrimidines join together (or bond) with a deoxyribose molecule that also contains a phosphate group. The combination of A, G, T, and C with a deoxyribose molecule leads to the formation of four different nucleotides, two purine nucleotides (A and G) and two pyrimidine nucleotides (T and C). Hundreds of thousands of individual nucleotides can link together to form a polynucleotide strand. Each DNA molecule is comprised of two strands of nucleotides that are attached together. This molecular structure and the ensuing three-dimensional molecular geometry of DNA lead to its characteristic double helix nature. Once formed, single strands of DNA are matched with and then attached to another strand in a nonrandom manner governed by these rules: An adenine base can only be matched with and attached to a thymine base, and a guanine base can only be matched with and attached to a cytosine base, leading to A-T and G-C pairings. The term "complementary bases" reflects that each of the four nucleotide bases has a complementary base to which it becomes attached. A critical consequence of this arrangement is that once the sequence of nucleotides in one strand is known, the sequence of nucleotides in the other strand is known.

Replication, the process by which DNA produces an exact copy of itself, is facilitated by this phenomenon. The two polynucleotide chains that comprise a DNA molecule split apart from each other, and each then becomes attached to a newly formed chain, that is, an exact replicate of its original partner. This creates two identical DNA molecules, which can then continue to create replicates in an exponential manner.

The Relevance of Genetics for Behavioral Medicine

While this encyclopedia is not focused on molecular genetics, genetic inheritance is of considerable importance in behavioral medicine, and therefore the information conveyed by this entry and other related genetic entries is deserving of inclusion. In some cases, inheriting certain alleles can be the sole and readily identifiable cause of a disease. A case in point is phenylketonuria (PKU), which is inherited in an autosomal recessive manner. Those who inherit the disease do not have the ability to create an enzyme called phenylalanine hydroxylase, which metabolizes the amino acid phenylalanine, a component of many foods. Accumulating levels of phenylalanine are harmful to the central nervous system. Fortunately, a very strict diet low in phenylalanine provides successful treatment.

In contrast, for complex diseases of interest in behavioral medicine, a multi-gene etiology is typical, and it has proved very difficult to isolate and identify individual genes/alleles that are responsible for sizeable amounts of variation in the dispersion of a disease. What has become apparent, however, is that environmental (behavioral) influences are of great importance in many such complex disorders. Consider the case of alcoholism, since it well exemplifies the interesting phenomenon of disposition rather than predetermination. Clinical data suggest that, when inherited, the genes that underlie alcoholism liability confer a vulnerability to alcoholism expression rather than the certainty of it. Genetic inheritance of alcoholism liability can thus be highly sensitive to environmental modulation (McGue, 2005).

The role of behavioral influences is important both in disease etiology and disease treatment. Given a certain genetic inheritance, some environments can tend to increase the likelihood of a disease's expression, while others tend to decrease it. This also means that behavioral interventions can be very successful at preventing or ameliorating behaviorally influenced conditions of clinical concern. Thus, behavioral medicine is concerned with both genetic predisposition to complex disease states and environmental influences that interact with them.

Cross-References

- ▶ Genetics
- ▶ Genomics
- ▶ Proteomics

References and Readings

Bryson, B. (2004). *A short history of nearly everything*. New York: Black Swan.

Watson, J. D. (2006). *DNA: The secret of life*. New York: Alfred A Knopf.

McGue, M. (2005). Mediators and moderators of alcoholism inheritance. In J. R. Turner, L. R. Cardon, & J. K. Hewitt (Eds.), *Behaviour genetic approaches in behavioral medicine*. New York: Plenum.

DNA-Methylation

- ▶ Methylation

DNR Order

Howard Sollins
Attorneys at Law, Ober, Kaler, Grimes & Shriver, Baltimore, MD, USA

Synonyms

End-of-life care preferences

Definition

Do Not Resuscitate Order: DNR

Description

Do Not Resuscitate Order: DNR

A do not resuscitate (DNR) order is a directive that cardiopulmonary resuscitation (CPR) should not be initiated if an individual's heart stops or if the individual stops breathing. State law determines who may issue a DNR order such as whether a health-care practitioner other than a physician is authorized to do so. Health-care facilities will have established protocols for resuscitating patients when there is no DNR order, in accordance with state law. Competent patients or their authorized decision maker or proxy should be consulted about any proposed DNR order. The authority of a decision maker or proxy to consent to a DNR order can be affected depending on (a) whether that person is an agent under an advance directive, representative under a durable health care power of attorney, surrogate under state law, or guardian appointed by a court and (b) the scope of any such authorizing document. State law also determines if or how a physician may or may not have the authority to enter a DNR order without a direction by a competent patient or authorized decision maker or proxy, based on a determination that CPR would be medically futile in a particular case. Hospitals may have special procedures, consistent with established policy and state law, on the circumstances under which a DNR order may be suspended during surgery.

Cross-References

► End-of-life Care Preferences

References and Readings

Kawana-Singer, M. (2011). *Overcoming cultural differences between patients, caregivers, and providers in providing quality palliative and end-of-life care: A multicultural experience*. Educational Book.

Doctor-Patient Communication: Why and How Communication Contributes to the Quality of Medical Care

Debra Roter[1] and Judith A. Hall[2]
[1]Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, USA
[2]Department of Psychology, Northeastern University, Boston, MA, USA

Synonyms

Medical dialogue; Medical interaction

Description

The patient–physician relationship and its expression through the medical dialogue have been described or alluded to in the history of medicine since the time of the Greeks. Nevertheless, historians of modern medicine have tracked an undeniable decline in the centrality of communication to the care process. In his study of the history of doctors and patients, Shorter (1985) attributes the denigration of communication through the twentieth century to the ascendancy of the molecular and chemistry-oriented sciences as the predominant medical paradigm. This change was fundamental in directing medical inquiry away from the person of the patient to the biochemical makeup and pathophysiology of the patient. It was not coincidental that it was during this period of scientific advance that the practice of interviewing patients from a written outline designed around a series of yes–no hypothesis-testing questions largely replaced unstructured medical histories. Conversation was largely curtailed by these changes; patients were restricted to answering the questions asked and the medical dialogue was recast as a medical interview with the exchange directed by the scientist-physician.

Professional dominance of physicians over patients, and scientific objectivity over the patient

perspective, was noted (and lamented) by influential authors in the medical and social sciences (Freidson, 1970; Szasz & Hollender, 1956), but the issue did not attract the attention of medical educators and policymakers until communication was convincingly linked to patient outcomes. It is in this light that the critical role of technical advances in the 1960s that made audio recording of the medical visit logistically possible may be seen and that the methods that allowed investigation of the relationships between the medical dialogue and outcomes may be appreciated.

The objectives of this essay are threefold: (1) to present evidence establishing the importance of medical visit communication to a variety of valued outcomes, (2) to provide insight into how medical communication is assessed by using a popular coding method, and (3) to briefly consider future directions for improving medical communication in light of current national initiatives in health-care policy and reform.

Why Medical Communication Is Important

Within 20 years of the groundbreaking study by Barbara Korsch and colleagues (1968) documenting pediatric visit communication was described and related to patient satisfaction and adherence with medical recommendations, a convincing body of literature had emerged linking medical communication to patient outcomes. As reflected in a meta-analysis of medical communication and its correlates, covering the period from 1964 to 1988, relationships between specific elements of medical visit communication and patient outcomes were apparent in regard to patient recall of medical information, patient satisfaction, adherence, and patient assessment of technical care quality (Hall, Roter, & Katz, 1988). These relationships, however, varied depending on which aspect of communication was measured. For instance, when the doctor offered more information, asked fewer questions overall, but more questions about compliance in particular, and was more positive and less negative (both verbally and nonverbally), the patient was significantly more adherent. Satisfaction was also found to be higher when the doctor offered more information, actively enlisted patient involvement in care, was more positive (verbally and nonverbally), engaged in more social conversation, and when there was more visit talk overall. Communication predictors of patient recall of information included more information, positive talk and partnership building, but less question asking. Thus, some elements of physician communication like information giving and positive talk were significantly correlated with all outcomes, while elements such as question asking, partnership building, and overall talk were only related to particular outcomes.

Although not as commonly studied as satisfaction and adherence, there is a small but extremely important body of work that has linked doctor-patient communication to other measures of outcome, including indicators of patient health status. Included among these measures are physiologic indicators such as levels of glycosylated hemoglobin (HbA1c) in the blood of diabetic patients and blood pressure in hypertensive patients. In addition, such measures as functional status (the patient's sense of his or her ability to perform usual daily routines) and a patient's overall sense of well-being and emotional coping have been linked to elements of the medical dialogue (Griffin et al., 2004).

Finally, there are a few studies that have explored how physicians are affected by factors associated with the way in which they relate to patients and perform their work. Among these outcomes are physician satisfaction and the likelihood of becoming involved in medical malpractice litigation. An appreciation for these outcomes is underscored by the relatively high levels of physician stress and burnout, particularly in specialties associated with rising malpractice rates, and the medical workforce shortages made worse by increasing numbers of physicians taking early retirement. It should not come as a surprise that many of the predictors of patient satisfaction also affect physician satisfaction as the communication of emotion is highly reciprocal. The positive regard associated with patient satisfaction with care and judgments of good performance, interpersonal rapport, and personal warmth and affection are all likely to inspire physician satisfaction and similar feelings

of liking. The opposite is true as well; critical judgments and perceptions of rejection or disregard also inspire similarly negative emotions. Not only are patient and physician satisfaction and liking related to one another, but when these measures of a positive interpersonal and professional relationship are absent, patient compliance is lowered, therapeutic effect is diminished, and physician risk for malpractice litigation is heightened (see Roter & Hall, 2006 for a review of this literature).

How Communication Is Assessed

A review of the methods used to analyze the medical communication in the 61 studies included in the meta-analysis described earlier found that 28 different coding systems were used (Roter, Hall, & Katz, 1988). Only three systems, Bales' Interaction Process Analysis, the Verbal Response Mode (VRM), and the Roter Interaction Analysis System (RIAS), were used in multiple studies, and these were applied in only a handful of studies (ranging from five to seven studies each). The Bales' system and the VRM taxonomy were originally devised as a general-purpose system for coding speech acts but applied within the medical context, while the RIAS was developed specifically to reflect communication dynamics of the medical dialogue. Many of the systems coded information exchange in some form, while others focused on particular kinds of expression like empathy or concern. A subsequent review of communication assessment instruments, covering the period 1986–1996, identified 44 unique instruments, but only four of these were used in multiple studies (Boon & Stewart, 1998).

While investigators continue to develop and apply new coding approaches, the RIAS has clearly gained prominence in the research literature with more than 250 published studies using the system as of 2011. Studies have been conducted in 23 countries in a variety of medical care contexts and provider types and specialties with translations to Spanish, French, German, Italian, Swedish, Norwegian, Danish, Dutch, Swiss-German, Portuguese, Japanese, Korean, Chinese, Arabic, Hebrew, and Swahili. (See https://riasworks.com for a bibliography of RIAS studies.) Because of its widespread use, a brief description of the RIAS and examples of coding categories will be presented.

Derived loosely from social exchange theories related to interpersonal influence and problem solving, the system takes a perspective of the medical encounter as a "meeting between experts," grounded in an egalitarian model of patient-provider partnership that accounts for the contributions of each speaker (Roter & Hall, 2006). The basic system is comprised of 40 mutually exclusive and exhaustive codes applied to all dialogue statements expressed by each speaker in the encounter. This is usually the patient and physician, but may also include one or more family members or friends accompanying the patient to the visit or multiple providers including consultant or attending physicians, nurses, or technicians. The codes are applied to the smallest unit of expression to which a meaningful code can be assigned, generally a complete thought, simple sentence, phrase, or clause in a compound statement but sometimes a single word. In addition to verbal exchange, RIAS coders globally assess the emotional tone of the visit at the close of a session for each speaker in terms of overall levels of irritation, anxiety, dominance, interest, and friendliness. The global ratings have been found to capture vocal qualities independent of literal verbal content and can thereby be considered as an indicator of nonverbal communication (Hall, Roter, & Rand, 1981).

A useful framework for organizing and grounding RIAS-coded communication in the clinical encounter is the four-function model of medical interviewing that includes data gathering, patient education and counseling, responding to emotions, and partnership and activation. Specific codes and dialogue examples in each of these areas are presented in Table 1.

Future Directions for Improving Medical Communication

The appearance of patient-centered medical care on the national health-care agenda reflects a sea change in the value attributed to communication by policymakers, medical educators, and the

Doctor-Patient Communication: Why and How Communication Contributes to the Quality of Medical Care, Table 1 Communication functions of the medical visit as reflected in RIAS codes and dialogue examples

Functional grouping	Communication behavior	Example of provider dialogue	Example of patient dialogue
Data gathering skills	Open-ended question (categories: medical condition, therapeutic regimen, lifestyle and self-care, psychosocial topics, other)	What can you tell me about the pain? How are the meds working for you? What are you doing to keep yourself healthy? What's happening with your son?	How dangerous is my blood pressure? How do the meds work? What can I do to keep myself healthy? Do you have any suggestions for getting my son to go along with the family program?
	Closed-ended question (categories: medical condition, therapeutic regimen, lifestyle and self-care, psychosocial topics)	Does it hurt now? Do you take your meds every day? Are you still smoking? Is your wife back?	Is my blood pressure too high now? Is that white pill the diuretic? Do you think the patch that can help me stop smoking?
Patient education and counseling skills	Information about medical condition and symptoms	A normal blood pressure for someone with diabetes would be less than 130/80	Last time I took it my blood pressure was 130/80
	Information about therapeutic regimen, procedures and tests	The medication may make you drowsy. You need to take it for 10 days	The medication made me drowsy
			I took the test and am waiting for the result
	Lifestyle/self-care information	Getting plenty of exercise is always a good idea. I can give you some tips on quitting	I get plenty of exercise. I have been trying to quit
	Psychosocial exchange about problems of daily living, issues about social relations, feelings, emotions	It's important to get out and do something daily. The community center is good for company	It's tough to quit when your wife smokes
	Counseling statements regarding medical condition/therapeutic regimen	The medication will not be effective if you don't take it as prescribed; I want you to set up a routine to take your pills the same time every day	Not applicable
	Counseling statements regarding psychosocial and lifestyle issues	It is very important for you to get out of the house everyday! Being social is good medicine	Not applicable
Relationship skills	Positive talk (categories: agreements, jokes/laughter, approvals)	You look fantastic, you are doing great	The new medicine works great
			More blood! You're a vampire!
	Negative talk (categories: disagreements, criticisms)	No, it doesn't look to me like you were careful about your salt. The local stores are just not very good about making fresh vegetables available	That new drug you gave me was useless
			How can you eat healthy when even the hospital cafeteria serves junk food?
	Social talk (nonmedical chit-chat)	How about them O's last night?	I follow the Ravens – I've given up on the Orioles.
	Emotional talk (categories: concern, reassurance/optimism, empathy, legitimation, partnership)	I'm worried about that. I'm sure it will get better. We'll get through this	I am really worried. I'm going to make it work!
			I can see how upset you get when you see me in this state
			I want to work with you until we get it right

(*continued*)

Doctor-Patient Communication: Why and How Communication Contributes to the Quality of Medical Care, Table 1 (continued)

Functional grouping	Communication behavior	Example of provider dialogue	Example of patient dialogue
Partnering skills	Facilitation (categories: asking for patient opinion, asking for understanding, paraphrase and interpretation, back-channel)	What do you think it is? What would help? Do you follow me? Let me make sure I've got it right. I heard you say you the meds didn't work for you	Do you follow what I'm saying? Let me make sure I've got it right. I heard you say you that the meds take time to work and I have to just keep taking it and be patient
		Uh-huh, right, go on, hmm	Uh-huh, right, go on, hmm
	Orientation (categories: transitions, directions)	Ok, well, let's see	Alright, now
		I'd like to do a physical now. Get up on the table. Now we'll check your back	I'll get started on filling this form out while you're gone

public. The influential Institute of Medicine report on Health Care Quality identified patient-centered care as key to any significant future improvements in health-care quality, alongside core medical care quality requisites of safety, timeliness, effectiveness, efficiency, and equity (Institute of Medicine [IOM], 2001). In a similar vein, patient-centered communication was recognized as a significant vehicle for the prevention of medical errors and malpractice litigation (Kohn, Corrigan & Donaldson, 1999). The scientific evidence reflected in these important reports not only has implication for the routine practice of medicine, but it has also influenced national health policy. The Surgeon General has targeted an increase in the proportion of persons who report that their health-care providers have satisfactory communication skills among the key objectives for the nation (Surgeon General Report, Healthy People 2010, Health Objective 11.6). This goal is integrated into objectives in screening, diagnosis, treatment, prevention, and hospice care applicable to chronic diseases and cancer. Most recently, patient-centered care has been included among the quality benchmarks for Accountable Care Organizations as part of the Patient Protection and Affordable Care Act, Public Law 111–148 (Levinson, Lesser, & Epstein, 2010).

Responding to these same pressures, the American Association of Medical Colleges (AAMC) and the Accreditation Council for Graduate Medical Education (ACGME) have required documentation of communication skills training as part of the accreditation criteria for undergraduate and graduate level medical training programs. Consequently, virtually all US medical schools now require that some portion of their curriculum be dedicated to this area. Despite this progress, medical education challenges remain as the intensity and format vary widely and training is often concentrated in the first 2 years of training before medical students begin to see patients (Levinson et al., 2010). Requirements for medical certification have also been expanded to include demonstration of proficiency in communication skills as part of the United States Medical Licensing Exam (USMLE). The clinical skills portion of the exam assesses candidates' performance using standardized patients (actors trained to portray patients) presenting cases that a physician is likely to encounter in clinics, doctors' offices, emergency departments, and hospital settings (http://www.usmle.org/index.html). Furthermore, recent changes by the American Board of Medical Specialties (ABMS) now require communication skills for recertification every 5 years (Levinson et al.).

This essay began by suggesting that medicine had lost its focus on the person of the patient in embracing the scientific advances of the twentieth century, but there is reason for optimism in anticipating that the scientific, educational, and policy advances of the twenty-first century will return the patient to the center of care. Recognition of communication's centrality to the heart and art of medicine as well as its science is well

reflected in the words of an early advocate of biopsychosocial medicine, George Engel, "It is not just that science is a human activity, it is also that the interpersonal engagement required in the clinical realm rests on complementary and basic human needs, especially the need to know and understand and the need to feel known and understood" (Engel, 1988, p. 136).

Cross-References

- ▶ Health Care
- ▶ Health Communication
- ▶ Patient Care
- ▶ Patient-Centered Care

References and Readings

Boon, H., & Stewart, M. (1998). Patient-physician communication assessment instruments: 1986 to 1996 in review. *Patient Education and Counseling, 35*, 161–176.

Engel, G. L. (1988). How much longer must medicine's science be bound by a seventeenth century world view? In K. White (Ed.), *The task of medicine: Dialogue at Wickenburg* (pp. 113–136). Menlo Park, CA: Henry J. Kaiser Family Foundation.

Freidson, E. (1970). *Professional dominance*. Chicago: Aldine.

Griffin, S. J., Kinmonth, A. L., Veltman, M. W. M., Gillard, S., Grant, J., & Stewart, M. (2004). Effect on health related outcomes of interventions to alter the interaction between patients and practitioners. A systematic review of 35 trials. *Annals of Family Medicine, 2*, 595–608.

Hall, J., Roter, D., & Katz, N. (1988). Meta-analysis of correlates of provider behavior in medical encounters. *Medical Care, 26*, 657–675.

Hall, J. A., Roter, D. L., & Rand, C. S. (1981). Communication of affect between patient and physician. *Journal of Health and Social Behavior, 11*, 18–30.

Institute of Medicine. (2001). *Crossing the quality chasm: A new health system*. Washington, DC: National Academy Press.

Kohn, L. T., Corrigan, J. M., Donaldson, MS. (1999). To err is human: building a safer health care system. Washington (DC): National Academies Press.

Korsch, B. M., Gozzi, E. K., & Francis, V. (1968). Gaps in doctor-patient communication: I. Doctor-patient interaction and patient satisfaction. *Pediatrics, 42*, 855–871.

Levinson, W., Lesser, C. S., & Epstein, R. M. (2010). Developing physician communication skills for patient-centered care. *Health Affairs, 29*, 1310–1318.

Roter, D. L. (2000). The enduring and evolving nature of the patient-physician relationship. *Patient Education and Counseling, 39*, 5–15.

Roter, D. L., & Hall, J. A. (2006). *Doctors talking with patients/patients talking with doctors: Improving communication in medical visits* (2nd ed.). Westport, CT: Praeger Publishers.

Roter, D. L., Hall, J. A., & Katz, N. R. (1988). Patient-physician communication: A descriptive summary of the literature. *Patient Education and Counseling, 12*, 99–119.

Roter, D., & Larson, S. (2002). The Roter Interaction Analysis System (RIAS): Utility and flexibility for analysis of medical interactions. *Patient Education and Counseling, 46*, 243–251.

Shorter, E. (1985). *Bedside manners*. New York: Simon and Schuster.

Szasz, P. S., & Hollender, M. H. (1956). A contribution to the philosophy of medicine: The basic model of the doctor? Patient relationship. *Archives of Internal Medicine, 97*, 585–592.

Domestic Violence

- ▶ Family Violence

Dominance

- ▶ Interpersonal Circumplex

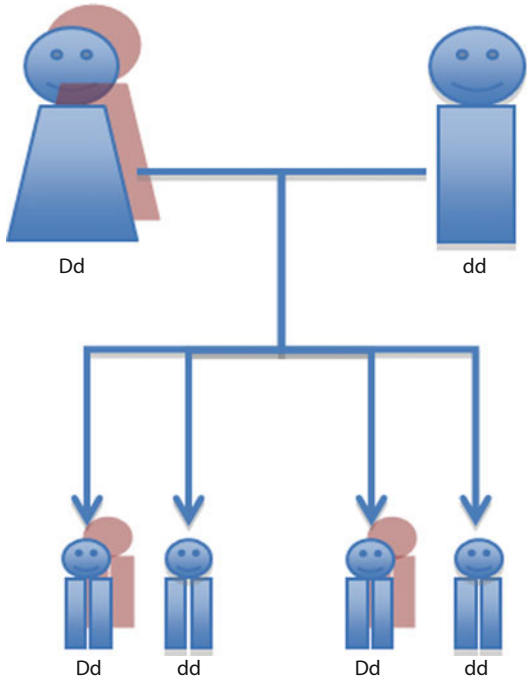
Dominant Inheritance

Laura Rodriguez-Murillo[1] and Rany M. Salem[2]
[1]Department of Psychiatry, Columbia University Medical Center, New York, NY, USA
[2]Broad Institute, Cambridge, MA, USA

Definition

A trait with dominant inheritance is expressed in the presence of the dominant allele, both in individuals who are homozygous for the locus of interest (DD, the same alleles) and in those who are heterozygous (Dd, differing alleles).

Humans have two versions of all autosomal genes, called alleles, one from each parent.



Dominant Inheritance, Fig. 1 Dominant inheritance diagram. Highlighted individuals are affected by the disease given by the dominant D allele

Dominant inheritance refers to the situation when an allele of a gene is expressed (dominant allele) over the alternate gene allele, which is masked (recessive allele). An example of a disease with dominant inheritance is Huntington's disease, where affected individuals carry at least one defective allele, leading to production of the defective protein and resulting in disease (Walker, 2007). To illustrate, the children of an affected heterozygous parent have a 50% chance of inheriting the disease allele and of being affected (see pedigree, Fig. 1). Children of a homozygous affected parent have a 100% chance of inheriting the allele and developing disease.

Cross-References

- ▶ Allele
- ▶ Gene
- ▶ Genotype
- ▶ Heterozygous
- ▶ Homozygous
- ▶ Recessive Inheritance

References and Readings

Lewis, R. (2005). *Human genetics. Concepts and applications* (7th ed.). Boston: McGraw-Hill Science/Engineering/Math.

Strachan, T., & Read, A. P. (2003). *Human molecular genetics* (3rd ed.). London/New York: Garland Science/Taylor & Francis Group.

Walker, F. O. (2007). Huntington's disease. *Seminars in Neurology, 27*(2), 143–150.

Dopamine

Michela (Micky) Marinelli
Department of Cellular and Molecular Pharmacology, Rosalind Franklin University of Medicine and Science, North Chicago, IL, USA

Definition

Dopamine is a catecholamine neurotransmitter produced in dopamine neurons of the brain.

Description

Dopamine is a catecholamine produced in dopamine neurons of the brain.

Anatomy

Approximately, 75% of all of the dopamine cells of the brain originate in the midbrain. From the midbrain, three main pathways project to mesocorticolimbic structures, where dopamine acts as a neurotransmitter (for review, see Zahm, 2006).

The mesocorticolimbic or A10 pathway: Dopamine neurons originate from the *ventral tegmental area* and project primarily to the ventral striatum, amygdala, hippocampus, and frontal cortex.

The nigrostriatal, *mesostriatal*, *or A9 pathway*: Dopamine neurons originate from the *substantia nigra pars compacta* and project primarily to the dorsal striatum.

The retrorubral or A8 pathway: Dopamine neurons originate from the *retrorubral field* project primarily to the dorsal and ventral striatum, hippocampus, and parts of the extended amygdala.

Another dopaminergic pathway originates from the *hypothalamus* and projects to the anterior lobe of the pituitary (*tuberoinfundibular pathway or A12 and A14 pathways*), where dopamine acts as a neurohormone.

Transmission

Dopamine is stored in synaptic vesicles and is released upon neuronal depolarization. Once released, it acts on dopamine receptors. Dopamine receptors are metabotropic (G-protein-coupled) receptors. They are divided in two classes: D1-like (D1, D5 receptors) and D2-like (D2, D3, and D4), which respectively stimulate or inhibit adenylyl cyclase and consequent formation of cAMP. Most released dopamine is cleared from the synapse by reuptake into the dopamine neurons, via dopamine transporters.

Function

While dopamine released from the tuberoinfundibular pathway inhibits prolactin release, dopamine released in mesocorticolimbic and motor structures serves to modulate movement, emotions, and reward.

In particular, the mesostriatal pathway is mostly involved in movement control: This is most notable in the neurodegeneration of dopamine neurons of this pathway, which is associated with ▶ Parkinson's disease.

Dopamine from the mesocorticolimbic and mesostriatal pathways plays an important role in reward. In particular, the activity of dopamine cells increases in response to unexpected rewarding events, or the cues that predict them (Schultz, 2002).

Dopamine is also one of the major players mediating the rewarding effects in ▶ drug abuse and drug dependence (Marinelli, Rudick, Hu, & White, 2006; Volkow, Fowler, Wang, & Goldstein, 2002). Addictive drugs have the common action of increasing dopamine levels in the striatal complex (Di Chiara & Imperato, 1988; Imperato et al., 1992). This effect is mediated by different mechanisms, such as blocking the reuptake of dopamine at the level of the dopamine transporter, reversing the transporter from reuptake to release, or increasing the activity of dopamine neurons. In addition, animal studies show that subjects with heightened dopaminergic transmission are more prone to self-administer drugs of abuse compared with subjects expressing reduced dopaminergic transmission. Furthermore, treatments that decrease dopaminergic transmission generally produce a decrease in drug responding and relapse, whereas treatments that increase it have opposite effects (Marinelli et al., 2006). Similarly, human studies show a positive correlation between dopamine levels and behavioral responses to psychostimulant drugs (Leyton et al., 2002; Oswald et al., 2005). While this suggests that *increased* dopaminergic tone is a facilitator of drug abuse, there is also evidence for *decreased* dopaminergic tone to play a role (Melis et al., 2005). Thus, withdrawal from long-term use of addictive drugs can lead to a hypo-dopaminergic state that could promote the search for drug, to counteract the decrease in dopaminergic tone. These views are not incompatible; thus, it has been proposed that drug craving and relapse in the could result from two separate phenomena: "chronic craving," which is an attempt to alleviate a state of hypo-dopaminergia and "instant craving," which is instead caused by a temporary increase in dopaminergic tone that triggers relapse (Childress & O'Brien, 2000; Franken et al., 2005; Pilla et al., 1999).

Antipsychotic drugs that block dopaminergic transmission are effective in treating some aspects of schizophrenia (mostly delusions and hallucinations), suggesting that hyperdopaminergia may underlie these conditions; however, the matter is controversial because it is still unclear if patients with schizophrenia have impaired dopaminergic transmission (Howes & Kapur, 2009; Moncrieff, 2009; van Os & Kapur, 2009).

Cross-References

- ▶ Drug Abuse
- ▶ Neurotransmitter
- ▶ Parkinson's Disease

References and Readings

Childress, A. R., & O'Brien, C. P. (2000). Dopamine receptor partial agonists could address the duality of cocaine craving. *Trends in Pharmacological Sciences, 21*, 6–9.

Di Chiara, G., & Imperato, A. (1988). Drugs abused by humans preferentially increase synaptic dopamine concentrations in the mesolimbic system of freely moving rats. *Proceedings of the National Academy of Sciences of the United States of America, 85*, 5274–5278.

Franken, I. H., Booij, J., & van Den, B. W. (2005). The role of dopamine in human addiction: From reward to motivated attention. *European Journal of Pharmacology, 526*, 199–206.

Howes, O. D., & Kapur, S. (2009). The dopamine hypothesis of schizophrenia: Version III–the final common pathway. *Schizophrenia Bulletin, 35*, 549–562.

Imperato, A., Mele, A., Scrocco, M. G., & Puglisi-Allegra, S. (1992). Chronic cocaine alters limbic extracellular dopamine. Neurochemical basis for addiction. *European Journal of Pharmacology, 212*, 299–300.

Leyton, M., Boileau, I., Benkelfat, C., Diksic, M., Baker, G., & Dagher, A. (2002). Amphetamine-induced increases in extracellular dopamine, drug wanting, and novelty seeking: A PET/[11C]raclopride study in healthy men. *Neuropsychopharmacology, 27*, 1027–1035.

Marinelli, M., Rudick, C. N., Hu, X. T., & White, F. J. (2006). Excitability of dopamine neurons: Modulation and physiological consequences. *CNS & Neurological Disorders Drug Targets, 5*, 79–97.

Melis, M., Spiga, S., & Diana, M. (2005). The dopamine hypothesis of drug addiction: Hypodopaminergic state. *International Review of Neurobiology, 63*, 101–154.

Moncrieff, J. (2009). A critique of the dopamine hypothesis of schizophrenia and psychosis. *Harvard Review of Psychiatry, 17*, 214–225.

Oswald, L. M., Wong, D. F., McCaul, M., Zhou, Y., Kuwabara, H., Choi, L., et al. (2005). Relationships among ventral striatal dopamine release, cortisol secretion, and subjective responses to amphetamine. *Neuropsychopharmacology, 30*, 821–832.

Pilla, M., Perachon, S., Sautel, F., Garrido, F., Mann, A., Wermuth, C. G., et al. (1999). Selective inhibition of cocaine-seeking behaviour by a partial dopamine D3 receptor agonist. *Nature, 400*, 371–375.

Schultz, W. (2002). Getting formal with dopamine and reward. *Neuron, 36*, 241–263.

van Os, J., & Kapur, S. (2009). Schizophrenia. *Lancet, 374*, 635–645.

Volkow, N. D., Fowler, J. S., Wang, G. J., & Goldstein, R. Z. (2002). Role of dopamine, the frontal cortex and memory circuits in drug addiction: Insight from imaging studies. *Neurobiology of Learning and Memory, 78*, 610–624.

Zahm, D. S. (2006). The evolving theory of basal forebrain functional-anatomical 'macrosystems'. *Neuroscience and Biobehavioral Reviews, 30*, 148–172.

Dorsal Hypothalamic Area

- ▶ Hypothalamus

Dorsalgia

- ▶ Back Pain

Dorsomedial Nucleus

- ▶ Hypothalamus

Dose: Intensity, Response

Nicole Brandt
School of Pharmacy, University of Maryland, Baltimore, MD, USA

Definition

The underlying mechanism of dose intensity and response deals with drug-receptor interactions. Drugs can bind to a receptor and enhance the effect of that receptor (agonist), inhibit the effect of that receptor (antagonist), or enhance the effect of that receptor at less than the maximum response (partial agonist). Clinically, the goal of a drug dose, or dose regimen, is to provide a patient with maximal benefit and minimal

toxicity. To accomplish this, one must know about a drug dose's pharmacological potency and maximal efficacy. Potency is defined as the dose of drug required to produce 50% of the drug's maximal effect. The more potent a drug is, the lower the dose needed to produce a desired clinical response. Drug potency depends on the affinity of receptors to bind the drug and how effectively the drug-receptor interaction leads to a clinical response. Drug potency is the main factor considered when deciding a dose to administer.

A dose's response relates to how effective a drug is at a particular dose in relation to clinical response. How effective a dose is and what type of response it can have in a patient varies based on the route of administration, the absorption, the distribution in the body, and the clearance from the blood or site of action. When deciding on a drug and dose, not only does the dose's efficacy response have to be taken into account, but the dose's toxic response also has to be considered. Each drug has a therapeutic window and the goal is to give a dose of drug that is above the bottom line of that window (clinical efficacy) but not over the top line of that window (clinical toxicity). In short, the overall aim is to give the smallest dose possible that has the greatest efficacy and least toxicity.

In addition to the characteristics of the drug (pharmacokinetics), the intensity and response are also based on the characteristics of the patient. The intensity of effect of a drug dose can be increased or decreased if the patient is hyperreactive or hyporeactive (compared to the majority of patients) to the drug. How reactive a patient is to a drug depends on multiple factors such as genetics, age, gender, body size, comorbidities, and concomitant medications (i.e., drug-drug interactions). There is also the issue of tolerance that may develop with some medication classes (e.g., narcotics). Basically, continued administration leads to decreased dose intensity, and when this happens, the patient is said to have built up a tolerance to the drug at that specific dose. All of these variables need to be taken into consideration when deciding on a drug dosing regimen.

Cross-References

▶ Agonist

References and Readings

Katzung, B. G., Masters, S. B., & Trevor, A. J. (2009). *Basic & clinical pharmacology* (11th ed.). New York: McGraw-Hill.

Shargel, L., Wu-Pong, S., & Yu, A. B. (2005). *Applied biopharmaceutics and pharmacokinetics* (5th ed.). New York: McGraw-Hill.

Double-Blind Study

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

A double-blind research study is one in which neither the subject nor the researcher knows which treatment a subject is receiving.

Employing the double-blind methodology is a hallmark of pharmaceutical clinical trials. In some trials, an investigational drug is compared with a placebo. Both drug products must therefore look, smell, and taste the same so that subjects cannot deduce from one of these characteristics which treatment they are receiving. These drug products must also be manufactured, and similarly shipped to the sites running the trials, in a manner that does not allow the physician investigators to know which treatment subjects are being administered. The same applies for a trial in which two active drugs are being compared. While the double-blind process takes considerable work, it is certainly possible to implement it successfully.

This methodology can be much more difficult to implement in studies of behavioral medicine interventions.

Cross-References

▶ Randomization

Drinking

▶ Alcohol Consumption

Drug Abuse

▶ Dependence, Drug

Drug Abuse: Treatment

▶ Substance Abuse: Treatment

Drug and Alcohol Treatment

▶ Substance Abuse: Treatment

Drug Dependence Treatment

▶ Substance Abuse: Treatment

Drug Development

▶ Pharmaceutical Industry: Research and Development

Drug Rehabilitation

▶ Substance Abuse: Treatment

Drug, Adverse Effects/Complications

Nicole Brandt
School of Pharmacy, University of Maryland, Baltimore, MD, USA

Synonyms

Adverse drug events; Adverse drug reactions

Definition

Adverse drug events (ADE) are noxious events that occur during the use of a medication, but there is not always a causal link (ICH, 1994). An ADE can be a direct injury from the medication, like an adverse drug effect such as a fall. It can also be some form of harm due to the way in which the medication is used, such as discontinuing the medication abruptly, causing an adverse drug withdrawal event such as confusion or increased blood pressure (VA, 2006).

Adverse drug reactions (ADR) are "unintended, harmful responses to a usual dose of a medication during normal administration, with a direct causal relationship" (Nebeker, et al., 2004). Adverse effects or reactions differ from side effects in that medication side effects can be beneficial or harmful, while adverse drug effects are always adverse or negative. Allergic reactions are a type of adverse effect that is elicited by the immune system, for example, hives or shortness of breath. It is important to monitor for and document drug adverse effects in order to provide the best possible care and to prevent subsequent harm (VA, 2006).

References and Readings

International Conference on Harmonization (ICH). (1994). *Clinical safety data management: Definitions and standards for expedited reporting The International Conference on Harmonization*. Report No.: E2A. Accessed at http://www.ich.org/fileadmin/Public_Web_Site/ICH_Products/Guidelines/Efficacy/E2A/Step4/E2A_Guideline.pdf

Nebeker, J. R., Barach, P., & Samore, M. H. (2004). Clarifying adverse drug events: A clinician's guide to

terminology, documentation, and reporting. *Annals of Internal Medicine, 140*, 795–801.
VA Center for Medication Safety, VHA Pharmacy Benefits Management Strategic Healthcare Group and the Medical Advisory Panel. (2006). *Adverse drug events, adverse drug reactions and medication errors, frequently asked questions*. The Department of Veterans Affairs. Accessed at http://www.pbm.va.gov/vamedsafe/Adverse%20Drug%20Reaction.pdf

DST

▶ Dexamethasone Suppression Test

Dual Process Models of Health Behavior

▶ Temporal Self-Regulation Theory

Dual Systems Models

▶ Temporal Self-Regulation Theory

Dunbar-Jacob, Jacqueline

Faith S. Luyster
School of Nursing, University of Pittsburgh, Pittsburgh, PA, USA

Biographical Information

Jacqueline Dunbar-Jacob was born in Detroit, Michigan. She received her BSN degree from Florida State University, earned her MS degree in Psychiatric Nursing from University of California, San Francisco, and earned her Ph.D. degree in Counseling Psychology from Stanford University. In 1984, Dunbar-Jacob joined the faculty at the University of Pittsburgh, Pittsburgh, PA, as assistant professor and director of Nursing at Western Psychiatric Institute and Clinic and later rose through the ranks in the School of Nursing to become professor in 1993. She subsequently received secondary appointments as professor of Psychology, Epidemiology, and Occupational Therapy. Since 2001, she has served as the dean at the University of Pittsburgh School of Nursing. She has been internationally recognized for her study of patient adherence to treatments across a variety of patient populations including rheumatologic conditions, cardiovascular disease, and diabetes.

Major Accomplishments

Dunbar-Jacob's research has been supported by the National Science Foundation and several institutes within the National Institutes of Health (NIH) including the National Institute of Nursing Research; National Heart, Lung, and Blood Institute; and National Institute of Diabetes and Digestive and Kidney Diseases.

Dunbar-Jacob is director of a P01 program project grant from the NIH on translating interventions related to patient adherence and quality of life. She has served as the director of the NIH-funded Center for Research in Chronic Disorders at the University of Pittsburgh. She has served on three NIH safety and data monitoring boards, as a behavioral scientist for three NIH-funded multicenter clinical trials, and on 20 NIH working groups addressing research agendas. She served on the NIH Prevention of Alzheimer's Disease Consensus Panel and is currently a member of the technical expert panel for the AHRQ comparative effectiveness project on

adherence interventions. Her work has been recognized with the PA Nightingale Award for research, the Pathfinders Award for research by the Friends of the NINR, and her induction into the Sigma Theta Tau International Inaugural Nurse Researcher Hall of Fame.

Her current leadership roles include president of Friends of the National Institute of Nursing Research, chair of the Advisory Board for the Institute for Health Care Communication, and cochair of the Pennsylvania Center for Health Careers Supply-Demand Committee. Dunbar-Jacob has also served as the chair of the AACN Task Force on the Future of the Research Focused Doctorate and chair of the Scientific Advisory Board for NIH Roadmap Initiatives for the Patient Reported Outcomes Measurement Information System (PROMIS). Recently, she was a fellow in the Robert Woods Johnson Executive Nurse Fellows Program and a member of the National Institute of Nursing Research Advisory Council. She is also past president of the Academy of Behavioral Medicine Research and past president of the Society for Behavioral Medicine.

Cross-References

- ▶ Adherence
- ▶ Compliance
- ▶ Nonadherence
- ▶ Noncompliance
- ▶ Patient Adherence

References and Readings

Chia, L., Schlenk, E., & Dunbar-Jacob, J. (2006). Effect of personal and cultural beliefs on medication adherence in the elderly. *Drugs & Aging, 23*(3), 191–202.

Dunbar-Jacob, J. (2007). Models for changing patient behavior. *The American Journal of Nursing, 107*(6 Suppl), 20–25.

Dunbar-Jacob, J., Bohachick, P., Mortimer-Stephens, M. K., Sereika, S., & Foley, S. (2003). Medication adherence in patients with cardiovascular disease. *Journal of Cardiovascular Nursing, 18*(3), 209–218.

Dunbar-Jacob, J., Burke, L. E., Rohay, J. M., Sereika, S., Schlenk, E. A., Lippello, A., et al. (1996). Comparability of self-report, pill count, and electronically monitored adherence data. *Controlled Clinical Trials, 17*(2S), 80S.

Dunbar-Jacob, J., Erlen, J. A., Schlenk, E., Ryan, C., Sereika, S., & Doswell, W. (2000). Adherence in chronic disease. In J. Fitzpatrick & J. Goeppinger (Eds.), *Annual review of nursing research* (Vol. 18, pp. 48–90). New York: Springer.

Dunbar-Jacob, J., Gemmel, L. A., & Schlenk, E. A. (2008). Predictors of patient adherence: Patient characteristics. In S. A. Shumaker, E. Schron, J. Ockene, & W. L. McBee (Eds.), *Handbook of health behavior change* (3rd ed., pp. 397–410). New York: Springer.

Dunbar-Jacob, J., Houze, M., Kramer, C., Luyster, F., & McCall, M. (2010). Adherence to medical advice: Processes and measurement. In A. Steptoe (Ed.), *Handbook of behavioral medicine: Methods and applications* (pp. 83–95). New York: Springer.

Dunbar-Jacob, J., & Mortimer-Stephens, M. (2001). Treatment adherence in chronic disease. *Journal of Clinical Epidemiology, 54*(1), S57–S60.

Dunbar-Jacob, J., & Schlenk, E. A. (1996). Treatment adherence and clinical outcome. Can we make a difference? In R. J. Resnick & R. H. Rozensky (Eds.), *Health psychology through the lifespan: Practice and research opportunities* (pp. 323–343). Washington, DC: American Psychological Association.

Dunbar-Jacob, J., & Schlenk, E. A. (2000). Patient adherence to treatment regimen. In A. Baum, T. A. Revenson, & J. E. Singer (Eds.), *Handbook of health psychology* (pp. 571–580). Hillsdale, NJ: Lawrence Erlbaum.

Dunbar-Jacob, J., Schlenk, E. A., Burke, L. E., & Matthews, J. T. (1998). Predictors of patient adherence: Patient characteristics. In S. A. Shumaker, E. Schron, J. Ockene, & W. L. McBee (Eds.), *Handbook of health behavior change* (2nd ed., pp. 491–511). New York: Springer.

Dunbar-Jacob, J., Sereika, S., Rohay, J., & Burke, L. (1998). Electronic methods in assessing adherence to medical regimens. In D. Krantz & A. Baum (Eds.), *Technology and methods in behavioral medicine* (pp. 95–113). Mahwah, NJ: Lawrence Erlbaum.

Martin, K. A., Bowen, D. J., Dunbar-Jacob, J., & Perri, M. G. (2000). Who will adhere? Key issues in the study and prediction of adherence in randomized controlled trials. *Controlled Clinical Trials, 21*(5), 195S–199S.

McCall, M. K., Dunbar-Jacob, J., & Puskar, K. (2009). Promoting medication adherence. *Nursing Made Incredibly Easy, 7*(5), 20–25.

Stilley, C., Bender, C., Dunbar-Jacob, J., & Ryan, C. (2010). The impact of cognitive function on medication management: Three studies. *Health Psychology, 1*, 50–55.

Dyadic Stress

- ▶ Family Stress

Dynorphins

▶ Endogenous Opioids/Endorphins/Enkephalin

Dysfunction Syndrome

▶ Chronic Fatigue Syndrome

Dysfunctional/Dysfunction

▶ Maladaptive/Maladjustment

Dyslipidemia

Ronald Goldberg
Diabetes Research Institute, University of Miami Miller School of Medicine, Miami, FL, USA

Synonyms

High cholesterol; Hypercholesterolemia; Hypertriglyceridemia

Definition

The term dyslipidemia refers to an abnormality of circulating lipoproteins, which are the protein-lipid complexes that transport the major blood lipids, cholesterol, and triglyceride through the circulation. The standard test for circulating lipids and lipoproteins consists of the measurement of the total serum cholesterol, its low-density (LDL-C) and high-density lipoprotein cholesterol (HDL-C) subfractions, and the fasting triglyceride level. Dyslipidemia is then defined as a higher than acceptable total cholesterol, LDL-C or triglyceride level, or a low HDL-C value, or various combinations of these. The clinical importance of these abnormalities relates to the association of elevated cholesterol, LDL-C, and in many instances of hypertriglyceridemia with an increased risk of cardiovascular disease (CVD). Conversely, HDL-C is inversely associated with the risk of CVD. Although not readily measured with standard testing, there are other circulating atherogenic lipoproteins, such as intermediate density lipoprotein and lipoprotein (a), that are believed to increase risk for CVD. Very high triglyceride values (>1,000 mg/dl) are also associated with an increased risk of pancreatitis.

Etiology

The etiology of dyslipidemia is complex. Primary forms of dyslipidemia may be due to monogenic or as yet mostly poorly defined polygenic abnormalities of lipoprotein metabolism. Clinically, these manifest as predominant (and sometimes severe) hypertriglyceridemia, moderate to severe elevations in LDL-C, or combinations of these two abnormalities. Hypertriglyceridemia is commonly associated with low HDL-C levels as well as smaller than normal LDL particles which may have increased atherogenicity. In addition, isolated HDL deficiencies may primarily be due to genetic disturbances of HDL metabolism. More commonly, dyslipidemia is due to effects of secondary factors acting on the particular genetic substrate of the individual to produce a range of lipid abnormalities. Common secondary causes of hypertriglyceridemia, small LDL particles, and low HDL-C include abdominal obesity, insulin resistance, and type 2 diabetes, while the most common reason for an elevated LDL-C is the high fat diet that is typical of Western societies.

Clinical Evaluation and Intervention

The clinical evaluation of dyslipidemia has been guided by the recommendations of the National Cholesterol Education Program (NCEP) which published its initial Adult Treatment Panel (ATP I) recommendations in 1987 for diagnosis and treatment of lipid disorders, and these were revised in 1994 (ATP II) and 2001 (ATP III) with an update in 2004. ATP IV will be published in due course. The current guidelines recommend that attention to LDL-C should be the first

priority because of its close association with CVD event rates, except in patients with severe hypertriglyceridemia (>500 mg/dl), where urgent triglyceride lowering to prevent pancreatitis should be the initial treatment.

The question of what constitutes an LDL-C level requiring treatment has undergone considerable evolution as clinical trial data showing benefit from statin drugs in different population subgroups have been reported. In essence, the population is divided into low-risk, moderate-risk, and high-risk subgroups. Cut points for dietary intervention or pharmacologic intervention have been defined for each group according to severity of CVD risk and in 30 mg/dl increments of LDL-C; however, because these cut points and the targets of treatment are somewhat arbitrarily defined and are also a subject of debate, these values are given as a range. Thus, for low-risk subjects (with no more than one major CVD risk factor), the cut point for therapeutic lifestyle changes, focusing largely on lowering cholesterol, and saturated fat intake is recommended at 130–160 mg/dl, while drug therapy is recommended at 160–190 mg/dl, aiming for an LDL-C of 130–160 mg/dl – the average for the general population.

For those with at least two major risk factors, but without diabetes or evident CVD, the cut points for pharmacologic and lifestyle change are 130–160 and 100–130 mg/dl respectively, and the goal is <100–130 mg/dl (an LDL-C of ≤100 mg/dl is considered optimal for the general population). For highest-risk subjects, a single cut point for combined lifestyle and drug therapy is set at 70–100 mg/dl, with a target of ≤70 mg/dl. Statin drugs are the first choice, with add-on agents such as ezetimibe and bile sequestrants available in the event that statin therapy is inadequate or not tolerable. Because LDL-C reflects only the cholesterol content of LDL and may not adequately reflect the full range and impact of atherogenic lipoproteins before and on treatment, the use of alternative or secondary measures such as non-HDL-C (calculated by total cholesterol minus HDL-C) or apolipoprotein B (the protein component of all atherogenic lipoproteins) has been proposed.

Elevation in triglyceride levels is less directly related to CVD risk, and it is likely that there is considerable heterogeneity in this relationship across the population. Subjects with triglyceride values >150 mg/dl are said to have borderline hypertriglyceridemia, and those with values >200 mg/dl have hypertriglyceridemia which should be considered for treatment. In this population, non-HDL-C or apolipoprotein B measurements may have special advantages because hypertriglyceridemia alters the LDL-C value. The initial approach is therapeutic lifestyle change in which overweight is an important target. Medications such as fibrate drugs, high doses of omega 3 fatty acids, or niacin may be added if lifestyle therapy is considered inadequate, although evidence for CVD benefit with these medications is weaker than that for statin drugs.

Lastly, low HDL-C, defined as <40 mg/dl in men and <50 mg/dl in women, is considered to be an independent CVD risk factor. Weight reduction and increased physical activity may modestly raise HDL-C and are prudent approaches to management of low HDL-C. However, drug therapy with HDL-raising drugs such as fibrates or niacin remains a challenge because of the lack of a full understanding of the relationship between HDL and CVD, side effects of medications, and the paucity of data that this approach clearly adds to the benefit of statin therapy. Considerable efforts are being made to improve our understanding and the management of dyslipidemia.

Cross-References

- ▶ Cardiovascular Disease
- ▶ Cholesterol
- ▶ Diabetes
- ▶ Insulin Resistance (IR) Syndrome
- ▶ Lipid Abnormalities

References and Readings

Lorenzo, C., Williams, K., Hunt, K., & Haffner, S. M. (2007). The national cholesterol education program-adult treatment panel III, international diabetes

federation, and world health organization definitions of the metabolic syndrome as predictors of incident cardiovascular disease and diabetes. *Diabetes Care, 30*, 8–13.

Scott, M., Grundy, S., Cleeman, J., Bairey Merz, C. N., Brewer, H. B., Clark, L. T., Hunninghake, D., Pasternak, R., Smith, S., Stone, N., & Coordinating Committee of the National Cholesterol Education Program. (2004). Implications of recent clinical trials for the national cholesterol education program adult treatment panel III guidelines. *Journal of the American College of Cardiology, 44*, 720–732.

Dyspnea

Valerie Sabol
School of Nursing, Duke University, Durham, NC, USA

Definition

Dyspnea, or shortness of breath, is a frequently reported symptom of unpleasant and/or uncomfortable respiratory sensations with many potential underlying etiologies (e.g., pulmonary disease, primary manifestation of cardiac disease, neuromuscular disease, obesity, anxiety). The American Thoracic Society (1999) defines dyspnea as a "subjective experience of breathing discomfort that is comprised of qualitatively distinct sensations that vary in intensity; the experience derives from interactions among multiple physiological, psychological, social, and environmental factors, and may induce secondary physiological and behavioral responses." Although older adults with chronic pulmonary and/or cardiac disease may have a blunted response to dyspnea, perhaps as a result of adaptation over time, it is still an important clinical history finding (e.g., acute exacerbation of a chronic disease process).

There are variations in the clinical reporting of dyspnea; words used by individuals to describe their breathing discomfort may provide insight into the underlying pathophysiology of their disease. For example, words used to describe difficulty inspiring is associated with upper airway obstruction (e.g., aspiration), and words used to describe difficulty with expiratory flow is associated with obstruction of the smaller bronchioles (e.g., asthma). The inability to breathe while in a recombinant position is known as orthopnea, and complaints of sudden onset of coughing or difficult breathing after sleeping in a recombinant position (typically after 1–2 h) is known as paroxysmal nocturnal dyspnea (PND), and is typically associated with heart failure. Dyspnea on exertion (DOE) is common in obstructive and restrictive pulmonary diseases. Dyspnea that is described as painful may have an underlying inflammatory or trauma-related etiology. Signs of air hunger include mouth breathing, use of accessory muscles, and/or inability to finish a sentence without pausing to breathe. Some individuals, however, are able to adjust their physical activities to limit or prevent dyspnea and more objective testing may be warranted.

Depending on the most likely underlying etiology, there are several diagnostic studies that could be used to evaluate dyspnea. For example, exercise testing (e.g., 6-min walk test), spirometry, and other pulmonary function tests (PFTs), pulse oximetry, arterial blood gas sampling, blood chemistries (e.g., b-type natriuretic peptide, anemia, renal function), and chest radiography may provide information to aid in a differential diagnosis and help target treatment strategies. Treatment strategies include self-help strategies (e.g., accurate self-assessment and regulation of breathing), smoking cessation, avoidance of infection, and environmental stressors (e.g., weather extremes, poor air quality, pollutants), pulmonary rehabilitation, and chronic disease management.

Cross-References

▶ Anxiety and Heart Disease

References and Readings

American Thoracic Society. (1999). Dyspnea. Mechanisms, assessment, and management: a consensus statement. *American Journal of Respiratory Critical Care Medicine, 159*(1), 321–340.

Marx, J., Hockberger, R., & Walls, R. (2009). *Rosen's emergency medicine: Concepts and clinical practice* (7th ed.). Philadelphia: Mosby-Elsevier.

Dysrhythmia

► Arrhythmia

Dysthymia

Nina Rieckmann
Berlin School of Public Health, Charité Universitätsmedizin, Berlin, Germany

Synonyms

Chronic depression; Chronic depressive disorder; Dysthymic disorder

Definition

Dysthymia is a form of chronic depression, characterized by persistent ("most of the days, for more days than not") depressed mood lasting for at least 2 years. It is a diagnostic category within the mood disorders in the current versions of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) and the International Classification of Diseases (ICD-10). In addition to a depressed mood, two or more of the following symptoms must have been present most of the time (i.e., the person must not have been symptom-free for more than 2 months at a time):

1. Poor appetite or overeating
2. Insomnia or hypersomnia
3. Low energy or fatigue
4. Low self-esteem
5. Poor concentration or difficulty making decisions
6. Feelings of hopelessness

In children and adolescents, a dysthymia diagnosis requires at least 1 year of depressed or irritable mood, and two of the above symptoms. Exclusion criteria are manic, hypomanic or mixed episodes, presence of a major depressive episode during the first 2 years, and psychosis.

Description

Dysthymia is a mood disorder of low symptom intensity. Nevertheless, it is associated with markedly decreased quality of life, and functional impairments are significant. While generally able to cope with everyday life, affected persons struggle with workplace or school demands, social and intimate relationships. Lack of energy, feelings of worthlessness, general negativity and pessimism are common and foster the stigma of "character weakness" and interfere with help-seeking.

The comorbidity with other mental as well as physical disorders is high. Because of under-recognition and under-treatment, persons with dysthymia are high-users of the health-care system, which results in substantial direct (health-care consumption) and indirect (absenteeism, loss of productivity) costs.

Persons with dysthymia have an increased lifetime risk of developing a major depressive episode. When dysthymia worsens into a major depressive episode, this is termed "double depression." Clinically, this form of depression is distinct from others as it is marked by extreme hopelessness and a poor prognosis.

Epidemiology and Risk Factors

Reports of the lifetime prevalence of dysthymia range from 0.1% to 6%, with higher rates in higher income countries and among females.

Persons with comorbid chronic medical disorders, anxiety disorders, a history of substance abuse, and personality disorders are at increased risk for dysthymia and other forms of chronic depression. A special risk group comprises persons who experience depressive symptoms early in life (before the age of 21).

Treatment

The most effective treatment for dysthymia consists of a combination of antidepressant medication and psychotherapy. Randomized controlled trials have shown that both the treatment duration and the intensity need to be higher for dysthymia than for major depressive episodes in order to achieve similar response rates. This is possibly due to the high rates of comorbidities as well as the chronic nature of dysthymia, which often results in yearlong struggles with everyday social and occupational life, which are not easily overturned (Dunner, 2001). Many factors influence the choice of treatment: comorbid illnesses, previous experience with similar medication or psychotherapies, interactions with other medications, side-effect profile, short- and long-term effects, and, most importantly, patient tolerability and individual preferences for type of treatment. Several guidelines and treatment algorithms are available to guide the initial treatment choices as well as the dosage augmentation or switches in therapy when symptom improvement is insufficient.

Since residual or subthreshold depressive symptoms carry a strong risk of depressive symptom relapse, the treatment goal is complete remission from all symptoms.

Importantly, any treatment should be followed by a maintenance phase, which can last as long as a patient's lifetime, in order to prevent the recurrence of depressive symptoms. Maintenance therapy can involve the long-term treatment with efficacious antidepressant medication, regular professional depression symptom monitoring, and patient education about medication side effects, the importance of medication adherence, and the connection between psychosocial stressors and symptom recurrence.

Cross-References

- ▶ Depression: Measurement
- ▶ Depression: Symptoms
- ▶ Depression: Treatment
- ▶ Mood

References and Readings

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Dunner, D. L. (2001). Acute and maintenance treatment of chronic depression. *The Journal of Clinical Psychiatry, 62*(Suppl. 6), 10–16.

Gureje, O. (2011). Dysthymia in a cross-cultural perspective. *Current Opinion in Psychiatry, 24*(1), 67–71.

Pettit, J. W., & Joiner, T. E. (2005). *Chronic depression: Interpersonal sources, therapeutic solutions*. Washington, DC: American Psychological Association.

Trivedi, M. H., & Kleiber, B. A. (2001). Algorithm for the treatment of chronic depression. *The Journal of Clinical Psychiatry, 62*(Suppl. 6), 22–29.

Dysthymic Disorder

▶ Dysthymia

E

Eating Behavior

Lara LaCaille
Department of Psychology, University of Minnesota Duluth, Duluth, MN, USA

Synonyms

Eating habits; Eating practices

Definition

Eating behavior is a broad term that encompasses food choice and motives, feeding practices, dieting, and eating-related problems such as obesity, eating disorders, and feeding disorders. Within the context of behavioral medicine, eating behavior research focuses on the etiology, prevention, and treatment of obesity and eating disorders, as well as the promotion of healthy eating patterns that help manage and prevent medical conditions such as diabetes, hypertension, and certain cancers.

Description

Eating behavior is complex; humans make hundreds of food decisions each day that are influenced by a variety of personal, social, cultural, environmental, and economic factors. What people eat and how much they eat has a considerable influence on their health. An ecological model that considers the impact of individual factors, social environments, physical environments, and macro-level environments on food choices is useful in understanding the multitude of determinants of eating behavior. Intraindividual factors influencing eating behavior and food choice include physiological processes (e.g., hunger, satiety, innate preference for sweet foods, brain mechanisms) and psychological processes (e.g., learned food preferences, knowledge, motivations, attitudes, values, personality traits, cognitive processes, self-regulation). The social environment has also been shown to have a substantial effect on eating behavior. Eating behavior is shaped indirectly through observing others and internalization of food rules, as well as directly (i.e., one eats more in the presence of others than when alone). The physical environment, including availability of foods, the context in which foods are provided, and the external cues, such as proximity to food, salience of food, packaging, plate/serving size, and variety of food assortments, have all been shown to affect the type and amount of food eaten. Finally, macro-level environments, including economic systems, food and agricultural policies, food production and distribution, food marketing, and cultural norms and values, may have a more indirect yet powerful impact on food choices and eating behavior. The research on determinants of eating behavior has largely emphasized intraindividual variables, whereas there is considerably less known about the environmental influences and the interaction

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

between these. In particular, there is a need to conduct multilevel research, among diverse subgroups, using better measures, in order to better understand the mechanisms involved in eating behavior (Larson & Story, 2009).

Cross-References

► Obesity

References and Readings

Baranowski, T., Cullen, K., & Baranowski, J. (1999). Psychosocial correlates of dietary intake: Advancing dietary intervention. *Annual Review of Nutrition, 19*, 17–40.

Just, D. R., & Payne, C. R. (2009). Obesity: Can behavioral economics help? *Annals of Behavioral Medicine, 38*(Suppl. 1), S47–S55.

Larson, N., & Story, M. (2009). A review of environmental influences on food choices. *Annals of Behavioral Medicine, 38*(Suppl. 1), S56–S73.

Rothman, A. J., Sheeran, P., & Wood, W. (2009). Reflective and automatic processes in the initiation and maintenance of dietary change. *Annals of Behavioral Medicine, 38*(Suppl. 1), S4–S17.

Savage, J., Orlet, F. J., & Birch, L. (2007). Parental influence on eating behavior: Conception to adolescence. *Journal of Law and Medical Ethics, 35*, 22–34.

Story, M., Kaphingst, K. M., Robinson-O'Brien, R., & Glanz, K. (2008). Creating healthy food and eating environments: Policy and environmental approaches. *Annual Review of Public Health, 29*, 253–272.

Wansink, B. (2010). From mindless eating to mindlessly eating better. *Physiology and Behavior, 100*, 454–463.

Eating Disorders: Anorexia and Bulimia Nervosa

Anna Maria Patino-Fernandez
Department of Pediatrics, University of Miami, Miami, FL, USA

Definition

Prevalence

Anorexia nervosa afflicts an estimated 0.9% of females, with 0.5% of men reporting having anorexia at some time in their lives (Hudson, Hiripi, Pope, & Kessler, 2007). Bulimia nervosa is estimated to occur in 1.5% of females and in 0.5% of men (Hudson et al., 2007). Approximately 3.5% of women and 2% of men reported having binge-eating disorder (BED) at some point in their lives (Hudson et al., 2007). BED affects about 8% of people who are obese. Eating disorders typically (86%) are reported by the age of 20, with the majority of those affected (43%) with anorexia reporting onset between the ages of 16 and 20. Thirty percent of those afflicted report duration from 1 to 5 years, and 31% report duration from 6 to 10 years. The full recovery rate of women with bulimia has been reported to be significantly higher than that of women with anorexia, with 74% of those with bulimia achieving full recovery, whereas only 33% of those with anorexia achieved full recovery (Herzog et al., 2009). Approximately one third of both women with anorexia and with bulimia relapse after full recovery (Herzog et al., 2009).

Adolescent and young women account for 90% of cases (women between the ages of 12 and 25) of eating disorders (American Psychiatric Association [APA], 2000). In anorexia, there is a female to male ratio of about 11 to 1. Bulimia has a female to male ratio of about 30 to 1. Anorexia nervosa may arise in children as young as 8 years of age, whereas bulimia rarely appears before the age of 12. Increasing numbers of older women and men have these disorders with up to 5–10% of all cases of eating disorders occurring in males (Academy of Pediatrics Committee on Adolescence [AAP], 2003). Teen boys have shown an increasing trend in dieting, use of diet products, and their use of exercise for weight control, with Hispanic boys being the most likely to practice weight control, followed by black and then white boys. Binge-eating disorder is more prevalent than both anorexia nervosa and bulimia nervosa, but the treatment outcome for individuals with BED is also more favorable (Fairburn, Cooper, Doll, Norman, & O'Connor, 2000).

There is relatively little data on the role of ethnicity or racial background in eating disorders. Some data show that ethnic minority women who

seek treatment for anorexia have lower admission weights than white women, suggesting that anorexia may go undetected or untreated longer in minority women. Typically a disorder of white affluent women, disordered eating appears to be increasing among nonwhite groups, including Hispanics and American Indians (Croll, Neumark-Sztainer, Story, & Ireland, 2002).

Etiology

There is no one single cause but rather a complex interaction between biological issues, such as genetics and metabolism; psychological issues, such as control; coping skills; personality factors; family issues; and social issues, such as a culture that promotes thinness and media that transmits this message.

Comorbidities

People with eating disorders suffer higher rates of other mental disorders including depression, anxiety, obsessive-compulsive disorder, and substance abuse (Hudson et al., 2007). There is an increased frequency of mood disorders in individuals with anorexia and bulimia nervosa, which may develop at the same time, or the mood disorder may precede the eating disorder (APA, 2000). Obsessive-compulsive features are prominent in individuals with anorexia nervosa, whereas increased frequency of more general anxiety symptoms is more common in those with bulimia (APA, 2000).

Cross-References

- ▶ Anorexia Nervosa
- ▶ Bulimia

References and Readings

Academy of Pediatrics Committee on Adolescence. (2003). Identifying and treating eating disorders. *Pediatrics, 111*, 204–211.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: APA.

Croll, J., Neumark-Sztainer, D., Story, M., & Ireland, M. (2002). Prevalence and risk and protective factors related to disordered eating behaviors among adolescents: Relationship to gender and ethnicity. *Journal of Adolescent Health, 31*, 166–175.

Fairburn, C. G., Cooper, Z., Doll, H. A., Norman, P., & O'Connor, M. (2000). The natural course of bulimia nervosa and binge eating disorder in young women. *Archives of General Psychiatry, 57*, 659–665.

http://www.mayoclinic.com/health/eating-disorders/DS00294

http://www.nimh.nih.gov/health/publications/eating-disorders/nimheatingdisorders.pdf

Herzog, D. B., Dorer, D. J., Keel, P. K., Selwyn, S. E., Ekeblad, E. R., Flores, A. T., et al. (2009). Recovery and relapse in bulimia and anorexia nervosa: A 7.5-year follow-up study. *Journal of the American Academy of Child and Adolescent Psychiatry, 38*, 829–837.

Hudson, J. I., Hiripi, E., Pope, H. G., & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the national comorbidity survey replication. *Biological Psychiatry, 61*, 348–358.

Rosen DS & The Committee on Adolescence (2010) Identification and management of eating disorders in children and adolescents. *Pediatrics, 126*, 1240–1253.

Eating Habits

▶ Eating Behavior

Eating Practices

▶ Eating Behavior

EBV

▶ Epstein-Barr Virus

ECG

▶ Electrocardiogram (EKG)

Ecologic Bias

▶ Ecological Fallacy

Ecological Fallacy

Jane Monaco
Department of Biostatistics, The University of North Carolina at Chapel Hill, Chapel Hill, NC, USA

Synonyms

Ecologic bias

Definition

Ecological fallacy is improperly inferring an association (or lack of association) at an individual-level based on a group-level relationship.

Ecologic studies use measures taken at the level of a group (such as a country, school, or hospital) rather than at the individual (such as patient) level. Ecologic studies are widespread in behavioral medicine literature due their low cost and convenience since ecologic data can often be obtained through census records or existing surveys and records. Two typical behavioral medicine ecologic examples are a study investigating the association between alcohol availability and violence (Gorman, Zhu, & Horel, 2005) in which data were collected at the census tract level and a study investigating the association between needle exchange programs and HIV seroprevalence among injecting drug users (Hurley, Jolley, Kaldor, 1997) in which data were collected at the city level.

When risk factors and outcomes are measured at an aggregate level, the relationship between the group-level variables may be different than the relationship between variables measured at the individual level. An often-cited example used to illustrate the issue involved a nineteenth century study which found higher suicide rates within Prussian provinces that had higher proportions of Protestant residents (Durkheim, 1951). The conclusion that Protestant individuals (rather than Catholic individuals) were more likely to commit suicide cannot be inferred based on the observed association among the provinces (Morgenstern, 1982; Robinson, 2009). One possible scenario is that Catholic residents within the largely Protestant provinces had the high suicide rates, resulting in a positive association between percent Protestant and suicide rate. Extrapolation of aggregate results to individuals is a mistake in logic which can lead to a potentially misleading conclusion.

Because of the many limitations of ecologic studies, including ecological fallacy, they are often used as exploratory or hypothesis-generating studies rather than as confirmatory.

Cross-References

▶ Aggregate Data
▶ Hypothesis Testing

References and Readings

Durkheim, E. (1951). *Suicide: A study in sociology* (Spaulding & Simpson, Trans.). Glencoe, IL: Free Press. (Original Work Published 1897).
Gorman, D., Zhu, L., & Horel, S. (2005). Drug "hot-spots", alcohol availability and violence. *Drug and Alcohol Review, 24*(6), 507–513.
Hurley, S. F., Jolley, D. J., & Kaldor, J. M. (1997). Effectiveness of needle-exchange programmes for prevention of HIV infection. *The Lancet, 349*(9068), 1797–1800.
Morgenstern, H. (1982). Uses of ecologic analysis in epidemiologic research. *American Journal of Public Health, 72*(12), 1336.
Robinson, W. S. (2009). Ecological correlations and the behavior of individuals. *International Journal of Epidemiology, 38*(2), 337.

Ecological Framework

▶ Ecological Models: Application to Physical Activity

Ecological Models: Application to Physical Activity

Ding Ding
Graduate School of Public Health/Department of Family Preventive Medicine, San Diego State University/University of California San Diego, San Diego, CA, USA

Synonyms

Behavioral ecological model; Ecological framework; Social ecological framework; Social ecological model

Definition

Ecological models are a series of models/frameworks that emphasize multiple levels of influences on behaviors. These influences usually include intrapersonal, interpersonal, organizational, community, physical environment, and policy (Sallis, Owen, & Fisher, 2008). Numerous ecological models have been created to explain specific behavior or to guide behavioral interventions. Although different ecological models may involve different terminology, they share two basic principles: (1) multiple levels of influence on behaviors and (2) interactions across levels of influence. In the context of behavioral change interventions, two additional principles apply as follows: (1) effectiveness of multilevel interventions and (2) emphasis on behavior-specific ecological model (Sallis et al., 2008).

Description

History of Ecological Models

The evolution of ecological models highlights a process of proliferation, diversification, and specification. At the early stage, the conceptual basis of ecological models was the general idea that the environment influenced behaviors. Skinner conceptualized most behaviors as a product of interactions between individuals and the environment (i.e., "operant behaviors") (Skinner, 1953). Barker was a founder of environmental psychology (a.k.a. "ecological psychology") and emphasized "behavior settings," where behaviors take place (Barker, 1968). Later on, Bronfenbrenner developed Ecological Systems Theory and defined the "microsystem," "mesosystem," and "macrosystem" as different levels of environmental influences (Bronfenbrenner, 1979). The more recent models were created specifically for health behaviors. McLeroy at al.'s Ecological Model of Health Behavior emphasized five sources of influences on health behaviors, including intrapersonal, interpersonal, institutional, community, and policy factors (McLeroy, Bibeau, Steckler, & Glanz, 1988). Stokols' Social Ecological Model for Health Promotion offered theoretical guidance for behavioral interventions (Stokols, Allen, & Bellingham, 1996; Stokols, Grzywacz, McMahan, & Phillips, 2003). Hovell et al.'s Behavioral Ecological Model (Hovell, Wahlgren, & Adams, 2009; Hovell, Wahlgren, & Gehrman, 2002) incorporated Skinner's Operant Learning Theory (Skinner, 1969) and emphasized multiple contingencies of reinforcement for behavior. Some models were created for a specific health behavior, such as Glanz and colleagues' Model of Community Food Environments (Glanz, Sallis, Saelens, & Frank, 2005), Sallis and colleagues' Ecological Model of Four Domains of Active Living (Sallis et al., 2006), and Fisher and colleagues' Resources and Skills for Self-Management Model (Fisher et al., 2005).

Ecological Models and Physical Activity

The development of ecological models parallels the conceptual evolution of the field of physical activity. Decades ago, "exercise" (i.e., planned physical activity for fitness purpose) was the main focus of physical activity research and most behavioral interventions applied cognition-based theories and targeted individual-level correlates (Sallis et al., 2006). The limitations of these interventions included small number of target individuals who benefited from interventions, small-to-moderate effect sizes

(Dishman & Buckworth, 1996; Dishman, Oldenburg, O'Neal, & Shephard, 1998), and lack of maintenance of behavioral change (Marcus et al., 2000).

More recently, the concept of "active living" emerged to expand previous understanding of physical activity by emphasizing different domains of physical activity, including occupational, leisure-time, household activities, and active transportation (Pratt, Macera, Sallis, O'Donnell, & Frank, 2004). Disciplines outside public health, such as urban planning, transportation, and leisure science, became involved in physical activity research because the multiple levels of influence and domains of activity highlighted needs for expanded expertise. As a result of multidisciplinary collaboration, ecological models have been widely accepted and applied in the field of physical activity.

Sallis et al. (2006) summarized empirical findings and conceptual associations from multidisciplinary research and developed Ecological Model of Four Domains of Active Living (Sallis et al.). In this model, factors influencing physical activity are multilevel and domain specific (Sallis, Adams, & Ding, 2011). Based on the model, physical activity is influenced by intrapersonal factors (e.g., demographic and psychosocial variables), interpersonal factors (e.g., social support and social modeling), perceived environment (e.g., safety, convenience, aesthetics), behavioral settings (e.g., home equipment, walking and biking facilities, parks), and policy environment that directly influences the built environment (e.g., zoning codes, park policies, transportation policies) (Sallis et al., 2006). Most environmental influence is domain specific, for example, bike lanes provide settings for bicycling (especially for transportation purpose), while parks provide settings for leisure-time physical activity. Similarly, transportation policies and parking regulations are more likely to influence transportation physical activity, while policies regarding parks and recreation facilities are more likely to influence leisure-time physical activity (Sallis & Glanz, 2009; Sallis et al., 2011).

Strengths and Limitations of Ecological Models in Physical Activity Research

A major strength of ecological models is the comprehensiveness. Unlike most cognition-based models that include mostly psychosocial variables, ecological models place an individual's behavior in a larger context and take into account multiple levels of influence external and internal to the individual. This approach offers a wide range of opportunities for interventions. Furthermore, ecological models emphasize the effects of the built environment and policies on physical activity and prioritize environment and policy changes to promote active life-styles. Once these changes have been implemented, they are likely to affect a large population and promote sustainable behavioral change.

Current ecological models have weaknesses. First, most models lack specificity and do not include behavior-specific or setting-specific factors; therefore, they cannot provide clear research hypotheses or intervention strategies (Sallis et al., 2008). Second, ecological models do not provide information about mechanisms of how specific factors affect behaviors and how different influences interact across levels. Third, although multilevel interventions have been recommended as an effective approach for producing behavioral change, such interventions are extremely difficult to implement and evaluate. Because it is not feasible to randomly assign individuals to neighborhoods, randomized controlled trials cannot normally be conducted to determine the effectiveness of a specific environment or policy intervention (Sallis & Glanz, 2009).

Cross-References

▶ Built Environment
▶ Physical Activity and Health

References and Readings

Barker, R. G. (1968). *Ecological psychology*. Stanford, CA: Stanford University Press.

Bronfenbrenner, U. (1979). *The ecology of human development*. Cambridge, MA: Harvard University Press.

Dishman, R. K., & Buckworth, J. (1996). Increasing physical activity: A quantitative synthesis. *Medicine and Science in Sports and Exercise, 28*(6), 706–719.

Dishman, R. K., Oldenburg, B., O'Neal, H., & Shephard, R. J. (1998). Worksite physical activity interventions. *American Journal of Preventive Medicine, 15*(4), 344–361.

Fisher, E. B., Brownson, C. A., O'Toole, M. L., Shetty, G., Anwuri, V. V., & Glasgow, R. E. (2005). Ecological approaches to self-management: The case of diabetes. *American Journal of Public Health, 95*(9), 1523–1535.

Glanz, K., Sallis, J. F., Saelens, B. E., & Frank, L. D. (2005). Healthy nutrition environments: Concepts and measures. *American Journal of Health Promotion, 19*(5), 330–333.

Hovell, M. F., Wahlgren, D. R., & Adams, M. (2009). The logical and empirical basis for the behavioral ecological model. In R. J. DiClemente, R. Crosby, & M. Kegler (Eds.), *Emerging theories and models in health promotion research and practice: Strategies for enhancing public health* (2nd ed.). San Francisco: Jossey-Bass.

Hovell, M. F., Wahlgren, D. R., & Gehrman, C. (2002). The behavioral ecological model: Integrating public health and behavioral science. In R. J. DiClemente, R. Crosby, & M. Kegler (Eds.), *New and emerging theories in health promotion practice & research*. San Francisco: Jossey-Bass.

Marcus, B. H., Dubbert, P. M., Forsyth, L. H., et al. (2000). Physical activity behavior change: Issues in adoption and maintenance. *Health Psychology, 19* (Suppl. 1), 32–41.

McLeroy, K. R., Bibeau, D., Steckler, A., & Glanz, K. (1988). An ecological perspective on health promotion programs. *Health Education Quarterly, 15*(4), 351–377.

Pratt, M., Macera, C. A., Sallis, J. F., O'Donnell, M., & Frank, L. D. (2004). Economic interventions to promote physical activity: Application of the SLOTH model. *American Journal of Preventive Medicine, 27* (Suppl. 3), 136–145.

Sallis, J. F., Adams, M. A., & Ding, D. (2011). Physical activity and the built environment. In J. Cawley (Ed.), *The oxford handbook of the social science of obesity*. New York: Oxford University Press.

Sallis, J. F., Cervero, R. B., Ascher, W., Henderson, K. A., Kraft, M. K., & Kerr, J. (2006). An ecological approach to creating active living communities. *Annual Review of Public Health, 27*, 297–322.

Sallis, J. F., & Glanz, K. (2009). Physical activity and food environments: Solutions to the obesity epidemic. *The Milbank Quarterly, 87*(1), 123–154.

Sallis, J. F., Owen, N., & Fisher, E. B. (2008). Ecological models of health behavior. In K. Glanz, B. K. Rimer, & K. Viswanath (Eds.), *Health behavior and health education: Theory, research, and practice* (4th ed.). San Francisco: Jossey-Bass.

Skinner, B. F. (1953). *Science and human behavior*. New York: Macmillan.

Skinner, B. F. (1969). *Contingencies of reinforcement: A theoretical analysis*. New York: Appleton-Century-Croft.

Stokols, D., Allen, J., & Bellingham, R. L. (1996). The social ecology of health promotion: Implications for research and practice. *American Journal of Health Promotion, 10*(4), 247–251.

Stokols, D., Grzywacz, J. G., McMahan, S., & Phillips, K. (2003). Increasing the health promotive capacity of human environments. *American Journal of Health Promotion, 18*(1), 4–13.

Ecological Momentary Assessment

C. Renn Upchurch Sweeney
VA Salt Lake City Healthcare System,
Salt Lake City, UT, USA

Definition

Ecological momentary assessment (EMA) refers to a collection of methods often used in behavioral medicine research by which a research participant repeatedly reports on symptoms, affect, behavior, and cognitions close in time to experience and in the participants' natural environment (Stone Shiffman, 1994). Technologies such as written diaries, electronic diaries, telephones, and physiological sensors are often utilized in EMA studies. EMA studies can be utilized to study a variety of topics such as depression, social support, relationships, diet, work activity and satisfaction, psychotherapy, drug use, allergies, psychological stress, medications, self-esteem, and asthma.

Description

EMA is not a single research method. Instead it encompasses a range of methods that differ in their particular design, assessment schedule, content, and technology. However, all EMA studies have four aspects in common. They all assess research subjects in their natural environments,

in their current or recent states, at selected times, and repeatedly over time (Shiffman, Stone, Hufford, 2008). First, in all EMA approaches, data are collected in real-world environments as subjects go about their normal lives. EMA recognizes that many behaviors and experiences are affected by the context in which they are studied. For an assessed experience or behavior to be representative, it has to be sampled in the context in which it naturally occurs. Therefore, with EMA, psychological processes are not studied in a laboratory environment, but in the natural setting of the subject. EMA allows for improved ecological validity (generalization to the subjects' real lives and real-world experience) because data are collected in the subject's natural setting.

Second, all EMA assessments focus on a subject's current state. For example, EMA self-report items ask about current feelings (or very recent ones), rather than asking for recall or summary over long periods. This aims to reduce biases associated with retrospection. That is, errors or inaccuracies in recalling information are not just random, but are often systematically biased and can change the data in systematic ways. For example, people are more likely to retrieve positively charged information when they are in a good mood, thus introducing biased reporting. Because EMA assesses behaviors, attitudes, emotions, and other characteristics at the moment they occur, it reduces cognitive biases that are often a part of retrospective recall reports.

Third, the moments that are assessed with EMA are strategically selected for assessment. This avoids pitfalls associated with allowing participants to choose when they will provide data. Strategic selection of assessment points can be based on particular features of interest (i.e., occasions when subjects smoked), by random sampling, or by other sampling schemes.

Finally, subjects complete multiple assessments over time. These multiple assessments provide the researcher with a rich picture of how subjects' experiences and behaviors vary over time and across various situational contexts in the participants' normal environment. EMA studies range in the frequency of assessments. Some studies may implement a very frequent schedule of assessment (i.e., assessing subjects every 30 min over a period of days). In other studies, subjects are assessed less frequently (e.g., daily) over periods as long as a year.

Although there are many advantages of EMA such as increasing ecological validity, avoiding retrospective recall, avoiding global summarizations, and being able to study dynamic processes that unfold over time, there are also drawbacks to EMA studies. For example, EMA methods are onerous for participants and require a tremendous level of compliance (Shiffman Stone, 1998). Depending on the design of the study, subjects can be required to stop what they are doing and complete an assessment multiple times a day. EMA studies also place demands on the investigator (Shiffman & Stone). For example, automated methods for prompting or data capture can be expensive. Additionally, the volume of data collected can make data management challenging.

References and Readings

Engle, S. G., Wonderlich, S. A., Crosby, R. D. (2005). Ecological momentary assessment. In J. Mitchell C. B. Peterson (Eds.), *Assessment of eating disorders* (pp. 203–220). New York: Guilford.

Hufford, M. R., Shiffman, S., Paty, J., Stone, A. (2001). Ecological momentary assessment: Real-world, real-time measurement of patient experience. In J. Fahrenberg M. Myrteck (Eds.), *Progress in ambulatory assessment: Computer-assisted psychological and psychophysiological methods in monitoring field studies* (pp. 69–92). Ashland, OH: Hogrefe Huber.

Shiffman, S., Stone, A. A. (1998). Ecological momentary assessment: A new tool for behavioral medicine research. In D. Krantz A. Baum (Eds.), *Technology and methods in behavioral medicine* (pp. 117–131). Mahwah, NJ: Lawrence Erlbaum.

Shiffman, S., Stone, A. A., Hufford, M. R. (2008). Ecological momentary assessment. *Annual Review of Clinical Psychology, 4*, 1–32.

Stone, A. A., Shiffman, S. (1994). Ecological momentary assessment in behavioral medicine. *Annuals of Behavioral Medicine, 16*, 199–202.

Stone, A. A., Shiffman, S. S., DeVries, M. W. (1999). Ecological momentary assessment. In D. Kahneman, E. Diener, N. Schwarz (Eds.), *Wellbeing: The foundations of hedonistic psychology* (pp. 6–39). New York: Russell Sage.

Ecology

► Ecosystems, Stable and Sustainable

Ecosocial Theory

► Social Epidemiology

Ecosystems, Stable and Sustainable

Colin D. Butler[1] and Colin L. Soskolne[2]
[1]Centre for Epidemiology and Population Health, Australian National University, Canberra, ACT, Australia
[2]Department of Public Health Services, School of Public Health, University of Alberta, Edmonton, AB, Canada

Synonyms

Ecology; Sustainability; Units of nature

Definition

Ecosystems: Basic units of nature within which living organisms continually interact with non-life forms, whose boundaries are arbitrary, and which exist in "dynamic equilibrium."

Ecosystem services: A way of thinking about the benefits provided to humans and other species by nature; examples range from obvious (oxygen, food, water) to subtle (regulation of the carbon cycle) and psychological (aesthetic beauty). Sometimes called "nature's services."

Biodiversity: Abundance of biological variety.

EcoHealth: A term that indicates linkages between human and ecological well-being.

Sustainability: A debate exists concerning the definition of "sustainability" (or its earlier formulation "permanence"). One pole is anthropocentric (humans first), the other is biocentric (life in its basic form first). A formulation that many would consider anthropocentric is: "capacity to meet long-term human needs enduring over many generations." In this definition, human coexistence with a sufficient quantity and quality of nature is only implicit. The definition risks interpretation as meaning that nature can be replaced with human-made substitutes. A biocentric definition is: "a process of living within the limits of available physical, natural, and social resources in ways that allow the living systems in which humans are embedded to thrive in perpetuity." This is explicit about the need for ecosystems to thrive but is vulnerable to the criticism that human well-being may be exchanged in order to preserve nature, whether for itself or for the benefit of future generations.

Description

The original definition of an "ecosystem" dates only to 1935 (Tansley, 1935). It linked nature with physics, describing "basic units of nature" within which living organisms continually interact with non-life (the abiotic), including the climate system. The "system" part of the term is linked with ideas of holism, synthesis, emergence, self-organization, cybernetics (from the Greek for steersman, or governor), and, more recently, complexity theory (Fauth, 1997). More recently, Lovelock has extended the ideas of Tansley and Vernadsky by conceptualizing the Earth system as a self-regulating organism floating in space. This insight recognizes that a continual interaction between life and non-life maintains planetary balance (homeostasis).

Tansley coined the term "ecosystem" to contrast alternatives such as "complex organism" and "biotic community." He suggested that ecosystems belong to a category of physical systems in a catalogue from "the universe to an atom." He recognized that the boundaries between these components, including differently configured ecosystems, are arbitrary but argued that such reductionism (i.e., thinking non-systemically) was essential for their analysis. Tansley also

recognized that ecosystems are in "dynamic equilibrium," an idea he traced to the Scottish philosopher Hume and the Roman philosopher Lucretius. Implicitly, this recognizes that ecosystems are never stable but are constantly evolving and changing, including sometimes between alternative new states. A related concept is biodiversity, a contraction of "biological diversity," a term now about 30 years old.

Ecosystem Services and Human Well-Being

Ecosystems are of more than philosophical and scientific interest. Ecosystems and their services have probably been recognized as essential (though not necessarily conceptualized in these terms) by indigenous populations since the time that concepts of any kind evolved (Berkes, Kislalioglu, Folke, & Gadgil, 1998). However, as humans became more urbanized and reliant on technology, the complete dependence of humans on nature's services has become more disguised and less direct. Indeed, since the industrial revolution some 175 years ago, civilization's selected path has led to whole cultures becoming disconnected from the ecosystems that fundamentally sustain them, leaning increasingly on technology as a substitute for the services formerly derived from nature.

A widely used classification of ecosystem services was developed in the 2001–2005 Millennium Ecosystem Assessment, which conceptualized four forms of ecosystem goods and services: provisioning, regulating, culturally enriching, and supporting (see Table 1) (Millennium Ecosystem Assessment, 2003). Some ecologists have criticized the idea of "services" as excessively anthropocentric. From an extreme biocentric (deep ecology) position, the intrinsic value of an algal bloom, jellyfish swarm, or school of cod may be considered proportional to its biomass; however, such arguments seem like sophistry to those concerned with sustainable human well-being. The framers of the Millennium Ecosystem Assessment classification argued that an anthropocentric perspective was a necessary strategy with which to better engage policy makers, most of whom were thought to prioritize monetary over other kinds of value.

Ecosystems influence the entire human enterprise and incorporate, for example, wilderness, cornfields, oceans, and palm oil plantations. The vast human population (now over seven billion) could not be fed solely by hunting and gathering of wild species. For at least ten millennia, it has been increasingly dependent on domesticated plants and animals, grown ever more intensively through agriculture. Even ecosystems that appear wild, with no apparent significant human modification, such as remote mountains, deserts, rainforests, and tundra, have been altered through invasive species, the environmental atmospheric transportation of pollutants (e.g., organochlorines), and via anthropogenic climate change. Indeed, humans have been called the single greatest patch disturbers of all species on the planet (Soskolne et al., 2008).

Extensively transformed ecosystems, such as farms, are today essential for human well-being in order to provide goods in huge quantities, including food, fiber, biofuel, timber, and medicinal agents. But wild and minimally transformed ecosystems are also vital. While both categories of ecosystems provide all four categories of ecosystem service (see Table 2), it would be hubristic if humanity were to imagine that it could successfully transform the whole planet into a farm or garden. Wild places have intrinsic ("existence") value, but perhaps of even more importance, they provide enormous ecosystem regulating services which benefit humanity on a scale that modified ecosystems cannot approach. They also hold a vast reservoir of poorly catalogued species, some of which will be discovered to hold important pharmaceutical and other uses (Chivian & Bernstein, 2008). In general, wild ecosystems are shrinking, with their cultural and regulating services being exchanged for greater provisioning services.

Current economic models ignore (externalize) the costs of harming or maintaining ecosystem services. This practice intensifies several forms of inequity, including polarization between rich and poor and between current and future generations. Those who purchase goods and services tend to underpay, while others, especially the poor, bear the burden of risk and remediation,

Ecosystems, Stable and Sustainable, Table 1 The Millennium Ecosystem Assessment classified ecosystem "services" into four kinds. The selected examples represent only a tiny fragment of a very rich and complex set. Many ecosystems provide multiple ecosystem services; for example, a forest may provide food, fiber, water regulation, water purification, and cultural services. Most employment is provided by provisioning services, including through the transformation of wilderness to farms and plantations

Ecosystem service	Examples of benefit	Ecosystem examples
Provisioning	Food (calories, nutrients), fresh water, fiber, medicinals	Rice fields, aquaculture ponds, bamboo groves, cattle feedlots, wild plants, and wild animals
Regulating	Soil erosion reduction, coastal storm protection, atmospheric carbon stabilization, some cases of infectious disease limitation (e.g., malaria, Lyme disease)	Forests (including on slopes), coastal wetlands, mangroves, extinction of the passenger pigeon contributed to a cascade of ecological changes that enhanced habitat for ticks that transmit Lyme disease
Culturally enriching	Inspiration, aesthetic beauty, spiritual refreshment, religious observation, ancestral links, ceremonial materials, tourism income	Sacred groves, charismatic landscapes and species e.g. coral reefs, tiger reserves, old growth forests, bird of paradise feathers
Supporting	Soil fertility, nutrient cycling, pollination, insect control; many indirect benefits for other services	Many species enhance soil fertility, pollinate, and disperse seeds; bats and birds help control insects; bacteria and fungi recycle nutrients

Ecosystems, Stable and Sustainable, Table 2 Ecosystems can be grouped between two extremes, minimally and extensively transformed; no ecosystem is entirely "natural." Both kinds perform valuable ecosystem services. In general, wild ecosystems are shrinking, with their cultural and regulating services being exchanged for greater provisioning services. To flourish, humanity requires all four kinds of service in abundance

Ecosystem service	Extensively transformed ecosystem	Wild or minimally transformed ecosystem
Provisioning	Farms, plantations, greenhouse vegetables, farmed fish	Game, bushmeat, ocean fish, timber, a reservoir of species with potential human benefit
Regulating	Trees planted to reduce soil erosion, carbon sink from a long lived tree plantation, artificial wetlands	As opposite, but benefits vastly larger in scale; e.g., carbon sink of Amazon forest, scavenging services by wild birds and mammals
Culturally enriching	Bonsai tree, flower garden, zoological garden, artificial wetland; some people find cultivated areas very attractive	Knowledge of the existence of wild areas and species, wilderness hiking, contact with wild birds and mammals adapted to urban ecosystems
Supporting	Earthworms in a garden, planted legumes that fix nitrogen, complementary plantings that reduce pesticide use	Species that pollinate and disseminate seeds, animals that improve soil water absorption, algal varieties that enhance water purification

such as hazardous exposures and waste disposal costs. Indeed, accumulating impacts increasingly approach thresholds which threaten the collapse of crucial ecosystems, harming both present and future generations. Internalization of such costs would motivate consumer behavior more conducive to sustainability (Daly, 1996). A powerful reason for the transformation of wild ecosystems to one which provides intense provisioning services is that greater monetary profits can be made and that more people can be employed. But this transformation is always at a cost: not only to the species which are altered or lost but also, in many cases, to indigenous populations who lack sufficient political and economic power to resist.

Human Health

All ecosystem services are essential, directly or indirectly, for health, a concept captured by the term "ecohealth" (Wilcox, Aguirre, & Horwitz, 2011). Some ecosystem properties, including biodiversity, influence the distribution of important human infectious diseases, including malaria, onchocerciasis, Lyme disease, Chagas disease, and sleeping sickness (Keesing et al., 2010).

Although there are claims that ecosystems that are less transformed by human action provide an infectious disease-regulating "service," such that intact ecosystems lower infectious diseases, the picture is more complex (Butler, 2008). For example, there are many cases in which ecosystem transformation has improved health, such as the clearing of swamps, which reduces mosquito habitat and may thus lower malaria transmission. Somewhat relatedly, "paddies paradox" describes how health can improve even where increased irrigation leads to more potential mosquito habitat in malarial areas. Increased malaria is not inevitable; for example, some of the increased wealth generated by irrigated agriculture can be used to promote technologies and behaviors which are health protective, such as treated bed nets, insecticides, and health services. Areas of high biodiversity may also harbor infectious agents, such as Ebola and HIV, in reservoir species including bats and some nonhuman primates.

Disease introduction can have profound effects on ecosystems, and indirect human health effects. In the late nineteenth century, the epizoonosis (a disease that infects only nonhuman animals) rinderpest entered eastern Africa via imported cattle, causing catastrophic harm to the ecology and human well-being. Immunologically naive oxen (domesticated and wild) died in huge numbers. The loss of oxen reduced plowing and thus agricultural productivity. Infection in wild animal species also reduced meat available for hunting. Exacerbated by periodic droughts, as many as one third of the Ethiopian population and two thirds of the Maasai people of East Africa died in this period, a time known to the Maasai as the *Emutai* ("to wipe out") (Gillson, 2006).

Psychological Health and Ecosystem Cultural Services

Ecosystems are also an important source of "cultural services," essential for good psychological health and thus for individual and community well-being. Ecosystems that help provide this vary from sacred groves (Ramakrishnan, Saxena, & Chandrashekara, 1998) that maintain species with spiritual or symbolic value to viable populations of charismatic species within national parks and tracts of roadless wilderness. Many cultural symbols, decorations, and ceremonies rely on materials from nature, including sacred plants, fungi, and animals or seasonal displays, such as animal migration.

There is increasing evidence that exposure to gardens and wild areas is beneficial for behavior and good mental health (Louv, 2008). This effect may be particularly strong among those with high biophilia (sensitivity to nature) (Wilson, 1984).

Supporting Ecosystem Services

The fourth category of service described by the Millennium Ecosystem Assessment is "supporting" ecosystem services. This category may be the least obvious, but brief reflection shows that they are fundamental because they underpin all the other forms of ecosystem services. Examples include pollination, seed dispersal, and the recycling of nutrients and the formation and aeration of soil by earthworms, ants, and termites. In Western Australia, the brush-tailed bettong *Betolgia pwicillata* (an endangered species) has been shown to improve the absorptive capacity of moisture in soil through its habit of digging for fungi. Many birds, bats, and other mammals assist in seed dispersal, forest maintenance, and insect control. White nose syndrome, a devastating fungal disease affecting several bat species in the USA will lead to increased insect populations, forcing increased reliance on pesticides and fossil fuels to maintain agricultural productivity. This illustrates the interdependency of life, including of our own species.

Prospects

Globally, the progression toward an increase in ecosystems which provide provisioning ecosystem services, such as the exchange of biodiverse forest ecosystems for monocultural plantations that provide food or biofuels, seems unstoppable (Danielsen et al., 2008). Fundamentally, this transformation has been driven by the enormous expansion of human populations since the industrial revolution supported by fossil fuels and agriculture. But there are now numerous warnings

that these processes are unsustainable and that this path places not only health and well-being, but civilization itself at increasing risk of grave harm (Soskolne et al., 2008). This risk occurs through multiple pathways, particularly the accumulation of greenhouse gases which worsen climate impacts and which threaten diverse and adverse feedbacks that could lead to the crossing of system thresholds with extreme danger (Lovelock, 2009).

Solutions

There is widespread denial about the extent, trend, and consequences of the relentless transformation of ecosystems. Many highly transformed ecosystems also risk degradation due to overuse, overgrazing, and contamination by chemicals, invasive species, and even landmines. Human well-being will inexorably decline if these trends are permitted to continue. Indeed, in some countries, constitutional law is changing in ways that will provide better ecosystem protection (Soskolne et al., 2008).

The tragedy of the commons can be overcome (Buck, 1985). But this will not happen without a vast amount of effort, exceeding that of the Space Race or even World War II. As Aldo Leopold wrote (using land as a synonym for ecosystems):

> We abuse land because we regard it as a commodity belonging to us. When we see land as a community to which we belong, we may begin to use it with love and respect. There is no other way for land to survive the impact of mechanized man, nor for us to reap from it the aesthetic harvest it is capable, under science, of contributing to culture (Leopold, 1949).

The question of why, collectively, humanity seems incapable of changing its economic and consumer models to ones that are more sustainable is beyond the scope of this entry. Suffice it to say that denial is made possible because humans are remarkably adaptive; many seem to live in hope of a technological solution to the crises that await under current trends. Ultimately, consciousness of humanity's inseparable dependence on nature's services is required if a functioning civilization is to be maintained.

Cross-References

- ▶ Ecosocial Theory
- ▶ General Population
- ▶ Health Economics
- ▶ Infectious Diseases
- ▶ Mental Illness
- ▶ Mental Stress

References and Readings

Berkes, F., Kislalioglu, M., Folke, C., & Gadgil, M. (1998). Exploring the basic ecological unit: Ecosystem-like concepts in traditional societies. *Ecosystems, 1*, 409–415.

Buck, S. J. (1985). No tragedy on the commons. *Environmental Ethics, 7*(Spring), 49–61.

Butler, C. D. (2008). Human health and forests: An overview. In C. J. P. Colfer (Ed.), *Human health and forests: A global overview of issues, practice and policy* (pp. 13–33). London: Earthscan.

Chivian, E., & Bernstein, A. (Eds.). (2008). *Sustaining life. How human health depends on biodiversity*. Oxford, UK: Oxford University Press.

Daly, H. (1996). *Beyond growth*. Boston: Beacon.

Danielsen, F., Beukema, H., Burgess, N. D., Parish, F., Brühl, C. A., Donald, P. F., et al. (2008). Biofuel plantations on forested lands: Double jeopardy for biodiversity and climate. *Conservation Biology, 23*(2), 348–358.

Fauth, J. E. (1997). Working toward operational definitions in ecology: Putting the system back into ecosystem. *Bulletin of the Ecological Society of America, 78*(4), 295–297.

Gillson, L. (2006). A "large infrequent disturbance" in an East African savanna. *African Journal of Ecology, 44*, 458–467.

Keesing, F., Belden, L. K., Daszak, P., Dobson, A., Harvell, C. D., Holt, R. D., et al. (2010). Impacts of biodiversity on the emergence and transmission of infectious diseases. *Nature, 468*, 647–652.

Leopold, A. (1949). *A sand country almanac. With essays on conservation from Round River*. Oxford, UK: Oxford University Press.

Louv, R. (2008). *Last child in the woods*. New York: Algonquin Books of Chapel Hill.

Lovelock, J. (2009). *The vanishing face of Gaia, a final warning*. London: Allen Lane.

Millennium Ecosystem Assessment. (2003). *Ecosystems and human well-being. A framework for assessment*. Washington, DC: Island Press.

Ramakrishnan, P., Saxena, K., & Chandrashekara, U. (1998). *Conserving the sacred: For biodiversity management*. New Delhi: UNESCO, Oxford, IBH.

Soskolne, C. L., Westra, L., Kotzé, L., Mackey, B., Rees, W., & Westra, R. R. (Eds.). (2008). *Sustaining life on earth*.

Environmental and human health through global governance. Lanham, MD: Lexington Books, a Division of Rowman & Littlefield Publishers.
Tansley, A. (1935). The use and abuse of vegetational concepts and terms. *Ecology, 16*, 284–307.
Wilcox, B. A., Aguirre, A. A., & Horwitz, P. (2011). EcoHealth: Connecting ecology, health and sustainability. In A. A. Aguirre, R. Ostfeld, et al. (Eds.), *Conservation medicine* (2nd ed.). Oxford, UK: Oxford University Press.
Wilson, E. O. (1984). *Biophilia*. Cambridge, MA: Harvard University Press.

Education, Health

▶ Health Education

Education, Lack Of: As a Risk Factor

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Synonyms

Socioeconomic status (SES)

Definition

Lack of education as a risk factor of health conditions is part of the category of factors termed socioeconomic status (SES). Level of education can be measured in several manners including years of education (e.g., 5, 10, 18 years), stage of education (e.g., primary school, secondary school, professional vocation, academic degree, graduate studies), as well as types of education (e.g., vocational, humanities, engineering, biomedical, social sciences). Low education has been shown to be a risk factor of multiple disease outcomes and can be construed as a source of health inequalities. For example, (Clegg et al. 2009) found that level of education below high-school education was a risk factor of cancer in men and women. In a Scottish study, lower education was associated with shorter height, high blood pressure, smoking, poorer lung functioning, and higher risk of death (Davey Smith et al., 1998). In the same study, occupational status however emerged as a more important risk factor than education. Nevertheless, these studies show that lack of education can be a risk factor of known disease risk factors (e.g., smoking, high blood pressure) and of actual illnesses (e.g., cancer).

Among the mechanisms suggested to link low education with poor health outcomes are poor health behaviors (e.g., smoking, poor diet), stress, and physiological factors (e.g., inflammation, cardiovascular reactivity, oxidative stress). For example, Finkelstein, Kubzansky, Capitman, and Goodman (2007) found an inverse correlation between levels of education (below 12 years, 12–15 years, college, professional) and stress levels. Janicki-Deverts, Cohen, Matthews, Gross, and Jacobs (2009) found that initially high education levels predicted lower levels of oxidative stress and higher levels of antioxidants. Oxidative stress is a major etiological factor of multiple chronic diseases including cancer, heart disease, and dementia. Taken together, low education level is a risk factor of poor health, possibly via various psychophysiological pathways, access to material and health resources, and inadequate health behaviors.

References and Readings

Clegg, L. X., Reichman, M. E., Miller, B. A., Hankey, B. F., Singh, G. K., Lin, Y. D., Goodman, M. T., Lynch, C. F., Schwartz, S. M., Chen, V. W., Bernstein, L., Gomez, S. L., Graff, J. J., Lin, C. C., Johnson, N. J., & Edwards, B. K. (2009). Impact of socioeconomic status on cancer incidence and stage at diagnosis: Selected findings from the surveillance, epidemiology, and end results: National Longitudinal Mortality Study. *Cancer Causes & Control, 20*, 417–435.
Davey Smith, G., Hart, C., Hole, D., MacKinnon, P., Gillis, C., Watt, G., Blane, D., & Hawthorne, V. (1998). Education and occupational social class: Which is the more important indicator of mortality risk? *Journal of Epidemiology and Community Health, 52*, 153–160.
Finkelstein, D. M., Kubzansky, L. D., Capitman, J., & Goodman, E. (2007). Socioeconomic differences in

adolescent stress: The role of psychological resources. *The Journal of Adolescent Health, 40*, 127–134.
Janicki-Deverts, D., Cohen, S., Matthews, K. A., Gross, M. D., & Jacobs, D. R., Jr. (2009). Socioeconomic status, antioxidant micronutrients, and correlates of oxidative damage: The Coronary Artery Risk Development in Young Adults (CARDIA) study. *Psychosomatic Medicine, 71*, 541–548.

Education, Patient

Yori Gidron
Faculty of Medicine and Pharmacy,
Free University of Brussels (VUB), Jette,
Belgium

Synonyms

Health education

Definition

Patient education is a basic step in medical care, reflecting empowering patients with knowledge on the risk of or nature of an illness, how to prevent it, how to perform self-care, and when to seek help. Patient education can be provided by physicians, nurses, physiotherapists, health psychologists, etc. and is a basic part of adequate clinician-patient communication. This can be seen as part of health education, where people learn to prevent, identify the signs, seek treatment for an illness, and perform self-care behaviors. This reflects the move in medicine from a hierarchical doctor-patient style toward a more self-managed and active patient role. Patient education can include, for example, information on the consequences of smoking and excessive alcohol consumption, use of condoms and the consequences of not using condoms, how to perform self-monitoring and management of insulin levels in diabetic patients, and adherence to medical treatment in cardiac patients after surgery. Lagger, Pataky, & Golay (2010) reviewed 35 meta-analyses of 598 studies on therapeutic patient education in asthma, cancer, and diabetes, among various chronic diseases. They found that in 64% of studies, improvements were found. However, unlike "therapeutic education," patient education alone relies mainly on increasing knowledge, but rarely addresses patients' psychological factors that impede healthy behaviors such as barriers and social pressures against adopting healthy behaviors. Studies have found multiple barriers in relation to healthy eating, physical activity, and in cardiac patients' medical adherence (e.g., Zunft et al., 1999). Furthermore, in the context of condom use, for example, studies have shown that education led to little or no increases in condom use (Gallant & Maticka-Tyndale, 2004). In contrast, use of the "psychological inoculation" method, which precisely trains people to break their own barriers, may have better effects than health education alone (Duryea, Ransom, & English, 1990). Thus, while patient education is an essential element of prevention and treatment, its effectiveness can be increased when accompanied by cognitive-behavior skills for reducing patient barriers and increasing self-efficacy, or by including simple behavioral tips for moving patients along different stages of behavior change.

Cross-References

► Self-care

References and Readings

Duryea, E. J., Ransom, M. V., & English, G. (1990). Psychological immunization: theory, research, and current health behavior applications. *Health Education Quarterly, 17*, 169–178.
Gallant, M., & Maticka-Tyndale, E. (2004). School-based HIV prevention programmes for African youth. *Social Science & Medicine, 58*, 1337–1351.
Lagger, G., Pataky, Z., & Golay, A. (2010). Efficacy of therapeutic patient education in chronic diseases and obesity. *Patient Education and Counseling, 79*, 283–286.
Zunft, H. J., Friebe, D., Seppelt, B., Widhalm, K., Remaut de Winter, A. M., Vaz de Almeida, M. D., et al. (1999). Perceived benefits and barriers to physical activity in a nationally representative sample in the European Union. *Public Health Nutrition, 2*, 153–160.

EEG

► Brain Wave

Effectiveness

► Efficacy

Effect Modification

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

Effect modification occurs when an effect modifier is associated with both an apparent case and an apparent effect and modifies the association of interest (Katz, 2001).

Consider the example of the association between vigorous exercise and risk for heart disease. This association is real, but its direction varies with level of fitness. An individual who is essentially fit will in all likelihood reduce his or her risk of heart disease by exercising vigorously. However, an individual who is unfit may acutely increase his or her risk by participating in such exercise, or by engaging in other vigorous physical activity. An unfortunately too frequent example of this occurs when unfit individuals attempt to shovel heavy snow, a very physically demanding task, and suffer a myocardial infarction. The association between vigorous exercise and heart disease, therefore, while real, is not unidirectional, but is modified by an individual's level of physical fitness.

Cross-References

► Cardiovascular Disease

References and Readings

Katz, D. L. (2001). *Clinical epidemiology and evidence-based medicine: Fundamental principles of clinical reasoning and research*. Thousand Oaks, CA: Sage.

Efficacy

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

The efficacy of a treatment is a measure of its influence on a clinical characteristic or outcome of interest when administered in tightly controlled, near-ideal circumstances. It is a measure of benefit.

The term "efficacy" can be meaningfully differentiated from the related term, "effectiveness." Effectiveness, which is of greater clinical interest, is a measure of the treatment's influence under real-world conditions (Katz, 2001).

Efficacy is typically assessed wherever possible in a randomized controlled clinical trial, since this provides the best approximation to "near-ideal circumstances." A paradox of such assessment, however, is that the deliberate (and, at this point in time, desirable) nature of the tightly controlled environment in which the treatment is assessed limits the generalizability of the therapeutic results obtained to interventional therapy administered outside that setting. There are many reasons for this, including the relatively homogenous nature of the subjects participating in the trial: Strict inclusion and exclusion criteria are typically employed. From a statistical perspective, this tightly controlled subject population is beneficial in that it reduces extraneous variation that may lead to a genuine treatment effect being difficult to detect. However, a clinically important effect seen in a well-controlled trial may not be reflected when the treatment is applied to a heterogeneous population in circumstances of real-world clinical practice.

This is why effectiveness assessments are so important. Various approaches can be employed. One is to conduct large-scale trials (sometimes called mega-trials or large simple trials) which employ much more simple measurement schedules that focus on the one aspect of interest and that are conducted in conditions much more akin to clinical practice.

Cross-References

- ▶ Baseline
- ▶ Comparative Effectiveness Research

References and Readings

Katz, D. L. (2001). *Clinical epidemiology and evidence-based medicine: Fundamental principles of clinical reasoning and research*. Thousand Oaks, CA: Sage.

Efficacy Cognitions

Jorie Butler
Department of Psychology, University of Utah, Salt Lake City, UT, USA

Definition

Efficacy cognitions: Efficacy cognitions are thoughts that develop from self-efficacy. Self-efficacy is the belief in personal ability to successfully perform challenging life tasks (Bandura, 1977a). Self-efficacy develops from mastery experiences, modeling, social persuasion, and physiology (Bandura, 1977b).

Description

Efficacy cognitions include thoughts about performance that are optimistic, pessimistic, productive, or self-debilitating. Efficacy cognitions are influenced by mastery experiences which promote cognitive expectations. Efficacy cognitions can be modeled similar to self-efficacy. Social persuasion, in the form of encouragement from others to achieve, or stated positive expectations, promotes positive efficacy cognitions. Negative persuasion in the form of discouragement or deflating comments promotes negative efficacy cognitions. Negative interpretations of physiological responses such as strong emotions (e.g., anxiety) and the physiological accompaniments (fast heart rate) influence efficacy cognitions as negative, "I'll never be able to do this, I'm terrified" or positive, "I've got this in the bag, I'll just enjoy it!" Efficacy cognitions spring from interpretations and can contribute to habitual interpretations.

Efficacy cognitions are not perfectly aligned with reality – hence the concert pianist overcome with doubts about their potential to produce beautiful music and the exuberant child overestimating the pleasantness in tone of their singing voice. Efficacy cognitions contribute to motivation to perform an activity, personal well-being, positive health behavior, and future achievement. Negative efficacy cognitions result in avoidance of the activity, whereas confident efficacy cognitions promote engagement in the task. Emotions related to activity performance will ally with the tone of the efficacy cognitions – stress and anxiety for tasks about which efficacy cognitions are negative, joyful immersion for activities about which efficacy cognitions are positive.

Productive efficacy cognitions contribute to a view that difficult tasks are challenges to be mastered. Armed with such thoughts, people are more likely to approach the difficult task at hand with zestful effort. Self-debilitating efficacy cognitions are centered on tasks as insurmountable or fearsome result in activities that are dropped after minimal obstacles intervene. Optimistic efficacy cognitions promote task enjoyment whereas pessimistic thoughts promote task-associated distress and early failure. Efficacy cognitions influence action planning for future goals. Individuals planning to implement a health behavior change – such as smoking cessation, eating a healthier diet, exercising, or adhering to recommendations for managing a chronic illness – are more likely to be successful at

implementation when efficacy cognitions are positive. In the health behavior change model, individuals progress through stages of change that culminate in maintaining lasting change (Prochaska & Vellicer, 1997). Early in the process, during the contemplation stage, efficacy cognitions are particularly important. Individuals who doubt they can make a successful health change during the contemplation may not reach the preparation stage – in which planning for change will occur – or the action stage when substantive changes are made.

The expectancies in efficacy cognitions may influence future performance more than objective measures such as past performance (Schunk & Pajares, 2005). Efficacy cognitions have wide-ranging implications as the perception that one can accomplish something can be as motivating as having accomplished the task in the past. Thus, positive efficacy cognitions can have a tremendous effect on personal motivation and willingness to experience activities. Conversely, negative efficacy cognitions – even in the face of contradictory objective evidence – can stymie efforts to engage in challenging tasks. Individuals expecting poor outcomes, "I'll never be able to quit smoking, I've tried and failed a dozen times" may indeed be more likely to experience poorer outcomes. In contrast, individuals facing health behavior change with positive expectations may be able to incorporate adaptive strategies for success into their planning more effectively (Gollwitzer, 1999; Schwarzer, 1992).

Efficacy cognitions do not develop in complete independence from objective evidence of past performance and therefore often are realistically correlated with outcomes. Multiple factors influence the development of efficacy cognitions in addition to experiences – such as environmental factors or propensity for anxiety. Individuals who might be expected to feel competent based on other predictors may not if efficacy cognitions have developed in the face of physiological state challenges (anxiety, stress, fear), negative expectations from the social environment (such as stated gendered expectations for performance in athletics or mathematics), or faulty interpretations of past experience (not recognizing a strong or poor performance) (Schunk & Pajares, 2005). Positive efficacy cognitions are strongly associated with performance because such cognitions promote action and use of skills. Negative efficacy cognitions hold individuals back from achieving all they might in their lives and for their health.

Cross-References

- ▶ Affect
- ▶ Cognitive Appraisal
- ▶ Cognitive Distortions
- ▶ Cognitive Restructuring
- ▶ Cognitive Strategies
- ▶ Efficacy
- ▶ Implementation Intentions
- ▶ Locus of Control
- ▶ Self-Efficacy

References and Readings

Bandura, A. (1977a). Self-efficacy: Toward a unifying theory of behavioral change. *Psychological Review, 84*, 191–215.

Bandura, A. (1977b). *Social learning theory*. Englewood Cliffs, NJ: Prentice-Hall.

Bandura, A. (1986). *Social foundations of thought and actions: A social cognitive theory*. Englewood Cliffs, NJ: Prenctice-Hall.

Gollwitzer, P. M. (1999). Implementation intentions. Strong effects of simple plans. *American Psychologist, 54*, 493–503.

Pajares, F. (2002). *Overview of social cognitive theory and of self-efficacy*. Retreived July 12, 2011, from http://www.emory.edu/EDUCATION/mfp/eff.html

Prochaska, J. O., & Velicer, W. F. (1997). The transtheoretical model of health behavior change. *American Journal of Health Promotion, 12*, 38–48.

Schunk, D. H., & Pajares, F. (2005). Competence perceptions and academic functioning. In A. J. Elliot & C. S. Dweck (Eds.), *Handbook of competence and motivation* (pp. 84–104). New York: Guilford Press.

Schwarzer, R. (1992). Self-efficacy in the adoption and maintenance of health behaviors: Theoretical approaches and a new model. In R. Schwarzer (Ed.), *Self-efficacy: Thought control of action* (pp. 217–243). Washington, DC: Hemisphere.

Schwarzer, R., & Renner, B. (2000). Social-cognitive predictors of health behaviour: Action self-efficacy and coping self-efficacy. *Health Psychology, 19*, 487–495.

Egg Donation

► In Vitro Fertilization, Assisted Reproductive Technology

Egg Donor

► In Vitro Fertilization, Assisted Reproductive Technology

Ego-Depletion

► Self-Regulatory Fatigue

eHealth and Behavioral Intervention Technologies

Michelle Nicole Burns and David C. Mohr
Feinberg School of Medicine, Department of Preventive Medicine, Center for Behavioral Intervention Technologies, Northwestern University, Chicago, IL, USA

Synonyms

Behavioral intervention technologies; Internet-based interventions

Definition

eHealth is a broad term that refers to the use of information and communications media to facilitate access to health-related information and to support or deliver healthcare. eHealth can include health informatics, health knowledge management, and health data management. Telemedicine and telehealth are subsets of eHealth. Telemedicine refers to the provision of clinical services via telecommunications technologies (e.g., phone, instant messaging), while telehealth is a more general term (Jordan-Marsh, 2011) that refers to the broader use of telecommunications in healthcare and health promotion (e.g., electronic access to personal health records, websites allowing patients to schedule appointments).

Although healthcare providers and administrators are also targets of eHealth, we will restrict this review to the use of communication technologies aimed at changing patients' behaviors, cognitions, and emotions in the service of better health outcomes. We refer to these interventions as Behavioral Intervention Technologies (BITs). BITs promote behavior change through electronic provision of didactic material, skill-building tasks, feedback, decision-making aids, risk self-assessments, or patient self-management tools.

Description

Remote Provision of Clinical Services. The use of videoconferencing, telephone, and instant messaging harnesses communications technologies to extend care geographically while preserving the traditional structure of behavioral treatments. Videoconferencing has been shown to be an effective treatment delivery medium for a variety of mental health problems, as well as teaching self-management strategies for chronic conditions and providing support to caregivers of older adults. Older studies used videoconferencing to extend care to remote clinics where there was an absence of specialized care, but newer studies are harnessing the capacity to videoconference directly into patients' homes.

Many trials have examined the telephone-based delivery of behavioral interventions to address a wide range of targets including preventive health behaviors such as weight loss and smoking cessation, self-management interventions for chronic illnesses, and mental health. Telephone treatments have generally been shown to be effective, and there is evidence that the use of the telephone may improve access and reduce attrition. A few studies have also indicated that the use of instant messaging to deliver

standard psychological interventions can be effective.

While extending care, each of these delivery modalities progressively decreases the "bandwidth" for social cues (e.g., nonverbal behavior; Mohr, Cuijpers, & Lehman, 2011), which has raised concerns that the reduction in cue bandwidth may reduce efficacy. There is no evidence to date that videoconferencing or telephone delivery reduces efficacy, although this has not been rigorously tested. The evidence for instant messaging is too preliminary to begin to speculate on its comparative efficacy.

Web-Based BITs. Web-based BITs have been evaluated for a growing number of health behavior and mental health problems, including weight loss, physical activity, insomnia, adjustment to illness, depression, and anxiety. While most, but not all, trials find evidence supporting the efficacy of web-based BITs, the effect sizes vary considerably and range from negligible to effects on par with traditional face-to-face care. This variability is likely due to a variety of factors, including website design, implementation, and support features.

The structure of web-based BITs can vary on a wide variety of dimensions, including the degree to which they present static information versus interactive features, the degree of personalization, the use of multimedia, the manner in which patients progress through the intervention (e.g., all material being completely available from the beginning, versus some or all material being presented according to criteria such as time or task completion), the expected length of engagement with the intervention, whether the website is freely available to the general public versus contingent on patient characteristics or healthcare system, and the degree of human support. Improvement in health behaviors is maximized by the incorporation of features such as automated text messages and email, personalized feedback, human support via email or telephone, use of multiple behavior change strategies, and a theoretically informed choice of participants and intervention content (Webb, Joseph, Yardley, & Michie, 2010).

Lower efficacy is often associated with poor patient adherence (e.g., few logins to the site) or treatment dropout (ranging from extremely high, >95% for free-standing depression websites with no human support, to minimal). Website usage may be increased by ensuring that the site is easily usable and navigable, provides tools and information that help the user to achieve his/her goals, is attractive, and conveys credibility (Fogg, 2003). Periodic updates to the website content are likely to draw users back to the site (Brouwer et al., 2011). In addition to website design, a fairly consistent body of literature has shown that the adherence and efficacy of web-based BITs are enhanced when the website is supported by human interaction (Andersson & Cuijpers, 2009; Brouwer et al., 2011). One theoretical model to explain the benefits of human support, called supportive accountability, posits that adherence is enhanced by accountability to a supportive coach or provider. Mutually-agreed-upon process goals (e.g., logging into the website or using website tools) are monitored by a supportive coach, and the user is expected to account for use or nonuse at pre-specified times through personal contact via brief telephone calls, email, or messaging (Mohr et al., 2011).

Variability in adherence may also be associated with the selection of research participants. Trials that have extensive screening processes likely select for patients who are more motivated and more likely to adhere. When screening involves contact with an evaluator, adherence may be even higher. Websites that are accessible to the general population with little or no entry processes can produce high rates of initial access, with few participants returning to the website after one or two visits. It is not yet clear the degree to which these low return rates are due to large numbers of potential users, for whom the website is not appropriate, being able to easily find and investigate the site, versus design and implementation flaws, or users finding the information or help they desired more quickly than expected.

Internet Support Groups. Internet support groups have proliferated, often with the aim of fostering empowerment and sense of community, decreasing illness-related stigma, promoting the sharing of information, and increasing social

support. There is some evidence that therapist-moderated internet support groups can reduce distress. However, findings regarding the efficacy of un-moderated internet support groups are mixed. Some trials demonstrate modest improvement, but many trials find no significant effect or even increased distress for some users. This suggests that assumptions as to why and how these groups might be helpful may be erroneous. Thus, while the appeal of social media is considerable, little is currently known about how to effectively harness online social networks to improve health outcomes.

Emerging BITs. Web-deployed virtual worlds offer a diverse range of health intervention and educational experiences. For example, Second Life is being used to provide health-related information, meetings, support groups, simulations of medical procedures or symptoms, discussion groups, appointments with human healthcare providers, movies, and opportunities to practice new skills (e.g., role plays; Beard, Wilson, Morra, & Keelan, 2009). Serious gaming is a field in which the entertainment value of games is harnessed for a purpose, such as improving health (Zyda, 2005). Serious health-related games have been developed, in particular to increase physical activity and improve diet in children. Although outcomes have been promising, more research is needed to demonstrate clinical efficacy.

Mobile BITs. Mobile electronic devices (e.g., handheld computers, mobile phones) and wireless technology can be used to establish a continuous connection with patients as they conduct their daily lives. This subset of eHealth is often referred to as mHealth. Real-time delivery of intervention (e.g., encouragement, information, therapeutic tools) can be provided to the patient in their own environment. mHealth BITs can also collect information about the patient's current state and provide tailored intervention based on that state. Finally, as Smartphones can access the web, web-based and mobile components can be integrated into the same intervention.

A growing number of studies have examined mobile phone BITs that target preventive health behaviors (smoking cessation, weight loss, and physical activity), self-management of chronic illnesses (e.g., diabetes, asthma), mood and anxiety disorders, schizophrenia, and medication adherence. Trials have found positive short-term benefits. However, literature on the effects of mHealth is still limited in many clinical areas, and not enough high-quality studies have been conducted to enable a reliable quantitative meta-analysis (Heron & Smyth, 2010).

Most mHealth BITs include SMS messages, which vary on a number of dimensions. Some studies send informational messages or reminders, while other interventions employ SMS dialogues which are commonly automated and lead to the provision of tailored information. SMS dialogues are often initiated by the intervention, but some focus on or allow patient-initiated SMS dialogues. For example, many of the disease self-management interventions require the patient to provide information (e.g., blood pressure for hypertension interventions), which then results in tailored SMS feedback. The frequency of the SMS messages can vary from 5+ per day to a little as once weekly, and is usually tied to the expected frequency of the targeted behavior. Degree of tailoring and personalization also varies, with some mHealth BITs providing highly tailored messages, while others provide more generic messages or tips. Finally, mHealth BITs vary to the degree to which they rely solely on the mobile intervention or are supplemented by other intervention strategies such as interactive websites or consultations with healthcare providers.

An emerging area in mHealth is the development of passive data collection methods that avoid problems related to patient's reluctance to log information. In this way, mHealth BITs can detect when intervention is needed, without requiring the patient to self-report their current state. Passive data collection uses sensors to automatically collect data that can help to infer patient states. Such sensors can be located within the mobile device itself (e.g., GPS, accelerometer), or through wirelessly connected external devices (e.g., heart rate or glucose monitors). There are two ways in which intervention delivery can be informed by sensor data. First, algorithms can be developed based on existing scientific knowledge (i.e., expert systems).

The algorithms are then applied to make inferences regarding the patient's state, and consequently their need for intervention, from the sensor data. As mobile devices can also allow patients to self-report their current states, the second approach is to use machine learning techniques (Witten & Eibe, 2005) to model the relationship between sensor data and patient states. These models are then used to predict patient's states solely from new sensor data, with the advantage being that the models were automatically generated and personalized. Use of machine learning in this way is a new and complex approach that has been applied to detect physical activities, mood, and social context with varying levels of accuracy (e.g., Burns et al., 2011).

Potential Benefits of BITs. The primary anticipated benefit of BITs is increased access to behavioral healthcare services. Telehealth can be used to assess and provide services to patients living in rural areas, those with medical conditions that affect mobility, or patients for whom travel to service providers is too time consuming given their employment, caregiving, or other responsibilities. Web-based and mHealth BITs are expected to deliver care at substantially reduced costs, and there are some preliminary studies suggesting that web-based BITs can be very cost effective. For example, BITs are usually designed to require less time burden on clinicians, and since BITs can be delivered remotely, they might also reduce the patient's transportation costs and lost work productivity due to time spent in transit. However, there is still a paucity of cost-effectiveness studies examining eHealth compared to usual care, and results may vary based on whether costs and benefits are calculated in terms of the individual patient, the healthcare system, or society as a whole.

Given the prevalence of obesity, smoking, chronic illness, and mental illness, there will never be enough behavioral health specialists to meet population needs for behavioral care. BITs offer the possibility of bridging the gap between behavioral health interventions, which have traditionally been delivered on an individual or small group basis, and population-level public health intervention. Access to the web is growing rapidly. Mobile phones have reduced the Digital Divide between racial/ethnic minority and majority groups in the United States, with African American and English-speaking Hispanic adults using mobile devices to access the web at greater rates than White, non-Hispanic adults (Smith, 2011). Thus, mobile BITs might be used to more equitably distribute healthcare services. Should BITs fulfill expectations to increase access to care in underserved populations, this may also facilitate a transition away from acute, crisis-based care toward preventive care.

eHealth interventions, particularly those involving remote patient monitoring or real-time outreach, may increase detection of emergency situations. Often, web-based and mobile BITs also allow patients a 24-h capability to send messages to providers. This convenience may encourage patients to report their difficulties in real time rather than at their next scheduled appointment, by which time the problem may have worsened or the patient may have forgotten important information. Many studies have demonstrated safe implementation of BITs in specific clinical areas.

Potential Risks of BITs. The Digital Divide refers to continuing disparities in internet access and familiarity based on age, race/ethnicity, and socioeconomic status. These disparities may be reduced by sensitivity to differences in the way technology is used between different populations. For example, only 2% of mobile phone owners 65+ years of age access social networking sites using their mobile phone, while 24% use text messaging (Smith, 2011). Trends suggest that text messaging will be used by increasing numbers of the elderly; thus, if peer support is involved in a BIT for older adults, the forum for peer communication at this time should likely be text messaging rather than a website. Community-based participatory research and careful usability testing of BITs should also be conducted with underserved populations to address issues of access, level of familiarity with the technology, and concordance with the ways in which the population already uses and perceives the technology.

Privacy and security are hotly debated issues in BITs research. Privacy refers to the prevention of

improper disclosure of personal information, while security refers to the technical and procedural mechanisms used to protect privacy. Security protocols are a necessary part of any BIT, and they require ongoing consultations with an IT expert who will remain current on security vulnerabilities in supporting components (e.g., the operating system and servers; Bennett, Bennett, & Griffiths, 2010). Encrypted data transmission and restricted access to research data should be standard protocols. Increasingly, however, the efficiency of BITs is being maximized by conducting much of the computing on the user's device. This introduces privacy risks when the patient uses devices that are shared or monitored by others, such as public or work computers. Protocols for wiping data on these remote devices should thus be in place.

Assuming adequate technical security measures, privacy is more likely to be compromised by procedures associated with the interventions, and by the users themselves not taking advantage of security measures such as passcodes. Research staff require ongoing training to avoid procedural errors (Bennett et al., 2010) and effectively teach patients to do the same. Another challenge is conveying to patients how their health information will be handled in terms that are clearly understood. This is an ethical responsibility, and may also help to gain the trust of individuals whose privacy concerns or unanswered questions may prevent them from accessing or fully utilizing potentially beneficial BITs.

Future Directions. BITs research integrates and absorbs methodology from many disciplines, including behavioral science, medicine, computer science, engineering, human computer interaction, computer-mediated communication, visual design, education, and public health. There is a need for individual researchers with expertise across a number of these areas, as well as multidisciplinary team science. New, integrated theoretical frameworks are also needed to describe interactions between use of technology and behavioral change processes. For example, given the virtually ubiquitous presence of information and communication technologies and their resulting ability to engage with individuals as they interact with multiple spheres of their daily lives (e.g., intrapersonal, interpersonal, institutional, the natural environment, and macro-social factors such as public policy and economic realities), an ecological intervention model has been created that would encompass expertise in each of these domains (Patrick, Intille, & Zabinski, 2005).

Due to the rapid development of new technological capabilities, new methods are evolving to evaluate the efficacy of BITs. The randomized controlled trials traditionally used to demonstrate efficacy are time intensive, and by the time such trials are concluded, the technology being evaluated is likely to be outdated. Disciplines outside of clinical science, in which the rapidity of technological advances has long been a common concern, may be well suited to help behavioral researchers address this challenge. Methodologies that borrow from engineering, such as Multiphase Optimization Strategies (Collins, Murphy, & Strecher, 2007), may be more appropriate in optimizing and evaluating new BITs.

Dissemination. Strategies to disseminate and integrate BITs into healthcare are largely unexplored. BITs can be deployed independent of healthcare delivery systems; this is evidenced by the growing number and use of websites aimed at supporting diet, weight loss, and health lifestyle, as well as the proliferation of mHealth Smartphone applications. There is little efficacy data for many of these BITs, or information on how their use impacts health, healthcare utilization, or healthcare cost. There is also considerable interest in integrating BITs into existing healthcare delivery systems. This integration will require research on at least four levels: (1) Research should determine how BITs will fit in with existing treatment options. For example, stepped care models may first provide the patient with a BIT, and reserve more clinician-intensive treatments for patients who fail to respond. (2) BITs can be integrated into electronic medical records and patient management systems to facilitate referrals, treatment monitoring, follow-up care, and integration of the

BIT into the patient's overall treatment plan. (3) Demonstrated cost-effectiveness of BITs relative to existing treatments will be required for adoption by healthcare systems. (4) Implementation research will be required to identify implementation barriers and opportunities, as well as develop implementation models that can optimize the uptake and use of efficacious BITs by both patients and healthcare providers.

Cross-References

- ▶ Behavior Change
- ▶ Electronic Health Record
- ▶ Health Care Access
- ▶ Medication Event Monitoring Systems
- ▶ Patient Adherence
- ▶ Research to Practice Translation
- ▶ Translational Behavioral Medicine

References and Readings

Andersson, G., & Cuijpers, P. (2009). Internet-based and other computerized psychological treatments for adult depression: A meta-analysis. *Cognitive Behaviour Therapy, 38*(4), 196–205.

Beard, L., Wilson, K., Morra, D., & Keelan, J. (2009). A survey of health-related activities on second life. *Journal of Medical Internet Research, 11*(2), e17.

Bennett, K., Bennett, A. J., & Griffiths, K. M. (2010). Security considerations for e-mental health interventions. *Journal of Medical Internet Research, 12*(5), e61.

Brouwer, W., Kroeze, W., Crutzen, R., de Nooijer, J., de Vries, N. K., Brug, J., & Oenema, A. (2011). Which intervention characteristics are related to more exposure to Internet-delivered healthy lifestyle promotion interventions? A systematic review. *Journal of Medical Internet Research, 13*(1), e2.

Burns, M. N., Begale, M., Duffecy, J., Gergle, D., Karr, C. J., Giangrande, E., & Mohr, D. C. (2011). Harnessing context sensing to develop a mobile intervention for depression. *Journal of Medical Internet Research, 13*(3), e55.

Collins, L. M., Murphy, S. A., & Strecher, V. (2007). The multiphase optimization strategy (MOST) and the sequential multiple assignment randomized trial (SMART) – New methods for more potent eHealth interventions. *American Journal of Preventive Medicine, 32*(5), S112–S118.

Fogg, B. J. (2003). *Persuasive technology*: *Using computers to change what we think and do*. San Francisco: Morgan Kaufmann.

Heron, K. E., & Smyth, J. M. (2010). Ecological momentary interventions: Incorporating mobile technology into psychosocial and health behaviour treatments. *British Journal of Health Psychology, 15*(Pt 1), 1–39.

Jordan-Marsh, M. (2011). *Health technology literacy*: *A transdisciplinary framework for consumer-oriented practice*. Sudbury, MA: Jones & Bartlett Learning.

Mohr, D. C., Cuijpers, P., & Lehman, K. (2011). Supportive accountability: A model for providing human support to enhance adherence to eHealth interventions. *Journal of Medical Internet Research, 13*(1), e30.

Patrick, K., Intille, S. S., & Zabinski, M. F. (2005). An ecological framework for cancer communication: Implications for research. *Journal of Medical Internet Research, 7*(3).

Smith, A. (2011). *Americans and their cell phones*. Pew Internet and American Life Project, Report Released August 15, 2011. Retrieved December 9, 2011, from http://www.pewinternet.org/~/media/Files/Reports/2011/Cell%20Phones%202011.pdf

Webb, T. L., Joseph, J., Yardley, L., & Michie, S. (2010). Using the Internet to promote health behavior change: A systematic review and meta-analysis of the impact of theoretical basis, use of behavior change techniques, and mode of delivery on efficacy. *Journal of Medical Internet Research, 12*(1), e4.

Witten, I. H., & Eibe, F. (2005). *Data mining*: *Practical machine learning tools and techniques* (2nd ed.). San Francisco: Morgan Kaufman.

Zyda, M. (2005). From visual simulation to virtual reality to games. *Computer, 38*(9), 25–32.

Elderly

Ivan Molton
Department of Rehabilitation Medicine, University of Washington, Seattle, WA, USA

Synonyms

Aged; Older adult; Senior

Definition

The term "elderly" is derived from the Middle English word *eald* (meaning old) and generally refers to an individual who is near or surpassing

the average life expectancy for his or her community, culture, and historical period. The term differs from clinical or medical language used to describe older adults (e.g., senescent or geriatric) in that it does not describe biological aspects of aging. Rather, "elderly" is used more broadly in the context of social gerontology, and carries the connotation of having achieved a certain degree of respect, status, expertise, or wisdom with advanced age (i.e., as an *elder*). For the purposes of research and policy efforts, the age cut off for "elderly" is often set in western countries at 65 or 70, based on the age at which individuals have historically been able to receive government retirement benefits. However, the term is descriptive rather than scientific, and does not typically denote a particular age band within older adulthood.

Cross-References

- ▶ Aging
- ▶ Gerontology

Electrocardiogram (EKG)

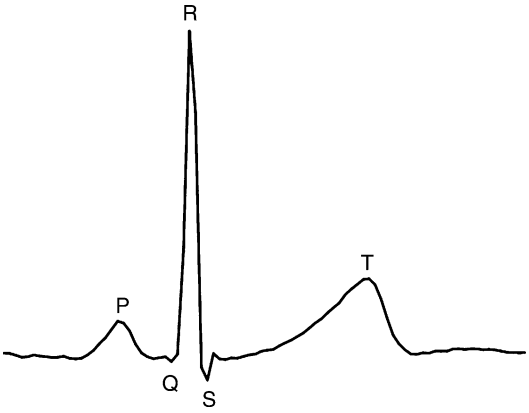
Douglas Carroll
School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

ECG

Definition

The electrocardiogram (EKG) is the noninvasive record of the electrical activity of the heart muscle, as reflected in tiny electrical changes on the skin, during the heart's active (systole) and passive (diastole) phases (Hampton, 2008). The EKG can be recorded easily from two electrodes placed on either side of the heart, for example, on the chest or on the left and right arms. For clinical and diagnostic purposes, however, it is usual to attach an array of 12 electrodes at various bodily sites so that the EKG can be recorded from different orientations. The characteristic wave form (see Fig. 1) that identifies the heart's active phase, i.e., when it beats and pumps blood into the systemic circulation, was first described in 1903 by Willem Einthoven, in Leiden in the Netherlands, although electrical records of heart beats had been made as early as 1872. In 1924, Einthoven was awarded the Nobel Prize for Medicine for his research in EKG. The first wave in the three-wave systolic portion of the EKG record is the P-wave, and it represents the depolarization, i.e., the contraction, of the atrial chamber of the heart. Next, the R-wave, usually considered at the whole QRS complex, reflects the depolarization of the ventricles. Finally, the T-wave represents the repolarization, i.e., recovery, of the ventricles. In behavioral medicine, interest in the EKG is usually confined to the derivation of heart rate, the number of systoles in a given minute, or to its reciprocal, heart period, the time between successive R-waves. The former indicates the speed at which the heart is beating under specified circumstances, such as during relaxed rest or psychological stress exposure. Unfortunately, this tells us nothing about volume of blood being pumped by the heart into the circulation: for that, we need



Electrocardiogram (EKG), Fig. 1 EKG waveform

other techniques, such as impedance cardiography and, more recently, Doppler echocardiography. The latter, heart period and its variability, can, particularly when subject to spectral analysis of frequency, tell us about the extent of activation of the heart by the main parasympathetic nerve, the vagus, and about the balance between parasympathetic and sympathetic neural activation of the heart. Finally, the precise configuration of the EKG can tell us other useful things, particularly in clinical settings. For example, a blunted or inverted T-wave is a reasonable indicator of cardiac ischemia, where the heart muscle is suffering from impaired blood flow and hence a restricted oxygen supply.

Cross-References

► Heart Rate

References and Readings

Hampton, J. R. (2008). *The ECG made easy* (7th ed.). London: Churchill Livingstone.

Electrodermal Activity (EDA)

Hugo Critchley and Yoko Nagai
Brighton and Sussex Medical School, University of Sussex, Brighton, East Sussex, UK

Definition

Electrodermal activity (EDA) reflects the output of integrated attentional and affective and motivational processes within the central nervous system acting on the body. EDA is a valuable tool in behavioral medicine as a biomarker of individual (state and trait) characteristics of emotional responsiveness, as an index for direct examination of axis of stress-related effects on bodily function, and as a potential avenue of treatment of psychosomatic conditions through biofeedback training. Below is an overview of EDA with examples of its application to behavioral medicine.

Description

What Is Electrodermal Activity?

Electrodermal activity (EDA) is a measure of neurally mediated effects on sweat gland permeability, observed as changes in the resistance of the skin to a small electrical current, or as differences in the electrical potential between different parts of the skin. The EDA signal reflects the action of sympathetic nerve traffic on eccrine sweat glands. There are two salient features of this sympathetic innervation that enhance the usefulness of EDA in psychophysiology and behavioral medicine. First, there is no antagonistic parasympathetic innervation of sweat glands (i.e., EDA reflects only sympathetic activity not sympathovagal balance), and second neurotransmission at the effector synapse is (almost completely in adults) cholinergic, i.e., mediated by acetylcholine release. This differs from the noradrenergic neurotransmission typical of other sympathetic effector synapses and further makes the EDA signal independent of circulating adrenaline and noradrenaline levels.

Sympathetic neural activity in skin is closely coupled to changes in mental state: In the laboratory setting, at rest and constant temperature, EDA indexes change in attention and cognitive and emotional states of arousal, expressed both as sustained shifts in tonic level (skin resistance level, SRL, or skin conductance level, SCL; see below) and transient responses evolving over the course of a few seconds (galvanic skin response, GSR, sympathetic skin response, SSR, skin resistance response, SRR, or skin conductance response, SCR) (Venables & Christie, 1973). Tonic and phasic aspects of EDA interrelate, yet are dissociable.

Within the brain, psychological influences on EDA are thought to emerge from activity within reticular formation centers within the brainstem and thalamus, in turn influenced by cortical mechanisms controlling orientation to salient

information (Luria & Homskaya, 1970). Frontal lobe regions strongly influence the orienting electrodermal reflex (Venables & Christie, 1973). Correspondingly, the magnitude of EDA responses in humans is reduced following discrete lesions to the prefrontal cortex and also related "attentional centers" within the anterior cingulate and right parietal lobe (Zahn, Grafman, & Tranel, 1999). Moreover, individual differences in the frequency of discrete EDA responses correlate with the size of prefrontal lobe regions (Raine, Lencz, Bihrle, LaCasse, & Colletti, 2000). Functional imaging studies implicate ventromedial prefrontal cortex (Critchley, Elliot, Mathias, & Dolan, 2000) and anterior cingulate and amygdala (Williams et al., 2000) in phasic EDA responses to motivationally important stimuli. Nagai, Critchley, Featherstone, Trimble, and Dolan (2004a), using EDA biofeedback during functional neuroimaging, showed differential coupling of brain regions to phasic EDA responses (widespread enhancement within the anterior cingulate/dorsolateral prefrontal cortices and subcortical thalamic and brainstem centers) when compared to the tonic EDA level (which was inversely correlated with activity in ventromedial prefrontal cortex and subgenual cingulate). This latter observation links the EDA level to processes ascribed to the "default mode network" of brain function (Raichle et al., 2001).

Applications of Electrodermal Activity

Among the autonomic nervous system responses, EDA is a particularly useful parameter because EDA responses are easy to measure and to elicit reliably (much more difficult to suppress). However, EDA is sensitive to a wide variety of stimuli, hence careful interpretation is required. Changes in EDA also typically occur as part of a complex, patterned autonomic reaction wherein EDA may serve as a circumstantial marker of an accompanying physiological response. As noted, a number of functional variables can be derived from recorded EDA. The basal (tonic) level of electrodermal arousal reflected in SRL/SCL can be used to track individual differences in the general level of arousal and the integrity of diurnal rhythms: The skin resistance level rises during sleep and drops sharply in the morning on awakening, returning to approximately the same level of presleep resistance by the evening. Phasic electrodermal responses such as SRR and SCR are easily elicited by emotional stimulation or a change in attention and interact with the tonic basal level of electrodermal arousal. Thus EDA in a combined form has been widely applied as an index of physiological and emotional arousal in studies of stress responsivity, including challenges with pain shock, emotional films, and mental and physical effort (e.g., Folkins, 1970).

Psychological and Psychosomatic Illness

This relevance of such studies to understanding the psychological and physical manifestations of chronic stress is based on the notion that individuals with increased physiological (EDA) reactivity to stressors are most at risk of long-term detrimental health consequences. In clinical populations, higher skin conductance level and increased phasic responsivity are reported in anxiety patients (Raskin, 1975). In this context, EDA may be a more specific biomarker; patients with panic disorder and agraphobia showed delayed habituation and greater nonspecific phasic fluctuation compared with other anxiety patients (Birket-Smith, Hasle, & Jensen, 1993); patients with posttraumatic stress disorder (PTSD) also show slower habituation even in response to neutral stimulation (this has been suggested as a trait marker for PTSD susceptibility; Rothbaum et al., 2001).

In contrast, EDA hypo-responsivity and hyporeactivity are reported with patients with depression (Williams, Iacono, & Remick, 1985) and related to higher occurrence of suicidal attempts (Thorell, 1987). Faster EDA habituation is reported in patients with a history of suicidal attempt compared to those with non-suicidal attempts (Jandl, Steyer, & Kaschkaet, 2010). Hyporesponsivity and risk is also a feature of psychopathy (Hare, 1978). While in some countries EDA is incorporated in polygraphy used in criminal justice proceedings and forensic evaluations, it is worth noting that hyporesponsivity may be present across constitutional, or developmentally acquired, disorders of empathy.

Personality factors are commonly linked to differences in vulnerability to stress-related disease, the concept of Type A personality being particularly studied. Type A behavior is characterized by excessive competitiveness, aggressiveness, impatience, chronic haste, and striving for achievement. People with characteristics of Type A behavior are reportedly more prone to coronary heart disease (CHD) related to psychological (emotional) stress and its impact is mediated by brain-triggered autonomic nervous reactions as well as neuroendocrine responses. It is hypothesized that Type A individuals are physiologically hyperresponsive to stress stimuli, evoking greater sympathetic activity (accompanied by shifts in sympathovagal cardiac responses and enhancement within both adrenomedullary and hypothalamic-pituitary-adrenocortical systems). Type A behaviors such as impatience and hostility have been proposed to reflect underlying sympathetic reactivity. However, while EDA shows some promise as a functional biomarker for cardiac vulnerability and Type A personality, findings are inconclusive (Steptoe & Ross, 1981). Thus in psychosomatic medicine, EDA can be used to quantify individual differences in autonomic reactivity to salient challenges, where enhanced reactivity in one sympathetic axis (EDA) may signal risks (e.g., of hypertension or arrhythmia) mediated through related autonomic pathways.

Therapeutic Applications of Electrodermal Activity

EDA biofeedback has been studied as a treatment tool for anxiety states and stress-sensitive physical disorders. Biofeedback is a biobehavioral treatment approach where an individual/patient learns to gain volitional control over an involuntary bodily process, e.g., the "emotional arousal" associated with sympathetic innervation of the skin. In contrast to other biofeedback approaches including neurofeedback with electroencephalography, EDA biofeedback is easy to implement. Typically EDA biofeedback has been used to train people to reduce their sympathetic arousal with the aim to induce psychological as well as physiological relaxation states that alleviate stress-related tension. One of the earliest applications was for the treatment of tension headaches, where a significant reduction in frequency and intensity of patients' headaches was attained. EDA biofeedback relaxation therapy has been shown to be successful in treating the irritable bowel syndrome (IBS) (Leahy, Clayman, Mason, Lloyd, & Epstein, 1998), where 4 weeks of biofeedback relaxation training improved scores across a range of IBS symptoms (Leahy et al.). EDA biofeedback relaxation training has been tried for other specific psychological (anxiety disorders, hyperkinesis) and physical (hyperhidrosis, bruxism, weight control, migraine, tics, tremor) conditions. However, the research evidence base is limited and inferences regarding the effectiveness of these approaches are constrained by variation in treatment delivery. In recent years, it has become clear that EDA biofeedback arousal, i.e., training to increase sympathetic arousal in the skin, has therapeutic value. In neurological patients with drug-resistant epilepsy, electrodermal biofeedback was applied to reduce seizure frequency (Nagai, Goldstein, Fenwick, & Trimble, 2004b). Electrodermal biofeedback to increase the sympathetic arousal level was neuroscientifically motivated from the observation of an inverse relationship between the EDA level and the central cortical arousability quantified using electroencephalography. In a small randomized controlled trial, 1 month of electrodermal biofeedback training was associated with a significant decrease in seizure frequency, with 6 of 10 actively treated patients exhibiting more than a 50% seizure reduction at 3 months (and a subset reporting sustained effects at follow up over 3 years).

Conclusion

Electrodermal activity provides an accessible index of the brain's neural influence on the bodily organs, and hence a measure of the emotional capacities and psychophysiological vulnerabilities of individuals. As a route for biobehavioral intervention EDA shows promise, with potential advantages of low cost and implementability.

References and Readings

Birket-Smith, M., Hasle, N., & Jensen, H. H. (1993). Electrodermal activity in anxiety disorders. *Acta Psychiatrica Scandinavica, 88*(5), 350–355.

Critchley, H. D., Elliot, R., Mathias, C. J., & Dolan, R. J. (2000). Neural activity relating to the generation and representation of galvanic skin conductance response: A functional magnetic imaging study. *The Journal of Neuroscience, 20*, 3033–3040.

Folkins, C. H. (1970). Temporal factors and the cognitive mediators of stress reaction. *Journal of Personality and Social Psychology, 14*, 173–184.

Hare, R. D. (1978). Psychopathy and electrodermal responses to nonsignal stimulation. *Biological Psychology, 6*(4), 237–246.

Jandl, M., Steyer, J., & Kaschka, W. P. (2010). Suicide risk markers in major depressive disorder: A study of electrodermal activity and event-related potentials. *Journal of Affective Disorders, 123*(1–3), 138–149.

Leahy, A., Clayman, C., Mason, I., Lloyd, G., & Epstein, O. (1998). Computerised biofeedback games: A new method for teaching stress management and its use in irritable bowel syndrome. *Journal of the Royal College of Physicians of London, 32*, 552–556.

Luria, A. R., & Homskaya, E. D. (1970). Frontal lobes and the regulation of arousal processes. In D. I. Mostofsky (Ed.), *Attention: Contemporary theory and analysis*. New York: Appelton.

Nagai, Y., Critchley, H. D., Featherstone, E., Trimble, M. R., & Dolan, R. J. (2004). Activity in ventromedial prefrontal cortex covaries with sympathetic skin conductance level (SCL): A physiological account of a "default mode" of brain function. *NeuroImage, 22*, 243–251.

Nagai, Y., Goldstein, L. H., Fenwick, P. B. C., & Trimble, M. R. (2004). Clinical efficacy of biofeedback treatment on reducing seizures in adult epilepsy: A preliminary randomized controlled study. *Epilepsy & Behaviour, 5*, 216–223.

Raichle, M. E., MacLeod, A. M., Snyder, A. X., Powers, W. J., Gusnard, D. A., & Shulman, G. L. (2001). A default mode of brain function. *Proceedings of the National Academy of Sciences of the United States of America, 16*, 676–682.

Raine, A., Lencz, T., Bihrle, S., LaCasse, L., & Colletti, P. (2000). Reduced prefrontal gray matter volume and reduced autonomic activity in antisocial personality disorder. *Archives of General Psychiatry, 57*(2), 119–127.

Raskin, M. (1975). Decreased skin conductance response habituation in chronically anxious patients. *Biological Psychology, 2*, 309–319.

Rothbaum, B. O., Kozak, M. J., Foa, E. B., & Whitaker, D. J. (2001) Posttraumatic stress disorder in rape victims: autonomic habituation to auditory stimuli. *Journal of Traumatic Stress, 14*, 283–293.

Steptoe, A. L., & Ross, A. (1981). Psychophysiological reactivity and the prediction of cardiovascular disorder. *Journal of Psychosomatic Research, 25*, 23–31.

Thorell, L. H. (1987). Electrodermal activity in suicidal and nonsuicidal depressive patients and in matched healthy subjects. *Acta Psychiatrica Scandinavica, 76*(4), 420–430.

Venables, P. H., & Christie, M. J. (1973). Mechanisms, instrumentation, recording techniques and quantification of responses. In W. F. Prokasy & D. C. Raskin (Eds.), *Electrodermal activity in psychological research* (pp. 1–124). New York: Academic.

Williams, L. M., Brammer, M. J., Skerrett, D., Lagopolous, J., Rennie, C., Kozek, K., et al. (2000). The neural correlates of orienting: an integration of fMRI and skin conductance orienting. *NeuroReport, 11*, 3011–3015.

Williams, K. M., Iacono, W. G., & Remick, R. A. (1985). Electrodermal activity among subtypes of depression. *Biological Psychiatry, 20*(2), 158–162.

Zahn, T. P., Grafman, J., & Tranel, D. (1999). Frontal lobe lesions and electrodermal activity: Effects of significance. *Neuropsychologia, 37*, 1227–1241.

Electronic Health Record

Linda C. Baumann and Alyssa Karel
School of Nursing, University of Wisconsin-Madison, Madison, WI, USA

Synonyms

Computer-based patient record; Electronic medical record; Electronic patient record; Health information record; Personal health record

Definition

The electronic health record (EHR) is a computer-based record of patient health information. It is generated by one or more encounters in any healthcare delivery setting. The EHR includes information on patient demographics, progress notes, medications, vital signs, clinical history, immunizations, laboratory results, and reports of diagnostic procedures. The EHR documents evidenced-based decision-making, quality management, and patient outcomes.

Although the term EHR is used interchangeably with the electronic medical record (EMR), there are differences. The EMR most often refers to a single healthcare event whereas the EHR includes the entire patient record of healthcare encounters.

Description

Computer-based health records have been shown to be far superior to paper records not only because they decrease error due to handwriting and documentation issues, but also allow for the portability of and timely access to data. Other benefits include the aggregation and privatization of health data to facilitate research and promote the further education and knowledge base of clinicians.

In addition to safe, efficient, and high quality care, patients expect privacy, rights to access, and the opportunity to give consent for research uses of their health information. An EHR system must satisfy its users regarding privacy, confidentiality, and security. In the United States, the Health Insurance Portability and Accountability Act (HIPAA) ensures that these goals are met (http://www.hhs.gov/ocr/hipaa/).

There are other practical, economic, political, and professional concerns that arise regarding the implementation of computerized documentation. Individual physicians and practice groups have concerns related to cost, time to implementation, and learning curves associated with the EHR. There are also questions about whether to convert current records retrospectively or prospectively.

Research has shown that the healthcare industry will save approximately 80$ billion dollars annually by adopting electronic documentation. National mandates dictate that by 2014 all health documentation be in a computerized form. The EHR has the potential to improve patient outcomes, improve coordinated care (optimally worldwide), automate adverse event and medical error disclosure, as well as to allow for more efficient diagnosis and treatment.

References and Readings

Gunter, T., & Terry, N. (2005). The emergence of national electronic health record architectures in the United States and Australia: Models, costs, and questions. *Journal of Medical Internet Research, 7*(1), 1–15.

Lamberg, L. (2001). Confidentiality and privacy of electronic medical records. *Journal of the American Medical Association, 285*(4), 3075–3076.

Murphy, E., Ferris, F., & O'Donnell, W. (2007). An electronic medical records system for clinical research and the EMR-EDC interface. *Investigative Ophthalmology and Visual Science, 48*(10), 4383–4389.

Skolnik, N. (2011). *Electronic medical records: A practical guide for primary care*. New York: Springer.

Electronic Medical Record

▶ Electronic Health Record

Electronic Patient Record

▶ Electronic Health Record

Elevated Blood Pressure

▶ Blood Pressure, Measurement of

Embryo Donation

▶ In Vitro Fertilization, Assisted Reproductive Technology

Emotion

▶ Affect
▶ Anger Management

Emotion Modulation

▶ Emotional Control

Emotion Regulation

▶ Anger Management
▶ Emotional Control

Emotional Control

Michelle Skinner
Department of Psychology, University of Utah, Salt Lake City, UT, USA

Synonyms

Emotion modulation; Emotion regulation

Definition

Emotional control can be thought of as a facet of emotion regulation, but refers primarily to attempts by an individual to manage the generation, experience, or expression of emotion, and/or one's emotional responses (Gross, 1999). Emotional control, like emotional expression, is tied the broader context of emotion regulation. Emotional control can occur as part of antecedent-focused regulation prior to generation of emotion or through response-focused regulation after an emotion has been generated (Gross, 1998a). Emotional control can refer to the ability to exercise influence over emotion, and modulate emotion through the use of cognitive or behavioral strategies (Gross, 1998b; Lazarus & Folkman, 1984). The ways in which individuals are able to achieve emotional control have implications for health and well-being (Beck, 1995; Berg, Skinner, & Ko, 2009).

Description

Emotional control has varied definitions in literature on stress and coping and emotion regulation. Emotional control includes efforts by the individual to alter the generation of emotion, emotional experience, and emotional expression. Strategies aimed at emotional control can impact health in both positive and negative ways and are contextually dependent on situation, individual differences in personality and social context, and well as demographic factors such as ethnicity, culture, gender, and age (Berg, Skinner, & Ko, 2009).

Emotional control is an important facet of emotion regulation and can be facilitated by types of emotion regulation. Antecedent-focused regulation can have an influence on emotional control (Gross, 1999). Antecedent-focused regulation refers to altering and regulating aspects of a situation and emotional experience prior to generation of emotion (Gross, 1998a). There are several ways that an individual may use antecedent-focused regulation for emotional control. Essential parts of antecedent-focused regulation are situation selection, selective attention, and cognitive appraisal. Situation selection is defined as deciding where to go, what to be exposed to, or who to be exposed to as a means of controlling emotion (Gross, 1998a). Selective attention is defined as choosing aspects of a situation to minimize emotional impact such as distraction or attending to less emotionally salient features of the situation (Gross, 1998a; Strecher & Rosenstock, 1997). Cognitive appraisal is defined as changing the meaning of situations so as to mitigate emotional impact such as looking at positive aspects or minimizing importance (Gross, 1999, 2007; Strecher & Rosenstock, 1997).

Use of antecedent-focused regulation strategies as a means of emotional control can lead to health outcomes, both positive and negative. For example, situation selection can reduce the likelihood that someone might encounter negative emotional experiences. However, selecting to avoid activities can potentially lead to decline in physical, emotional, and social functioning that may be associated with morbidity and mortality (Gross, 2007). Shifting attention, such as use of distraction, may be adaptive in the short-term (e.g., pain management) (Berg et al., 2009; Gross, 1998a). Prolonged use of distraction may not allow individuals to address aspects of problems that they can control or may prevent

accurate detection of symptoms (Gross, 1998a, 2007). Cognitive appraisal can prevent misinterpretations of situations known to impact emotional and physical health such as catastrophizing, emotional reasoning, or black and white thinking (Beck, 1995). However, if one appraises problems as nonthreatening then appraisals may not translate to appropriate emotional reaction or "over control" when it may be appropriate to react leading to worse outcomes (Lazarus & Folkman, 1984; Strecher & Rosenstock, 1997).

In contrast to antecedent-focused regulation, response-focused regulation occurs after emotion has been generated and includes direct attempts to alter experiential, physiological, and behavioral responses to the experience of emotion after the emotion has occurred (Gross, 1998a, 1999, 2002). Response-focused regulation is also a means of emotional control. Emotion can be controlled by situation modification. Situation modification is defined as changing aspects of a situation to reduce emotional impact. Situation modification relies on coping strategies such as generation of multiple solutions and problem solving (Gross, 1998a). Once an emotion has been generated, individuals may choose to actively solve the problem which can allow for emotional arousal to subside. Use of problem-solving skills may provide health benefit when problems are well-defined and controllable (Berg et al., 2009). However, when problems are ill-defined, ambiguous, and perceived as uncontrollable, use of problem solving may prolong stress, reactivity, and negative emotion. Thus, the way an individual chooses to control emotions can depend on the context of the problem.

Emotional control is an important facet of emotion regulation and can occur through efforts to minimize negative emotional experience prior to emotion generation (e.g., antecedent-focused regulation) or following an event through the use of coping strategies (e.g., response-focused regulation). Such skills are taught to individuals engaging in cognitive behavioral therapy for management of illness, psychopathology, and everyday problems (Beck, 1995). Cognitive and behavioral strategies can help individuals have greater control over emotional arousal that produces ill health effects (Gross, 1999). Emotional control can be achieved through provision of skills related to problem solving (Gross, 1998a, 1999). These include behavioral skills to facilitate emotional control such as stress management skills (e.g., deep breathing, progressive muscle relaxation), exercise, and/or, engaging in regular healthy behaviors such as sleep hygiene and diet. Similarly, using cognitive appraisal skills to alter ongoing emotional experience may be helpful. Use of antecedent- and response-focused regulation for emotional control can help downregulate negative emotion and reduce physiological reactivity which may confer health benefits (Gross, 1999, 2002). Inability to effectively control emotion can have detrimental effects on health and well-being. Difficulty in controlling emotional reactions has been linked to psychopathology such as personality disorders, anxiety disorders, as well as risky behavior all of which have adverse association with health (e.g., poor social support, prolonged interpersonal stress, substance abuse, risk-taking behaviors) and may have a neural basis (Gross, 2007; Strecher & Rosenstock, 1997).

Cross-References

- ▶ Cognitive Appraisal
- ▶ Cognitive Behavioral Therapy (CBT)
- ▶ Emotional Expression
- ▶ Physiological Reactivity
- ▶ Problem-Focused Coping
- ▶ Stress
- ▶ Stress Management

References and Readings

Beck, J. S. (1995). *Cognitive therapy: Basics and beyond*. New York: Guilford Press.

Berg, C. A., Skinner, M. A., & Ko, K. K. (2009). An integrative model of everyday problem solving across the adult life span. In M. C. Smith (Ed.), *Handbook of research on adult learning and development* (pp. 524–552). Mahwah, NJ: Erlbaum.

Gross, J. J. (1998a). Antecedent- and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology, 74*, 224–237.

Gross, J. J. (1998b). The emerging field of emotion regulation: An integrative review. *Review of General Psychology, Special Issue: New directions in research on emotion, 2*(3), 271–299.
Gross, J. J. (1999). Emotion regulation: Past, present, and future. *Cognition & Emotion, 13*(5), 551–573.
Gross, J. J. (2002). Emotion regulation: Affective, cognitive, and social consequences. *Psychophysiology, 39*, 281–291.
Gross, J. J. (Ed.). (2007). *Handbook of emotion regulation*. New York: Guilford Press.
Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.
Oshsner, K. N., & Gross, J. J. (2005). The cognitive control of emotion. *Trends in Cognitive Sciences, 9*(5), 242–249.
Strecher, V. J., & Rosenstock, I. M. (1997). The health belief model. In K. Glanz, F. M. Lewis, & B. K. Rimer (Eds.), *Health behavior and health education: Theory, research, and practice*. San Francisco: Jossey-Bass.

Emotional Disclosure

▶ Disclosure

Emotional Disorder

▶ Psychological Disorder

Emotional Distress

▶ Negative Affect
▶ Stress, Emotional

Emotional Expression

Michelle Skinner
Department of Psychology, University of Utah, Salt Lake City, UT, USA

Definition

Emotional expression refers to how one conveys emotional experience through both verbal and nonverbal behavior (Gross, 1998b, 1999). Emotional expression should be distinguished from emotional experience in that it is possible to experience emotions without expressing them. Emotional expression is an important part of emotion regulation and can affect health outcomes. Emotional expression is embedded in the broader domain of emotion regulation, which is defined as how individuals, either consciously or unconsciously, influence, experience, and express emotions (Gross, 1999). Emotion regulation unfolds over time in a given situation either before emotional experience, during emotional experience, or in response to emotional experience (Gross, 1998a). Emotion regulation involves coordination of several systems including how one thinks about emotion, physiological reactivity elicited by emotion, and behavioral responses such as emotional expression and utilizing coping strategies that either promote health or contribute to poorer health. Emotional expression can be adaptive or maladaptive and may be dependent on context (Gross, 1998a, 2002).

Description

Emotional expression is part of the emotion regulation process and functions as a way to communicate internal states to others. Emotional expression can include behavioral, nonverbal, and/or verbal expressions (Gross, 1998a). Emotional expression can be beneficial when adaptive and fit to a given situation. For example, suppression of emotion may be inappropriate in some instances such as displays of anger or sadness while at work. However, prolonged suppression of emotion can result in poorer health. Links between maladaptive emotional expression and prolonged suppression have been made to cardiovascular disease. Similarly, "venting" negative emotion may perpetuate negative emotion via physiological and social responses to venting. Thus, the popular idea that "letting it out" may be beneficial for well-being or health may be inaccurate (Gottman, 2000; Gross, 2002; Hatfield, Cacioppo, & Rapson, 1994). Expression of positive emotions may also help to in buffering

negative emotional experience and has been shown to impact the affiliative quality of marital relationships (Gottman, 2000). It is important to acknowledge that emotional expression involves many components of the emotion regulation process and that health effects can be dependent on contextual factors of the situation and individual difference characteristics such as age, ethnicity, and gender (Gross, 1999).

Emotional expression as a means of emotion regulation has its roots in the stress and coping paradigm originally put forth by Lazarus and Folkman (Gross, 1999; Lazarus, 1991). The stress and coping paradigm asserts that emotional expression can act as a coping strategy and thus may impact health and well-being. Lazarus and Folkman made a distinction between emotion-focused coping, defined as changing the internal state to meet the demands of the stressor (e.g., altering emotions associated with stressor, reappraisals of stressor) and problem-focused coping, defined as changing the environment to meet the demands of the stressor such as finding a problem solution (Lazarus). Emotion-focused strategies (e.g., controlling emotional expression, changing the way one thinks about a stressor, acceptance) were thought to be associated with poorer health outcomes (Lazarus). However, recent models of emotion regulation have recognized nuances of regulatory process as emotion regulation unfolds at points over time in a given situation.

Points of regulation specifically linked to emotional expression are defined as antecedent-focused regulation or as response-focused regulation (Gross, 1998a). Antecedent-focused regulation refers to altering and regulating aspects of a situation and emotional experience prior to generation of emotion (Gross, 1998a). In contrast, response-focused regulation occurs after emotion has been generated and includes direct attempts to alter experiential, physiological, and behavioral responses to the experience of emotion after the emotion has occurred (Gross, 1998a, 1999, 2002). Emotional expression can be altered through both antecedent-focused coping and response-focused coping.

The clearest link between emotional expression and health outcomes occur as a function of response-focused coping and how one chooses to express emotion after an emotion has been generated. Individuals may choose to express emotions in a productive way (conveying how they feel or felt through communication with others, journaling), in an aggressive manner (punching, kicking, self-mutilation), or suppress emotions all together (Gross, 2002; Kennedy-Moore & Watson, 1999). Emotional expression can include behavioral expressions of emotion such as engaging in risky health behaviors (e.g., substance use, overeating). Risky health behaviors may be useful for altering emotion and physiological reactivity in the short term but can damage health over time. The ability to use adaptive coping strategies can attenuate physiological reactivity (Gross, 1998b, 1999, 2007) and can confer health benefits. For example, adaptive coping mechanisms might reduce cardiovascular risk, promote feelings of control, and self-efficacy which are important in choosing positive health behaviors. Adaptive coping responses can protect against prolonged negative mood states associated with metabolic dysregulation, poor immune response, inflammatory processes, and insomnia (Gross, 2007). Conversely, maladaptive coping strategies such as substance abuse and risky behaviors can be associated with physical injury, poorer health status, morbidity, and mortality (Gross, 2007; Kennedy-Moore & Watson, 1999).

The effect of emotional expression on health may be contextually bound. Emotional suppression can decrease emotionally expressive behavior but simultaneously may not impact physiological responding. In certain instances, suppression of emotion may be effective as a coping mechanism but long-term suppression can negatively impact life satisfaction and depression. Emotional suppression can dampen emotional expression in the context of social interactions resulting in less positive social support which may lead to poorer health. Expression of emotion may also convey health benefits. For example, expression of emotion through writing

has been associated with better adjustment in cancer patients, especially in patients that may prefer to avoid or deny managing cancer-related problems. Emotional expression in the context of support groups for health problems may help patients to tolerate and find benefit through others' emotional expression as they react to an illness (Kennedy-Moore & Watson, 1999). In close relationships, being able to express emotions in a less negative way may reduce the negative emotional arousal that can affect members of a couple (Gottman, 2000; Hatfield, Cacioppo, & Rapson, 1994). It should also be noted that the expression of positive emotion can also contribute greater satisfaction in relationships and can be associated with more affiliation and less hostility as couples interact (Gottman, 2000).

Cross-References

- ▶ Comorbidity
- ▶ Coping
- ▶ Emotional Responses
- ▶ Emotions: Positive and Negative
- ▶ Expressive Writing and Health
- ▶ Mortality
- ▶ Physiological Reactivity
- ▶ Problem-Focused Coping
- ▶ Stress

References and Readings

Baumeister, R. F., & Vohs, K. D. (Eds.). (2004). *Handbook of self-regulation: Research, theory, and applications*. New York: Guilford Press.

Gottman, J. M. (2000). *The seven principles of making marriage work*. New York: Three Rivers Press.

Gross, J. J. (1998a). Antecedent- and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology, 74*, 224–237.

Gross, J. J. (1998b). The emerging field of emotion regulation: An integrative review. *Review of General Psychology, Special Issue: New directions in research on emotion, 2*(3), 271–299.

Gross, J. J. (1999). Emotion regulation: Past, present, and future. *Cognition & Emotion, 13*(5), 551–573.

Gross, J. J. (2002). Emotion regulation: Affective, cognitive, and social consequences. *Psychophysiology, 39*, 281–291.

Gross, J. J. (Ed.). (2007). *Handbook of emotion regulation*. New York: Guilford Press.

Hatfield, E., Cacioppo, J. T., & Rapson, R. L. (1994). *Emotional contagion*. New York: Cambridge University Press.

Kennedy-Moore, E., & Watson, J. C. (1999). *Expressing emotion: Myths, realities, and therapeutic strategies*. New York: Guilford Press.

Lazarus, R. S. (1991). *Emotion and adaptation*. Oxford, UK: Oxford University Press.

Emotional Reactions

▶ Emotional Responses

Emotional Responses

Pamela S. King
Pediatric Prevention Research Center, Department of Pediatrics, Wayne State University School of Medicine, Detroit, MI, USA

Synonyms

Affective responses; Emotional reactions

Definition

Emotions are defined as multicomponent response tendencies that unfold over a relatively short span of time. Emotions occur in response to a stimulus or event. The emotional response consists of an appraisal process, during which individuals determine the personal significance of the stimulus or event (e.g., is it harmful or beneficial, does it affect personal goals). The emotional response also includes the subjective experience of emotion, cognitive processing, and physiological changes (e.g., activation of the amygdala and hypothalamus, and subsequent release of

epinephrine, norepinephrine, dopamine, and cortisol). Emotions are believed to have evolved to promote behaviors necessary to survive and thrive. Researchers often conceptualize emotions as varying along two dimensions: (1) valence (i.e., negative to positive); and (2) activation (aroused to unaroused). Discrete emotion theorists, in contrast, consider each emotion as a distinct entity. Researchers who examine emotional responses to a stimulus or event (e.g., emotional responses to stress) commonly measure multiple aspects of the multicomponent response. For example, when examining emotional responses to a laboratory stressor, researchers may measure cognitive appraisal components (appraisals of threat or harm), affective components (e.g., reports of anxiety and fear), and physiological components (e.g., rising cortisol levels).

Sometimes, the terms emotion, affect, and mood are used interchangeably. Many researchers, however, make distinctions among these terms. Whereas emotions are short-lived responses to stimuli, moods are relatively long-lasting emotional states, and are not always linked to a stimulus or event. Affect is a term used to describe the conscious, subjective aspect of an emotion, separable from any physiological response.

Cross-References

- ▶ Affect
- ▶ Affect Arousal
- ▶ Anger
- ▶ Anger Expression
- ▶ Emotional Expression
- ▶ Emotions: Positive and Negative
- ▶ Mood
- ▶ Negative Affect

References and Readings

Diener, E., & Emmons, R. A. (1984). The independence of positive and negative affect. *Journal of Personality and Social Psychology, 47*, 1105–1117.

James, G. J. (1998). The emerging field of emotion regulation: An integrative review. *Review of General Psychology, 2*(3), 271–299.

Lewis, M., Haviland-Jones, J. M., & Barrett, L. F. (2008). *Handbook of emotions* (3rd ed.). New York: The Guilford Press.

Russell, J. A., & Carroll, J. M. (1999). On the bipolarity of positive and negative affect. *Psychological Bulletin, 125*(1), 3–30.

Emotions: Positive and Negative

Pamela S. King
Pediatric Prevention Research Center, Department of Pediatrics, Wayne State University School of Medicine, Detroit, MI, USA

Synonyms

Affect; Mood; Positive and negative affect

Definition

Emotions are defined as multicomponent response tendencies that unfold over a short span of time, and include cognitive processing, physiological responses, and the subjective experience of emotion (i.e., affect). Emotions are often conceptualized as varying in valence, from positive (e.g., happiness, excitement, contentment, curiosity) to negative (e.g., sadness, anger, anxiety, disgust). Subjectively, people experience positive emotions as feelings that reflect a level of pleasurable engagement with the environment. Negative emotions, in contrast, reflect a general feeling of distress. Emotions are thought to have evolved to promote the behaviors necessary to survive and thrive. Positive emotions facilitate approach behavior or continued action; experiences of positive affect prompt people to engage with their environments and partake in activities which are adaptive. Negative emotions, on the other hand, prompt withdrawal behavior and signal when a particular behavior or course of action may not be adaptive.

Description

There is some debate in the emotion literature about whether positive emotions and negative emotions are bipolar extremes of the same factor, or whether they are orthogonal or independent factors. Evidence suggests that positive and negative emotions may be managed by different structures in the nervous system (i.e., positive emotion activation in left frontal cortex; negative emotion activation in right frontal cortex), and that neurotransmitters may respond differently to negative versus positive emotions, which supports the notion that negative and positive emotions are orthogonal factors. In addition, in many studies, researchers have noted the unique contribution of negative and positive emotions (i.e., each contributed to outcomes after controlling for the other), and the low correlation between negative and positive emotion. Some studies, however, have observed moderate to strong (negative) correlations between positive and negative emotion (particularly when "state" vs. "trait" emotions are measured), and many studies do not find unique effects of negative and positive emotion. Research on the distinction between negative and positive emotions is ongoing; thus, while most consider positive and negative emotions to be orthogonal factors, some researchers consider positive and negative emotions to be opposite ends of the same scale.

There are several measures currently used to assess positive and negative emotions. Among them are two commonly used self-report measures that ask people to rate their experience of positive and negative emotions. Because these measures are assessing the subjective experience of emotion, they are more appropriately labeled measures of affect. The PANAS (Positive and Negative Affect Schedule) includes a 10-item Positive Affect (PA) scale and a 10-item Negative Affect (NA) scale. High scores on the PA scale reflect high energy and concentration (e.g., attentive, interested, alert), whereas high NA reflects a state of general distress (e.g., guilty, hostile, irritable). The POMS (Profile of Mood States) is another measure commonly used to assess positive and negative affect. The POMS has one PA scale reflecting "vigor" (e.g., alert, energetic, cheerful, active, lively), and four NA subscales measuring depression, anger, fatigue, and tension-confusion. For both the PANAS and the POMS, instructions can be modified to ask for current state (which is most likely to reflect "emotion"), as well as mood in the last day, general mood, or mood over the last few weeks or longer (note that mood is longer lasting compared to emotions, which are short-lived). Researchers often distinguish "state" PA and NA (current emotion, or mood in the last day) from "trait" PA (general mood or mood over the last few weeks or months). In addition to measuring the subjective experience of emotion, researchers have also used mood induction procedures to generate positive and negative emotions in the laboratory and observe their effects on outcomes. In general, research (largely correlational) suggests that positive emotions are beneficial for health, and that negative emotions are detrimental for health. Researchers urge caution in interpreting these findings, however, as most studies linking negative emotion and health do not control for the effect of positive emotions, and most studies linking positive emotion and health do not control for the effect of negative emotion.

There is some debate in the literature about the value of distinguishing individual positive and negative emotions (e.g., happiness, excitement, sadness, anger) versus aggregating positive emotions and aggregating negative emotions. Some research suggests that people are not sensitive to individual emotions and experience similar responses across emotions within a valence (i.e., positive or negative). However, there is also research to suggest that different emotions within a valence have different associations with outcomes (e.g., distinct positive and negative emotions are associated with distinct immune responses). Most researchers currently examine aggregated emotions, distinguishing only positive from negative, but increasingly research suggests that there may be value in distinguishing among emotions within a valence.

Cross-References

- ▶ Affect
- ▶ Anger
- ▶ Emotional Expression
- ▶ Happiness and Health
- ▶ Mood
- ▶ Negative Affect
- ▶ Negative Affectivity
- ▶ Positive Affect Negative Affect Scale (PANAS)
- ▶ Positive Affectivity

References and Readings

Diener, E., & Emmons, R. A. (1984). The independence of positive and negative affect. *Journal of Personality and Social Psychology, 47*, 1105–1117.

Fredrickson, B. L. (2001). The role of positive emotions in positive psychology: The broaden-and-build theory of positive emotions. *American Psychologist, 56*(3), 218–226.

Krantz, D. S., & McCeney, M. K. (2002). Effects of psychological and social factors on organic disease: A critical assessment of research on coronary heart disease. *Annual Review of Psychology, 53*, 341–369.

Lewis, M., Haviland-Jones, J. M., & Barrett, L. F. (2008). *Handbook of emotions* (3rd ed.). New York: The Guilford Press.

Pressman, S. D., & Cohen, S. (2005). Does positive affect influence health? *Psychological Bulletin, 131*(6), 925–971.

Russell, J. A., & Carroll, J. M. (1999). On the bipolarity of positive and negative affect. *Psychological Bulletin, 125*(1), 3–30.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*, 1063–1070.

Empathy

Linda C. Baumann and Alyssa Karel
School of Nursing, University of Wisconsin-Madison, Madison, WI, USA

Definition

Empathy is the ability of a person to perceive, understand, and accept the experiences of another. It is having the capacity to identify with another's feelings without actually experiencing the situation. In a healthcare setting it is often therapeutic for clients going through difficult situations to have healthcare professionals that can be empathetic to their situations.

Empathy is different than sympathy, which is concern or pity for another person generated by a subjective perspective. Oftentimes this subjective perspective is a barrier to problem solving. The most therapeutic approach to clinical situations is often an objective empathetic approach.

References and Readings

Lamm, C., Batson, C. D., & Decety, J. (2007). The neural basis of human empathy: Effects of perspective-taking and cognitive appraisal. *Journal of Cognitive Neuroscience, 19*, 42–58.

Lewis, S. L., Heitkemper, M. M., Dirksen, S. R., O'Brien, P. G., & Bucher, L. (2007). *Medical surgical nursing: Assessment and management of clinical problems* (7th ed.). St. Louis, MO: Mosby Elsevier.

Potter, P. A., & Perry, A. G. (2009). *Fundamentals of nursing* (7th ed.). St. Louis, MO: Mosby Elsevier.

Emphysema

Siqin Ye
Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Synonyms

Chronic bronchitis; Chronic obstructive pulmonary disease

Definition

Emphysema is defined as the pathological enlargement and destruction of lung alveoli. Along with chronic bronchitis, which describes the clinical manifestation of chronic cough with

sputum production, these two terms have traditionally been used to refer to the two phenotypes of chronic obstructive pulmonary disease (COPD). Recently, however, the Global Initiative for Chronic Obstructive Lung Disease (GOLD) has advocated defining COPD based on airflow limitation that is not fully reversible, is progressive, and is associated with an abnormal inflammatory response of the lung to noxious particles or gases. As patients with COPD usually have overlapping features of both emphysema and chronic bronchitis, the distinction is rarely of clinical significance.

Description

COPD remains one of the most common causes of morbidity and mortality globally. In the United States in 2000, it accounted for eight million outpatient visits, 1.5 million emergency room visit, and 673,000 hospitalizations. COPD is currently the fourth leading cause of death in the USA and is projected to become the third most common cause of death worldwide in 2020 (Global Initiative for Chronic Obstructive Lung Disease, 2008). One reason for this rise is the strong, dose-dependent relationship between cigarette smoking/exposure and the prevalence of COPD, although other genetic and environmental factors also play important roles, since many smokers never develop clinically significant disease. It has been known, for instance, that genetic defects causing severe deficiency of the protease inhibitor α1-antitrypsin lead to a form of early-onset COPD, especially in those who are also smokers. Other known risk factors include exposure to occupational dust and chemicals, indoor and outdoor air particle pollutants, as well as low birth weight. On a cellular level, it has been demonstrated that inhaled cigarette smoke and other noxious particles promote inflammation through the recruitment of neutrophils, macrophages, lymphocytes, and eosinophils. This in turn activates proteinases that degrade lung parenchyma and cause mucus hypersecretion, leading to impaired gas exchange, fibrosis of small airways, expiratory flow obstruction, and hyperinflation (Barnes, Shapiro, & Pauwels, 2003; Eisner et al., 2010).

Patients with COPD typically present with cough, sputum production, and exertional dyspnea. The hallmark of the disease, expiratory airflow obstruction, may be present for years before medical attention is sought (Hogg, 2004). While early on the physical examination may be normal, most patients will demonstrate diminished air movement with a prolonged expiratory phase and wheeze on exam. Pulmonary function testing with spirometry is used to characterize the degree of airflow obstruction and provide prognostic information. When airflow obstruction becomes severe, cyanosis may develop as a manifestation of hypoxemia, and the patient may adopt pursed-lip breathing and the classic "tripod" position to recruit accessory muscles and improve expiratory flow. Another marker of poor prognosis is the development of pulmonary hypertension from chronic hypoxemia, which can result in right heart failure or cor pulmonale. Death from respiratory failure is unfortunately a common outcome for patients with end-stage COPD (Reilly, Silverman, & Shapiro, 2006).

Many patients with COPD will also experience episodic exacerbations, characterized by increased shortness of breath and changes in pattern and quantity of sputum. These are often triggered by viral or bacterial infections and, depending on severity, may require hospitalization for treatment. For exacerbations, inhaled β-agonists and anticholinergic agents, antibiotics, glucocorticoids, and supplement oxygen for hypoxemia are the mainstay of pharmacological therapy. Noninvasive positive pressure ventilation and conventional mechanical ventilation can be used to stabilize patients in severe respiratory distress. For treatment of stable COPD, only smoking cessation and oxygen therapy in those with chronic hypoxemia have been shown to improve survival. Inhaled β-agonists and anticholinergic agents can provide symptomatic benefit, while inhaled glucocorticoids can be used to reduce exacerbations. Pulmonary rehabilitation has also been shown to improve quality of life and exercise capacity as well as reduce

hospitalizations. Finally, in select patients with severe COPD but limited comorbidities, lung transplantation can be pursued and provides significant symptomatic and survival benefit (American Thoracic Society, 2004).

Cross-References

► Chronic Obstructive Pulmonary Disease

References and Readings

American Thoracic Society/European Respiratory Society Task Force. (2004). *Standards for the diagnosis and management of patients with COPD* (Internet). Version 1.2. New York: American Thoracic Society, (Updated September 8, 2005). Available from http://www.thoracic.org/go/copd

Barnes, P. J., Shapiro, S. D., & Pauwels, R. A. (2003). Chronic obstructive pulmonary disease: Molecular and cellular mechanisms. *European Respiratory Journal, 22*(4), 672–688.

Eisner, M. D., Anthonisen, N., Coultas, D., Kuenzli, N., Perez-Padilla, R., Postma, D., Romieu, I., Silverman, E. K., Balmes, J. R., & On behalf of the Environmental and Occupational Health Assembly Committee on Nonsmoking COPD. (2010). An official American thoracic society public policy statement: Novel risk factors and the global burden of chronic obstructive pulmonary disease. *American Journal of Respiratory and Critical Care Medicine, 182*, 693–718.

Global Initiative for Chronic Obstructive Lung Disease (GOLD). (2008). *Global strategy for the diagnosis, management and prevention of COPD*. Available from http://www.goldcopd.org

Hogg, J. C. (2004). Pathophysiology of airflow limitation on chronic obstructive pulmonary disease. *The Lancet, 364*(9435), 709–721.

Reilly, J. L., Silverman, E. K., & Shapiro, S. D. (2006). Chronic obstructive pulmonary disease. In D. L. Kasper, E. Braunwald, A. S. Fauci, S. L. Hauser, D. L. Longo, & J. L. Jameson (Eds.), *Harrison's principles of internal medicine* (16th ed., pp. 1547–1554). New York: McGraw-Hill.

Employee Appraisal

► Job Performance

Employee Assistance Programs (EAP)

Karen Jacobs[1], Miranda Hellman[2], Jacqueline Markowitz[2] and Ellen Wuest[2]
[1]Occupational Therapy, College of Health and Rehabilitation Science Sargent College, Boston University, Boston, MA, USA
[2]Boston University, Boston, MA, USA

Synonyms

Employer-sponsored assistance programs

Definition

Employee Assistance Programs (EAPs) are programs offered to employees; they include policies and procedures for identifying or responding to employee difficulties that may or may not interfere with job performance (Walsh, 1982). These programs often provide counseling or treatment to those who require these services, and can also be provided to the employee's family members. EAPs are aimed to be preventative services, and these services can address psychological issues, alcohol, and drug abuse (Muto, Fujimori, & Suzuki, 2004). Other areas include, but are not limited to, health, marital, family, financial, legal, or stress issues that may influence job performance (EAPA, 2010). EAPs are beneficial in helping employees balance demands while meeting employer's goals of workplace productivity (Jacobson, 2010).

Description

Employee benefit assistance programs typically include programs that address a variety of personal and workplace issues that impact job performance such as stress management, weight reduction, workplace violence, and financial management.

EAPs were modeled after Alcoholics Anonymous (AA) programs, and both AA and EAPs understand the importance of acknowledging the problem as the initial step of treatment (Walsh, 1982). Since alcoholism has negative impacts on job performance, alcoholism was the first problem addressed by EAPs, followed by substance abuse. It was later recognized that many substance abuse problems have roots in psychosocial problems, which further expanded the outreach of EAPs. EAPs use the importance of retaining the job as a motivating factor to have employees seek help. That is, the services can help fix a problem or difficulty that could threaten employment.

The services offered by EAPs have many positive impacts on the employees receiving them, as well as the organization providing them. Reported benefits include a reduction in expenses associated with medical claims, accident benefits, mental health care costs, absenteeism, lost wages, medical costs, and employee turnover (Hargrave, Hiatt, Alexander, & Shaffer, 2008).

EAPs have a variety of components, including written policies and procedures, labor and management cooperation in program development, referral systems, program information conveyed to the work force, health insurance covering the treatment, and total confidentiality (Walsh, 1982). These factors are integral in the success of the EAP (Richard, Emener, & Hutchison, 2009). The policy statement, a statement in which the institution states what the philosophy and the intent of the program is, keeps the EAP on target of the goals. This statement should make it clear that human problems can interfere with work performance, but are inevitable; assistance is available to aid these problems, and the employee will not be terminated on the basis of that problem. The services should be administered through the EAP confidentially and professionally, which helps the employees feel secure in their recovery. The EAPs should be accessible, and employees should be able to receive the services in a timely and efficient manner (Richard et al., 2009).

There are various methods of delivery of services depending on the corporation and EAP model. In "internal" EAPs, the professionals delivering the services are employed with the company offering the EAP, whereas "external" EAPs hire service professionals who are outside contractors from the company. "External" methods are most commonly found. However, a third method, "combination" or "hybrid" EAP, is a delivery system in which the professionals began as internal employees, and then expand services to other workplaces. The "hybrid" EAP is a way in which smaller companies can share the cost of an EAP. Additionally, services can be accessed in a variety of ways, including in-person, via phone, or via the Internet (Jacobson, 2010).

EAPs have become popular within businesses. Within USA state and local government in 2008, more than 75% of employees have access to EAPs. About 39% of employees working within the public sector, part-time, had access to EAPs, and 54% of full-time workers in the public sector had access. Of those working in the private sector, 15% of part-time workers and 28% of full-time workers had access to EAPs (U.S. Department of Labor, Bureau of Labor Statistics, 2008).

Cross-References

- ▶ Diabetes Education
- ▶ Education, Patient
- ▶ Exercise-General Category
- ▶ Exercise Testing
- ▶ Exercise, Benefits of
- ▶ Health Promotion and Disease Prevention
- ▶ Lifestyle, Healthy
- ▶ Lifestyle, Modification
- ▶ Smoking Cessation
- ▶ Substance Abuse: Treatment

References and Readings

Employee Assistance Professionals Association (EAPA). (2010). *What is an employee assistance program (EAP)*? Arlington, VA: Employee Assistance Professionals Association (EAPA). Retrieved May 1, 2010, from http://www.eapassn.org/i4a/pages/index.cfm?pageid=869

Hargrave, G. E., Hiatt, D., Alexander, R., & Shaffer, I. A. (2008). EAP treatment impact on presenteeism and absenteeism: Implications for return on investment.

Journal of Workplace Behavioral Health, 23, 283–295. doi:10.1080/15555240802242999.
Jacobson, J. (2010). Employee assistance programs (EAPs): An allied profession for work/life. Retrieved February 22, 2011, from http://wfnetwork.bc.edu/encyclopedia_entry.php?id=17296&area=All
Muto, T., Fujimori, Y., & Suzuki, K. (2004). Characteristics of an external employee assistance program in Japan. *Occupational Medicine, 54*, 570–575. doi:10.1093/occmed/kqh124.
Richard, M. A., Emener, W. G., & Hutchison, W. S. (Eds.). (2009). *Employee assistance programs* (4th ed.). Springfield: Charles C Thomas.
U.S. Department of Labor, Bureau of Labor Statistics. (2008). *National compensation survey: Employee benefits in the United States*. Washington, DC: U.S. Government Printing Office. Retrieved February 22, 2011, from http://www.bls.gov/opub/cwc/cm20090416ar01p1.htm
Walsh, D. C. (1982). Employee assistance programs. *The Milbank Memorial Fund Quarterly. Health and Society, 60*, 492–517.

Employer-Sponsored Assistance Programs

▶ Employee Assistance Programs (EAP)

Employment

▶ Job Classification

Employment Status

▶ Occupational Status

Empowerment

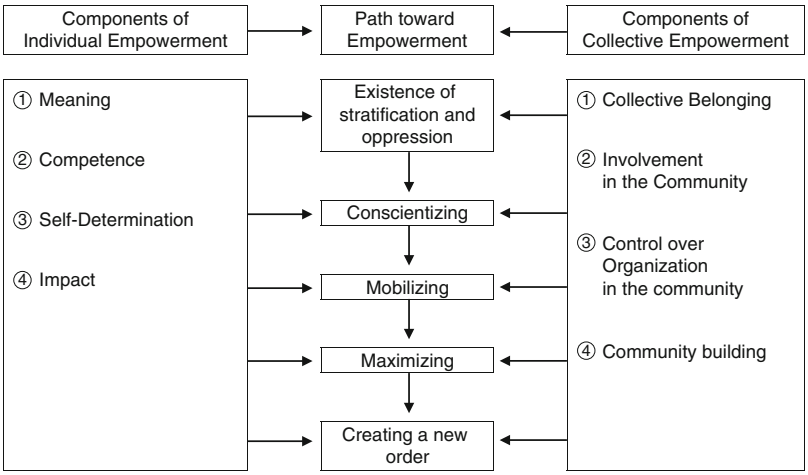
Mann Hyung Hur
Public Administration, Chung-Ang University, Seoul, Korea

Definition

The origin of empowerment as a form of theory is traced back to the Brazilian educator Freire (1971), when he suggested a plan for liberating the oppressed people through education. Empowerment is a form of power that helps people gain control over their own lives. It is described as a social process that fosters power in people, their communities, and in their society (Page & Czuba, 1999).

The process of empowerment can be synthesized into five progressive stages: an existing social disturbance, conscientizing, mobilizing, maximizing, and creating a new order, as seen in Fig. 1. Empowerment has two interrelated forms such as individual empowerment and collective empowerment. Each form has its own components. A set of four components, including meaning, competence, self-determination, and impact, are found in individual empowerment. A set of four components, including collective belonging, involvement in the community, control over organization in the community, and community building, are explored in collective empowerment. The goal of individual empowerment is to achieve a state of liberation strong enough to impact one's power in life, community, and society. The goal of collective empowerment is to establish community building so that members of a given community can feel a sense of freedom, belonging, and power that can lead to constructive social change (Hur, 2006).

The term, empowerment, has become a widely used word in the social sciences in the last decade across a broad variety of disciplines, such as community psychology, political theory, social work, education, women studies, and sociology. Community psychology is one of the disciplines in which the word *empowerment* is most frequently used. The typological approach to the study of empowerment is useful for field workers, social workers, community psychologists, and educators who help the disadvantaged (Hur, 2006). These people, including the disadvantaged, the aged, and the young, can actualize the latent powers that an individual or group possesses, or enable them and use their capacities and power more effectively (Weil & Kruzich, 1990). The process and components can be guidelines for practitioners who hope to develop the latent power of the "have-nots," actualize



Empowerment, Fig. 1 Paths toward and components of empowerment

their upward mobility, and finally establish a value of justice in a given society.

Cross-References

- ▶ Behavior Change
- ▶ Behavioral Intervention
- ▶ Health Behavior Change
- ▶ Health Education
- ▶ Health Promotion and Disease Prevention
- ▶ Intervention Theories
- ▶ Lifestyle Changes
- ▶ Protective Factors
- ▶ Social Capital and Health

References and Readings

Freire, P. (1971). *Pedagogy of the oppressed*. New York: Seabury Press.
Hur, M. H. (2006). Empowerment in terms of theoretical perspectives: Exploring a typology of the process and components across disciplines. *Journal of Community Psychology, 34*(5), 523–540.
Page, N., & Czuba, C. E. (1999). Empowerment: What is it? *Journal of Extension, 37*(5), 24–32.
Weil, M., & Kruzich, J. (1990). Introduction to the special issue. *Administration in Social Work, 14*(2), 1–12.

Endocrinologist

- ▶ Diabetologist (Diabetes Specialist)

Endocrinology

Janine Sanchez
Department of Pediatrics, University of Miami, Miami, FL, USA

Synonyms

Hormone system

Definition

Endocrinology is the study of the endocrine system and its diseases. The endocrine system includes hormones (chemical mediators) and the organs/cells which secrete them. Endocrinology includes the study of the biosynthesis, storage, chemistry, and physiological function of hormones and the tissues that secrete them. The endocrine system consists of different parts of the body that secrete hormones directly into the blood rather than into a duct system. Hormones have many different functions and modes of action. They may act locally or away from their site of origin. They often interact with other biological systems.

Cross-References

- ▶ Diabetes

References and Readings

Sperling, M. A. (2009). *Pediatric endocrinology* (3rd ed.). Philadelphia: WB Saunders.
Wilson, J. D. (2008). *Williams textbook of endocrinology* (11th ed.). Philadelphia: WB Saunders.

End-of-Life Care

Andrea Croom
Department of Psychology, University of Texas Southwestern Medical Center, Dallas, TX, USA

Synonyms

End-of-life issues; Terminal care

Definition

End-of-life care is a general term used to describe all aspects of care received by patients with a terminal illness or terminal condition that has become advanced, progressive, and/or incurable. End-of-life care has become increasingly important in the past century as life expectancies have increased and causes of death have predominantly moved from acute illnesses (e.g., infections) to chronic and terminal illnesses (e.g., cancer and heart disease). The general goal of end-of-life care is to help patients achieve a "good death" as they define it. End-of-life care is provided through palliative care and hospice services and frequently incorporates complementary and alternative medicines (e.g., massage therapy, pet therapy, music therapy, aromatherapy, acupuncture, etc.). These services aim to improve patient *quality of life* through reducing pain and managing symptoms, addressing spiritual and emotional needs, and providing family/caregiver support.

Description

Issues for Patients (Advance Care Planning)

End-of-life care emphasizes the importance of patient autonomy through advance care planning, which is the process of patients, healthcare professionals, and caregivers discussing and formally documenting the patients' preferences for healthcare treatment as death approaches. The Patient Self Determination Act (PSDA) passed by the United States Congress in 1990 requires healthcare facilities that receive federal funding to educate patients and the community about advance directives. Advance directives are oral and written instructions about the patients' goals and wishes concerning future medical care that becomes effective only when a person cannot speak for him or herself. Decisions are commonly made about desires for mechanical ventilation (e.g., respirator), nutrition, and hydration (e.g., feeding tubes), kidney dialysis, and antibiotic treatments. Advance care planning also involves making decisions about receiving cardiopulmonary resuscitation (CPR) when vital functions cease. Patients who do not wish to receive CPR can complete a Do Not Resuscitate (DNR) order, which is kept as part of their medical file. Finally, patients are able to appoint a durable medical power of attorney (sometimes referred to as a healthcare proxy). This is the person who will be responsible for making decisions for the patient about healthcare treatment after the patient lacks the capacity to do so for him or herself. Advance care planning should be formally documented as well as verbally communicated between patients, caregivers, and healthcare professionals to ensure that the patients' wishes are known and understood. All states legally recognize some form of advance directives.

Issues for Healthcare Professionals

Healthcare professionals are responsible for many important aspects of end-of-life care. Healthcare professionals must formulate and communicate information to patients about their prognosis (i.e., how long the patient is expected to live). Developing an accurate prognosis is difficult to do considering the unpredictability of disease, the large number of life-extending technologies available, and the great number of unknown and unmeasureable variables that influence how and when a person will die. Communicating this information to

patients is equally difficult due to concerns about over- or under-estimating life expectancy, instilling or destroying hope, and cultural differences about discussing death. Healthcare professionals are also responsible for helping patients to engage in advance care planning and determining when specific treatments are not likely to benefit the patient (i.e., medically futile treatments). Healthcare professionals' major responsibility is to identify and manage symptoms, which typically become more severe as the illness or condition progresses. These symptoms commonly include: (1) pain, (2) increased sleep, drowsiness, or unresponsiveness, (3) decreased needs for food and fluids, loss of appetite, nausea or vomiting, (4) decreased socialization and increased withdrawal, (5) depression, (6) confusion about time, place, or identity (i.e., delirium), (7) changes in bladder or bowel control, (8) changes in temperature regulation (e.g., skin feels cool), and (9) respiratory changes (e.g., irregular and shallow breaths). Finally, healthcare professionals aid in assessing the patients' capacity to make healthcare decisions (i.e., capacity assessment).

Issues for Caregivers

Caregivers play an important role in end-of-life care as family members and friends are often responsible for most of the day-to-day care of patients during the end of life. The risk for caregiver stress is high as caregivers have to balance their normal daily activities, additional care giving responsibilities, efforts to help the patient adjust to the illness, and their personal emotional reactions to and fears about the illness. Even though family members report high levels of satisfaction with the care-giving experience, they also report more depressive symptoms and psychosocial stress than the general public. As the illness progresses, there are additional caregiving needs and the psychosocial distress of caregivers becomes more prevalent. Untreated psychosocial distress in caregivers is associated with poorer patient care, increased health problems for caregivers, and more severe grief reactions after patient death. Caregivers must learn how to care for the patient while continuing to practice good self-care. Caregivers also have the additional stress of surrogate decision making when the patients lacks the capacity to make their own healthcare decisions.

Ethical Issues

End-of-life care is an area of medicine that frequently involves ethical dilemmas. The majority of laws related to end-of-life care are governed by individual states and there is wide variation in how the states approach these issues.

Early debates focused on determining when a patient is legally dead. Death was traditionally considered the point at which a patient's vital physical functions cease; however, advances in life support technology have made it more difficult to determine when someone's body is no longer functioning. The Uniform Determination of Death Act (UDDA), written by the President's Commission on Bioethics in 1981, confronts the complexities concerning the declaration of death. The UDDA states that a person can be declared dead when *either* the heart and lungs *or* the brain and brain stem stop functioning permanently, but specific guidelines are determined by individual states. Declaring the point at which a patient has died can be an important issue in organ donation.

One of the most prominent debates related to end-of-life care has been the issue of euthanasia (also referred to as "hastened death"). Euthanasia is an act where a third party, usually implied to be a physician, terminates the life of a person either passively or actively. Physician-assisted suicide is a specific form of euthanasia where a doctor provides a patient with a prescription for drugs that the patient can choose to voluntarily use to end his or her life. The main distinction between physician-assisted suicide and active euthanasia is that the physician is not the person physically administering the drugs. The Oregon Death with Dignity Act (1997) and the Washington Death with Dignity Act (2008) made it legal in these two states for patients to hasten their own death with a prescribed lethal dose of medication from a physician.

There are several unique patient populations which carry their own ethical concerns. First, until recently children with terminal conditions have been granted limited autonomy in making decisions about their end-of-life care. As recently as the 1960s, the consensus was that children should not be informed of a terminal diagnosis because they would not be able to understand and would find the news too upsetting. Parents have legal rights to make decisions for their children, but many healthcare professionals now feel that it is beneficial for children to be included in healthcare discussions and to be permitted to make their wishes known. Ethical dilemmas arise when parents and children disagree about healthcare decisions, when two parents with equal custodial rights disagree about healthcare decisions, or when parents refuse physician treatment or do not appear to be acting in the "best interest" of their child. Second, culturally diverse populations have been found to favor different treatment preferences (e.g., African Americans and Hispanics are more likely than European Americans to express a preference for life-sustaining treatment), to engage less frequently in advance care planning, to use services such as hospice less frequently, and to report higher levels of insufficient pain management. Many cultures do not prioritize patient autonomy and prefer to pass decision-making responsibilities to others in the family. Healthcare professionals must balance respecting cultural differences while still sufficiently providing end-of-life care and informing patients of their options.

Cross-References

- ▶ Capacity Assessment
- ▶ Complementary and Alternative Medicine
- ▶ Euthanasia
- ▶ Hospice
- ▶ Palliative Care
- ▶ Physician-Assisted Suicide
- ▶ Self-Care
- ▶ Surrogate Decision Making
- ▶ Symptoms

References and Readings

American Psychological Association. (1998). *Report of the APA working group on assisted suicide and end-of-life decisions*. Retrieved January 15, 2011 from http://www.apa.org/pubs/info/reports/aseol.aspx.

American Psychological Association. (2002). *End-of-life issues and care brochure*. Retrieved January 15, 2011 from http://www.apa.org/topics/death/end-of-life.aspx.

Association of Death Education and Counseling (ADEC). Published in 2012 in Deerfield, IL. Retrieved January 15, 2011 from www.adec.org.

Callanan, M., & Kelley, P. (1992). *Final gifts: Understanding the special awareness, needs, and communications of the dying*. New York: Bantam Books.

Feldman, D. B., & Lasher, S. A. (2007). *The end-of-life handbook: A compassionate guide to connecting with and caring for a dying loved one*. Oakland, CA: New Harbinger.

Ingram, D. J. (2003). *A good death: A guide to life's last voyage*. Bloomington, IN: AuthorHouse.

Kessler, D. (1997). *The needs of the dying: A guide for bringing hope, comfort, and love to life's final chapter*. New York: HarperCollins.

Kinzbrunner, B., & Policzer, J. (2010). *End of life care: A practical guide* (2nd ed.). New York: McGraw Hill.

Kubler-Ross, E. (1969). *On death and dying*. New York: Scribner.

University of Minnesota Center for Bioethics. (2005). *End-of-life care: An ethical overview*. Retrieved December 16, 2010 from www.ahc.umn.edu/img/assets/26104/End_of_Life.pdf

Werth, J., & Blevins, D. (2009). *Decision-making near the end of life: Issues, developments, and future directions*. New York: Taylor & Francis Group LLC.

End-of-Life Care Preferences

- ▶ DNR Order

End-of-Life Issues

- ▶ End-of-Life Care

Endogenous Morphine

- ▶ Endogenous Opioids/Endorphins/Enkephalin

Endogenous Opioids/Endorphins/Enkephalin

James A. McCubbin
Department of Psychology, Clemson University, Clemson, SC, USA

Synonyms

Dynorphins; Endogenous morphine; Endomorphins; Opiate neuropeptides; Opiate peptides; Opiate receptors

Definition

Endogenous opioids are neuropeptides with morphine-like activity that are naturally synthesized within the body. These neuropeptides have widespread distribution throughout the central and peripheral nervous systems, and various endocrine and other tissues. Opioids function as neurotransmitters and hormones, with a wide variety of biobehavioral effects in health and disease. Their effects on physiological and psychological responses to intense aversive and appetitive stimuli suggest potentially important roles in the etiology and treatment of self-regulatory disorders of appetite, affect, and adaptation to stress.

Description

Classification of Opioid Peptides and Receptors

Endogenous opioids systems include several different neuroactive peptides that are linked, in turn, to a matrix of distinctive receptor systems. The opioid peptides are divided into basic subgroups, e.g., endorphins, enkephalins, and dynorphins, based on their biosynthetic parent molecules. A separate group of endomorphins has been identified, but these peptides are not yet well characterized. Opioid receptors are part of the family of G-protein-coupled receptors and are classified into multiple receptor types and subtypes based on relative affinity for selective agonists and antagonists. For example, μ (mu) receptors demonstrate high affinity for morphine and endomorphins, while δ (delta) receptors are highly selective for enkephalins, and κ (kappa) receptors show high affinity and activity for dynorphins. Another putative receptor type, the ε (epsilon) receptor, has been postulated to explain beta-endorphin activity not mediated via the other receptor types.

Distribution of Opioid Peptides and Receptors

Endogenous opioids and receptors are localized in the central and peripheral nervous systems, including neuroendocrine stress pathways, and in brain areas mediating reward and reinforcement. For example, opioid peptides and/or receptors are found in afferent and integrative pain nuclei, as well as in the two major stress effector pathways, the hypothalamic-pituitary-adrenocortical (HPA) axis and the hypothalamic-sympatho-adrenomedullary (SAM) axis. Enkephalins have an abundant distribution throughout the limbic and sympathetic systems, while endorphin-containing cells are prominent in the hypothalamus and in the anterior pituitary. Dynorphins are widely distributed throughout both central and peripheral nervous systems. Opioid systems are intimately incorporated into peripheral organs including the heart and the gastrointestinal system. The diversity of opioidergic molecular representation yields, in turn, a diversity of functions, with important behavioral and physiological effects.

Role of Opioids in Stress, Neuroendocrine Reactivity, and Homeostasis

Endogenous opioids are important regulators of both the anterior and the posterior pituitary. Endogenous opioid mechanisms inhibit both the SAM and the HPA axes, suggesting opioidergic input to corticotropin-releasing factor neurons in the paraventricular hypothalamus. Opioids influence stress-induced pituitary release of adrenocorticotropic hormone (ACTH) and prolactin, as well as release of growth hormone and luteinizing

hormone. In the posterior pituitary, endogenous opioids inhibit release of both vasopressin and oxytocin. Therefore, regulation of the HPA axis and other important neuroendocrine pathways is mediated, in part, via endogenous opioids.

Peripheral opioid peptides and receptors are especially prominent in pituitary systems intimately involved in maintenance of homeostasis during stress. For example, beta-endorphin is localized in anterior and intermediate pituitary and is co-stored and co-released with ACTH. Therefore, activation of the HPA cascade is associated with pituitary release of beta-endorphin into the systemic circulation, where it has critical roles in the integrated response to psychological stressors.

The SAM axis, including the peripheral sympathetic nervous system and the adrenal medullae, is subject to central opioidergic control in the hypothalamus and elsewhere (McCubbin, 1993). CNS opioids are capable of both excitatory and inhibitory functions, and these effects are especially pertinent to biobehavioral function and dysfunction. Opioids have been shown to inhibit sympathetic and adrenomedullary responses at multiple levels of the SAM axis. For example, peripheral enkephalins are found in autonomic ganglia and in the spinal sympathetic cell columns. Enkephalins have been shown to inhibit release of catecholamines from peripheral sympathetic nerve endings as well as from the adrenal medullae.

Opioids in Health and Disease

Endogenous opioids play important roles in motivational integration of appetitive and aversive behavior and are critical in the maintenance of visceral homeostasis. The importance of these basic mechanisms of adaptation suggests that opioid dysfunction could underlie a variety of disorders involving dysregulation of appetite, affect, and neuroendocrine reactivity.

Appetitive Mechanisms Maintaining Chemical and Behavioral Dependencies

Opioid input to mesolimbic dopaminergic and other CNS systems suggests a potentially important role in appetitive reward and reinforcement mechanisms that maintain behavioral and chemical dependencies (Koob & Le Moal, 1997). Moreover, the important role of endogenous opioids in mediation of CNS reward systems may point to better treatment strategies in substance abuse and other disorders of appetite regulation (Reece, 2011). One overarching theory is that if opioids mediate the CNS reward mechanisms in dependency, then pharmacological opioid blockade may disengage brain mechanisms that reinforce and maintain a variety of different chemical and behavioral dependencies. For example, clinical trials are currently underway to examine the potential therapeutic effects of opioid antagonists, either alone or in combination with other drugs, in several appetitive disorders, including nicotine and alcohol dependence, and obesity. (Note: The efficacy of opioid antagonists in treatment of heroin/morphine dependencies operates via multiple complex mechanisms.) Therefore, opioid brain mechanisms may point to novel strategies for development of new behavioral and pharmacological treatments for dependencies and other diseases of appetite regulation.

Acute and Chronic Pain

Opioid systems contribute to important endogenous analgesic mechanisms, such as stress-induced analgesia and the hypoalgesia accompanying elevated blood pressure. These effects on pain sensitivity may be intimately associated with higher CNS integration of pain perception. For example, endogenous opioids do much more than simply inhibit pain perception; they may also have significant effects on regulation of affective responses to both painful and nonpainful aversive stressors. This observation reinforces the notion that endogenous opioid systems play an important role in the higher-level integration of affect in the appraisal of emotionally meaningful stimuli.

Endogenous opioidergic analgesia appears to be important for coping with both acute and chronic pain. Evidence suggests that chronic pain patients have reduced opioid levels in

plasma and cerebrospinal fluid. This is consistent with the opioid depletion hypothesis, which proposes that chronic pain is associated with progressive depletion of endogenous opioid analgesic neurochemicals and/or downregulation of opioid receptors (Bruehl, McCubbin, & Harden, 1999). This depletion of analgesic opioids results in dysfunction of an important endogenous mechanism for coping with chronic pain. Opioids may also have a role in the expression or maintenance of self-injurious behavior.

Depression and Posttraumatic Stress Disorder (PTSD)

Endogenous opioids play an important role as physiological mechanisms for coping with psychological stress, and these systems have been implicated in stress-related disorders, including depression and PTSD (Merenlender-Wagner, Dikshtein, & Yadid, 2009). Opioid blockers can reverse stress analgesia and performance deficits in learned helplessness, and can worsen symptoms of PTSD. Persons exposed to traumatic stress may utilize their endogenous opioid analgesic and/or affect regulatory mechanisms to cope. Thus, the role of opioids in HPA axis regulation, endogenous analgesia, and multiple CNS pathways provide a neurobiological rationale for role of opioids in regulation of affect and coping with traumatic stress.

Cardiovascular Disease

A biobehavioral link between opioids and risk for cardiovascular disease is found in work with the opioid antagonists, naloxone and naltrexone. These studies suggest that opioids can inhibit sympatho-adrenomedullary and blood pressure responses to psychological stress in young persons with normal circulatory risk profiles. However, young persons at increased risk for hypertension show reduced opioid inhibition of sympathetic, HPA, and circulatory responses to stress (McCubbin, 1993). This apparent dysfunction of inhibitory opioids may underlie exaggerated blood pressure reactivity to stress and its attendant cardiovascular health consequences. Moreover, there is some evidence to suggest that opioid hypoalgesia is associated, at least in part, with blood pressure elevations and increased risk for hypertension development. Recent evidence points to gender differences in opioid effects on blood pressure control that are dependent, in part, on estrogen. The relationship between reduced opioid inhibition of the HPA and SAM axes, blood pressure dysregulation, and hypoalgesia requires additional work to better characterize these complex interactions.

Opioids and Behavioral Therapies

Behavioral control of endogenous opioid tone may become an important strategy in prevention and treatment of self-regulatory disorders of appetite and adaptation to stress. Interestingly, studies of aerobic fitness, relaxation, and systematic desensitization suggest that these forms of stress management operate, at least in part, via activation of endogenous opioid mechanisms (McCubbin et al., 1996). For example, opioid blockade with naltrexone can reverse the reductions in cardiovascular stress reactivity associated with aerobic fitness and progressive relaxation training. Thus, in persons at risk for hypertension who lack robust opioidergic inhibition of the SAM axis, some behavioral stress-management interventions can restore normal opioid inhibitory function. Behavioral prevention and treatment strategies that target normalization of endogenous opioid tone may become more common as sophistication of these peptide systems grows.

Summary

The endogenous opioid neuropeptides and receptors form a diverse set of basic neuroendocrine systems that modulate behavioral and physiological reactions to aversive and appetitive stimuli. These systems have become critical for understanding integrated responses to psychological stress in health and disease. Better understanding of these neuropeptide systems will provide insight into the developmental etiology and new

treatment strategies for self-regulatory disorders of appetite, affect, and adaptation to stress.

Cross-References

- Addiction
- Affect
- Analgesia
- Appetite
- Autonomic Nervous System (ANS)
- Behavioral Therapy
- Blood Pressure
- Depression
- Exercise
- Homeostasis
- Hypertension
- Hypothalamus
- Obesity
- Pain
- Posttraumatic Stress Disorder
- Relaxation
- Smoking
- Stress
- Substance Abuse
- Sympathetic Nervous System (SNS)
- Systematic Desensitization

References and Readings

Bruehl, S., McCubbin, J. A., & Harden, R. N. (1999). Theoretical review: Altered pain regulatory systems in chronic pain. *Neuroscience and Biobehavioral Reviews, 23*, 877–890.

Koob, G. F., & Le Moal, M. (1997). Drug abuse: Hedonic homeostatic dysregulation. *Science, 278*, 52–57.

McCubbin, J. A. (1993). Stress and endogenous opioids: Behavioral and circulatory interactions. *Biological Psychology, 35*(2), 91–122.

McCubbin, J. A., Wilson, J. F., Bruehl, S., Ibarra, P., Carlson, C. R., Norton, J. A., et al. (1996). Relaxation training and opioid inhibition of blood pressure response to stress. *Journal of Consulting and Clinical Psychology, 64*(3), 593–601.

Merenlender-Wagner, A., Dikshtein, Y., & Yadid, G. (2009). The beta-endorphin role in stress-related psychiatric disorders. *Current Drug Targets, 10*(11), 1096–1108.

Reece, A. S. (2011). Hypothalamic opioid-melanocortin appetitive balance and addictive craving. *Medical Hypotheses, 76*(1), 132–137. doi:10.1016/j.mehy.2010.09.002.

Endometriosis

Beate Ditzen[1], Friedrich Wieser[2] and Robert N. Taylor[3]
[1]Division of Clinical Psychology and Psychotherapy, Department of Psychology, University of Zurich, Binzmuhlestrasse, Zurich, Switzerland
[2]Department of Gynecology and Obstetrics, Emory University School of Medicine, Atlanta, GA, USA
[3]Department of Obstetrics and Gynecology, Wake Forest School of Medicine, Winston-Salem, NC, USA

Definition

Endometriosis (EM) is a common, benign disorder defined by the presence of endometrial tissue outside of the uterus (Giudice, 2010). EM can be asymptomatic, but it may be associated with severe dysmenorrhea (painful menstruation or "cramps"), pelvic pain (intermittent non-menstrual or continuous pain in the pelvic area), dyspareunia (pain during sexual intercourse), and infertility in affected women. The prevalence of pelvic EM is 6–10% in the general population whereas it approaches 35–50% in women with pain, infertility, or both (Houston, 1984; Sensky & Liu, 1980). The etiology of the disease appears to involve a complex interplay of multiple genetic, environmental, immunologic, and potential psychological factors (Guo, 2009); however, the ultimate pathogenesis of EM is unknown to date. Surgical assessment, by laparoscopy or laparotomy, remains the diagnostic gold standard in order to diagnose EM. The extent of the disease is classified by the American Society for Reproductive Medicine (ASRM) revised classification system for EM (rAFS score, American Society for Reproductive Medicine [ASRM], 1997). The rAFS scoring system categorizes EM into four stages (I through IV), with the higher stages representing more extensive disease. While rAFS staging tends to correlate with infertility, the severity of EM does not show consistent

associations with pain ratings, suggesting that further mechanisms mediate pain perception in women with EM (Asante & Taylor, 2011).

The successful treatment of EM-associated symptoms typically requires medical as well surgical interventions: Medical therapies include agents that suppress ovarian function and limit the growth of endometriosis, such as androgens, progestagens, GnRH agonists, and contraceptive steroids. The treatment of EM-associated pain has been well studied and most major medical therapies appear to be superior to placebo. EM-associated infertility, however, does not respond to medical therapies alone. Surgical treatment (via laparotomy or laparoscopy) as well as assisted reproduction techniques were found to be beneficial in restoring fertility in EM (Giudice & Kao, 2004; Practice Committee of the American Society for Reproductive Medicine, 2004). Surgery commonly provides temporary pain relief, but symptoms recur in 50% of the women within 2 years. Because of the associated pain and due to the fact that so far there is no cure for EM, affected women often report higher levels of distress than healthy controls (cf., Kaatz, Solari-Twadell, Cameron, & Schultz, 2010). In line with this, increased rates of anxiety and depressive symptoms as well as impaired overall quality of life were found in EM. Also, women suffering from EM have an increased risk of several medical conditions, including hypothyroidism, fibromyalgia and chronic fatigue syndrome, autoimmune inflammatory diseases, allergies, and asthma (Sinaii, Cleary, Ballweg, Nieman, & Stratton, 2002). These data suggest a common immunological and endocrinological aspect to EM and these conditions, which in turn might further increase disease burden. As a consequence, a multilevel approach to EM should include an evaluation of psychological distress, and in some women psychological interventions can be helpful in order to reduce impairment.

Cross-References

► Chronic Pain, Types of (Cancer, Musculoskeletal, Pelvic), Management of

References and Readings

American Society for Reproductive Medicine. (1997). Revised American Society for Reproductive Medicine classification of endometriosis: 1996. *Fertility and Sterility, 67*(5), 817–821.

Asante, A., & Taylor, R. N. (2011). Endometriosis: The role of neuroangiogenesis. *Annual Review of Physiology, 73*, 163–182.

Giudice, L. C. (2010). Clinical practice: Endometriosis. *The New England Journal of Medicine, 362*(25), 2389–2398.

Giudice, L. C., & Kao, L. C. (2004). Endometriosis. *The Lancet, 364*(9447), 1789–1799.

Guo, S. W. (2009). Epigenetics of endometriosis. *Molecular Human Reproduction, 15*(10), 587–607.

Houston, D. E. (1984). Evidence for the risk of pelvic endometriosis by age, race and socioeconomic status. *Epidemiologic Reviews, 6*, 167–191.

Kaatz, J., Solari-Twadell, P. A., Cameron, J., & Schultz, R. (2010). Coping with endometriosis. *Journal of Obstetric, Gynecologic, and Neonatal Nursing, 39*(2), 220–225. Quiz 225–226.

Practice Committee of the American Society for Reproductive Medicine. (2004). Endometriosis and infertility. *Fertility and Sterility, 82*(Suppl. 1), S40–S45.

Sensky, T. E., & Liu, D. T. (1980). Endometriosis: associations with menorrhagia, infertility and oral contraceptives. *International Journal of Gynaecology and Obstetrics, 17*(6), 573–576.

Sinaii, N., Cleary, S. D., Ballweg, M. L., Nieman, L. K., & Stratton, P. (2002). High rates of autoimmune and endocrine disorders, fibromyalgia, chronic fatigue syndrome and atopic diseases among women with endometriosis: a survey analysis. *Human Reproduction (Oxford, England), 17*(10), 2715–2724.

Endomorphins

► Endogenous Opioids/Endorphins/Enkephalin

Endothelial Function

Jet Veldhuijzen van Zanten
School of Sport and Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

Synonyms

Blood vessel wall

Definition

Blood vessels consist of three layers. The inner layer is the intima, which is made up from endothelial cells. The middle layer, the tunica media, consists of mainly smooth muscle cells, which are important for the maintenance of the vessel diameter. Finally, the outer layer, the tunica externa, consists of connective tissue. The endothelium is a dynamic organ with several functions, such as regulation of the vascular tone, platelet aggregation, thrombosis, and adhesion of leucocytes, which are vasoprotective.

Description

The peripheral vascular system consists of arteries, capillaries, and the veins. The arteries supply the organs with blood, the capillaries allow for the exchange of metabolites between the blood and the organs, and the veins facilitate the return of the blood to the heart. Blood vessels consist of three layers. The inner layer is the intima, which is made up from endothelial cells. The middle layer, the tunica media, consists of mainly smooth muscle cells, which are important for the maintenance of the vessel diameter. Finally, the outer layer, the tunica externa, consists of connective tissue. The relative thickness of these individual layers is dependent on the position of the vascular tree of the vessel wall. For example, in capillaries, the vessel wall mainly consists of endothelial cells to optimize metabolite exchange (Vander, Sherman, & Luciano, 2006).

The endothelium is a dynamic organ with several functions, such as regulation of the vascular tone, platelet aggregation, thrombosis, and adhesion of leucocytes, which are vasoprotective. However, when damage to the endothelial cells occurs, this can result in endothelial dysfunction, which is a precursor of atherosclerosis (Lerman & Zeiher, 2005). The activity of the endothelial cells can be influenced by sympathetic nerve activity, hormones, and inflammatory molecules among other factors (Levick, 2003). From a behavioral medicine perspective, endothelial function has been associated with health behaviors such as physical activity (Green et al., 2003) and smoking (Toda & Toda, 2010). In addition, sympathetic activation through acute exercise or mental stress can influence vascular function (Joyner & Halliwill, 2000).

Assessing Endothelial Function

An important function of endothelial cells is the maintenance of vascular tone, which is emphasized by the release of several vasoactive substances by the endothelial cells. Therefore, it is not surprising that most in vivo endothelial function assessments are concerned with the capacity of the endothelium to vasodilate. A principal vasodilator released by the endothelial cells is nitric oxide (NO). Following the conversion from L-arginine under the influence of nitric oxide synthase, NO starts a cascade of conversions which leads to smooth muscle cell relaxation, i.e., vasodilation (Sandoo, Veldhuijzen van Zanten, Metsios, Carroll, & Kitas, 2010). It is outside the scope of this section to describe the mechanisms of NO and other vasoactive substances in detail.

Various methods are available for the assessment of endothelial function in the peripheral circulation. Most functional assessments examine vasodilation in response to a standardized stimulus, with an attenuated vasodilatory response indicative of endothelial dysfunction. As described above, a major contributor to vasodilation bioavailability of NO starts a cascade of events, which results in the relaxation of the vascular smooth muscle cells. Therefore, impaired vasodilation can be due to both reduced NO bioavailability in the endothelium or impaired capacity of the vascular smooth muscle to dilate. Consequently, most endothelial function assessments involve both measures of endothelial-dependent vasodilation (related to NO bioavailability) and endothelial-independent vasodilation (related to vascular smooth muscle function) (Sandoo et al., 2010). Given the difference in anatomy and function of arteries, the functional assessments vary depending on their position in the arterial tree. The microvasculature involves conduit arteries, such as the brachial and femoral artery, whereas microvasculature entails arterioles or resistance vessels. In addition to the functional assessment described below, the structure of the

vessel walls can be examined with intima medial thickness (for conducting artery, such as carotid) and nailfold capillaroscopy (for capillaries) (Sandoo et al., 2010).

Microvascular function can be assessed using iontophoresis or forearm blood flow. Iontophoresis assessment involves the administration of vasoactive substances through the skin by applying a small electrical current. The most commonly used substances are acetylcholine (ACh) for the assessment of endothelium-dependent vasodilation and sodium nitroprusside (SNP) for endothelium-independent vasodilation. Perfusion of the vessels in the skin is assessed using either laser Doppler flowmetry, when examining a single point, or laser Doppler imaging, when the area of interest is a larger area of skin (Turner, Belch, & Khan, 2008).

Forearm blood flow is most commonly used together with venous occlusion strain gauge plethysmography. For this assessment, venous outflow of the vessels is occluded, while allowing arterial inflow (Joyner, Dietz, & Shepherd, 2001). Changes in arm circumference are assessed using strain gauge plethysmography, with the slope of the increase in arm circumference reflecting of blood flow. The advantage of this assessment is that it can be carried out at several time points throughout a testing session, so immediate changes of blood flow in response to mental stress or exercise can be investigated using this method. Strain gauge forearm blood flow assessments are also carried out in response to intravenous infusion of vasoactive substances, such as ACh and bradykinin.

Macrovascular function can be assessed using flow-mediated dilation. Blood flow to the arm will be occluded for a period of 5 min by inflating a brachial cuff placed around the arm to at least 50 mmHg above systolic blood pressure. Release of the cuff will result in a sudden inflow of blood into the arm. The increase in shear stress as a result of the surge of blood (reactive hyperemia) will induce vasodilation in healthy arteries, which is dependent on NO production of the endothelium. Endothelial-independent vasodilation is assessed by investigating the vasodilation in response to the administration of glyceryl trinitrate (GTN). Macrovascular dilation is most commonly quantified by recording the vessel diameter using high-resolution ultrasound (Corretti et al., 2002).

Finally, arterial stiffness is related to the compliance of the vessel wall and can be classified as both a structural as well as a functional measure of endothelial function. This assessment explores the capacity of the vasculature to accommodate pressure pulsations. Reduced elasticity will increase the afterload on the heart, which means that the strain on the heart is increased. Applanation tonometry is used to record the arterial pressure waveforms. For pulse-wave analyses, the waveforms of one artery are explored and this results into the calculation of the augmentation index, which is derived from the first and second systolic peak in pressure. For pulse-wave velocity, waveforms are recorded on two sites on the arterial tree and the combination of the transit time between the waveforms and the distance between assessment points will be used to calculate pulse-wave velocity. An increase in augmentation index and an increase in pulse-wave velocity are indicative of arterial stiffness (Sandoo et al., 2010).

It is worth noting that substantial training is necessary in order to carry out these vascular assessments to a sufficient standard. In addition, all these assessments are influenced by several factors such as timing of assessment, fasting, caffeine consumption, and smoking. Therefore, it is important that all assessments are carried out following published guidelines (Corretti et al., 2002; Turner et al., 2008).

Cross-References

- ▶ Arteries
- ▶ Atherosclerosis
- ▶ Intima-Media Thickness (IMT)
- ▶ Nitric Oxide Synthase (NOS)
- ▶ Vasoconstriction
- ▶ Vasodilation, Vasodilatory Functions

References and Readings

Corretti, M. C., Anderson, T. J., Benjamin, E. J., Celermajer, D., Charbonneau, F., Creager, M. A.,

et al. (2002). Guidelines for the ultrasound assessment of endothelial-dependent flow-mediated vasodilation of the brachial artery: A report of the International Brachial Artery Reactivity Task Force. *Journal of the American College of Cardiology, 39*, 257–265.
Green, D. J., Walsh, J. H., Maiorana, A., Best, M. J., Taylor, R. R., & O'Driscoll, J. G. (2003). Exercise-induced improvement in endothelial dysfunction is not mediated by changes in CV risk factors: Pooled analysis of diverse patient populations. *AJP – Heart and Circulatory Physiology, 285*, H2679–H2687.
Joyner, M. J., Dietz, N. M., & Shepherd, J. T. (2001). From Belfast to Mayo and beyond: The use and future of plethysmography to study blood flow in human limbs. *Journal of Applied Physiology, 91*, 2431–2441.
Joyner, M. J., & Halliwill, J. R. (2000). Sympathetic vasodilatation in human limbs. *The Journal of Physiology, 526*(Pt 3), 471–480.
Lerman, A., & Zeiher, A. M. (2005). Endothelial function: Cardiac events. *Circulation, 111*, 363–368.
Levick, J. R. (2003). *An introduction to cardiovascular physiology* (4th ed.). Oxford, USA: Oxford University Press.
Sandoo, A., Veldhuijzen van Zanten, J. J. C. S., Metsios, G. S., Carroll, D., & Kitas, G. D. (2010). The endothelium and its role in regulating vascular tone. *Open Cardiovascular Medicine Journal, 4*, 302–312.
Toda, N., & Toda, H. (2010). Nitric oxide-mediated blood flow regulation as affected by smoking and nicotine. *European Journal of Pharmacology, 649*, 1–13.
Turner, J., Belch, J. J. F., & Khan, F. (2008). Current concepts in assessment of microvascular endothelial function using laser Doppler imaging and iontophoresis. *Trends in Cardiovascular Medicine, 18*, 109–116.
Vander, A., Sherman, J., & Luciano, D. (2006). *Human physiology* (10th ed.). New York: McGraw Hill.

Endothelial Nitric Oxide Synthase (eNOS)

► Nitric Oxide Synthase (NOS)

End-Stage Renal Disease

Quinn D. Kellerman
Department of Psychology, University of Iowa, Iowa City, IA, USA

Synonyms

Chronic kidney disease (CKD)

Definition

The kidneys serve four primary functions: (1) to clean the blood of toxins, (2) to remove excess fluid and waste, (3) to balance chemicals (i.e., sodium, potassium, phosphorus), (4) and to release hormones that control blood pressure, the production of red blood cells, and contribute to bone strength. End-stage renal disease (ESRD) is reached when the capacity of the kidneys declines such that they are no longer able to adequately perform these functions, ultimately requiring the affected individual to initiate treatment in the form of renal replacement therapy to sustain life.

Description

Cause, Symptoms, and Diagnosis of ESRD

ESRD most commonly manifests as a secondary condition resulting from poorly managed diabetes or hypertension. Chronic elevations in blood glucose and blood pressure cause damage to the small blood vessels in the kidneys, which over time can progress to ESRD. Other causes of ESRD include autoimmune diseases such as lupus, complications of infection such as glomerulonephritis, and genetic abnormalities such as polycystic kidney disease.

Many symptoms are associated with the progression of kidney disease to ESRD, including weakness, fatigue, lack of energy, appetite and weight loss, nausea and vomiting, metallic taste in the mouth, breath smelling like ammonia, changes in skin color, rash or itching, cognitive impairment, changes in urination, swelling, shortness of breath, feeling cold, and leg or flank pain.

According to the National Kidney Foundation's Kidney Disease Outcomes Quality Initiative (KDOQI), chronic kidney disease (CKD) can

Note: Some of the data reported here have been supplied by the United States Renal Data System (USRDS). The interpretation and reporting of these data are the responsibility of the author(s) and in no way should be seen as an official policy or interpretation of the U.S. government.

progress through stages of severity, with stage 5 typically denoting a diagnosis of ESRD and a need for treatment initiation. The stages of disease are determined by the level of kidney damage (i.e., pathologic abnormalities) and/or the degree of deficiency in the individual's estimated glomerular filtration rate (eGFR), a commonly used biomarker to diagnose ESRD. The eGFR is calculated based on serum creatinine, age, race, and gender; values less than 15 mL/min/1.73 m^2 are suggestive of stage 5 kidney disease, or ESRD. These values indicate that the kidneys are performing at less than 15% of normal functioning. In addition, increases in blood urea nitrogen (BUN) and protein in the urine (proteinuria) are markers of ESRD.

Prevalence of ESRD in the US Population

The Annual Data Report from the United States Renal Data System suggests that the number of individuals affected by ESRD increases annually, with a record high of 571, 414 patients receiving treatment in 2009. There exist significant racial and ethnic disparities in ESRD, with African Americans nearly four times more likely to develop ESRD than Whites. Native Americans and Asians are at least twice as likely to be diagnosed with ESRD compared to Whites, and the rate of ESRD in the Hispanic population is 1.5 greater than that of non-Hispanics. With regard to age and gender, the ESRD rates are higher among older adults and males. Recent reports suggest that the growing number of new ESRD patients has been driven by a linear increase in diagnoses among individuals aged 45–64; in contrast, there has been minimal change in the incidence rates of patients age 65 and older over the last several years.

Treatments for ESRD

Hemodialysis is the most common type of treatment for ESRD, with 65% of the affected population (approximately 372,000 patients) utilizing this treatment modality. Individuals who participate in hemodialysis typically come to a hospital or clinic 3 days per week for 3–5 h treatments. During this time, they are connected to a machine via an insertion site such as arteriovenous fistula or graft surgically configured in the forearm or a port catheter in the chest. The hemodialysis machine removes blood from the body, filtering accumulated toxins and removing excess fluids, and then returns the cleaned blood. This process is primarily directed by a nurse or dialysis technician, leaving the patient a relatively passive recipient of treatment. Hemodialysis can also be conducted in the home environment, though this is less commonly implemented due to expense and caregiver burden.

Peritoneal dialysis is an intervention for ESRD that requires the patient to be a more active participant in the treatment process. There are two forms of this treatment: continuous ambulatory peritoneal dialysis (CAPD) and continuous cycler-assisted peritoneal dialysis (CCPD). In CAPD, a permanent catheter is inserted into the patient's abdomen, which allows for a bag of sterile dialysis solution called dialysate to be connected to the body. The patient is responsible for performing "exchanges" which involve draining the dialysate into the peritoneal cavity via a sterile tube, allowing for the blood to filter through the peritoneal membrane leaving the excess fluid and toxins behind in the dialysate, and then discarding the used solution before reinitiating the procedure. Patients usually perform 3–4 exchanges throughout the course of the day while ambulatory and one longer overnight exchange while they are sleeping. In contrast, CCPD utilizes an automated cycler to perform the exchanges, with 3–5 cycles overnight while the patient sleeps and one long exchange during the day being the typical prescription. Recent reports indicate that approximately 6–7% of the ESRD population utilizes peritoneal dialysis as their treatment, which is a notable decrease from the 12–18% prevalence in the 1980s and 1990s. However, there is some evidence to suggest that the number of peritoneal dialysis users will increase in upcoming years.

Transplantation is often considered the preferred option for treatment of ESRD as it offers advantages including increased survival time and improvements in quality of life. Due to the continued shortage of donor organs,

contraindications to surgery in some patients, and concerns about rejection, however, this treatment is less commonly prescribed for ESRD compared to dialysis. Approximately 30% of the ESRD population undergoes renal transplantation with organs from either a deceased or living donor. According to the Organ Procurement and Transplantation Network (OPTN), an average of 17,000 renal transplants have been performed annually over the last 5 years, with approximately 65% from deceased donors and 35% from living donors. Survival of the renal graft across donor type is relatively high for renal transplant recipients (i.e., 1-year = 92%, 3-year = 82%, and 5-year = 71%), and living donor grafts tend to fare better than deceased donor grafts.

Adherence to ESRD Treatments

All patients undergoing treatment for ESRD are required to follow a lifelong regimen that necessitates ongoing behavioral involvement to ensure that the medical intervention remains safe and effective. For patients whose ESRD is treated with renal transplantation, adherence to an immunosuppressant medication regimen for the remainder of life is required to prevent the body from rejecting the transplanted organ. Individuals receiving hemodialysis as their method of ESRD treatment have an arguably more complex behavioral regimen to follow. Although patients undergo lengthy treatments several days per week, this does not fully compensate for normative kidney function; specifically, excess fluid and toxins build up and remain in the body between hemodialysis sessions. Fluid overload can lead to deleterious consequences, including congestive heart failure, pulmonary edema, cramping on dialysis, hypertension, fatigue, and decreased life expectancy. Similarly, buildup of chemicals that are dysregulated in ESRD can lead to complications such as myocardial infarction, stroke, heart arrhythmias, increased mortality, and bone demineralization. Thus, it is necessary for patients to restrict the amount of fluid ingested, and also their sodium, phosphorus, and potassium intake while their ESRD is being treated with hemodialysis.

Given the multifaceted and complex nature of the ESRD treatment regimen, the majority of patients have difficulty adhering to these recommendations. Research indicates that approximately 40–60% of ESRD patients do not adhere to one or more central aspects of the medical regimen. Adherence to fluid intake restrictions is most commonly measured by documented interdialytic weight gains (IWG). Individuals with ESRD are weighed before and after their hemodialysis treatments. The amount of weight gained *between* treatment sessions, calculated, for example, by subtracting the post-dialysis weight on a Monday from the pre-dialysis weight on a Wednesday, is considered a proxy for the amount of fluid the individual ingested during that time. One kilogram (kg) of weight is equivalent to 1 L of fluid; the recommended limitation is 1 L of fluid per day (including fluid in solid foods), which would equate to 2–3 kg of weight gained between sessions. Though patients tend to have the most difficulty with fluid restrictions, dietary adherence is also problematic. Sodium, potassium, and phosphorus intake is typically measured by serum levels drawn each month. There is modest evidence to suggest that in nearly half of cardiac-related ESRD patient deaths, nonadherence to dietary restrictions is the most significant contributor to mortality.

Researchers have also documented that patients experience the extreme restrictions on fluid intake as the most stressful and behaviorally challenging aspect of the ESRD hemodialysis regimen. There are a number of factors that likely contribute to this experience. First, individuals with ESRD tend to have increased thirst at baseline, often related to high blood glucose levels in those with diabetes and/or medication side effects. Second, the hemodialysis process itself, which rapidly removes excess fluid and toxins, leads to an electrolyte imbalance, increasing sodium appetite and thirst. Finally, contextual and behavioral factors impact patients' ability to follow fluid recommendations. For example, restricting fluid intake contradicts social norms about the health benefits of consuming large amounts of water, drinking has become habitual

for many individuals and habits are difficult to break, and there exist substantial environmental cues and social pressures to consume fluid in many different contexts. The fluid intake adherence problem often becomes cyclical in nature: increased thirst leads to greater fluid consumption, which leads to larger interdialytic weight gains and longer dialysis sessions, which further increases electrolyte imbalance and thirst, maintaining and increasing the severity of nonadherence.

Determinants and Interventions Related to ESRD Adherence

As might be expected based on reviews of the general adherence literature, a comprehensive understanding of the factors that contribute to nonadherence among ESRD patients has proven difficult to attain. Researchers have studied several factors thought to influence adherence in this population, and the results have been mixed. For example, some findings indicated that family support and marital adjustment were predictors of improved fluid intake adherence, while other studies found no evidence of an association between social support and fluid or dietary adherence among dialysis patients. The impact of cognitive and personality factors on adherence in ESRD has also been examined, including self-efficacy, health locus of control, perceived barriers, conscientiousness, and hostility. Higher self-efficacy expectations have been associated with improved fluid adherence in dialysis and better medication adherence in both dialysis and transplantation, whereas greater perceived barriers were related to poorer medication adherence. The findings relating health locus of control and adherence have been inconsistent. Personality characteristics such as conscientiousness and hostility have been significantly associated with adherence in ESRD patients in some work.

Some researchers have posited that the examining the interaction between patient characteristics or preferred style of coping with stress and the contextual features of the treatment regimen might help us to better understand adherence in this population. For example, individuals who endorse avoidant coping styles or prefer to have less control/involvement in their treatment have been found to display better adherence to hemodialysis performed in a center or hospital where the contextual demands (i.e., staff-directed, passive patient role) match the individual's preferences.

Some researchers have theorized that difficulties with adherence are related to deficits in self-regulation skills, suggesting that building these skills through interventions focused on self-monitoring, goal-setting, self-reinforcement, and increasing individuals' ability to delay gratification may be an effective strategy. Educational, cognitive, and cognitive behavioral interventions have been cited most frequently in the literature, though the results have been mixed. A recent review of randomized controlled trials designed to improve adherence in hemodialysis patients found that interventions utilizing cognitive or cognitive behavioral techniques showed the largest effects and warrant future research.

Depression and ESRD

Mood disorders have been documented as one of the most common psychiatric diagnoses in patients with ESRD. The prevalence of depression varies based on the type of assessment used; approximately 20–45% of patients endorse symptoms of depression on self-report instruments, and 15–20% may be diagnosed with a depressive disorder following a clinical interview. While depression is recognized in a large number of individuals with ESRD, underdiagnosis and lack of adequate psychological treatment remain significant problems in this population. One of the difficulties in diagnosing depression in ESRD relates to the overlap in somatic depression symptoms with the uremic symptoms of kidney disease. Fatigue, loss of interest in sex, difficulty sleeping, loss of appetite, and problems with concentration and attention could be attributed to both depression and ESRD; thus, the etiology of these symptoms is often unclear. As a result, it has been suggested that assessing the cognitive or nonsomatic symptoms may enable

researchers and clinicians to more accurately identify depression in patients with ESRD.

Several factors have been studied in order to better understand contributors to depression in individuals with kidney disease. The research suggests that perceptions of control and of how intrusive the illness is in disrupting important life domains are related to depression in this population. More specifically, incongruence between beliefs about control or illness intrusiveness and the relevant contextual or situational factors are predictors of depression in ESRD. The effects of social support on moderating depression symptoms have also been examined, and the results have been inconsistent.

Depression has been found to have deleterious consequences for patients with ESRD and earlier stages of CKD, including increased nonadherence to treatment recommendations, morbidity, and mortality. Some research has also suggested that depression is associated with decisions to prematurely terminate dialysis treatment. Thus, adequate treatment of depression in ESRD is essential. A review of the literature suggests that pharmacologic treatment with certain serotonin-selective reuptake inhibitors may be safe and effective for patients with later stage CKD and ESRD. Cognitive behavioral therapy was also found to be one of the most promising interventions for depression in this population. Future behavioral medicine research is necessary to expand our understanding of ESRD, particularly as the prevalence of this chronic illness is projected to increase over time.

Cross-References

- ▶ Adherence
- ▶ Depression
- ▶ Health Behaviors
- ▶ Locus of Control

References and Readings

Christensen, A. J., & Ehlers, S. L. (2002). Psychological factors in end-stage renal disease: An emerging context for behavioral medicine research. *Journal of Consulting and Clinical Psychology, 70*, 712–724. doi:10.1037/0022-006X.70.3.712.

Cvengros, J. A., & Christensen, A. J. (2006). Adherence to dialysis treatment in end-stage renal disease. In W. T. O'Donohue & E. R. Levensky (Eds.), *Promoting treatment adherence: A practical handbook for health care providers* (pp. 331–340). Thousand Oaks, CA: Sage Publications, Inc. ix, 458 pp.

Hedayati, S. S., Yalamanchili, V., & Finkelstein, F. O. (2011). A practical approach to the treatment of depression in patients with chronic kidney disease and end-stage renal disease. *Kidney International* advanced online publication 19 October 2011. doi: 10.1038/ki.2011.358.

Khalil, A. A., & Frazier, S. K. (2010). Depressive symptoms and dietary nonadherence in patients with end-stage renal disease receiving hemodialysis: A review of quantitative evidence. *Issues in Mental Health Nursing, 324–330*. doi:10.3109/01612840903384008.

Mattheson, M. L., & Russell, C. (2010). Interventions to improve hemodialysis adherence: A systematic review of randomized-controlled trials. *Hemodialysis International, 14*, 370–382. doi:10.1111/j.1542-4758.2010.00462.x.

National Kidney Foundation. (2009). *Kidney disease facts*. Retrieved March 7, 2009 from http://www.kidney.org

U.S. Renal Data System, (USRDS). (2009). *Annual data report: Atlas of end-stage renal disease in the United States, National Institutes of Health*. Bethesda, MD: National Institute of Diabetes and Digestive and Kidney Diseases.

Energy

- ▶ Affect Arousal
- ▶ Fatigue

Energy In

- ▶ Caloric Intake

Energy Intake

- ▶ Caloric Intake

Energy: Expenditure, Intake, Lack of

Jennifer Heaney
School of Sport and Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

Definition

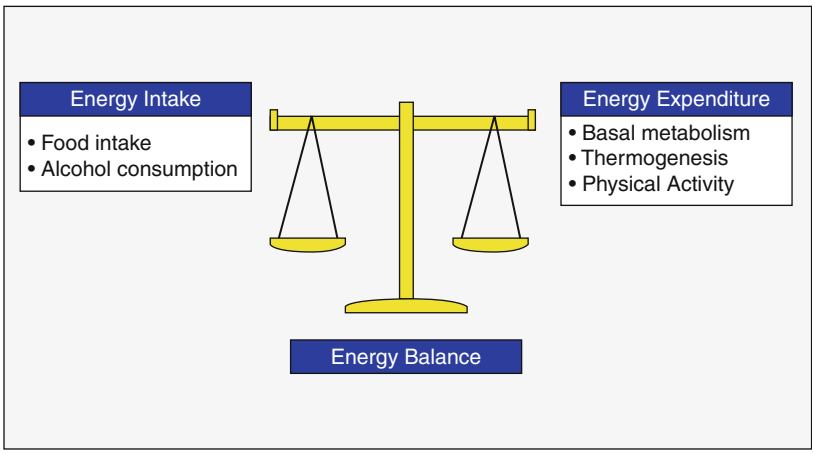
Energy expenditure refers to the amount of energy an individual uses to maintain essential body functions (respiration, circulation, digestion) and as a result of physical activity. Total daily energy expenditure is determined by resting or basal metabolic rate (BMR), food-induced thermogenesis, and energy expended as a result of physical activity.

BMR is the minimum amount of energy that the body requires for essential organ and cellular function when lying in a state of physiological and mental rest. BMR accounts for typically 65–75% of total energy expenditure. Differences in BMR exist between genders and across ages. Females tend to have a lower BMR than males, and BMR decreases with age. These differences can largely be accounted for by differences in fat-free mass, which is proportional to BMR.

Food-induced thermogenesis refers to the increase in energy expenditure following the ingestion of food. This increase in energy expenditure is a result of digestion, absorption, and transportation of nutrients and accounts for approximately 10% of total energy expenditure.

Physical activity refers to energy expended when carrying out everyday tasks and exercise. It typically accounts for 15–30% of energy expenditure, but can vary greatly between individuals. For example, energy expenditure expended through physical activity would be greater in an individual who exercises regularly or is an athlete, compared to someone who is sedentary.

Energy intake is the amount of energy produced by an individual taken in from food consumption; this is typically measured in calories (kcal). Energy intake must be matched with energy expenditure to ensure *energy balance*. If food intake exceeds energy expenditure, through overeating or sedentary behavior, then energy storage occurs resulting in weight gain. This potentially can lead to an individual becoming overweight and at risk of obesity. Alternatively, a negative imbalance can occur where energy expenditure exceeds energy intake. This can occur as a result of undereating, possibly as a result of an eating disorder, or when an individual is involved in a high level of physical activity but failing to match this expenditure with food intake. A negative energy balance subsequently results in weight loss. Although a state of negative energy balance is desirable for overweight individuals in order to lose weight, in the long term if energy intake does not match energy expenditure, this may cause an individual to



Energy: Expenditure, Intake, Lack of, Fig. 1 Energy balance: energy intake should be equal to energy expenditure in order to achieve energy balance

become underweight. The above information has been compiled from the following sources, where more detail of energy expenditure can be found (McArdle et al., 2001) (Fig. 1).

References and Readings

McArdle, W. D., Katch, F. I., & Katch, V. L. (2001). *Exercise physiology: Energy, nutrition and human performance* (5th ed.). Philadelphia: Lippincott Williams & Wilkins.

Widmaier, E. P., Raff, H., & Strang, K. T. (2004). *Vander, Sherman, & Luciano's human physiology: The mechanism of body function*. New York: McGraw-Hill.

Engel, George

Marc D. Gellman
Behavioral Medicine Research Center, Department of Psychology, University of Miami, Miami, FL, USA

Biographical Information

George Libman Engel was born in New York City in 1913. He completed his undergraduate studies in chemistry at Dartmouth College, graduating in 1934. He then studied medicine at the Johns Hopkins University School of medicine, graduating in 1938. He was an intern at Mount Sinai Hospital (New York City), a research fellow at Harvard Medical School, and a graduate assistant in medicine at the Peter Bent Brigham Hospital (now Brigham and Women's Hospital).

In 1942, Engel moved to Cincinnati at the invitation of John Romano, who left Harvard to become the chair of the department of psychiatry at the University of Cincinnati. Both Engel and Romano then moved to the University of Rochester Medical School in 1946.

When commencing his medical career, Engel believed strongly in physical explanations of disease processes, even though some colleagues were incorporating psychosomatics into clinical practice. However, during his time at the University of Cincinnati, he slowly but surely became "converted" to the psychosomatic school. During his career, he became a prominent member of the American Psychosomatic Society, being elected as president and also serving as the editor of its journal, *Psychosomatic Medicine*.

Major Accomplishments

Engel published numerous books and articles on the relation of emotion and disease and on the incorporation of these ideas into medical training and clinical practice. Under his direction, the program at the University of Rochester became a leading center in the development of psychosomatic theory and training. Over time, he developed the "biopsychosocial model," which posits that health and illness are consequences of the interaction of biological, psychological, and social factors. This model was described in his 1977 paper entitled "The need for a new medical model: a challenge for biomedicine," published in the journal *Science*. The abstract of this paper reads as follows:

The dominant model of disease today is biomedical, and it leaves little room within its framework for the social, psychological, and behavioral dimensions of illness. A biopsychosocial model provides a blueprint for research, a framework for teaching, and a design for action in the real world of health care.

The literature is now replete with work addressing the biopsychosocial model, both as a theoretical framework and an approach to clinical practice. As Borrell-Carrio, Suchman, and Epstein (2004) observed, "The biopsychosocial

model is both a philosophy of clinical care and a practical clinical guide. Philosophically, it is a way of understanding how suffering, disease, and illness are affected by multiple levels of organization, from the societal to the molecular. At the practical level, it is a way of understanding the patient's subjective experience as an essential contributor to accurate diagnosis, health outcomes, and humane care."

During the 1980s and 1990s, the biopsychosocial model and biopsychosocial medicine "became the watchword of progressive unification of the medical and behavioral sciences, including psychiatry, in a search for etiological and preventive factors in human health and disease" (Dowling, 2005). Perhaps not surprisingly, given its eminence, various authors since then have suggested modifications and emphasized the importance, too, of other approaches. For example, Kontos (2011) commented that recognizing that medicine is made up of heterogeneous tasks, "no one model, including the biopsychosocial model, tends to all of them." Nonetheless, a quote from Dowling (2005) reviewing Engel's life is an appropriate way to conclude this entry: "He would appreciate the fact that some of us have taken on a bit of his flintiness, attempt his wry humor, and retain his determination to see our patients as 'united, biopsychosocial persons' rather than as 'biomedical persons' divorced from their psychological and social dimensions."

Editors' Note: Dr. Engel passed away in 1999.

Cross-References

► Biopsychosocial Model

References and Readings

Adler, R. H. (2009). Engel's biopsychosocial model is still relevant today. *Journal of Psychosomatic Research, 67*, 607–611.

Borrell-Carrio, F., Suchman, A. L., & Epstein, R. M. (2004). The biopsychosocial model 25 years later: Principles, practice, and scientific inquiry. *Annals of Family Medicine, 2*, 576–582.

Brown, T. M., (2000). The growth of George Engel's biopsychosocial model. Retrieved December 24, 2011, from http://human-nature.com/free-associations/engel1.html

Dowling, A. S. (2005). George Engel, M.D. (1913–1999). *The American Journal of Psychiatry, 162*(11), 2039.

Engel, G. L. (1968). A life setting conducive to illness. *Annals of Internal Medicine, 69*, 293–300.

Engel, G. L. (1977). The need for a new medical model: A challenge for biomedicine. *Science, 196*, 129–136.

Engel, G. L. (1980). The clinical application of the biopsychosocial model. *The American Journal of Psychiatry, 137*, 535–544.

Engel, G. L. (1997). From biomedical to biopsychosocial: Being scientific in the human domain. *Psychosomatics, 38*, 521–528.

Engel, P. A. (2001). George L Engel, MD, 1913–1999: Remembering his life work; Rediscovering his soul. *Psychosomatics, 42*, 94–99.

Frankel, R. M., Quill, T. E., & McDaniel, S. H. (Eds.). (2003). *The biopsychosocial approach: Past, present, future*. Rochester, NY: University of Rochester Press.

Kontos, N. (2011). Perspective: Biomedicine–menace or straw man? Reexamining the biopsychosocial argument. *Academic Medicine, 86*, 509–515.

Engineering Psychology

► Human Factors/Ergonomics

Enteritis Regionalis Crohn

► Crohn's Disease (CD)

Enterocolitis Regionalis

► Crohn's Disease (CD)

Environmental Tobacco Smoke

► Secondhand Smoke

EPA

► Omega-3 Fatty Acids

Epidemiological Studies

▶ Hispanic Community Health Study/Study of Latinos

Epidemiology

G. David Batty
Department of Epidemiology and Public Health, University College London, London, UK

Definition

Derived from the term epidemic, epidemiology is the study of the distribution and determinants of health-related states or events, particularly disease (communicable or noncommunicable). The discipline of epidemiology is eclectic, comprising facets of sociology, statistics, medicine, and demography. Although its development may be traced back to the scientific revolution of the 1600s, it was not until the nineteenth century that it was recognized as a subject area in its own right.

The goal of the discipline of epidemiology is disease control and prevention. As such, various methods can be used to carry out epidemiological investigations: descriptive studies (usually based on a cross-sectional design) can be used to investigate distribution of disease; analytical studies (e.g., case control, cohort, randomized controlled trial) can be used to examine determinants of disease.

Knowledge of the occurrence, etiology, and subsequent control of communicable diseases such as typhoid fever, smallpox, and cholera stemmed from early epidemiological studies, such as John Snow's investigation of the infamous 1854 cholera epidemic in London. The emergence of noncommunicable diseases such as coronary heart disease earlier last century led to epidemiology occupying a wider remit. Modifiable determinants of the growing epidemic of coronary heart disease were identified from large-scale epidemiological investigations beginning with the Framingham, the Seven Countries and Whitehall cohort studies.

Today, a growing body of professionals from health education, environmental and occupational health, and health service administration, in addition to medical science students, are required to have some knowledge of the foundations of epidemiology. Further details can be found in Hennekens and Buring (1987) and Rothman (2002).

Cross-References

▶ Medical Sociology
▶ Mortality
▶ Mortality Rates
▶ Occupational Health
▶ Social Epidemiology

References and Readings

Hennekens, C. H., & Buring, J. E. (1987). *Epidemiology in medicine*. Philadelphia: Lippincott Williams & Wilkins.
Rothman, K. J. (2002). *Epidemiology: An introduction*. Oxford, NY: Oxford University Press.

Epigenetics

Alicia K. Smith[1] and James W. Schroeder[2]
[1]Psychiatry & Behavioral Sciences, Emory University SOM, Atlanta, GA, USA
[2]Genetics and Molecular Biology Program, Emory University, Atlanta, GA, USA

Definition

Epigenetics is the study of changes in gene expression that cannot be attributed to variation in DNA sequence. The etymology of this term comes from the Greek "epi-," meaning "above" genetics and refers to covalent modifications

of the DNA, its associated proteins, or mRNA transcripts.

Description

All somatic cells within an organism contain the same DNA sequence, but epigenetic patterns regulate the timing and magnitude of gene expression by restricting the areas of the genome available for transcription and translation. This allows cells with the same genome to differentiate into specialized cells that perform a variety of functions (Jaenisch & Bird, 2003).

Epigenetic regulation participates in vital developmental processes. For example, one of the two X chromosomes in each cell of a female is permanently silenced through a series of epigenetic changes in a process called X-chromosome inactivation (Jaenisch & Bird, 2003). Epigenetic mechanisms also regulate genomic imprinting, a process in which an organism's parents contribute distinct epigenetic patterns that result in expression of only the maternally or paternally derived alleles in their offspring. Failure of these regulatory mechanisms can lead to developmental disorders such as Prader-Willi Syndrome or Angelman Syndrome (Feinberg, 2007).

Previously established epigenetic patterns responsible for cellular differentiation, X-chromosome inactivation, and imprinting are generally maintained through mitosis. However, some aspects of the epigenome are labile such that they may respond to environmental conditions and change over the course of an organism's lifespan (Feinberg, 2007).

DNA interacts with packaging proteins known as histones, which can be posttranslationally modified and facilitate dynamic gene regulation. Both the core (H2A, H2B, H3, and H4) and linker (H1 and H5) histones can be modified through methylation, acetylation, phosphorylation, ubiquitination, sumoylation, or citrullination. Each element of this histone code has a specific function. For example, histone acetylation typically promotes gene transcription while histone methylation can promote or repress transcription based on the where it occurs (Bannister & Kouzarides, 2011).

Histone modifications often correspond to changes in methylation of DNA at the 5′ position of the pyrimidine ring of cytosines within CpG dinucleotides (also called CpG sites). DNA methylation is the most widely studied epigenetic modification. It is concentrated in repetitive elements of the genome such as Alu sequences to repress transcription of latent retroviral elements. CpG sites are overrepresented in the promoter region of many genes, and when they cluster in sufficient density, the region is called a CpG island. Methylation of cytosines regulates gene expression by influencing the recruitment and binding of regulatory proteins to DNA. Specifically, gene expression typically increases when CpG methylation of that gene decreases and vice versa (Jaenisch & Bird, 2003).

Epigenetic regulation of gene expression can also be accomplished by a variety of non-protein coding RNA molecules (ncRNAs), which are continuously being discovered and characterized. RNA interference (RNAi) is a process by which ncRNA molecules bind to messenger RNA (mRNA) to regulate its translation into protein. As part of this process, small microRNA (miRNA) can bind a complementary strand of mRNA and repress its expression by targeting it for degradation or by directly preventing its translation. Similarly, small interfering RNA (siRNA) promotes mRNA cleavage and posttranscriptional silencing of a gene through induction of the RNA-induced silencing complex known as RISC (Taft, Pang, Mercer, Dinger, & Mattick, 2010).

Many epigenetic changes can occur over the course of an organism's lifetime as part of normal development, randomly as the organism ages or in response to environmental insults. However, if epigenetic changes occur in germ cells that participate in fertilization, epigenetic changes can be inherited from one generation to the next and may persist through multiple generations. With these and other recent discoveries, the role of epigenetic mechanisms in health and disease is being illustrated (Richards, 2006).

Cross-References

- ▶ DNA
- ▶ Gene Expression
- ▶ Methylation
- ▶ RNA

References and Readings

Bannister, A. J., & Kouzarides, T. (2011). Regulation of chromatin by histone modifications. *Cell Research, 21*(3), 381–395.

Feinberg, A. P. (2007). Phenotypic plasticity and the epigenetics of human disease. *Nature, 447*(7143), 433–440.

Jaenisch, R., & Bird, A. (2003). Epigenetic regulation of gene expression: How the genome integrates intrinsic and environmental signals. *Nature Genetics, 33*(Suppl), 245–254.

Richards, E. J. (2006). Inherited epigenetic variation–revisiting soft inheritance. *Nature Reviews Genetics, 7*(5), 395–401.

Taft, R. J., Pang, K. C., Mercer, T. R., Dinger, M., & Mattick, J. S. (2010). Non-coding RNAs: Regulators of disease. *The Journal of Pathology, 220*(2), 126–139.

www.ncbi.nlm.nih.gov/epigenomics/

Epinephrine

George J. Trachte
Academic Health Center, School of Medicine-Duluth Campus, University of Minnesota, Duluth, MN, USA

Synonyms

Adrenaline

Definition

Epinephrine is a major neurotransmitter of the sympathetic nervous system. Epinephrine partially mediates the body's reaction to stress by elevating heart rate, blood vessel tone, sweating, tremor, and blood pressure. Epinephrine is released primarily from the central region (medulla) of the adrenal gland in response to stressful situations. An alternate name for epinephrine is adrenaline.

Epinephrine interacts with at least five major protein receptors to produce a plethora of biological responses, typically characterized by an elevation of blood pressure and mobilization of energy stores. The major receptor interactions are with the following: α1 to both constrict blood vessels, resulting in increased vascular resistance, and an elevation of blood pressure and activate sweat glands to promote nervous sweating; α2 receptors to reduce the release of other catecholamines, such as norepinephrine, but also to constrict blood vessels; ß1 receptors to elevate heart rate and renin secretion, both resulting in elevated blood pressure; ß2 receptors on smooth muscle, particularly in bronchioles to ease breathing, and on skeletal muscle to sensitize the muscle to other stimuli resulting in tremor; and ß3 receptors on adipose tissue to stimulate the breakdown of fat stores. The stimulation of both α1 and ß2 receptors also mobilizes energy stores by activating glycogenolysis.

The physiological actions of epinephrine primarily involve augmentation of the sympathetic nervous system to promote rapid heart rate, arterial constriction, higher blood pressure, mobilization of fuel stores, sweating, and dilation of bronchioles and pupils of the lung and eye, respectively.

Epinephrine is a catecholamine synthesized from norepinephrine primarily in the central portion (medulla) of the adrenal gland. The entire synthetic pathway involves conversion of an amino acid, tyrosine, to dihydroxyphenylalanine (DOPA), followed by conversion of DOPA to dopamine, and dopamine to norepinephrine and the final step is the conversion of norepinephrine to epinephrine. The enzymes involved in the pathway are the following: tyrosine hydroxylase, DOPA decarboxylase, dopamine ß hydroxylase, and phenylethanolamine *N*-methyl transferase. The latter strictly controls the synthesis of epinephrine and is most abundant in the adrenal medulla.

In addition to the adrenal medulla, other tissues capable of synthesizing epinephrine are the following: brain stem, retina, and left atrium of the heart. The distinguishing feature of the tissues

synthesizing epinephrine is the presence of phenylethanolamine *N*-methyl transferase. The gene controlling synthesis of this enzyme is located on chromosome 17 in humans. The enzyme consists of 282 amino acids and has a molecular mass of 30,835 g.

The primary behavioral role for epinephrine involves mediation of stress responses such as tachycardia, vasoconstriction, hypertension, sweating, shaking, piloerection, and liberation of energy stores such as glucose and fatty acids. Epinephrine levels in the brain are depleted in Alzheimer's disease brains. Epinephrine has been shown to increase memory in humans but the effect is mimicked by exogenous epinephrine infusions which cannot penetrate the brain; therefore, the effect must be caused by peripheral actions of epinephrine and not central brain effects. Intracerebral epinephrine infusions in animals also can produce excitation or depression.

Cross-References

- ▶ Blood Pressure
- ▶ Catecholamines
- ▶ Heart Rate
- ▶ Norepinephrine/Noradrenaline

Epistasis

- ▶ Gene-Gene Interaction

Epstein-Barr Virus

Deidre Pereira[1] and Seema M. Patidar[2]
[1]Department of Clinical and Health Psychology, University of Florida, College of Public Health and Health Professions, Gainesville, FL, USA
[2]Department of Clinical and Health Psychology, University of Florida, Gainesville, FL, USA

Synonyms

EBV; Human herpesvirus-4 (HHV-4); Kissing disease; "Mono" or mononucleosis

Definition

EBV was formally identified by M.A. Epstein and Y.M. Barr in 1964 while examining tissue from a Burkitt's lymphoma patient. EBV is a very common type of herpesvirus found in humans. About 95% of Americans will have contracted the virus by the age of 40. The virus is highly contagious and is difficult, if not impossible, to prevent. It is often called the "kissing disease" due to its ease of transmission between individuals through saliva. Children often acquire EBV through close contact with family members and the people around them who are likely to have the virus. In adolescents and young adults, EBV leads to infectious mononucleosis 30–50% of the time. Symptoms of active EBV, or infectious mononucleosis, commonly include swollen lymph nodes, swollen throat, and fever. Treatment is limited and focuses on minimizing symptoms of the infection. The incubation period from contraction to presentation of symptoms can range from 4 to 6 weeks. Symptoms of infectious mononucleosis can last up to 1–2 months, but EBV remains dormant in the body for the rest of a person's life. EBV is present in the saliva and blood of infected persons and remains in some bodily cells after contraction. Since it functions as a virus, the body will develop antibodies to help fight off the virus. A "mono spot" test, which looks for these antibodies, is often administered for a formal diagnosis. Additionally, an elevated white blood cell count is indicative of active infection in the body. At times of immunosuppression, such as during cancer treatment, patients may experience reactivation of the virus, with or without associated symptoms. In some people, EBV may play a role in the development of Burkitt's lymphoma and nasopharyngeal carcinoma. Other people may live with the latent form of EBV for a number of years without reactivation.

References and Readings

Centers for Disease Control and Prevention and National Center for Infectious Diseases (2006, May 16). *Epstein-Barr virus and infectious mononucleosis*. Retrieved February 28, 2011, from http://www.cdc.gov/ncidod/diseases/ebv.htm

Epstein, M. A., & Achong, B. G. (1979). *The Epstein–Barr virus*. Berlin: Springer.
The Patient Education Institute, Inc. (1995–2008). *X-Plain: Epstein–Barr virus/mono* [Last reviewed on November 9, 2007]. Retrieved February 28, 2011, from http://www.nlm.nih.gov/medlineplus/tutorials/epsteinbarrvirusmono/id299103.pdf

Equilibrium

▶ Homeostasis

Equipoise

▶ Principle of Equipoise

Erectile Dysfunction

Catherine Benedict
Department of Psychology, University of Miami, Coral Gables, FL, USA

Synonyms

Impotence

Definition

Erectile dysfunction is a sexual dysfunction characterized by the consistent inability to develop or maintain an erection of the penis firm enough for satisfactory sexual performance. Symptoms of erectile dysfunction include trouble getting an erection, trouble keeping an erection, and/or reduced sexual desire.

Description

In the United States, it is estimated that erectile dysfunction affects 20–30 million men. The prevalence of erectile dysfunction of any degree is estimated to be approximately 39% in men 40 years old and up to 78% in men 75 years and older. Comorbid medical conditions such as obesity, diabetes, heart disease, or hypertension may increase the risk of developing erectile dysfunction. In men older than 50 years, approximately 40% of erectile dysfunction is due to atherosclerotic complications. The most common conditions associated with the development of erectile dysfunction include cigarette smoking, high blood pressure, lipid problems (cholesterol, triglycerides), and diabetes. Among diabetes patients, the prevalence of erectile dysfunction is approximately 50%, depending on age, duration, and severity of the diabetes. A high prevalence of erectile dysfunction is also observed with chronic renal failure, hepatic failure, sleep apnea, chronic obstructive pulmonary disease, multiple sclerosis, Alzheimer's disease, and endocrine disorders such as low testosterone and thyroid problems. Pelvic or perineal trauma such as pelvic surgery (major prostate, bladder, and bowel operations) and pelvic radiation therapy are associated with erectile dysfunction, as is direct trauma to the perineum, which can lead to vascular problems.

Pathophysiology

Erectile dysfunction can be classified as psychogenic, organic, or mixed psychogenic and organic. That is, psychologic, neurologic, hormonal, arterial, or cavernosal impairment factors alone or in combination may cause erectile dysfunction. The mixed psychogenic and organic form of erectile dysfunction is the most common. Common psychological factors include performance anxiety and personal and/or relationship distress, which often result in lack of sexual arousal, overinhibition, and decreased libido. Erectile dysfunction may occur despite experiencing sexual desire and maintaining the ability to have an orgasm and ejaculate. Psychiatric disorders such as depression and schizophrenia have been related to increased risk of erectile dysfunction. Neurogenic causes of erectile dysfunction include the presence of disorders such as Parkinson's disease, Alzheimer's disease,

stroke, and cerebral trauma, which often lead to decreased libido or failure to initiate nerve impulses or interrupted neural transmission that lead to an inability to develop an erection. Hormonal factors associated with erectile dysfunction include hypogonadism and hyperprolactinemia. Androgen deficiency may also result in loss of libido and decreased nocturnal erections. Vasculogenic factors include generalized penile arterial insufficiency and veno-occlusive dysfunction. Inadequate arterial flow may be the result of hypertension, hyperlipidemia, cigarette smoking, diabetes, and pelvic or perineal trauma. Impaired veno-occlusion is associated with old age, Peyronie's disease, structural damage to the cavernous muscle and endothelium, and poor relaxation of the trabecular muscle, as well as diabetes and pelvic or perineal trauma. Drug-induced erectile dysfunction may result from a number of antipsychotic, antidepressant, and antihypertensive drugs that affect central neurotransmitter pathways involving serotonin, androgen, and dopamine. Additionally, cigarette smoking is associated with vasoconstriction and penile venous leakage and chronic alcohol abuse is associated with hypogonadism and polyneuropathy. Finally, old age is associated with a progressive decline in overall sexual function such that older men report decreased penile sensitivity, decreased testosterone levels, less turgid erections, less forceful erections, decreased ejaculation volume, and lengthened refractory period between erections. Age-related declines may be exacerbated with comorbid medical conditions such as diabetes, coronary heart disease, and chronic renal failure that lead to neural and/or vascular dysfunction.

Diagnosis

Initial evaluation of a man presenting with erectile difficulties includes a thorough examination of medical, sexual, and psychosocial histories, physical examination, and appropriate laboratory tests. Psychosexual factors to consider include alterations of sexual desire, ejaculation, and orgasm, presence of genital pain, and lifestyle factors, such as sexual orientation, presence of spouse or partner, and quality of the relationship with the partner. Risk factors including smoking, trauma, or surgery to the pelvic, perineal, or penile areas, and prescription or recreational drug use should be considered. A detailed medical history should be taken and evaluation may also include laboratory tests to detect and rule out medical conditions that may be the cause of or comorbid with erectile difficulties and that may contraindicate certain therapies. Testing of testosterone levels, vascular and/or neurologic functioning, and monitoring of nocturnal erections may also be indicated in some patients. A physical examination should be conducted of the abdomen, penis, testicles, secondary sexual characteristics, and lower extremity pulses. The purpose of the initial evaluation is to identify psychosocial dysfunctions and organic comorbidities that contribute to erectile dysfunction. Assessment of patient's (and partner's) goals of treatment and preferences should also be conducted.

Treatment

Treatment of erectile dysfunction should address all the contributing factors associated with erectile difficulties. Appropriate treatment options should be utilized in a stepwise fashion according to medical expertise and patient preference. Healthcare professionals should carefully assess patients' (and their partners') goals for treatment; patients should be made aware of the risk involved with increasingly invasive treatments so that well-informed decisions are made with regard to the likelihood of treatment efficacy.

Cross-References

► Aging

References and Readings

American Urological Association. (2006). *Management of erectile dysfunction*. Retrieved from http://www.auanet.org/guidelines

Bacon, C. G., Mittleman, M. A., Kawachi, I., Giovannucci, E., Glasser, D. B., & Rimm, E. B. (2003). Sexual function in men older than 50 years of

age: Results from the health professionals' follow-up study. *Annals of International Medicine, 139*(3), 161–168.
Benet, A. E., & Melman, A. (1995). The epidemiology of erectile dysfunction. *Urology Clinic of North America, 22*, 699–709.
Eid, J. F., Nehra, A., Andersson, K. E., Heaton, J., Lewis, R. W., Morales, A., et al. (2000). First international conference on the management of erectile dysfunction overview consensus statement. *International Journal of Impotence Research, 12*(Suppl. 4), S2–S5.
Feldman, H. A., Goldstein, I., Hatzichristou, D. G., Krane, R. J., & McKinlay, J. B. (1994). Impotence and its medical and psychosocial correlates: Results of the Massachusetts male aging study. *Journal of Urology, 151*, 54–61.
Johannes, C. B., Araujo, A. B., Feldman, H. A., Derby, C. A., Kleinman, K. P., & McKinlay, J. B. (2000). Incidence of erectile dysfunction in men 40 to 69 years old: Longitudinal results from the Massachusetts male aging study. *Journal of Urology, 163*(2), 460–463.
Kloner, R. A., Mullin, S. H., Shook, T., Matthews, R., Mayeda, G., Burstein, S., et al. (2003). Erectile dysfunction in the cardiac patient: How common and should we treat? *Journal of Urology, 170*, S46–S50.
Lizza, E. F., & Rosen, R. C. (1999). Definition and classification of erectile dysfunction: Report of the nomenclature committee of the International Society of Impotence Research. *International Journal of Impotence Research, 11*, 141–143.
Saigal, C. S., Wessells, H., Pace, J., Schonlau, M., Wilt, T. J., Urologic Diseases in America Project, et al. (2006). Predictors and prevalence of erectile dysfunction in a racially diverse population. *Archives of International Medicine, 166*(2), 207–212.

Ergonomics

► Human Factors/Ergonomics

Ergotherapist

► Therapy, Occupational

Ergotherapy

► Occupational Therapy

Escape-Avoidance Coping

Urs M. Nater
Department of Psychology, University of Marburg, Marburg, Germany

Definition

Coping is a cognitive-behavioral process that takes place in the context of a situation or condition perceived as personally relevant, challenging, or that exceeds an individual's resources to adequately deal with a problem. Coping styles may be dysfunctional or maladaptive in various contexts. Particularly in various patient groups, it has been shown that patients tend to use significantly more maladaptive strategies than healthy controls. Maladaptive coping styles have been shown to be associated with clinical features (e.g., fatigue, impairment, illness burden, psychosocial problems, or psychiatric comorbidity).

One of the best examined maladaptive coping styles is escape-avoidance coping. Escape-avoidance coping involves disengaging or staying away from a stressful situation and its behavioral and cognitive/emotional consequences. Typical strategies in response to a stressful situation might encompass cognitive avoidance ("Refused to believe that it had happened"), avoidant actions ("Slept more than usual"), denial ("Refused to believe that it had happened"), or wishful thinking ("Wished that the situation would go away or somehow be over with it") (examples are items of the Ways of Coping Checklist which is the most widely used instrument for assessment of coping styles).

It should be noted that, according to Lazarus, coping strategies are not inherently adaptive or maladaptive, but their effectiveness depends on individuals' personal circumstances, goals, and expectations. Coping styles should be considered in the context of stress-related cognitions and their consequences in everyday life. Escape-avoidant coping in a stress context may result in an inadequate regulatory adaptation to stress

as well as in exaggerated or prolonged stress responses that may in turn be associated with increased neuroendocrine, autonomic and immune activation. Escape-avoidant coping (and other coping styles) must be considered in studies of risk factors, clinical course, pathophysiology, and therapy of illnesses relevant in behavioral medicine.

References and Readings

Folkman, S., & Lazarus, R. (1988). *Manual for the ways of coping questionnaire*. Palo Alto, CA: Consulting Psychologists Press.

Folkman, S., & Moskowitz, J. T. (2004). Coping: Pitfalls and promise. *Annual Review of Psychology, 55*, 745–774.

Lazarus, R. S. (1993). Coping theory and research: past, present, and future. *Psychosomatic Medicine, 55*(3), 234–247.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.

Escitalopram

► Selective Serotonin Reuptake Inhibitors (SSRIs)

ESM

► Experience Sampling

Essential Fatty Acids

Sheah Rarback
Department of Pediatrics, University of Miami, Miami, FL, USA

Synonyms

Dietary fatty acids

Definition

Essential fatty acids (EFA) are fats that cannot be synthesized by the body and must be obtained through diet. The two types are omega-3 fatty acids from alpha linolenic acid and omega-6 fatty acid from linoleic acid. Omega-9 fatty acid is necessary yet "nonessential" because the body can manufacture a small amount on its own, provided essential EFAs are present. EFAs are used to support the cardiovascular, reproductive, immune, and nervous systems.

The body has a very limited capacity for making the omega-3 fatty acids eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) from linolenic acid, so these are often classified with essential fatty acids. EPA is believed to play a role in the prevention of cardiovascular disease, while DHA is necessary for proper brain and nerve development.

The richest sources of omega-6 fatty acids are safflower, sunflower, corn, and sesame oil. The richest sources of omega-3 fatty acids are flaxseed, sardines, salmon, cooked soybeans, and halibut.

The current ratio of omega-6 to omega-3 fatty acids in the typical American diet is approximately 11:1. However, the recommended ratio is 4:1. High doses of supplemental EPA/DHA have been shown to lower triglycerides in patients with elevated triglycerides. The American Heart Association recommends two servings of fatty fish a week to increase intake of EPA and DHA.

Cross-References

► Fat, Dietary Intake
► Fat: Saturated, Unsaturated

References and Readings

Aterburn, A., Hall, E., & Oken, H. (2006). Distribution, interconversion, and dose response of omega-3 fatty acids in human. *American Journal of Clinical Nutrition, 83*, S1467–S1476.

Kris-Etherton, P. M., Innis, S., Ammerican Dietetic Assocition, & Dietitians of Canada. (2007). Position of the American Dietetic Association and Dietitians of Canada: Dietary fatty acids. *Journal of the American Dietetic Association, 107*, 1599.e1–1599.e15.

Tribole, E. (2007). *The ultimate omega-3 diet: Maximize the power of omega-3s to supercharge your health, battle inflammation, and keep your mind sharp*. New York: McGraw-Hill.

www.heart.org

Estrogen

Maurizio Cutolo
Department of Internal Medicine, Research Laboratories and Academic Unit of Clinical Rheumatology, University of Genova, Genova, Italy

Synonyms

Gonadal female hormones; Sex hormones; Steroid hormones

Definition

Estrogens represent a group of steroid hormones that primarily influence the female reproductive tract in its development, maturation, and function. There are three major hormones – estradiol, estrone, and estriol – and estradiol is the predominant one (Blair, 2010).

The major sources of estrogens are the ovaries and the placenta (the temporary organ that serves to nourish the fetus and remove its wastes); additional small amounts are secreted by the adrenal glands, by the male testes, and by intracrine synthesis in several peripheral cells/tissues (i.e., adipose tissue, which is also an important source of estrogen in postmenopausal women; macrophages in inflamed tissues) (Cutolo et al., 2004; Simpson, 2003).

Cholesterol is the parent molecule from which all ovarian steroid hormones are formed. Cholesterol is converted to pregnenolone, and pregnenolone is converted to progesterone. The steps in the conversion of progesterone to the main estrogens – estradiol and estrone – include the intermediate formation of several androgens (male sex hormones): dehydroepiandrosterone, androstenedione, and testosterone (Blair, 2010).

Practically, androgens are precursors of estrogens: they are converted to estrogens through the action of an enzyme known as aromatase (Chumsri, Howes, Bao, Sabnis, & Brodie, 2011). The ovaries are the richest source of aromatase. Estradiol, the most potent estrogen, is synthesized from testosterone. Estrone can be formed from estradiol, but its major precursor is androstenedione. Estriol, the weakest of the estrogens, is formed from both estrone and estradiol.

At its target tissues, the free hormone penetrates the cell surface and then binds to a protein known as an estrogen receptor in the cytoplasm of the cells (Gibson & Saunders, 2012). The estrogen-receptor complexes enter the cell nucleus, where they influence the rate at which particular genes are transcribed.

Recently, chemicals like xenoestrogens, which can mimic endogenous hormones or interfere with endocrine processes, may affect normal estrogen signaling (Singleton & Khan, 2003).

Bone metabolic actions of estrogens are related to bone development and bone maintenance including the stimulation of bone formation and the closure of bone epiphyses, which causes linear growth to cease at the end of puberty, and the maintenance of bone throughout the reproductive years, which limits bone resorption and preserves bone strength (Callewaert, Sinnesael, Gielen, Boonen, & Vanderschueren, 2010).

However, estrogen replacement therapy to treat menopause is not recommended, since The National Cancer Institute found in 2003 a very significant drop in the rate of hormone-dependent breast cancers among women, related to the fact that millions of women stopped taking

hormone therapy in 2002 after the results of a major government study found the treatment slightly increased a woman's risk for breast cancer, heart disease, and stroke (Rossouw et al., 2002).

Estrogens exert enhancing activities on the immune humoral response (B-cell activities) and are considered among risk factors involved in the higher frequency of autoimmune diseases in females (Schmidt et al., 2009). Estrogen administration is contraindicated in patients with active autoimmune diseases (i.e., systemic lupus erythematosus); therefore, progestogen-only pills offer a convenient and readily reversible method of contraception that is suitable for women with contraindications for estrogens (Ahrendt, Adolf, & Buhling, 2010).

Increased intracrine synthesis (action of local aromatases) and levels of peripheral estrogen metabolites (i.e., alpha-hydroxylated) are observed in fluids of patients affected by autoimmune diseases (i.e., synovial fluid of rheumatoid arthritis patients of both sexes) and in tissues affected by cancer (i.e., both breast and prostate cancer) (Cutolo, Sulli, & Straub, 2011; Cutolo, Straub, & Bijlsma, 2007; Nelles, Hu, & Prins, 2011).

Cross-References

► Immune Function

References and Readings

Ahrendt, H. J., Adolf, D., & Buhling, K. J. (2010). Advantages and challenges of oestrogen-free hormonal contraception. *Current Medical Research and Opinion, 26*, 1947–1955.

Blair, I. A. (2010). Analysis of estrogens in serum and plasma from postmenopausal women: past present, and future. *Steroids, 75*, 297–306.

Callewaert, F., Sinnesael, M., Gielen, E., Boonen, S., & Vanderschueren, D. (2010). Skeletal sexual dimorphism: relative contribution of sex steroids, GH – IGF1, and mechanical loading. *Journal of Endocrinology, 207*, 127–134.

Chumsri, S., Howes, T., Bao, T., Sabnis, G., & Brodie, A. (2011). Aromatase, aromatase inhibitors, and breast cancer. *The Journal of Steroid Biochemistry and Molecular Biology, 125*, 13–22.

Cutolo, M., Straub, R. H., & Bijlsma, J. W. (2007). Neuroendocrine-immune interactions in synovitis. *Nature Clinical Practice Rheumatology, 3*, 627–634.

Cutolo, M., Sulli, A., & Straub, R. H. (2011). Estrogen metabolism and autoimmunity. *Autoimmunity Reviews*. [Epub ahead of print].

Cutolo, M., Villaggio, B., Seriolo, B., Montagna, P., Capellino, S., Straub, R. H., et al. (2004). Synovial fluid estrogens in rheumatoid arthritis. *Autoimmunity Reviews, 3*, 193–198.

Gibson, D., & Saunders, P. T. (2012). Estrogen dependent signaling in reproductive tissues – A role for estrogen receptors and estrogen related receptors. *Molecular and Cellular Endocrinology, 348*(2), 361–372.

Key, T. J., Appleby, P. N., Reeves, G. K., Roddam, A. W., Helzlsouer, K. J., Alberg, A. J., et al. (2011). Circulating sex hormones and breast cancer risk factors in postmenopausal women: reanalysis of 13 studies. *British Journal of Cancer, 105*(5), 709–722. Endogenous Hormones and Breast Cancer Collaborative Group.

Nelles, J. L., Hu, W. Y., & Prins, G. S. (2011). Estrogen action and prostate cancer. *Expert Review of Endocrinology and Metabolism, 6*, 437–451.

Ostensen, M., Brucato, A., Carp, H., Chambers, C., Dolhain, R. J., Doria, A., et al. (2011). Pregnancy and reproduction in autoimmune rheumatic diseases. *Rheumatology (Oxford, England), 50*, 657–664.

Rossouw, J. E., Anderson, G. L., Prentice, R. L., Writing Group for the Women's Health Initiative Investigators, et al. (2002). Risks and benefits of estrogen plus progestin in healthy postmenopausal women: principal results from the Women's Health Initiative randomized controlled trial. *Journal of the American Medical Association, 288*(3), 321–333.

Schmidt, M., Hartung, R., Capellino, S., Cutolo, M., Pfeifer-Leeg, A., & Straub, R. H. (2009). Estrone/17beta-estradiol conversion to, and tumor necrosis factor inhibition by, estrogen metabolites in synovial cells of patients with rheumatoid arthritis and patients with osteoarthritis. *Arthritis and Rheumatism, 60*, 2913–2922.

Simpson, E. R. (2003). Sources of estrogen and their importance. *The Journal of Steroid Biochemistry and Molecular Biology, 86*, 225–230.

Singleton, D. W., & Khan, S. A. (2003). Xenoestrogen exposure and mechanisms of endocrine disruption. *Frontiers in Bioscience, 8*, s110–s118.

Straub, R. H. (2007). The complex role of estrogens in inflammation. *Endocrine Reviews, 28*, 521–574.

Ethical Issues

Yoshiyuki Takimoto
Department of Stress Science and Psychosomatic Medicine, Graduate School of Medicine, The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Synonyms

Clinical ethics

Definition

Ethical issues arise in the situation where values conflict. There are several ethical issues in the area of behavioral medicine. One of typical ethical issues is the problem of informed consent in the clinical settings and the research involving human subjects.

Ethics of behavioral therapy is a topic of ethical issues in behavioral medicine to be proposed. Ethics has been a priority among behavior therapists. If the application of a technique can inflict pain or clients are relatively powerless or are involuntarily the subjects of treatment, ethical concerns arise. The aversion technique is one of major techniques causing behavioral modification. However, using an aversion procedure becomes one focus of ethical criticism in behavioral therapy. In the case that clients cannot offer informed consent due to lack their competency, desirability of treatment outcome goals has to be weighed against the rights of the client, because using an aversion technique opposes nonmalficience which is major principle of biomedical ethics. Behavior therapists ethically ought to give positive consideration to reduce the target behavior through nonaversive means before applying an aversion procedure. Only when the target behavior has been conclusively shown to be impervious to other means, aversion therapy should be used.

Cross-References

▶ Informed Consent

Ethics

Yoshiyuki Takimoto
Department of Stress Science and Psychosomatic Medicine, Graduate School of Medicine, The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Definition

Ethics is the study or examination of morality and moral life. The concepts of ethics fall into two main categories. The first category comprises notions having to do with morality, virtue, rationality, and other ideals or standards of conduct and motivation; second, notions pertaining to human good or well-being and the "good life" generally. Several major approaches to the study of morality are encompassed under the broad term "ethics." Perhaps, the best-known approach is normative ethics, which attempts to identify those moral norms, values, or traits that should be accepted as standards or guides for moral behavior and moral judgment. Famous ethical theories of normative ethics are deontology, consequentialism, or virtue ethics. Deontology treats moral obligations as requirements that bind us to act, in large measure, independent of the effects our actions may have on our own good or well-being and, to a substantial extent, even independent of the effects of our actions on the well-being of others. Consequentialism contrasts with deontology. In consequentialism, all moral obligation and virtue are to be understood in terms of good or desirable consequences. Virtue ethics is conceiving what is admirable about individuals in terms of traits of character, rather than in terms of individual obedience to set of moral or ethical rules or requirement.

Cross-References

- ▶ Ethical Issues
- ▶ Informed Consent

References and Readings

Mithcam, C. (2005). Ethics. In *Encyclopaedia of science, technology, and ethics* (pp. 700–704). Detroit: Macmillan Reference.

Slote, M. A. (2003). Ethics. In S. G. Post (Ed.), *Encyclopedia of bioethics* (3rd ed., pp. 795–802). New York: Macmillan Reference.

Ethics Committee

Jane Upton
School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Research ethics committee

Definition

An ethics committee is a committee dedicated to the rights and well-being of research subjects and makes decisions regarding whether or not proposed research studies are ethical to permit to go ahead.

The emphasis on ethical research arose out of concern regarding unethical experiments on humans that occurred during the Second World War. This led to the "Nuremberg Code," which continues to inform current day ethics statements. This includes the ten basic principles that must be observed when performing medical experiments in order to satisfy moral, ethical, and legal concepts (BMJa, 1996). The Nuremberg Code was informed the Declaration of Helsinki, which was devised in 1964 in order to meet the needs of the biomedical community (BMJb, 1996). This has been revised six times; the most recent version was written in 2008. The Declaration of Helsinki stipulates that research protocols should be reviewed by specially appointed committee independent of the investigator and the sponsor. It also stipulates that research that does not directly benefit the patient is restricted to healthy volunteers or for individuals where the experimental design is not related to their illness. Importantly, it also states that any research that does not have ethical approval should not be accepted for publication. Researchers are now increasingly assessed for Good Clinical Practice as developed by the International Conference on Harmonization (http://www.ich.org/home.html), which developed from the Declaration of Helsinki.

In the UK, most research concerning human subjects is required to be approved by ethics committees prior to being conducted. This is overseen by the National Research Ethics Service (http://www.nres.npsa.nhs.uk/), which has the following dual mission:

- To protect the rights, safety, dignity, and well-being of research participants
- To facilitate and promote ethical research that is of potential benefit to participants, science, and society

A list of national ethics committees specializing in the ethical aspects of the life sciences, biotechnology, agriculture, food safety, and health can be found at: http://ec.europa.eu/research/biosociety/bioethics/bioethics_ethics_en.htm.

Links for key documents relating to ethics committees can be found at the following site: http://www.privireal.org/content/rec/documents.php.

References and Readings

BMJa. (1996). Nuremberg. *BMJ*, *313*, 1448. BMJ. Retrieved from http://www.bmj.com/content/313/7070/1448.1.full

BMJb. (1996). Nuremberg: Declaration of Helsinki (1964). *BMJ*, *313*, 1448. Retrieved from http://www.bmj.com/content/313/7070/1448.2.extract

Ethnic Differences

▶ Minority Subgroups

Ethnic Identities and Health Care

▶ Health Disparities

Ethnic Identity

▶ Ethnicity

Ethnic Minorities

▶ Ethnicity

Ethnicity

Kristine M. Molina and
Orit Birnbaum-Weitzman
Department of Psychology, University of Miami, Miami, FL, USA

Synonyms

Ethnic identity; Subethnic groups

Definition

Although there is no standardized definition for ethnicity, it has generally been agreed upon that ethnicity encompasses, but is not limited to, elements such as shared cultural background, customs and practices, values and norms, and common language or religious traditions that are usually maintained across generations and tied to a collective sense of identity (Dein, 2006; Lee, 2009). Moreover, ethnicity is a complex and dynamic concept that is dependent on both context and time (Boykin & Williams, 2010; Dein, 2006; Lee, 2009).

Description

The concept of ethnicity is commonly used interchangeably with the term "race" within the health literature, although both concepts are different from one another. A clear distinction between the two terms is that race is a scientifically unfounded taxonomy that categorizes individuals based on phenotypical characteristics (e.g., skin color, facial features) and geographic origin; it is a socially and ideologically constructed category (Sheldon & Parker, 1992; Williams, Lavizzo-Mourey, & Warren, 1994). Moreover, race has poor predictive validity for biological differences, and the amount of genetic variation that exists for any one particular ethnic group is larger than that found between "racial" groups (Sheldon & Parker, 1992), providing evidence for the lack of biologic or genetic basis for racial categorizations (Lin & Kelsey, 2000). On the other hand, despite that ethnicity is sometimes used in research as a fixed term, a distinguishable feature of it from that of race is that ethnicity is a much broader concept that also captures notions of self- and group identity (Sheldon & Parker, 1992). Although the fluidity of boundary demarcations as it relates to ethnicity (as can be noted, for example, throughout the different U.S. Decennial Census forms) is a limitation of the concept, many social scientists advocate for the use of "ethnicity" over "race" in order to avoid biological reductionism. Indeed, many researchers argue that ethnicity as a concept can better capture the environmental, cultural, behavioral, and sociopolitical experiences that affect health and illness (Dein, 2006). The term "ethnicity" is more commonly used in other countries (e.g., the UK and Canada) compared to the United States (Boykin & Williams, 2010). Significantly, despite that both majority and minority social groups have an ethnicity, the term "ethnicity"

has mostly been used (erroneously) to refer to ethnic minorities (Sheldon & Parker, 1992).

Ethnic and racial health disparities have been widely documented. However, the focus on ethnicity (and race) as fixed demographic categories and etiological factors has limited researchers' ability to adequately identify and delineate underlying mechanisms of these disparities (Sheldon & Parker, 1992). For example, despite that scientists are increasingly using ethnicity (and race) in biomedical and genetics research, when ethnic (or racial) differences are found, researchers typically fail to define the mechanisms through which these social categories operate in their statistical models or in actual life (Dein, 2006; Williams et al., 1994). Such a practice runs the risk of erroneously attributing noted differences in health status to ethnic minorities themselves, which may further contribute to pathologizing already socially marginalized groups (Sheldon & Parker, 1992; Williams et al., 1994). Similarly, ethnic differences in disease processes and/or health outcomes are usually attributed to culture, particularly to aspects of diet, lifestyle, and behavioral practices (Sheldon & Parker, 1992). However, researchers have noted that ethnic differences in health are not only due to behavioral and cultural factors but also due to larger social processes and structures, including historical, political, socioeconomic, environmental, and contextual factors, as well as discrimination and racism (Boykin & Williams, 2010; Pierce, Foliaki, Sporle, & Cunningham, 2004; Sheldon & Parker, 1992).

Methodological assessments of ethnicity in health research have some limitations. For example, a common methodological approach is that ethnic groups are usually grouped together to represent an ethnicity based on their national origin. However, ethnic differences may exist within national origin groups based on a number of factors, including cultural and linguistic ones. For example, whereas Mexican persons might be grouped together, differences exist in ethnic groups in Mexico (e.g., indigenous persons who might also speak a different language). This may apply to many other national origin groups that are typically collapsed into one ethnic category. Likewise, although there are certainly some advantages to grouping all Latino or Asian subgroups into one ethnic category, for example, this practice can simultaneously obscure the heterogeneity that exists among subgroups across social, contextual, political, and historical contexts, which in turn may come to differentially affect the health of such groups.

Given the diverging definitions, classifications, and use of ethnicity across geographic contexts and time, understanding disease processes and outcomes as well as health/health service disparities will require further and more explicit clarification and assessment of ethnicity (and race), as well as of mechanisms through which both ethnicity and race may come to affect health (e.g., discrimination, social class, access to care) (Boykin & Williams, 2010; Lin & Kelsey, 2000).

Cross-References

- ▶ Ethnic Identity
- ▶ Health Disparities
- ▶ Hispanic Community Health Study/Study of Latinos
- ▶ Minority Subgroups

References and Readings

Boykin, S. D., & Williams, D. R. (2010). Race, ethnicity, and health in global context. In A. Steptoe, K. E. Freedland, J. Richard Jennings, M. M. Labre, & S. B. Manuck (Eds.), *Handbook of behavioral medicine: Methods and applications* (pp. 321–339). New York: Springer.

Dein, S. (2006). Race, culture and ethnicity in minority research: A critical discussion. *Journal of Cultural Diversity, 13*(2), 68–75.

Lee, C. (2009). "Race" and "ethnicity" in biomedical research: How do scientists construct and explain differences in health? *Social Science and Medicine, 68*, 1183–1190.

Lin, S. S., & Kelsey, J. L. (2000). Use of race and ethnicity in epidemiologic research: Concepts, methodological issues, and suggestions for research. *Epidemiologic Reviews, 22*(2), 187–202.

Pierce, N., Foliaki, S., Sporle, A., & Cunningham, C. (2004). Genetics, race, ethnicity, and health. *British Medical Journal, 328*, 1070–1072.

Sheldon, T. A., & Parker, H. (1992). Race and ethnicity in health research. *Journal of Public Health Medicine, 14*(2), 104–110.
Williams, D. R., Lavizzo-Mourey, R., & Warren, R. C. (1994). The concept of race and health status in America. *Public Health Reports, 109*(1), 26–41.

Ethnicity Subgroups

▶ Minority Subgroups

Etiology/Pathogenesis

Michael Witthöft
Psychologisches Institut Abteilung Klinische Psychologie und Psychotherapie, Johannes Gutenberg Universität Mainz, Mainz, Germany

Definition

The terms "etiology" and "pathogenesis" are closely related to the questions of *why* and *how* a certain disease or disorder develops. Models of *etiology* and pathogenesis therefore try to account for the processes that initiate (*etiology*) and maintain (*pathogenesis*) a certain disorder or disease.

Etiology

Etiology (consisting of two Greek terms for "origin" and "study of") refers to the study of the causes of a mental or physical disease. As parts of the *etiology* of a respective disease, only causes that directly initiate the disease process (and therefore necessarily temporarily have to precede the onset of the disease) are considered as *etiological* factors. *Etiological* factors can thus be considered as *necessary conditions* for the development of a disease. The *etiology* of a certain condition is mostly defined not only by one but rather by the interplay of many different conditions (biological, environmental, etc.). As an example, the *etiology* of the common cold is based on an infection by Rhinoviruses causing a viral upper respiratory infection (e.g., Eccles & Weber, 2009). Additionally, multiple environmental and immunological factors modulate the infectious *etiology* of the common cold (Eccles & Weber, 2009). In the realm of mental disorders, dysregulations of the endocrinological stress system, especially the hypothalamic-pituitary-adrenal axis (e.g., caused by early traumatic experiences and early life stress), are considered as *etiological* factors relevant for the development of certain disorders (e.g., depression, posttraumatic stress disorder, somatoform disorders) (Ehlert, Gaab, & Heinrichs, 2001). However, in many complex mental and physical disorders, the exact *etiology* is still either entirely or partly unknown (e.g., depression, obesity). Especially regarding mental disorders, the causes of certain symptoms and syndromes remain vague and speculative. An exception is the diagnosis of a posttraumatic stress disorder, in which case the *etiology* is unambiguously defined by the occurrence of a traumatic event.

Pathogenesis

Pathogenesis, in turn, refers to the process and factors associated with the perpetuation and maintenance of a respective mental or physical disorder. Factors associated with the *pathogenesis* also comprise behavioral changes (e.g., avoidance of normal physical activities in chronic pain conditions) that may maintain and even worsen a specific condition (e.g., physical inactivity often aggravates chronic pain conditions). Accordingly, *pathogenetic* factors, as opposed to *etiological* factors, do not necessarily have to precede the onset of a certain mental or physical disorder. As an example, a depressive disorder might initially develop as the result of early dysregulation in the stress system paired with acute adverse or stressful life events, but might be maintained and even exacerbated due to cognitive and behavioral *pathogenetic* factors (e.g., social isolation, lack of physical activity, and ongoing self-blame).

Cross-References

- ▶ Pathophysiology
- ▶ Somatoform Disorders

References and Readings

Eccles, R., & Weber, O. (2009). *Common cold*. Basel, Switzerland: Birkhäuser Verlag.

Ehlert, U., Gaab, J., & Heinrichs, M. (2001). Psychoneuroendocrinological contributions to the etiology of depression, post-traumatic stress disorder, and stress related bodily disorders: The role of the hypothalamus-pituitary-adrenal axis. *Biological Psychology, 57*, 141–152.

Euthanasia

Kristin Kilbourn and Shannon Madore
Department of Psychology, University of Colorado Denver, Denver, CO, USA

Synonyms

Assisted suicide; Physician-assisted suicide

Definition

Euthanasia is broadly defined as the practice of ending a life as a means of relieving pain and suffering. Assisted suicide refers to actions by which an individual helps another person voluntarily bring about his or her own death. Despite the fact that physician assisted suicide (PAS) is illegal in most of the states in the USA, medical practitioners often receive requests from patients and their families to perform euthanasia, and many clinicians honor these requests. The reasons for these requests have not been well studied, but it appears that it involves a complex combination of physical and psychosocial symptoms and concerns. Euthanasia is currently legal in a small number of US states (Montana, Oregon, and Washington) and a limited number of European countries. The American College of Medical Quality (2001) has published guidelines for physicians confronted with a patient's request for physician-assisted suicide.

Euthanasia and PAS remain extremely controversial due to the moral, ethical, and religious issues that surround them. There are strong arguments for and against the legalization of PAS and euthanasia in the USA. One concern regarding the practice of euthanasia and PAS is the established association between depression and requests for euthanasia. Research suggests that euthanasia requests from depressed patients are often transitory, while in nondepressed, severely ill patients, the desire to hasten death tends to be enduring.

References and Readings

Asch, D. A. (1996). The role of critical care nurses in euthanasia and assisted suicide. *The New England Journal of Medicine, 334*(21), 1374–1379.

Emanuel, E. J., Fairclough, D. L., & Emanuel, L. L. (2000). Attitudes and desires related to euthanasia and physician-assisted suicide among terminally ill patients and their caregivers. *Journal of the American Medical Association, 284*(19), 2460–2468.

Hudson, P. L., Kristjanson, L. J., Ashby, M., Kelly, B., Schofield, P., Hudson, R., Aranda, S., O'Connor, M., & Street, A. (2006). Desire for hastened death in patients with advanced disease and the evidence base of clinical guidelines: A systematic review. *Palliative Medicine, 20*(7), 693–701.

Levene, I., & Parker, M. (2011). Prevalence of depression in granted and refused requests for euthanasia and assisted suicide: A systematic review. *Journal of Medical Ethics, 37*(4), 205–211.

Meier, D., Emmons, C., Wallstein, S., Quill, T., Morrison, S., & Cassel, C. (1998). A national survey of physician-assisted suicide and euthanasia. *The New England Journal of Medicine, 338*(17), 1193–1201.

Rosenfeld, B. (2000). Assisted suicide, depression, and the right to die. *Psychol Public Policy Law, 6*(2), 467–488.

The American College of Medical Quality (ACMQ). (2001). *Policy 34: Physician-assisted suicide and end of life care*. Retrieved May 4, 2012, http://www.acmq.org/policies/policy34.pdf

Wilson, K. G., Scott, J. F., Graham, I. D., Kozak, J. F., Chater, S., Viola, R. A., de Faye, B. J.,

Weaver, L. A., & Curran, D. (2000). Attitudes of terminally ill patients toward euthanasia and physician-assisted suicide. *Archives of Internal Medicine, 160*(16), 2454–2460.

Wilson, K. G., Chochinov, H. M., McPherson, C. J., Skirko, M. G., Allard, P., Chary, S., Gagnon, P. R., Macmillan, K., De Luca, M., O'Shea, F., Kuhl, D., Fainsinger, R. L., Karam, A. M., & Clinch, J. J. (2007). Desire for euthanasia or physician-assisted suicide in palliative cancer care. *Health Psychology, 26*(3), 314–323.

Evaluation of Potential Public Health Impact

▶ RE-AIM Guidelines

Evaluations

▶ Attitudes

Event Sampling

▶ Diaries

Event-Related Optical Imaging (EROI)

▶ Brain, Imaging
▶ Neuroimaging

Everyday Problems

▶ Daily Stress

Evidence Hierarchy

▶ Hierarchy of Evidence

Evidence-Based Behavioral Medicine (EBBM)

Bonnie Spring, Molly Ferguson, Stephanie Russell, Alyson Sularz and Megan Roehrig
Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Synonyms

Evidence-based behavioral practice; Evidence-based medicine; Evidence-based practice; Evidence-based psychological practice

Definition

Evidence-based behavioral medicine (EBBM) is that branch of the evidence-based practice (EBP) movement that addresses behavioral interventions to promote health and mitigate the impact of illness. Evidence is comprised of research findings derived from the systematic collection of data through observation and experiment and the formulation of questions and testing of hypotheses. Evidence-based practice is an approach that aims to improve the process through which high quality scientific research evidence can be obtained and translated into best practical decisions to improve health. The evidence-based practice movement establishes consensus on the standards used to conduct, report, evaluate, and disseminate research results so as to increase their uptake and impact on practice and policy. Best available research is defined as best in quality, according to consensually accepted scientific standards for different kinds of questions, and most contextually relevant. Evidence-based practice entails the use of conscientious, explicit decision making that integrates consideration of best available research evidence, client characteristics, and resources. Many practical decisions relevant to evidence-based practice concern the selection of

an assessment or intervention. Whereas the treatments evaluated in evidence-based medicine usually involve drugs or devices, those appraised in evidence-based behavioral medicine more often comprise non-drug, non-device, behavioral or psychosocial interventions.

Description

History of Evidence-Based Practice

The evidence-based practice movement began as an effort to distinguish valid health practices from illegitimate ones. By now, all major health professions endorse evidence-based practice, and the Institute of Medicine identifies EBP as a core competence for all twenty-first-century health professionals (Greiner & Knebel, 2003). The EBP movement emerged from evidence-based medicine (EBM) and can be traced to three influences on the history of medicine in the twentieth century: the Flexner Report, the Cochrane Collaboration, and the clinical epidemiology group at McMaster University.

First, the Flexner Report, commissioned by the American Medical Association and the Carnegie Foundation and published in 1910, represented a major effort to reform medical education by placing it on a scientific foundation. To prepare the Report, Abraham Flexner, a research scholar at the Carnegie Foundation for the Advancement of Teaching, surveyed 155 medical schools that were in operation in the United States and Canada. Flexner severely criticized the training offered by many medical schools. He described a curriculum that was not based on science, lax clinical training, and a predominant profit rather than public service motivation for many schools' existence (Flexner, 1910). The Flexner Report established an educational quality standard that many existing medical schools could not meet. In consequence, by 1935, more than half of all medical schools had been closed (Beck, 2004).

A second main catalyst for the EBM movement arose from British epidemiologist, Archibald Cochrane's efforts to establish a rational, systematic basis for determining health care coverage (Cochrane, 1972). Cochrane argued that because resources for health care are inevitably limited, it is essential that scarce dollars only be allocated for procedures of demonstrable worth. His conclusion that Randomized Controlled Trials (RCTs) offer the most reliable, unbiased method to evaluate the effectiveness of treatments led others to formulate a hierarchy of evidence. According to this evidence hierarchy, findings from high quality RCTs are given greater credence than those from observational studies, case studies, and expert opinion when making determinations about whether a treatment works. To aggregate and disseminate findings from RCTs, Cochrane's followers established the worldwide network known as the Cochrane Collaboration (www.cochrane.org), whose contributors track, critically appraise, synthesize, and disseminate RCT findings via the internet.

A third engine that drove EBM forward was the group of clinical epidemiologists working at Canada's McMaster University in the 1990s under the leadership of David Sackett and Gordon Guyatt. This group's ambitious agenda was to close the research-to-practice gap by socializing physicians to engage in lifelong learning about new research findings (Sackett & Rosenberg, 1995a, 1995b). The McMaster team was motivated by evidence that clinicians primarily implement practices learned during training but neglect alternative, new, and often more efficacious treatments (Isaacs & Fitzgerald, 1999). In order to encourage physicians to routinely ask questions and consult research, the group developed methods that allowed practitioners to find, appraise, and apply research results during the actual clinical encounter. They encountered resistance from clinicians who felt that an exclusive emphasis on research devalued the importance of clinical expertise to quality care provision (Haynes et al., 1996). To overcome that barrier and encourage clinical implementation, Gordon Guyatt renamed the approach "evidence-based medicine," in place of the earlier phrase "scientific medicine" (Guyatt et al., 1992). Instead of a single circle (research), subsequent models of evidence-based medicine have used a "three circles" or "three legged stool" model of EBM.

That is to say, they depict evidence-based practice as tying together research, patient characteristics, and expertise (Haynes et al., 1996; Sackett, Rosenberg, Gray, Haynes, & Richardson, 1996).

From EBM to EBBM

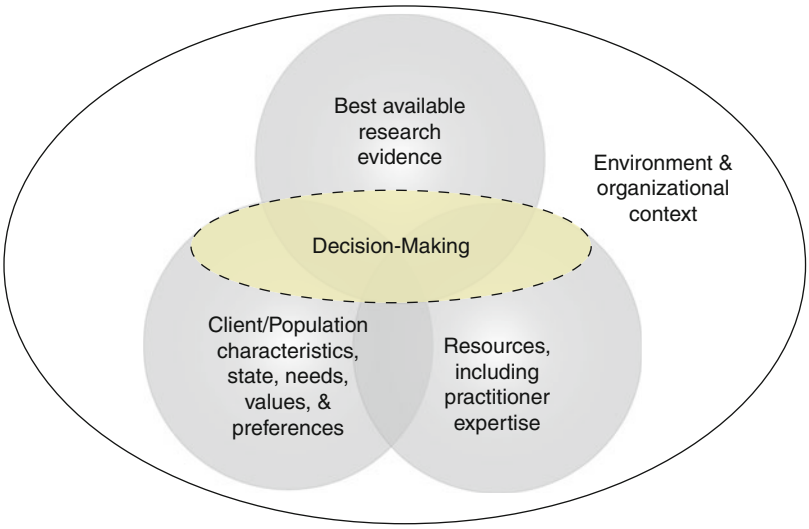
A necessary precondition for EBBM was to consider carefully how well the core principles of evidence-based medicine apply to research on behavioral (non-drug, non-device) interventions. That evaluation was undertaken initially by the Society of Behavioral Medicine's EBBM Committee, established in 2000 with support from the National Institutes of Health (NIH) Office of Behavioral and Social Science Research (OBSSR) under Acting Director, Peter Kaufmann. The EBBM Committee, first chaired by Karina Davidson, defined its scope to include behavioral interventions that prevent disease, promote health and adherence to treatment, or change biological determinants of behavioral conditions (Davidson et al., 2003). Initial efforts were dedicated to familiarizing behavioral medicine researchers with the CONSORT guidelines that support comprehensive, transparent reporting of RCTs in medical journals (Schulz et al., 2010a). Partly as a result of the Committee's efforts, the CONSORT guidelines were adopted by journals that publish behavioral medicine RCTs, including the *Annals of Behavioral Medicine, Health Psychology*, *International Journal of Behavioral Medicine*, and *Journal of Consulting and Clinical Psychology*. Additional efforts to improve the quality of behavioral medicine clinical trials addressed a frequent weakness in their analytic approach, i.e., failure to use the intent to treat principle whereby all randomized participants are included in study analyses according to the condition to which they were assigned (Pagoto et al., 2009; Spring, Pagoto, Knatterud, Kozak, & Hedeker, 2007).

Still other efforts of the EBBM Committee addressed common fears and misperceptions about what evidence-based practice entails (Spring et al., 2005). One frequent misunderstanding is that the approach neglects all but RCT evidence. Actually, the principle is that the optimal research design depends upon the question being asked. For example, a prognostic question about the likely course of a patient's condition is answered more effectively by an observational cohort study than by an RCT. Also addressed was the misperception that evidence-based practice equates to cookbook treatment and the false belief that RCTs inevitably exclude complex patients in real world settings.

From EBBM to EBBP to, Simply, EBP: The Conceptual Model

By 2006, the U.S. health care crisis had arrived. With it came the need for and promise of a better integrated system of care that addresses mental as well as physical health and prevention as well as care of the sick, and that accomplishes all this by the coordinated efforts of an interprofessional team. A horizon scan made apparent the need to upgrade and harmonize the approach to evidence-based behavioral practice across the health professions that offer behavioral interventions. Accordingly, OBSSR sponsored the Council on Evidence-Based Behavioral Practice (EBBP), chaired by Bonnie Spring, and its scientific and practitioner advisory boards. Composition of these groups was determinedly interprofessional, combining representatives from medicine, nursing, psychology, social work, public health, and information sciences (www.ebbp.org). The Council's first task was to formulate a conceptual model that could accommodate the diverse historic traditions as well as the individual and population level behavioral interventions that different health professions implement.

Medicine's initial conceptual model of evidence-based practice had emphasized a single parameter: research. EBM was defined simply as "the conscientious and judicious use of current best evidence in making decisions about the care of individual patients" (Sackett et al., 1996). Subsequent EBM definitions emphasized the need to balance considerations in addition to research: For example, "Evidence-based medicine requires the integration of the best research evidence with clinical expertise and the patient's unique values and circumstances" (Strauss, Richardson, Glasziou, & Haynes, 2005).



Evidence-Based Behavioral Medicine (EBBM), Fig. 1 The three circles of interprofessional evidence-based practice (Source: Spring & Hitchcock, 2009)

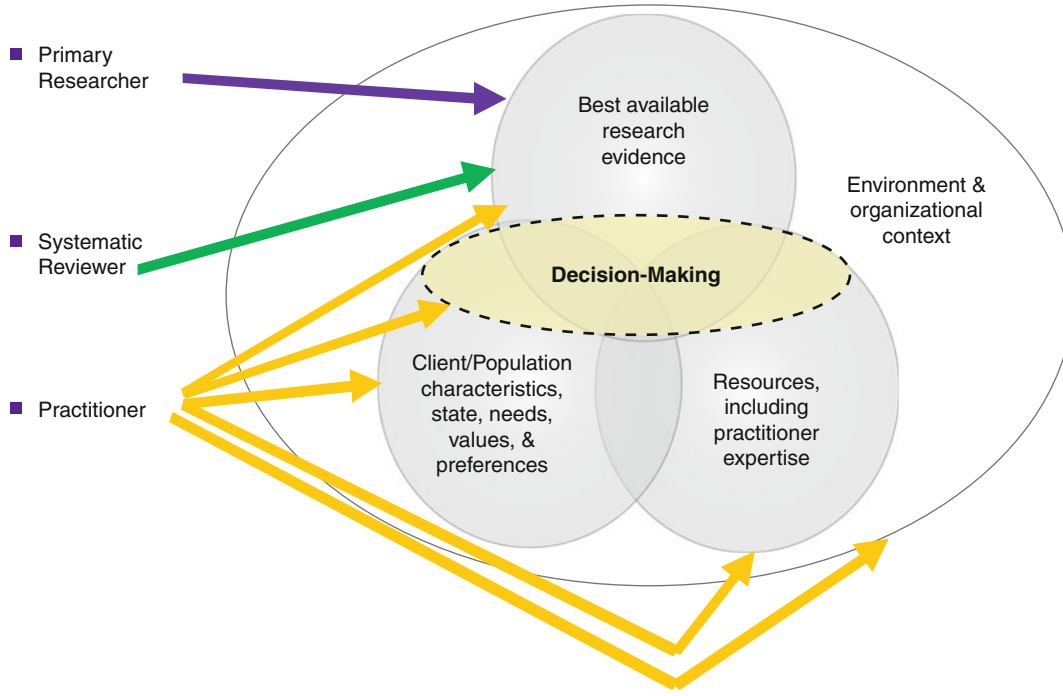
The EBBP Council worked to integrate the historic conceptualizations of evidence-based practice developed in medicine, nursing, psychology, public health, and social work (Satterfield et al., 2009). The aim was to develop a new, harmonized conceptual model suitable to be shared by the more diverse interprofessional health care teams of the future, whose members all require core competency in EBP (Greiner & Knebel, 2003). Because the shared conceptual model supports jointly held vocabulary, foundational assumptions, and practice principles that unite the team, a unified model of evidence-based practice supplants the need to have separate models for different disciplines or for behavioral versus medical interventions (Satterfield et al., 2009; Spring & Hitchcock, 2009).

The interprofessional model of evidence-based practice appears in Fig. 1.

Note that the model depicts three circles (data strands) that need to be integrated in EBP and that evidence-based decision making is shown as the skill that knits these strands together. As in all prior EBP models, best available research evidence remains a circle, occupying the top position because it reflects the cumulative, unbiased body of knowledge about what is effective for the health issue. The client's characteristics, including current state, prior history, values, and preferences, are a second key data strand that needs to be integrated by engaging the client in shared decision making. The third data strand involves resources because these can constrain the available treatment options. Resources include influences such as insurance coverage, other financial resources, available trained interventionists, community facilities, and time or capability to access treatment. The surrounding, outer circle acknowledges that, inevitably, EBP occurs in a particular organizational and environmental context that will influence what interventions are endorsed and how readily they can be implemented.

Health Professionals' Roles in EBP

It is no understatement to say that "it takes a village" to sustain the infrastructure of evidence-based practice. As shown in Fig. 2, health professionals can play three different and essential roles in relation to EBP. First, as primary researchers, they directly contribute to creating the evidence base. They develop new interventions, and they design, conduct, analyze, and report research that evaluates the efficacy and effectiveness of treatments. If random assignment to treatments is feasible, they will usually conduct RCTs. If it is not, as is often the case for policy interventions, they may use alternative designs, such as intermittent time series to evaluate whether a treatment works.



Evidence-Based Behavioral Medicine (EBBM), Fig. 2 Health professionals' roles in EBP

In the second role, as systematic reviewers, health professionals are evidence synthesizers. They aggregate primary research that was conducted by others to create and disseminate research syntheses that can be accessed efficiently and used by practitioners. The research synthesizer role is a critical underpinning in the infrastructure of EBP. Because of the rapid pace at which the scientific literature proliferates, few full-time practitioners can stay comprehensively abreast of new research, while also managing their patient care responsibilities. Systematic reviewers play a critically important role in EBP. By culling and analyzing the full body of new and old studies that address clinically important questions and disseminating their findings to clinicians in the form of pithy summaries, they make the EBP enterprise feasible for practitioners. To accomplish this, systematic reviewers first develop a comprehensive and unbiased protocol to locate the primary research that addresses a practical question. Having acquired the relevant studies, they critically appraise, extract, and synthesize the data to provide an answer. Depending upon the heterogeneity of the interventions and study designs included in the review, they may synthesize and report the findings solely qualitatively or also quantitatively, using meta-analysis. Systematic reviewing is itself a sophisticated and evolving form of research methodology that is increasingly used as the basis for health policies, including practice guidelines.

Finally, in the third role as practitioners, health professionals play the most complex and challenging role in EBP. The clinician extracts and uses data from each of the three EBP circles. Clinicians are research consumers: They access research evidence and appraise its quality and relevance for their context. For efficiency in a busy practice context, clinicians are advised to turn first to the secondary, synthesized, critically pre-appraised sources of evidence such as systematic reviews or the evidence-based practice guidelines to be found on www.guidelines.gov. However, in some instances, there may be no applicable treatment guidelines or systematic review, requiring the clinician to search the primary literature to locate relevant original

research. Moreover, the health professional adopting the practitioner role interacts more directly than either primary researchers or evidence synthesizers with the remaining two circles of the EBP model: client characteristics and resources. To simplify the practitioner's complex job, the 5-step evidence-based practice process, shown in Fig. 3, maps out a recommended series of steps that practitioners can follow in order to address each of the three circles.

The Five Steps of Evidence-Based Practice

Evidence-based practice entails both a conceptual model (shown in Fig. 1) and the 5-step process shown in Fig. 3. Each step represents an integral part of the EBP process and a competency to be mastered by the clinician. Note that assessment is assumed to precede the onset of the EBP process and to recur throughout it, rather than being considered a formal step. Step 1 is to ask questions that are formulated and structured in a manner that facilitates finding the relevant research. Step 2 is to acquire the best available evidence regarding the question. Step 3 is to critically appraise the evidence on two parameters: its validity and its applicability to the problem at hand. Step 4, Apply, is the most complex phase in the EBP process and the step that links most directly to the conceptual model depicted in Fig. 1. Apply is the step during which the practitioner engages in evidence-based decision making that balances and integrates best available research evidence, client characteristics, and resources to determine a treatment approach. Moreover, to increase the likelihood of treatment uptake and adherence, it is important for the decision-making process to be shared with the affected individual(s) and/or group(s). Choosing and collaboratively applying the best research-supported intervention represents a beginning rather than an end to the EBP process. The treatment supported by best available evidence will be the one that has proved cumulatively most effective for the average patient in an average environment. But what matters most for the evidence-based practice process is the single (N = 1) patient under the clinician's care. Step 5, the final step in the EBP process is to assess that client's response to the intervention, analyze progress, and, if warranted, adjust the course of treatment.

Evidence-Based Behavioral Medicine (EBBM), Fig. 3 Five steps of evidence-based practice (Source: www.ebbp.org)

Educational Resources

The www.ebbp.org website created by the NIH-sponsored EBBP project offers access to information, tools, and training modules that facilitate research-to-practice translation for behavioral medicine. Nine online modules are available free of charge, covering a mixture of content relevant to learning objectives in research or practice. Free registration in the online portal conveys access to pre- and post-tests for each module and enables learners to stop and save their progress on a partially completed module. Of particular relevance to primary researchers are the learning modules about Randomized Controlled Trials, as well as the module on Critical Appraisal. The Critical Appraisal module offers an overview of the strengths and weaknesses of alternative research designs for evaluating whether a treatment works. A module on Systematic Evidence Reviews provides an introduction to research synthesis for learners interested in exploring that activity. The module on Searching for Evidence offers useful tips about search strategies for practitioners as well as primary researchers. Its overview of the many available online research databases is of particular relevance for systematic reviewers.

The remaining modules are geared particularly toward those trying to conduct research to

practice translation in behavioral medicine. Of these, three are oriented toward clinicians who work with individuals or practitioners who work with communities or populations. The EBBP Process module provides an overview of how to perform the 5-step evidence-based practice process. The learner is given a choice about whether to work with either an individual or a community. Two separate modules on Shared or Collaborative Decision Making illustrate how to engage individuals or communities, respectively, in the Apply step of the evidence-based practice process. These Process and Shared Decision-Making modules are highly interactive and experiential, giving learners an opportunity to experiment, make mistakes, and ask experts while practicing new learning in a safe environment. Finally, the Stakeholder and Implementation Modules are intended to be used by both researchers and practitioners wishing to engage in collaborative translational research about evidence-based practice. Featuring a series of interview clips with academic researchers, clinicians, and community advocates, the Stakeholder module presents a glimpse of widely varying worldviews about research. The final Implementation module presents a conceptual model of implementation science and offers two different case studies of successful implementation of evidence-based behavioral programs: one involving in-person training and the other involving internet-delivered training.

The resources at www.ebbp.org are meant to be used by either individual learners or by instructors as part of a course on research methods, interventions, or evidence-based practice. In addition to tools provided by the EBBP project, other online tutorials about evidence-based practice are made available by both University of North Carolina and Vanderbilt University.

Cross-References

- ▶ Research to Practice Translation
- ▶ Translational Behavioral Medicine

References and Readings

Beck, A. H. (2004). The Flexner report and the standardization of American medical education. *Journal of the American Medical Association, 291*(17), 2139–2140.

Cochrane, A. (1972). *Eff ectiveness and efficiency: Random reflections on health services*. London: Royal Society of Medicine Press.

Davidson, K. W., Goldstein, M., Kaplan, R. M., Kaufmann, P. G., Knatterud, G. L., Orleans, C. T., et al. (2003). Evidence-based behavioral medicine: What is it and how do we achieve it? *Annals of Behavioral Medicine, 26*(3), 161–171.

Flexner, A. (1910). *Medical education in the United States and Canada: A report to the Carnegie foundation for the advancement of teaching* (Bulletin No. 4). New York City: The Carnegie Foundation for the Advancement of Teaching, pp. 346, OCLC 9795002.

Greiner, A. C., & Knebel, E. (Eds.). (2003). *Health professions education: A bridge to quality*. Washington, DC: National Academy Press.

Guyatt, G., Cairns, J., Churchill, D., Cook, D., Haynes, B., Hirsch, J., et al. (1992). Evidence-based medicine. A new approach to teaching the practice of medicine ['Evidence-Based Medicine Working Group']. *Journal of the American Medical Association, 268*, 2420–5.

Haynes, R. B., Devereaux, P. J., & Guyatt, G. H. (2002). Clinical expertise in the era of evidence-based medicine and patient choice. *Evidence-Based Medicine, 7*, 36–38.

Haynes, R.B., Sackett, D.L., Gray, J.M., Cook, D.J., Guyatt, G.H. (1996) Transferring evidence from research into practice: 1. The role of clinical care research evidence in clinical decisions. *ACP Journal Club*, 125(3):A14–6.

Institute of Medicine. (2001). *Crossing the quality chasm: A new health system for the 21st century*. Washington, DC: National Academy Press.

Isaacs, D., & Fitzgerald, D. (1999). Seven alternatives to evidence based medicine. *British Medical Journal, 319*(7225), 1618.

Pagoto, S., Kozak, A. T., John, P., Bodenlos, J., Hedeker, D., & Spring, B. (2009). Intention-to-treat analyses in behavioral medicine randomized clinical trials: The impact of CONSORT. *International Journal of Behavioral Medicine, 16*(4), 316–322.

Sackett, D. L., & Rosenberg, W. M. (1995a). On the need for evidence-based medicine. *Health Economics, 4*, 249–254.

Sackett, D. L., & Rosenberg, W. M. (1995b). The need for evidence-based medicine. *Journal of the Royal Society of Medicine, 88*, 620–624.

Sackett, D. L., Rosenberg, W. M., Gray, J. A., Haynes, R. B., & Richardson, W. (1996). Evidence-based medicine: What it is and what it isn't. *BMJ, 312*, 71–72.

Satterfield, J. M., Spring, B., Brownson, R. C., Mullen, E. J., Newhouse, R. P., & Walker, B. B. (2009).

Toward a transdisciplinary model of evidence-based practice. *The Milbank Quarterly, 87*(2), 368–390.
Schulz, K. F., Altman, D. G., & Moher, D. (2010a). CONSORT 2010 statement: Updated guidelines for reporting parallel group randomized trials. *Annals of Internal Medicine, 154*(11), 1–11.
Schulz, K.F., Altman, D.G., Moher, D. CONSORT Group (2010b). CONSORT 2010 statement: Updated guidelines for reporting parallel group randomized trials. *Annals of Internal Medicine*, *152*(11), 726–32.
Spring, B. (2007). Evidence-based practice in clinical psychology: What it is; why it matters; what you need to know. *Journal of Clinical Psychology, 63*(7), 611–631.
Spring, B. (2008). Health decision-making: Lynchpin of evidence-based practice. *Medical Decision Making, 28*, 866–874.
Spring, B., & Hitchcock, K. (2009). Evidence-based practice in psychology. In I. B. Weiner & W. E. Craighead (Eds.), *Corsini's encyclopedia of psychology* (4th ed., pp. 603–607). New York: Wiley.
Spring, B., & Neville, K. (2010). Evidence-based practice in clinical psychology. In D. Barlow (Ed.), *Oxford handbook of clinical psychology* (pp. 128–149). New York: Oxford University Press.
Spring, B., Pagoto, S., Knatterud, G., Kozak, A., & Hedeker, D. (2007). Examination of the analytic quality of behavioral health randomized clinical trials. *Journal of Clinical Psychology, 63*, 53–71.
Spring, B., Pagoto, S., Whitlock, E., Kaufmann, P., Glasgow, R., Smith, K., Trudeau, K., & Davidson, K. (2005). Invitation to a dialogue between researchers and clinicians about evidence-based behavioral medicine. *Annals of Behavioral Medicine*, 30(2), 125–137.
Strauss, S. E., Richardson, W. S., Glasziou, P., & Haynes, R. B. (2005). *Evidence-based medicine: How to practice and teach EBM* (3rd ed.). New York: Elsevier.

Evidence-Based Behavioral Practice

▶ Evidence-Based Behavioral Medicine (EBBM)

Evidence-Based Medicine

▶ Clinical Trial
▶ Evidence-Based Behavioral Medicine (EBBM)

Evidence-Based Practice

▶ Evidence-Based Behavioral Medicine (EBBM)

Evidence-Based Psychological Practice

▶ Evidence-Based Behavioral Medicine (EBBM)

Excess Weight

▶ Overweight

Excessive Drinking

▶ Binge Drinking

Executive Control

▶ Executive Function

Executive Control Resources

▶ Executive Function

Executive Function

Peter A. Hall
Faculty of Applied Health Sciences, University of Waterloo, Waterloo, ON, Canada

Synonyms

Executive control resources; Supervisory attentional system

Definition

Executive function (EF) is an emergent quality of cognitive function that arises from the operation of several brain structures situated at least partly

in the prefrontal cortex of the human frontal lobes. These cognitive operations are minimally reducible to the ability to inhibit prepotent responses, hold items in working memory, and direct attention. These core abilities in turn potentiate a variety of behavioral tendencies that include the capacity to avoid impulsive responding, the ability to remain mindful of and effectively pursue goals, the ability to avoid distraction, and the capacity to delay gratification.

Description

Interest in EFs emerged predominantly from the study of "frontal lobe" patients, or individuals who have sustained damage to the frontal lobes of the brain. Such individuals were initially observed to demonstrate intact general cognitive abilities, but evidenced specific impairments in several areas of function that were thought to be central to notions of personality and everyday social functioning. Early clinical descriptions of these patients in the neurological literature led to more specific formulations of the operations of the frontal lobes (see Stuss & Knight, 2002) and to vigorous inquiry regarding the nature of the concept of executive function itself in cognitive psychology.

EFs are thought to be a set of related cognitive operations that are housed within the frontal lobe of the brain, and they are specifically associated with the operation of the prefrontal cortex. As a group of related cognitive processes, EFs exhibit both unitary and diversity of function and so can be understood in relation to both their general level of operation, or the operation of the specific subfacets (Miyake et al., 2000). Some of the subfacets of general EF include behavioral inhibition, working memory, and task switching abilities. However, there are several conceptualizations of the structure of the executive system, which give prominence to one or more of these components (see Shallice, 1988).

Executive functions are frequently measured using a variety of neuropsychological tests. These include, but are not limited to, the following: the Stroop test, the go-no go test, trails B, digit symbol (subtest of the Wechsler Adult Intelligence Scales), Tower of London/Hanoi, Iowa gambling task, stop signal, and the flanker task. Many of these have been used for decades and represent a class of tasks with similar characteristics, rather than single tasks with exactly specified parameters. For example, the Stroop task involves viewing a series of color names (i.e., the word "red") displayed one at a time. The respondent is required to name the color of font – ignoring the word itself – as quickly and accurately as possible. On some trials, the font color matches the word (i.e., the word "red" is presented in red font; "concordant trials"), and on other trials, the font color is inconsistent with the word itself (i.e., the word "red" is presented in blue font; "discordant trials"). The dependent measure may be any number of parameters including the reaction time on correct trials, error rate, or ratio of reaction times on discordant versus concordant trials. The actual modality of presentation of the stimuli and responses varies depending on the specific requirements of the researcher, as do the number of trials. The Stroop test is thought to measure predominantly the inhibition facet of EF, though strong performance would naturally also correlate with working memory and attention as well.

In addition to such "behavioral" measures of EF, there is considerable interest in measurement of the activation of the underlying brain structures that give rise to EFs. Such approaches to imaging EF engagement include functional magnetic resonance imaging (fMRI), electroencephalogram (EEG), positron emission tomography (PET), and functional near-infrared spectroscopy (fNIRS).

Individual differences in EF are subject to both strong dispositional influences and potential for state-like fluctuation. For instance, EFs are among the most sensitive cognitive functions to the adverse effects of chronic health conditions, as well as the effect of both natural and pathological aging processes (e.g., Alzheimer's disease and other dementias). Nonetheless, in the absence of disease-related cognitive decline, individual differences in EF among cognitively intact individuals are subject to substantial genetic loading (the latter including both genetic and gene x environment interactions).

Interest in EFs in the field of behavioral medicine has increased partly as a function of the significance of EFs for self-regulatory processes in health behavior performance, emotional regulation, and mortality (Hall & Fong, 2007).

Cross-References

- ▶ Behavioral Inhibition
- ▶ Cognition
- ▶ Disinhibition
- ▶ Working Memory

References and Readings

Hall, P. A., & Fong, G. T. (2007). Temporal self-regulation theory: A model for individual health behavior. *Health Psychology Review, 1*, 6–52.

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: A latent variable analysis. *Cognitive Psychology, 41*, 49–100.

Shallice, T. (1988). *From neuropsychology to mental structure*. New York: Cambridge University Press.

Stuss, D. T., & Knight, R. T. (2002). *The principles of frontal lobe function*. New York: Oxford University Press.

Exercise

Jordan Carlson
Public Health, San Diego State University, University of California San Diego, San Diego, CA, USA

Synonyms

Leisure physical activity; Physical activity

Definition

Any bodily movement produced by skeletal muscles that results in an expenditure of energy (Caspersen, Powell, & Christenson, 1985).

Description

The term exercise is often used interchangeably with the term physical activity but is more appropriately used to refer to leisure physical activity performed for fitness or pleasure.

Domains of Physical Activity

The way people spend their time can be categorized into five domains: sleep, leisure, occupational, transportation, and household (SLOTH; Pratt, Macera, Sallis, O'Donnell, & Frank, 2004). Physical activity can occur in each domain but is not likely to occur during sleep. Most efforts to promote physical activity have focused on leisure physical activity, which is activity engaged in during free time, often for fitness or pleasure. This includes working out, playing sports, and recreational walking. Occupational physical activity is any movement resulting in energy expenditure that occurs while working. A person working in manual labor likely engages in more occupational physical activity than someone working a desk job. Transportation physical activity is activity engaged in for the purpose of getting from one place to another, such as walking or bicycling to a destination (e.g., grocery store). Transportation walking declined drastically during the twentieth century and is viewed as a promising domain for physical activity promotion (Handy, Boarnet, Ewing, & Killingsworth, 2002). Household physical activity includes household chores, such as washing dishes, and is not typically a focus of physical activity promotion.

Types of Physical Activity

There are four primary types of physical activity (Sallis & Owen, 1998). Aerobic activity (also called cardiovascular exercise) involves large muscle movement for a sustained period of time. Examples include walking, running, bicycling, and swimming. Aerobic activity is often the focus of physical activity promotion efforts. Anaerobic activity involves muscles working against an applied force, such as resistance training and weightlifting. Bone-strengthening activity involves weight-bearing

exercises that strengthen the body's bones, such as squats and body extensions. Flexibility exercises, such as stretching, are those that increase the range of movements of joints and muscles.

Physical Activity Intensity levels

Movement is commonly classified into four intensity levels based on the amount of energy used by the body per minute of activity. Sedentary activity refers to no or little body movement and often involves sitting (for more information, see "► Sedentary Behaviors"). Light-intensity physical activity refers to slight body movements that lead to energy expenditure but are not strong enough to be considered moderate or vigorous. Moderate-intensity physical activity is the primary intensity level promoted in behavioral medicine. Moderate-intensity physical activity is often defined as activity during which a person's heart rate is 50–70% of his or her maximum heart rate (obtained by subtracting the person's age from 220). This generally includes brisk walking, dancing, gardening, and bicycling. Vigorous-intensity physical activity occurs when a person's heart rate is 70–85% of his or her maximum heart rate. Examples of vigorous-intensity activities include race walking, jogging, running, and hiking (Centers for Disease Control and Prevention (CDC), 2010).

Benefits of Physical Activity

Considerable evidence suggests that regular physical activity reduces the risk of many adverse health outcomes, such as cardiovascular disease, type 2 diabetes, some cancers, and mortality (Blair et al., 1996; Haskell, Blair, & Hill, 2009). Physical activity is also beneficial for mental health and quality of life and has been successfully used as a treatment for mental health disorders such as depression (Dunn, Trivedi, & O'Neal, 2001). Most health benefits occur with at least 150 min a week of moderate-intensity physical activity; additional benefits occur with more physical activity, and some physical activity is better than none (U.S. Department of Health and Human Services, 2008). For more information on health-related benefits of physical activity, see "► Physical Activity and Health, ► Physical Activity."

Physical Activity Recommendations

The US Department of Health and Human Services produced physical activity guidelines for children/adolescents, adults, and older adults (U.S. Department of Health and Human Services, 2008). The recommendations are as follows:

- Children and adolescents should engage in at least 60 min of physical activity daily, including vigorous-intensity physical activity at least three days a week. As part of their 60 min or more of daily physical activity, children and adolescents should include muscle-strengthening physical activity on at least three days of the week and bone-strengthening physical activity on at least three days of the week.
- Adults should engage in at least 150 min of moderate intensity, or at least 75 min of vigorous-intensity physical activity per week. Aerobic activity should be performed in episodes of at least 10 min. Adults should also do muscle-strengthening activities that are moderate or high intensity and involve all major muscle groups on two or more days a week.
- Older adults ($\geq$ age 65) have the same guidelines as other adults, except when older adults cannot do 150 min of moderate-intensity aerobic activity a week because of chronic conditions, they should be as physically active as their abilities and conditions allow.

Prevalence of Physical Activity in the United States

Nationally representative data showed that in 2007, 64.5% of US adults met the US Department of Health and Human Services physical activity guidelines. Women, older adults, people of racial/ethnic minority, less educated people, and those who were obese reported lower amounts of physical activity (Carlson et al., 2008). This data is based on how much physical activity people report they are doing. Objective physical activity rates were measured in 2004 using physical activity monitoring devices. Results were that fewer than 42% of children, 8% of adolescents, and 4% of adults met physical activity guidelines (Troiano et al., 2008). Although physical activity prevalence rates vary drastically when measured

with self-report versus physical activity monitors, prevalence rates found using each method point to a need for increasing physical activity.

Cross-References

- ▶ Aerobic Exercise
- ▶ Benefits of Exercise
- ▶ Exercise Testing
- ▶ Physical Activity and Health
- ▶ Physical Activity Interventions
- ▶ Physical Inactivity

References and Readings

Blair, S. N., Kampert, J. B., Kohl, H. W., 3rd, Barlow, C. E., Macera, C. A., Paffenbarger, R. S., Jr., & Gibbons, L. W. (1996). Influences of cardiorespiratory fitness and other precursors on cardiovascular disease and all-cause mortality in men and women. *Journal of the American Medical Association, 276*(3), 205.

Carlson, S. A., Fulton, J. E., Galuska, D. A., Kruger, J., Lobelo, F., & Loustalot, F. V. (2008). Prevalence of self-reported physically active adults-United states, 2007. *MMWR. Morbidity and Mortality Weekly Report, 57*, 1297–1300.

Caspersen, C. J., Powell, K. E., & Christenson, G. M. (1985). Physical activity, exercise, and physical fitness: Definitions and distinctions for health-related research. *Public Health Reports, 100*(2), 126.

Centers for Disease Control and Prevention (CDC). (2010). *Physical activity for everyone*. Retrieved December 1, 2010, from http://www.cdc.gov/physicalactivity/everyone/measuring/index.html

Dunn, A. L., Trivedi, M. H., & O'Neal, H. A. (2001). Physical activity dose-response effects on outcomes of depression and anxiety. *Medicine & Science in Sports & Exercise, 33*(6), S587.

Handy, S. L., Boarnet, M. G., Ewing, R., & Killingsworth, R. E. (2002). How the built environment affects physical activity. *American Journal of Preventive Medicine, 23*(2 S), 64–73.

Haskell, W. L., Blair, S. N., & Hill, J. O. (2009). Physical activity: Health outcomes and importance for public health policy. *Preventive Medicine, 49*(4), 280–282.

Pratt, M., Macera, C. A., Sallis, J. F., O'Donnell, M., & Frank, L. D. (2004). Economic interventions to promote physical activity: Application of the SLOTH model. *American Journal of Preventive Medicine, 27*(3), 136–145.

Sallis, J., & Owen, N. (1998). *Physical activity and behavioral medicine*. Thousand Oaks, CA: Sage.

Troiano, R. P., Berrigan, D., Dodd, K. W., Masse, L. C., Tilert, T., & McDowell, M. (2008). Physical activity in the united states measured by accelerometer. *Medicine & Science in Sports & Exercise, 40*(1), 181–188.

U.S. Department of Health and Human Services. (2008). *2008 physical activity guidelines for Americans*. Retrieved April 14, 2012 from http://www.health.gov/paguidelines/guidelines/default.aspx

Exercise and Cancer

- ▶ Cancer and Physical Activity

Exercise Testing

Alexandre Morizio and Simon Bacon
Department of Exercise Science, Concordia University, Montreal Behavioral Medicine Centre, Montreal, QC, Canada

Synonyms

Physical fitness testing; Stress testing

Definition

Exercise testing is a method used to evaluate a number of physiological parameters and conditions, such as heart and lung capacities and pathologies, as well as physical ability. Clinical exercise tests, which evaluate vital organ functioning, are typically designed to incorporate large muscle groups, and these tests use modalities such as treadmills or cycle ergometers. Maximal exercise testing protocols are structured to be progressive to the point of exhaustion, whereas submaximal exercise tests are conducted at a lower exercise intensity in a single-stage or multistage protocol and are terminated at a predetermined point.

An individual performs an exercise test, and data is collected by the test administrator. The collected data/information can then be analyzed to assess the nature of the physiological measure or disease/condition that the test was specifically designed to evaluate. Exercise tests can not only be used as diagnostic tests, such as in the case

of evaluating heart conditions, but are also an important part of designing safe exercise programs for patients with a chronic disorder. In addition, exercise tests can also be used to monitor the improvements or detriments resulting from the individual's treatments or physical training: an exercise test is conducted before the treatment to establish a baseline and then conducted again after the treatment; the effects of the treatment can then be evaluated.

It is also important to note that, in the case of exercise testing batteries, where more than one exercise test will be administered at a given time, the order of the tests may alter the results. For example, nonfatiguing exercise tests and exercise tests requiring muscular coordination should be conducted before fatiguing tests so as to not tire out the individual prematurely. By carefully selecting the order of the tests in the exercise testing battery, one can ensure more accurate results.

Cross-References

- ▶ Exercise
- ▶ Graded Exercise
- ▶ Isometric/Isotonic Exercise

References and Readings

American College of Sports Medicine, Thompson, W. R., Gordon, N. F., & Pescatello, L. S. (2010). *ACSM's guidelines for exercise testing and prescription* (8th ed.). Philadelphia: Wolters Kluwer Health/ Lippincott Williams & Wilkins.

Baechle, T. R., Earle, R. W., & National Strength & Conditioning Association. (2008). *Essentials of strength training and conditioning* (3rd ed.). Champaign, IL: Human Kinetics.

Froelicher, V. F., & Myers, J. (2007). *Manual of exercise testing* (3rd ed.). Philadelphia: Mosby.

Weisman, I. M., & Zeballos, R. J. (2002). *Clinical exercise testing*. Basel/New York: Karger.

Exercise Tolerance Test

- ▶ Maximal Exercise Stress Test

Exercise, Benefits of

- ▶ Isometric/Isotonic Exercise

Exercise-General Category

- ▶ Isometric/Isotonic Exercise

Exhaustion

- ▶ Fatigue

Expanded Attributional Style Questionnaire (EASQ)

- ▶ Optimism and Pessimism: Measurement

Expectancy

- ▶ Nocebo and Nocebo Effect

Expectancy Effect

- ▶ Placebo and Placebo Effect

Experience Sampling

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

ESM; Experience sampling method

Definition

The experience sampling method (ESM) is an attempt to provide a valid instrument to describe variations in self-reports of mental processes. It can be used to obtain empirical data on the following types of variables: (a) frequency and patterning of daily activity, social interaction, and changes in location; (b) frequency, intensity, and patterning of psychological states, i.e., emotional, cognitive, and conative dimensions of experience; (c) frequency and patterning of thoughts, including quality and intensity of thought disturbance (Csikszentmihalyi & Larson, 1987).

ESM represents a valuable way of assessing clinical phenomena in real-world settings and across time. It can be used in various settings. In ESM studies, participants are required to fill in questions about their current thoughts, feelings, and experiences when prompted by an electronic device (e.g., a wristwatch, PDA). Entries are typically made at fixed or random intervals over a period of days (a week is a typical period). Briefing, debriefing, which sampling procedure to use, adherence, data management, and analytical issues must be considered carefully in the study design phase to ensure optimum data collection and hence optimum results from the study.

A growing body of research suggests that momentary assessment technologies that sample experiences in the context of daily life represent a useful and productive approach in the study of behavioral phenotypes, and a powerful addition to mainstream cross-sectional research paradigms (Myin-Germeys et al., 2009). These authors described momentary assessment strategies for psychopathology and presented a comprehensive review of research findings illustrating the added value of daily life research for the study of (1) phenomenology, (2) etiology, (3) psychological models, (4) biological mechanisms, (5) treatment, and (6) gene-environment interactions in psychopathology. They concluded that variability over time and dynamic patterns of reactivity to the environment are essential features of psychopathological experiences that need to be captured for a better understanding of their phenomenology and underlying mechanisms (Myin-Germeys et al.).

The last decade has seen an increase in the number of studies employing the ESM in clinical research (see Trull & Ebner-Priemer, 2009). Further research is needed to examine the optimal equipment and procedure for different clinical groups. Consider for example psychiatric studies. Despite its theoretical advantages, using this methodology in psychiatric populations is challenging (Palmier-Claus et al., 2011).

Cross-References

- ▶ Adherence
- ▶ Gene-Environment Interaction
- ▶ Phenotype

References and Readings

Csikszentmihalyi, M., & Larson, R. (1987). Validity and reliability of the experience-sampling method. *Journal of Nervous and Mental disease*, *175*, 526–536.

Myin-Germeys, I., Oorschot, M., Collip, D., Lataster, J., Delespaul, P., & van Os, J. (2009). Experience sampling research in psychopathology: Opening the black box of daily life. *Psychological Medicine, 39*, 1533–1547.

Palmier-Claus, J. E., Myin-Germeys, I., Barkus, E., Bentley, L., Udachina, A., Delespaul, P. A., et al. (2011). Experience sampling research in individuals with mental illness: Reflections and guidance. *Acta Psychiatrica Scandinavica, 123*, 12–20.

Sullivan, T. P., Khondkaryan, E., Dos Santos, N. P., & Peters, E. N. (2011). Applying experience sampling methods to partner violence research: Safety and feasibility in a 90-day study of community women. *Violence Against Women, 17*, 251–266.

Trull, T. J., & Ebner-Priemer, U. W. (2009). Using experience sampling methods/ecological momentary assessment (ESM/EMA) in clinical assessment and clinical research: Introduction to the special section. *Psychological Assessment, 21*, 457–462.

Experience Sampling Method

- ▶ Experience Sampling

Experimental Analyses

▶ Hypothesis Testing

Experimental Designs

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

There are two fundamental types of study design: experimental and nonexperimental (Piantadosi, 2005). Piantadosi defined an experiment (an experimental design) as a series of observations made under conditions in which the influences of interest are controlled by the research scientist. In contrast, in nonexperimental studies, the research scientist collects observations but does not exert control over the influences of interest.

The classic example of an experimental design is the randomized clinical trial, in which the subjects (participants) are randomized to one of two or more experimental groups, thus receiving the intervention given to all members of each group. The simplest form of this design contains an experimental group receiving the intervention of interest (e.g., a behavioral intervention to lower blood pressure) while a second group receives a control intervention. The results obtained for each group are then compared to examine any statistically significant and clinically significant differences between the groups.

Two commonly used designs are the crossover design and the parallel groups design. In the first, each subject will receive each intervention, while in the second different groups of subjects receive just one of the interventions. When possible, the crossover design is preferable since each subject acts as his or her control subject.

Cross-References

▶ Crossover Design
▶ Nonexperimental Designs
▶ Parallel Group Design
▶ Randomized Clinical Trial

References and Readings

Piantadosi, S. (2005). *Clinical trials: A methodologic perspective* (2nd ed.). Hoboken, NJ: Wiley.

Experimental Group

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

As noted in the ▶ Experimental Designs entry, experimental designs are those in which the influence(s) of interest are controlled by the research scientist. An experimental group is a group of subjects who receive a particular treatment or intervention. Experimental subjects are randomly assigned to one of the treatment groups so that many potential influences that cannot be controlled for (e.g., sex, height, and weight) are likely to be as frequent in one experimental group as they are in the other.

It should be noted that the term "treatment group" is related to the term "experimental group," but they are not synonymous. Experimental groups can be thought of as a subset of treatment groups, i.e., groups formed by research scientists before administering the treatment or intervention of interest. Treatment groups can be formed retrospectively. For example, a research scientist may wish to collect follow-up data for patients who received two kinds of intervention for the same illness or condition. A simple example might be to determine the percentages of patients still alive 10 years following the

cessation of Treatment A and Treatment B, two treatments given for the same serious condition. Such individuals could be classified as Treatment Group A and Treatment Group B. A meaningful comparison in this case would require the identification of groups of patients who were as similar as possible in every other regard except which treatment they received, a challenge common to many retrospective research strategies.

Cross-References

▶ Experimental Designs

References and Readings

Piantadosi, S. (2005). *Clinical trials: A methodologic perspective* (2nd ed.). Hoboken, NJ: Wiley.

Explanations

▶ Attribution Theory

Explanatory Models of Illness

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

This term refers to the manner in which patients explain their health conditions and consequences. Much theoretical work has been done in health psychology/behavior medicine on this topic, and it has been applied to prediction of patient coping and adherence with treatments and to prediction of disease outcomes. An important part of appraising an illness is making sense of it, and patients often search for causes to which they attribute the onset of an illness. As such, these "explanations" reflect patients' explanatory models of illness, in contrast with scientifically based models of illness. One may contrast explanatory models that attribute diseases to physical causes with those that attribute disease to psychosocial causes. They first may include one's genetic profile, an underlying pathophysiological process (e.g., inflammation), injury, or infectious agents, to mention but a few examples. The second include "stress," often a generic term referring to a life event or events, one's mental state (e.g., depression, anxiety, anger) or psychosocial context such as exposure to violence, poverty, or solitude, to mention but a few examples. Murdock (1980) found that among 139 societies worldwide, the prevailing causal attribution of illnesses was psychosocial.

One of the most scientifically tested explanatory models of illness is the Common Sense Model of Leventhal, Diefenbach, and Leventhal (1992). This model is a self-regulation model – people react to and regulate their behavior in light of an illness. The model's core is a patient's illness representation – a set of cognitions which guide patients in coping with, making sense of, and adapting to an illness. These cognitions include illness identity (label of a condition and its symptoms), cause (perceived or attributed cause, not necessarily biomedically-based), timeline (expected duration of an illness), consequences (physical and social consequences), and curability/controllability (extent of doctor and patient control over the illness). Illness representations, the core of patients' explanatory model, are dynamic and change over the course of one's experience with an illness. This model has been tested in relation to coping and outcomes of arthritic patients, psoriasis, multiple sclerosis, cardiac surgery, to name but a few examples (e.g., Hale, Treharne, & Kitas, 2007). Other investigators have proposed that explanatory models of illness are part of a cultural context of making sense of illness, deriving from one's personal and social experiences. Yet, these models can often exacerbate rather than ameliorate, especially medically unexplained somatic symptoms (Kirmayer & Sartorius, 2007). Thus, while explanatory models of illness are pivotal to

understanding how people understand and cope with their illnesses, these can have important implications for their well-being. Hence, explanatory models of illness can also be targets of therapeutic interventions.

Cross-References

- ▶ Common-Sense Model of Self-regulation
- ▶ Illness Perceptions Questionnaire (IPQ-R)

References and Readings

Hale, E. D., Treharne, G. J., & Kitas, G. D. (2007). The common-sense model of self-regulation of health and illness: How can we use it to understand and respond to our patients' needs? *Rheumatology, 46*, 904–906.

Kirmayer, L. J., & Sartorius, N. (2007). Cultural models and somatic syndromes. *Psychosomatic Medicine, 69*, 832–840.

Leventhal, H., Diefenbach, M., & Leventhal, E. A. (1992). Illness cognition: Using common sense to understand treatment adherence and affect cognition treatment. *Cognitive Therapy and Research, 16*(2), 143–163.

Murdock, G. P. (1980). *Theories of illness: A world survey*. Pittsburgh, PA: University of Pittsburgh Press.

Explanatory Style

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

This term refers to the manner in which people explain events in their lives and is considered a trait characteristic. Stemming from the pioneering work on learned helplessness by Overmier and Seligman (1967), this phenomenon became important for its relevance to learning and to the understanding of the etiology of depression. Animals initially exposed to uncontrollable stress generalized their lack of control to a controllable stressful situation, manifesting what was termed "learned helplessness." Such animals even continued to receive adversities (e.g., small shocks) with little attempt to control them, though they were in a novel but controllable situation. However, when extrapolating similar studies to humans (mostly using uncontrollable noise stress), it was found that not all people developed learned helplessness. In an attempt to explain this outcome, Abramson, Seligman, and Teasdale (1978) found that people exposed to uncontrollable stress, and who attribute this stress to internal and stable causes which have global effects on their lives, develop learned helplessness and later depression. This pattern of cognitions including internal, stable, and global attributions for negative events was termed "explanatory style." This theoretical change reflected the consideration of a situation × personality interaction in relation to outcomes.

In 1986, Sweeney, Anderson, and Bailey (1986) reviewed over 100 studies with various methodologies on explanatory style and depression. They found that following a negative uncontrollable stressor, people attributing its cause to an internal (rather than external cause), a stable (rather than unstable) cause, of global implications (rather than specific implications), had greater risk of depression. Conversely, people attributing a positive event to external and unstable causes of specific implications are also at risk for depression. The construct can be assessed in several manners, namely, by the Attribution Style Questionnaire (Peterson et al., 1982) or by using the Content Analysis for Verbatim Explanations (CAVE, Schulman et al., 1989) to analyze written texts.

The concept of explanatory style has received important predictive validity, some of crucial relevance to behavior medicine. For example, a study by Peterson, Seligman, and Vaillant (1988) found that a negative explanatory style predicted risk of physical illnesses, over a 35-year follow-up period. In that study, explanatory style was based on open-ended questions, completed by Harvard University students when they were at age 25. Pessimistic explanatory style predicted more risk of poor physical health at the ages of 45–60, after controlling statistically for baseline physical and mental health.

Another study found that a negative explanatory style predicted higher risk of nonfatal and fatal myocardial infarction (MI) in the Normative Aging Study (Kubzansky, Sparrow, Vokonas, & Kawachi, 2001). In contrast, Tomakowsky, Lumley, Markowitz, and Frank (2001) found that a negative explanatory style predicted a smaller decline in CD4 counts but more subjective symptoms in HIV patients. Thus, more evidence supports the notion that a negative explanatory style predicts poor health; though in some samples, this is not the case. A negative explanatory style can also serve as a framework for cognitive interventions in treating depressive people, given the relative consistent role of explanatory style in depression and given its clear structure and functions.

Cross-References

► Depression: Symptoms

References and Readings

Abramson, L. Y., Seligman, M. E. P., & Teasdale, J. D. (1978). Learned helplessness in humans: Critique and reformulation. *Journal of Abnormal Psychology, 87*, 49–74.

Kubzansky, L. D., Sparrow, D., Vokonas, P., & Kawachi, I. (2001). Is the glass half empty or half full? A prospective study of optimism and coronary heart disease in the normative aging study. *Psychosomatic Medicine, 63*, 910–916.

Overmier, J. B., & Seligman, M. E. P. (1967). Effects of inescapable shock upon subsequent escape and avoidance responding. *Journal of Comparative and Physiological Psychology, 63*, 28–33.

Peterson, C., Semmel, A., von Baeyer, C., Abramson, L. T, Metalsky, G. I., and Seligman, M. E. P. (1982). The attributional style questionnaire. *Cognitive Therapy and Research*, *6*, 287–300.

Peterson, C., Seligman, M. E. P., & Vaillant, G. (1988). Pessimistic explanatory style as a risk factor for physical illness: A thirty-five year longitudinal study. *Journal of Personality and Social Pychology, 55*, 23–27.

Schulman, P., Casetellon, C., and Seligman, M. E. P. (1989). Assessing explanatory style: The content analysis of verbatim explanations and the attributional style questionnaire. *Behaviour Research and Therapy*, *27*, 505–512.

Sweeney, P. D., Anderson, K., & Bailey, S. (1986). Attributional style in depression: A meta-analytic review. *Journal of Personality and Social Psychology, 50*, 974–991.

Tomakowsky, J., Lumley, M. A., Markowitz, N., & Frank, C. (2001). Optimistic explanatory style and dispositional optimism in HIV-infected men. *Journal of Psychosomatic Research, 51*, 577–587.

Expression Pattern

► Gene Expression

Expressive Writing and Health

Stephen J. Lepore[1] and Wendy Kliewer[2]
[1]Department of Public Health, Temple University, Philadelphia, PA, USA
[2]Department of Psychology, Virginia Commonwealth University, Richmond, VA, USA

Synonyms

Written disclosure

Definition

Expressive writing is a form of therapy in which individuals write about their thoughts and feelings related to a personally stressful or traumatic life experience. Expressive writing is sometimes referred to as written disclosure, because writers are instructed to disclose personal information, thoughts, and feelings. Unlike communicative forms of writing, expressive writing is personal, free flowing, and informal, often without concern for style, spelling, punctuation, or grammar.

Description

Origins

Expressive writing resembles journaling, which had its heyday the 1970s following the publication

of Ira Progoff's book, *At a Journal Workshop*. In the late 1980s, researchers James Pennebaker and Sandra Klihr Beall conducted one of the earliest controlled scientific investigations into the therapeutic effects of expressive writing. In that study, college students in an expressive writing intervention condition wrote for 15 min on 4 consecutive days about the "most traumatic or upsetting experiences" of their lives, while their counterparts in a control group wrote about superficial topics. Students who wrote about the facts and their associated feelings surrounding a life trauma evidenced short-term increases in arousal and negative mood, but also evidenced fewer health center visits months after the writing intervention relative to controls. Subsequently, dozens of studies have investigated the potential power of writing to bring about benefits in behavioral, psychological, and physical health outcomes. In the 1990s and early 2000s, investigators began to focus more on understanding how expressive writing influenced such a broad array of outcomes ranging from preventing depressive symptoms and health center visits for illness to altering immune functioning and working memory. Work on identifying theoretical mechanisms continues to this day, but mostly current research focuses on testing the efficacy of expressive writing for mitigating problems in an ever widening range of populations, including children and various high-risk or clinical populations (Lepore & Smyth, 2002).

Expressive Writing Interventions and Variations

Expressive writing interventions are usually quite brief, consisting of several 15- to 20-min writing sessions spread over multiple days. Benefits have been observed in interventions using just a single writing session or as many as 8 weekly sessions, but there is some evidence that effects are most powerful when the writing sessions last at least 15 min and are repeated at least three times. Typically, investigators instruct participants to write about nonspecific traumas of their choosing, but a growing number of studies have focused on evaluating the potential benefits of writing about specific traumas and stressful life events (see Box 1 for sample instructions). Whereas writing about major upheavals can be broadly beneficial, focused writing about a specific topic also appears to confer benefits. For example, writing about an upcoming graduate school examination has been shown to reduce depressive symptoms, and writing about breast cancer resulted in greater declines in physical symptoms when compared with control writing.

Whereas the core instructions of writing about one's deepest thoughts and feelings surrounding a traumatic or stressful life event are apparent in most expressive writing studies, investigators have experimented with the procedure in an effort to isolate mechanisms of action or to improve upon the intervention. For example, the writing might be private or shared with an investigator, conducted in a single writing session or multiple sessions, possibly include booster sessions, focus on past or ongoing events, consider either positive or negative aspects of life stressors. In addition, there have been variations in location of writing (e.g., home versus laboratory), as well as mode of writing (e.g., longhand versus typing). It appears that the beneficial effects of expressive writing are robust, resulting in benefits despite variations in instructions, settings, and procedures. The effects of expressive writing are somewhat stronger when people write at home or in a private setting, the outcomes are measured within a month after writing rather than later in time, the writing focuses on recent or previously undisclosed stressors, and the instructions provide directed questions, information on switching topics, and specific examples of what to disclose in writing. Some variations in the writing instructions have little or no effect on the benefits of writing, including the spacing between the writing, the positive or negative valence of the writing, whether the writer or the experimenter selects the topic of writing, or the mode of writing.

Effects of Expressive Writing

As shown in Box 2, the effects of expressive writing interventions are quite broad. Scholars have examined the impact of expressive writing on physiological functioning, self-reported health

Box 1. Sample Expressive Writing Instructions

A. Writing About Self-identified Stressors

For this writing exercise, please write for 15 min about your very deepest thoughts and feelings about the most traumatic experience of your life or an extremely important emotional issue that has affected you and your life. In your writing, really let go and explore your deepest thoughts and feelings. You might tie your topic to your relationships with others, including parents, lovers, friends or relatives; to your past, your present or your future; or to who you have been, who you would like to be, or who you are now. You may write about the same or different issues, experiences and topics each day. All of your writings will be confidential. Don't worry about spelling, grammar or sentence structure. The only rule is that once you begin writing, you continue until the time is up.

B. Writing About Specific Stressors

For this writing exercise, please write for 15 min about your deepest thoughts and feelings concerning your cancer. For example, you might write about the various ways the cancer has changed your life, what your life was like before the diagnosis, after diagnosis, during treatment and now. For some people, dealing with a cancer diagnosis is just one of many stressors in their life. You do not have to limit your writing to how cancer has affected your life. You may focus on other highly upsetting experiences in your life. The most important thing is that you should explore your very deepest emotions and thoughts. All of your writings will be confidential. Don't worry about spelling, grammar or sentence structure. The only rule is that once you begin writing, you continue until the time is up.

and health behaviors, psychological well-being, attitudes, and general life functioning. Meta-analytic reviews have revealed modest but significant effects of expressive writing across diverse outcomes and populations (Frattaroli, 2006; Frisina, Borod, & Lepore, 2004). Recent work has extended earlier investigations by studying the effects of expressive writing on outcomes as varied as emotional intelligence, workplace incivility, homesickness, caregiving stress, and gay-related stress.

Both objective and subjective indicators of physical health have been examined in response to expressive writing. Some of the most striking effects of expressive writing interventions are on physiological outcomes, including immune parameters (e.g., IL-8, CD8, T-helper lymphocytes, T-cytotoxic lymphocytes, Epstein-Barr antibodies), HIV viral load, and liver functioning. What is not yet clear is whether the changes in the immune parameters and other biomarkers are clinically meaningful. The evidence linking expressive writing to other theoretically plausible and objective biological outcomes has not always been positive, with some studies failing to find significant effects on outcomes such as blood lipids, blood pressure, lung capacity, heart rate, strength, joint condition, and body composition. Effects on self-reported physical health are fairly robust. Expressive writing reduces health-care utilization, pain, somatic illness symptoms (e.g., upper respiratory illness symptoms), disease severity ratings, and illness behaviors. However, with the exception of some studies that have found effects of writing on a healthy diet, most analyses have failed to show effects on health behaviors, including physical activity, substance use, sleep, and adherence to medical treatment. Thus, health behaviors are not likely mediators of the health benefits of expressive writing interventions.

In general, the effects of expressive writing on psychological health and well-being are weaker

Box 2. Observed Benefits of Expressive Writing

A. Physical health and physiological functioning.
   1. Objective measures
      Immune parameters
      HIV viral load
      Liver functioning
   2. Subjective measures
      Health care utilization
      Pain
      Disease severity ratings
      Illness behaviors

B. Psychological well-being.
   Depression
   Positive attitudes
   Positive mood

C. Role functioning and related outcomes.
   1. Work-related behaviors
      Re-employment
      Absenteeism
      Incivility in the workplace
   2. Social relationships
   3. Cognitive functioning
      Working memory
      Reaction time
   4. School outcomes
      Grade point average
      Adjustment to college life
      Adjustment to high school

than for physical health. Studies of psychological well-being have included assessments of depression, positive and negative mood, anger, aggression, grief, distress, anxiety, post-traumatic stress and growth, dissociation, adjustment to school, coping, cognitive schemas, and emotion regulation. Consistently strong effects have been observed for depression and positive attitudes and mood; effects are equivocal for post-traumatic growth and for anxiety, with some studies showing heightened anxiety in response to writing and others showing reductions in anxiety. There is no evidence that expressive writing has reliable effects on coping, cognitive schemas, or self-regulation.

As research on expressive writing has blossomed, scholars have extended outcomes beyond physical and psychological well-being to include broader indicators of functioning. There is strong evidence that expressive writing interventions affect work-related behaviors such as reemployment, absenteeism, and incivility in the workplace; the quality of social relationships, including forgiveness; cognitive functioning such as working memory and reaction time; and school outcomes, such as grade point averages and adjustment to college life.

Who Benefits from Expressive Writing?

One way to understand who benefits from expressive writing is to examine the kinds of populations that respond favorably to the intervention. Many of the early studies on expressive writing focused on nonclinical populations. Indeed, healthy college students have been the subjects in numerous expressive writing studies. In the past 15 years, though, an impressive number of clinical trials have been conducted involving high-risk and clinical populations, and even youth. Another way to understand who benefits from expressive writing is to look for subgroups of writers in intervention studies who benefit relatively more or less than other writers in the intervention.

Expressive writing interventions have been applied to populations confronting a wide range of clinical health problems, including cancer, arthritis, asthma, post-traumatic stress disorder, HIV infection, cystic fibrosis, chronic pain, and sleep disorders. There is evidence of benefits of writing in clinical populations, but it is mixed. For example, one expressive writing intervention study showed improvements in lung function and physician-rated disease severity, respectively, in participant with asthma or rheumatoid arthritis. However, these findings have not been replicated in follow-up studies. Perhaps the most frequent clinical population targeted for expressive writing interventions has been cancer survivors. The results have been mixed. Some studies have reported select benefits in cancer populations, such as reduced postoperative medical illness visits in good prognosis breast cancer survivors,

better sleep quality among renal cancer patients, and reduced pain perception among prostate cancer survivors; yet just as many studies have reported absolutely no significant benefits of expressive writing. In only a few trials with clinical populations, specifically participants with post-traumatic stress disorder, have possibly serious adverse effects been identified. It is possible that for psychiatric populations writing should be guided by a therapist and used only as an adjuvant to more traditional therapies. The vast majority of studies with clinical populations suggest that it is a safe intervention, if not particularly powerful and reliable. The quality of the interventions, writing instructions, measured outcomes, settings, and time to follow-up vary tremendously from one clinical trial to the next, so the evidence of the efficacy of this intervention with clinical populations is still inconclusive.

Although most expressive writing interventions are conducted with adults, there are a growing number of randomized clinical trials that have been conducted with children and adolescents. Approximately half of these trials have been conducted with clinical populations outside of the school context; the remaining trials have been conducted in a school context with general populations of youth. The findings with youth have not been as promising as findings with adults. Across studies, effects of writing on internalizing symptoms have been equivocal, with fewer than half of the trials reporting improvements on measures of psychological well-being. With respect to indicators of physical health, no study has reported improvements in somatic complaints due to writing, but several studies with small samples have reported improvements in functional ability and declines in medical and emergency room visits. Some of the equivocal findings with youth may be due to use of writing with youth who do not have the cognitive capacity to process their stressful experiences. Additionally, youth who are not dealing with significant stressors may see their anxiety increase in response to writing interventions. Because writing uses few resources and fits into normal school activities, there is a potential for writing interventions to have a large impact on school populations provided that it is used with youth who have experienced significant stress and have the cognitive capacity to process their stressful experiences.

Individual studies and meta-analytic reviews have investigated whether specific subgroups of writers benefit more or less from the intervention, or whether identifiable factors alter or moderate the effects of expressive writing on outcomes. One problem with such analyses is that they cannot establish cause-effect relationships because other unmeasured variables might explain any observed differences between subgroups. Nonetheless, as evidence on subgroup differences and moderators accumulates, it might suggest feasible targets for intervention and methods for improving upon the intervention. Analyses in this vein have shown that a number of factors do not appear to alter the effects of expressive writing, including participant age, ethnicity, education level, severity of stressor or trauma, baseline psychological health levels, negative affectivity, and level of inhibition or prior disclosure status. One caveat, however, is that not all studies that have examined individual moderators have had adequate representation within all the levels of the subgroups, so it is possible that future research will derive different conclusions. Other factors do appear to make a difference, but the evidence is mixed, since effects fail to replicate or have not yet been investigated in multiple studies. There is some evidence that the individuals who benefit the most are male, have higher stress or physical health problems prior to writing, have lower optimism, perceive that they are socially constrained in talking about their stress, have no difficulty in identifying and labeling emotions, and do not habitually repress negative emotions.

How Does Expressive Writing Work?

There are two dominant theoretical models to explain the array of beneficial effects of expressive writing, the disinhibition model and the self-regulation model. Relatively few studies have directly tested the theoretical mechanisms explaining the benefits of expressive writing, and evidence on the validity of all of the

mechanisms is mixed (Lepore & Smyth, 2002; Sloan & Marx, 2004).

The disinhibition model is based on the notion that individuals inhibit or avoid thoughts, reminders, and feelings of traumatic life events because they are distressing and can evoke negative social responses. Inhibition potentially influences health via the chronic physiological strain and arousal caused by the work of inhibition. Expressive writing theoretically counteracts the adverse effects of inhibition by encouraging individuals to disinhibit themselves by disclosing their deepest trauma-related experiences and associated thoughts and feelings. Numerous writing studies have challenged this model. For example, individuals writing about non-inhibited future events, such as an upcoming graduate school entrance examination, reported significantly lower depressive symptoms than controls. In addition, the benefits of expressive writing appear to be equivalent whether individuals write about previously disclosed or non-disclosed traumas, or write about positive or negative aspects of past traumas.

The self-regulation model is based on the notion that individuals who have excessively high or low levels of control over their emotions have an elevated risk for health problems due to the pathophysiological effects of emotion dysregulation. Research supports the notion that emotion regulation relates to health outcomes. For example, there is evidence that the inhibition, or non-expression, of anger is associated with heightened physiological arousal, which appears relevant to cardiovascular health. However, there is also evidence that individuals with little control over their expression of anger have heightened levels of physiological arousal and risk for cardiovascular disease. Individuals who are optimally regulated in their expression of anger may be at the lowest risk for health problems. According to the self-regulation model, individuals experiencing stressful life events need to strike a balance between emotionally overreacting and underreacting. Expressive writing is thought to facilitate emotion regulation processes by directing attention, facilitating habituation, and aiding in cognitive restructuring. Briefly, by directing attention to different aspects of a stressful experience and one's emotional response and thoughts, expressive writing increases habituation (desensitization) to the negative thoughts and feelings associated with the stressor and potentially allows for the creation of new and less-threatening appraisals and feelings to be attached to the memories of the stressor. Consistent with this theory, there is evidence that expressive writing desensitizes individuals to stress-related thoughts. In addition, there is evidence that expressive writing can reduce stress-related intrusive thoughts, which may be symptomatic of incomplete cognitive processing of stressors. This evidence, however, is not consistent across studies.

Additional research is needed to better understand how expressive writing results in positive social, behavioral, and health outcomes. Although scholars have posited a variety of plausible social, psychological, and biological mechanisms, empirical evidence does not strongly support or rule out any particular explanation. All of the identified mechanisms may be sufficient to influence the outcomes linked to expressive writing, either directly or indirectly. It is likely that there is no single mechanism of action given the diversity of outcomes studied and the mixed evidence on each mechanism.

Cross-References

► Stress

References and Readings

Frattaroli, J. (2006). Experimental disclosure and its moderators: A meta-analysis. *Psychological Bulletin, 132*, 823–865.

Frisina, P. G., Borod, J., & Lepore, S. J. (2004). A meta-analysis of the effects of written emotional disclosure on health outcomes of clinical populations. *Journal of Nervous and Mental Disease, 192*, 629–634.

Lepore, S. J., & Smyth, J. (Eds.). (2002). *The writing cure: How expressive writing influences health and well-being*. Washington, DC: American Psychological Association.

Nyklicek, I., Temoshok, L., & Vingerhoets, A. (Eds.). (2004). *Emotional expression and health: Advances in theory, assessment and clinical applications*. New York: Taylor & Francis.

Pennebaker, J. W. (Ed.). (2002). *Emotion, disclosure, & health*. Washington, DC: American Psychological Association.

Sloan, D. M., & Marx, B. P. (2004). Taking pen to hand: Evaluating theories underlying the written disclosure paradigm. *Clinical Psychology: Science and Practice, 11*, 121–137.

Ex-Smokers

Marcia D. McNutt and Monica Webb Hooper
Department of Psychology, University of Miami, Coral Gables, FL, USA

Synonyms

Former smokers; Past smokers; Previous smokers

Definition

The term ex-smoker refers to an individual who has given up (i.e., quit) cigarette and/or tobacco smoking. Ex-smokers were previous current smokers, but are no longer smoking.

Tobacco smoking is defined as the practice of burning and inhaling tobacco, and cigarette smoking is the most common form of tobacco smoking. National surveys define a current smoker as an individual who has smoked at least 100 cigarettes in their lifetime and currently smokes on at least some days.

Description

Cigarette smoking is the most important modifiable risk factor for chronic disease; yet, there is not a consensus in the way smoking status is classified. The harmful effects of cigarette smoking on various health outcomes have been determined by comparing individuals who are (1) current smokers, (2) ex-smokers, and/or (3) never smokers. Therefore, the definition of an ex-smoker is important for making cross-study comparisons regarding the health consequences of smoking and cessation. Most national surveys ask whether a person has a history of smoking 100 lifetime cigarettes *and* whether they currently smoke on some days. Respondents who indicate "yes" to the first question and "no" to the second are categorized as ex-smokers. Other research specifies a time period of smoking cessation needed for ex-smoker classification; however, that time (e.g., 1 day, 1 week, 3 months, 5 years, etc.) varies across studies. Nonetheless, results of studies comparing current smokers with ex-smokers have shown unequivocally that quitting smoking, for even a relatively short time period, decreases the risk of chronic disease.

Because there is unquestionable evidence that cigarette smoking is the leading preventable cause of multiple cancers (e.g., lung and esophageal), heart disease, and stroke, attention has focused on the specific effect quitting smoking has on health. The evidence shows that quitting, even after an extended period of smoking, decreases the risk of associated illnesses. Moreover, the disease risk decreases as the number of years since quitting increases. The 1989 US Surgeon General's report indicated that after 10 years of smoking cessation, the risk of lung cancer is decreased by almost 50% (Centers for Disease Control and Prevention, 1989). Still, ex-smokers continue to have an increased risk of developing a chronic disease compared to never smokers. In comparing the three groups on chronic disease risk (i.e., current smokers, ex-smokers, and never smokers), ex-smokers have a reduced risk compared to current smokers, but they have approximately twice the risk compared to never smokers (Ebbert et al., 2003). The absolute risk of lung cancer remains higher among ex-smokers than never smokers even after smoking cessation (Halpern, Gillespie, & Warner, 1993). However, the excess chronic disease risk for an ex-smoker is reduced to that of a never smoker after 15 years of abstinence. Additionally, Thornton, Lee, and Fry (1994) found that recent ex-smokers were similar to current smokers in the prevalence of chronic

disease risk factors (e.g., low vegetable consumption); and ex-smokers who were smoke-free for 20+ years (i.e., long-term ex-smokers) were similar to never smokers. Compared to current smokers, ex-smokers are more likely to engage in healthy lifestyle behaviors (e.g., attempting to lose weight, cutting down on fatty foods, and increasing vegetable consumption; Thornton et al., 1994), which may further reduce chronic disease risk.

Relapse prevention is important for ex-smokers, considering that approximately 90% of people who quit return to smoking within one year (Brandon, Tiffany, Obremski, & Baker, 1990; Garvey, Bliss, Hitchcock, Heinold, & Rosner, 1992). Although the risk of relapse after a long period of time (i.e., 6–12 months) is relatively low, smoking even one cigarette after quitting is likely to lead to a full relapse. Even with the vacillating status of many ex-smokers, approximately 10% of those who relapse are able to quit again permanently in the future (Wetter et al., 2004). Therefore, interventions for relapse prevention among ex-smokers have been designed and evaluated. A systematic review by Agboola, McNeill, Coleman, and Bee (2010) found that bupropion and nicotine replacement therapy (e.g., the nicotine patch, nicotine gum, and nicotine lozenge) are efficacious in preventing relapse among ex-smokers who quit smoking using such aids. Self-help materials may also be useful in preventing relapse among ex-smokers who quit smoking on their own (Brandon et al., 2004).

In summary, ex-smokers reduce their risk of chronic disease the longer they abstain from smoking. Because exposure to smoke causes significant harm to the body, ex-smokers still have an increased risk of several diseases compared to those who have never smoked. Over time, however, successful ex-smokers are comparable to never smokers on several risk factors for chronic disease.

Cross-References

- ▶ Smoking and Health
- ▶ Smoking Cessation

References and Readings

Agboola, S., McNeill, A., Coleman, T., & Bee, J. L. (2010). A systematic review of the effectiveness of smoking relapse prevention interventions for abstinent smokers. *Addiction, 105*, 1362–1380.

Brandon, T. H., Meade, C. D., Herzog, T. A., Chirikos, T. N., Webb, M. S., & Cantor, A. B. (2004). Efficacy and cost-effectiveness of a minimal intervention to prevent smoking relapse: Dismantling the effects of amount of content versus contact. *Journal of Consulting and Clinical Psychology*, *72*(5), 797–808. doi:10.1037/0022-006X.72.5.797

Brandon, T. H., Tiffany, S. T., Obremski, K. M., & Baker, T. B. (1990). Postcessation cigarette use: The process of relapse. *Addictive Behaviors, 15*(2), 105–114. doi:10.1016/0306-4603(90)90013-N

Centers for Disease Control and Prevention. (1989). Reducing the health consequences of smoking: 25 years of progress-a report of the Surgeon General. Washington, DC: US Department of Health and Human Services, Public Health Service, Centers for Disease Control. DHHS Publication No. (CDC) 89–8411.

Ebbert, J. O., Yang, P., Vachon, C. M., Vierkant, R. A., Cerhan, J. R., Folsom, A. R., et al. (2003). Lung cancer risk reduction after smoking cessation: Observations from a prospective cohort of women. *Journal of Clinical Oncology: Official Journal of the American Society of Clinical Oncology, 21*(5), 921–926.

Garvey, A. J., Bliss, R. E., Hitchcock, J. L., Heinold, J. W., & Rosner, B. (1992). Predictors of smoking relapse among self-quitters: A report from the normative aging study. *Addictive Behaviors, 17*(4), 367–377. doi:10.1016/0306-4603(92)90042-T

Halpern, M. T., Gillespie, B. W., & Warner, K. E. (1993). Patterns of absolute risk of lung cancer mortality in former smokers. *Journal of the National Cancer Institute, 85*(6), 457–464.

Thornton, A., Lee, P., & Fry, J. (1994). Differences between smokers, ex-smokers, passive smokers and non-smokers. *Journal of Clinical Epidemiology, 47*(10), 1143–1162.

Wetter, D. W., Cofta-Gunn, L., Fouladi, R. T., Cinciripini, P. M., Sui, D., & Gritz, E. R. (2004). Late relapse/sustained abstinence among former smokers: A longitudinal study. *Preventive Medicine, 39*(6), 1156–1163. doi:10.1016/j.ypmed.2004.04.02

Extended Life Orientation Test (E-LOT)

- ▶ Optimism and Pessimism: Measurement

External Locus of Control

Gary Davis
Medical School Duluth, University of Minnesota, Duluth, MN, USA

Synonyms

Helplessness; Low self-efficacy

Definition

External locus of control is the belief that one's behavior will not lead to valued reinforcement that is available in the environment and therefore not under one's control. The occurrence of reinforcement is believed to be a function of factors out of one's control such as luck, chance, or randomness.

Description

External Locus of Control

External locus of control anchors one end of a continuum of the locus of control construct with the other end anchored by internal locus of control. The construct developed out of work by E. Jerry Phares and Julian Rotter in the 1950s at Ohio State University and was influenced strongly by Alfred Adler's earlier work on striving for superiority. Feelings of inferiority were thought to be associated with externality. Rotter published his initial paper containing the external locus of control construct in 1966 that included the now famous Internal-External Locus of Control Scale (I-E) to measure the locus of control construct. It has subsequently become one of the most frequently cited papers ever published in psychology. The construct was embedded in Rotter's social learning theory and reflects his strong belief in the importance of theoretical frameworks. Some research over the years has been criticized because of the tendency to use the scale and the construct in a manner disconnected from their theoretical home.

External locus of control is defined as the belief or expectation that one's behavior will not lead to valued reinforcement that is available in one's environment; rather, the occurrence of reinforcement is a function of factors out of one's control such as luck, chance, or randomness. In Rotter's terms, external locus of control is a generalized expectancy, meaning that across a range of situations, people who are externally oriented, would have the expectation that control over reinforcements lies outside of their control. In its simplest form, the potential for any behavior to occur is a function of the expectation for reinforcement and the value of that reinforcement. Early in his work, Rotter represented that by the equation BP (behavior potential) $= f$ (E (expectation) $\times$ RV (reinforcement value)).

External locus of control is commonly measured or assessed by the Internal-External Locus of Control Scale. The original scale has 29 items, including 6 filler items, in a forced choice format that requires subjects to agree or disagree with statements. High scores are in the direction of externality. Two examples of "external items" are: *I have often found that what is going to happen will happen* and *Getting a good job depends mainly on being in the right place at the right time*.

Not surprisingly, since creating new tests seems to be one of the things that psychologists do best, many scales have been spawned to measure the construct of locus of control since Rotter's original scale was published. Some of these have been developed as a result of dissatisfaction with the forced choice format and other methodological issues. Others have been developed for use with specific populations or environments such as with children, medical patients, and organizational settings.

Many dissertations and published research papers have included the external locus of control construct. This widespread interest probably reflects our almost natural inclination to be interested in our fate and factors that influence it. Examples of some of the research results on externality are that external locus of control has

been related to low self-efficacy, low self-esteem, helplessness, depression, low achievement motivation, low risk-taking, less independent thinking and greater conformity, and less creativity.

Of particular interest to researchers has been the question of the role that external locus of control plays in the maintenance of health and the adjustment to illness. For the past 30 years, numerous patient populations, both acutely and chronically ill, have been studied to learn about the impact of externality on their illness experiences. The findings generally lend support to the notion that externality influences illness experiences, but the results have not been consistent across patient groups. For example, externals have been found to ask fewer questions of health-care staff and have less information about their illnesses. But other research has found that at least with certain chronic illnesses, such as, diabetes, externals are about as informed as internals. An interesting line of research pursued the idea of matching treatment approaches in a congruent manner with locus of control orientation. For example, it has been found that there is little difference in cardiac rehabilitation outcomes when externals who are in a highly structured and regimented program are compared to internals who are involved in a more self-directed program. However, the inconsistency in research outcomes and very modest association of locus of control to health and illness behaviors clearly suggests that many factors, including the nature of the illness itself, interact with locus of control and influence health behaviors. This conclusion, that many variables interact with locus of control, also holds for health maintenance behaviors for which it may be intuitive to think that externals would be less likely to engage in preventive measures.

A notable contribution to this literature, and the subject itself of considerable investigation, is the Multidimensional Health Locus of Control Scales by Wallston. The MHLC is a group of three scales intended to assess beliefs about health status control and beliefs about control over illness and disease. Numerous other disease-specific scales (e.g., cancer, diabetes, pain) to measure external locus of control have been developed in recent years.

Cross-References

▶ Locus of Control

References and Readings

Hand, M. P. (2008). *Psychological resilience*: *The impact of positive and negative life events upon optimism, hope, and perceived locus of control*. Germany: VDM Verlag.

Lefcourt, H. M. (1983). *Research with the locus of control construct* (Developments and social problems, Vol. 2). New York: Academic Press.

Rotter, J. B. (1966). Generalized expectancies for internal versus external control of reinforcement. *Psychological Monographs, 80*(1), 1–28, Whole No. 609.

Wallston, K. A. (2005). The validity of the multidimensional health locus of control scales. *Journal of Health Psychology, 10*, 623–631.

Extrinsic Religiousness (Religiosity)

Kevin S. Masters
Department of Psychology, University of Colorado, Denver, CO, USA

Definition

Extrinsic religiousness (initially and still sometimes referred to as extrinsic religiosity) is characterized as religion that primarily serves other more ultimate ends rather than central religious beliefs per se. Thus, individuals described by extrinsic religiousness use their religion to fulfill more basic needs such as social relations or personal comfort, but "the embraced creed is lightly held or else selectively shaped to fit more primary needs" (Allport & Ross, 1967, p. 434).

Description

Extrinsic religiousness was first described by Gordon Allport and colleagues in the 1960s (see Allport & Ross, 1967) when investigating the possible reasons for discrepant findings in the area of religiousness and prejudice. At that time,

some studies demonstrated that religiousness was positively associated with prejudice, whereas other studies found the opposite. Allport hypothesized that one's religious orientation, or sentiment, may provide guidance in sorting out these findings. The construct of religious orientation was later clarified by Gorsuch (1994) to be a motivational variable.

Extrinsic religiousness has often been measured by the Religious Orientation Scale (Allport & Ross, 1967). More recently, Gorsuch and McPherson (1989) developed the I/E-Revised scale as a more psychometrically sound instrument. Based on previous work (Kirkpatrick, 1989), Gorsuch and McPherson anticipated, and subsequently verified, two subscales on the extrinsic scale, extrinsic-personal (Ep) and extrinsic-social (Es). A prototypic Ep item is "What religion offers me most is comfort in times of trouble and sorrow" and for Es "I go to church mainly because I enjoy seeing people I know there." Scholars in the psychology of religion have debated the relative strengths and weaknesses of the religious orientation construct (e.g., Kirkpatrick & Hood 1990; Masters, 1991), but it remains the most empirically investigated and heuristic construct in this area of work. Investigators attempting to determine the relations between religion and health have also turned to religious orientation. Smith, McCullough, and Poll (2003) in a meta-analytic study found that extrinsic religiousness was associated with higher levels of depressive symptoms. Similarly, Masters and Bergin (1992) provided a narrative review of the literature and found extrinsic religiousness to be related positively with depression, anxiety, and obsessive-compulsive symptoms, whereas McCullough and Willoughby (2009) reported that extrinsic religiousness may be negatively related to self-control. Recent investigations found extrinsic religiousness related to greater laboratory-induced cardiovascular reactivity in older adults (Masters, Hill, Kircher, Lensegrav Benson, & Fallon, 2004), and Masters and Knestel (2011) found that among a random sample of community dwelling adults, those characterized by extrinsic religiousness were more likely to be divorced, reported overall worse health, higher body mass index, greater cigarette use, and a higher number of daily drinks of alcohol than did those characterized as intrinsically religious. There were no differences in the percentages of individuals who were classified as extrinsically religious based on religious denomination. Nevertheless, it is not entirely clear how extrinsic religiousness may interact with religious denomination, and some have suggested that the construct, as currently conceptualized and measured, is more congruent with Protestant notions of religiosity and perhaps most appropriately applied to this religious group (Cohen, Pierce Jr., Chambers, Meade, Gorvine, & Koenig, 2005).

Cross-References

▶ Intrinsic Religiousness (Religiosity)

References and Readings

Allport, G. W., & Ross, J. M. (1967). Personal religious orientation and prejudice. *Journal of Personality and Social Psychology, 5*, 432–443.

Cohen, A. B., Pierce, J. D., Jr., Chambers, J., Meade, R., Gorvine, B. J., & Koenig, H. G. (2005). Intrinsic and extrinsic religiosity, belief in the afterlife, death anxiety, and life satisfaction in young Catholics and Protestants. *Journal of Research in Personality, 39*, 307–324.

Gorcush, R. L., & McPherson, S. E. (1989). Intrinsic/extrinsic measurement: I/E-revised and single-item scales. *Journal for the Scientific Study of Religion, 28*, 348–354.

Gorsuch, R. L. (1994). Toward motivational theories of intrinsic religious commitment. *Journal for the Scientific Study of Religion, 33*, 315–325.

Kirkpatrick, L. A. (1989). A psychometric analysis of the Allport-Ross and Feagin measures of intrinsic-extrinsic religious orientation. In D. O. Moberg and M. L. Lynn (Eds.), *Research in the social scientific study of religion, 1*, (pp. 1–31). Greenwich, CT: JAI Press.

Kirkpatrick, L. A., & Hood, R. W., Jr. (1990). Intrinsic-extrinsic religious orientation: The boon or bane of contemporary psychology of religion? *Journal for the Scientific Study of Religion, 29*, 442–462.

Masters, K. S. (1991). Of boons, banes, babies, and bath water: A reply to the Kirkpatrick and Hood discussion of intrinsic-extrinsic religious orientation. *Journal for the Scientific Study of Religion, 30*, 312–317.

Masters, K. S., & Bergin, A. E. (1992). Religious orientation and mental health. In J. F. Schumaker (Ed.), *Religion and mental health* (pp. 221–232). New York: Oxford University Press.
Masters, K. S., Hill, R. D., Kircher, J. C., Lensegrav-Benson, T. L., & Fallon, J. A. (2004). Religious orientation, aging, and blood pressure reactivity to interpersonal and cognitive stressors. *Annals of Behavioral Medicine, 28*, 171–178.
Masters, K. S., & Knestel, A. (2011). Religious orientation among a random sample of community dwelling adults: Relations with health status and health relevant behaviors. *The International Journal for the Psychology of Religion, 21*, 63–76.
McCullough, M. E., & Willoughby, B. L. B. (2009). Religion, self-regulation, and self-control: Associations, explanations, and implications. *Psychological Bulletin, 135*, 69–93.
Smith, T. B., McCullough, M. E., & Poll, J. (2003). Religiousness and depression: Evidence for a main effect and the moderating influence of stressful life events. *Psychological Bulletin, 129*, 614–636.

Eye Tracker

► Eye Tracking

Eye Tracking

Naum Purits[1] and Ingrid Söderback[2]
[1]Stockholm, Sweden
[2]Department of Public Health and Caring Science, Uppsala University, Uppsala, Sweden

Synonyms

Eye tracker; Gaze tracking

Definition

An eye tracker is a computerized system used to record the activity of eye movements and visual overt attention, hence making it possible to study human behavior. Data obtained from an eye tracker session is useful for testing everything that could be visually observed.

Description

Eye tracking as a means of measuring and monitoring eye movements is widely used in different fields of human behavior research, for example, cognitive psychology, psycholinguistics and reading research, neurophysiology, ophthalmology, usability and human-computer interaction studies, and market research.

Eye Movement Measurement Methodologies

There are four eye movement measurement techniques (Duchowski, 2003) as follows:

- Electrooculography (EOG) – eye movement recording method commonly used in the 1970s. It is based on measuring potential differences of electrodes placed on the skin close to the eye.
- Sclera Contact Lens/Search Coil Lens – an old and very precise eye movement–measuring method based on a contact lens placed on the eye with a reference object, for example, wire coil, attached to the lens.
- Photo-oculography (POG) or Video-oculography (VOG) – a number of eye movement–recording methods based on measurement of different features of the eyes such as shape of the pupil, position of the limbus, etc. The above mentioned methods mainly measure the position of the eye relative to the head.
- Video-Based Combined Pupil-Corneal Reflection (Remote Eye Tracking) – noninvasive eye tracking methods providing measurement of position of the eye in space (not relative to the head). This technique is widely available and most suitable for eye tracking in real time.

Basic Operating Principles of Remote Eye Trackers

Remote eye trackers, that is, Pupil-Corneal reflection eye trackers, use infrared diodes to generate reflection patterns on the corneas of the studied person's eyes. The system uses image sensors to collect images of the eyes and

Eye Tracking,
Fig. 1 Remote eye tracker Tobii T60XL

the reflection patterns. Sophisticated image processing algorithms identify relevant features, including the eyes and the corneal reflection patterns. Complex mathematics is used to calculate the position of each eyeball and finally the gaze point, in other words, where and on what the person is looking. The development of eye trackers is based on present neuroscience knowledge. Here the brain's physiological and functional processes represented of the Mango- and Parvo-cellular pathways are of great importance (Duchowski, 2003).

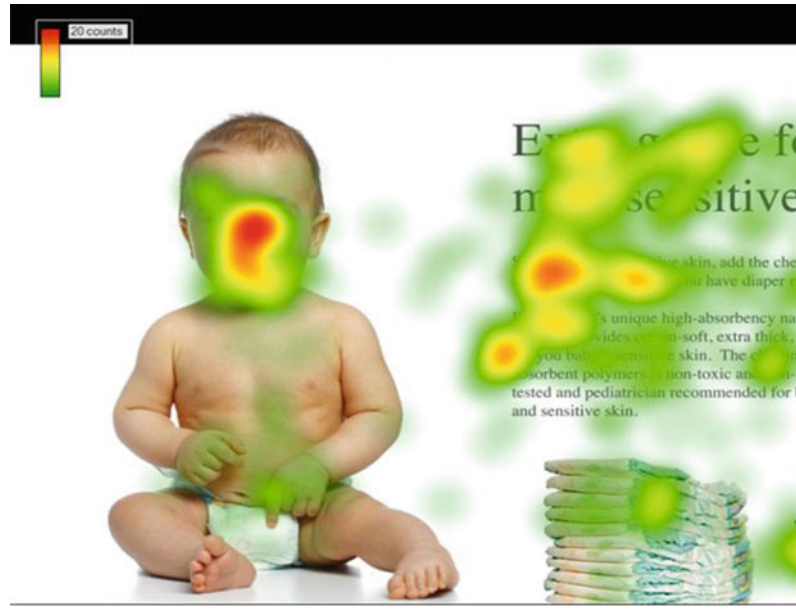
The performance of an eye tracker can be described in terms of gaze accuracy and precision, and track robustness. Accuracy describes the angular average distance from the actual gaze point to the one measured by the eye tracker. Gaze precision describes the spatial variation between successive samples collected when the person fixates at a specific point on a stimulus, for example, an image on the screen (Duchowski, 2003). Sampling rate and Latency are also important characteristics: the first one determines how fast the movement of the eye is measured; the second determines how fast the gaze point information from the eye tracker is obtained (important for gaze contingency and interaction).

The most commonly used systems for investigation of eye movements are categorized into eye tracking systems for human-computer interaction and diagnostic use of eye movement analysis. The interactive systems include selective and gaze-contingent systems and the latter are either screen-based or model-based. Examples of eye tracking systems usage are shown in Figs. 1–3.

Applications

Eye tracker systems are used to study human visual behavior by measuring gaze parameters like (a) fixation duration in milliseconds (gaze time and/or gaze fixation time), (b) average number of fixation points lasting between 200 and 300 ms, (c) saccade duration in milliseconds, that is, quick, simultaneous movements of both eyes, (d) proportion of left-to-right saccades, (e) proportion of saccade regressions and proportion of vertical saccades, (f) pupil dilation, (g) number of blinks, (h) smooth pursuit, (i) occurrence of nystagmus, (j) attention, and (k) inattention priority. The recorded data is statistically analyzed and graphically rendered and applied to measure visual search efficiency, priority, navigation usability, and real observing

Eye Tracking,
Fig. 2 Head-mounted eye tracker Tobii glasses



Eye Tracking,
Fig. 3 Example of a result of a marketing study: heat map

time of signs, letters, pictures, and figures in various communication systems or revealing inattention during work or driving.

Eye tracking is used to answer an endless array of *scientific research* questions regarding animal habits, for example, for studying chimpanzees' face scanning patterns (Kano & Tomonaga, 2010) and human visual habits. The human research is performed in the fields of behavioral medicine; linguistics; ophthalmology; cognitive, developmental, and behavioral psychology; and neurophysiology sometimes integrated with electroencephalography (EEG) in real time. For example, eye tracking technology is applied in developmental research, used as a diagnostic tool, for example, to children suffering from dyslexia, attention deficit hyperactivity disorder (ADHD), and autism, for oculomotor differential diagnosis in neurological disorders, discriminating between Parkinson's and Alzheimer's disease, diagnosis and treatment of neurological disorders, for example, mild traumatic brain injury, schizophrenia, and occurrence of macular degeneration, and in linguistics studies. Studies of fatigue and gaze attention are helpful to understand the effectiveness of work performed by

Eye Tracking, Table 1 Examples of the use of eye tracking systems for scientific investigation of human eye behavior, media and marketing research, usability studies, and as an assistive device

	Subject	Human eye behavior studied	Study summary	References
Human scientific research	Neuroscience developmental research	Search efficiency	The *speed of gaze fixations* was investigated during manual feeding and self-propelled feeding demonstrated on video films among 6-month- and 10-month-old babies and adults. The gaze had faster goal fixations when manual feeding was performed. The gaze direction among 10-month babies demonstrated that they were able to understand when the spoon was directed to their mouth and not to an adult feeding herself. Tobii X120 eye tracker, Tobii technology. Retrieved 20110525 http://www.toibii.com[1]	Kochukkova and Gredebäck (2010)
	Neuroscience developmental research	Visual real time: gaze time/gaze fixation time	A comparative study revealed that children with developmental dyslexia when reading sentences in a Cyrillic alphabet language had more than five times longer *gaze fixations* to the target words, affecting the reading frequencies and length compared to matched children without reading problems. Tobii X120 eye tracker, Tobii technology. Retrieved 20110525 http://www.toibii.com[1]	Hristova, Gerganov, Georigieva, and Todorova (2010)
	Linguistic research Brain physiology	Eye behavior investigation of attention	Seven eye movement variables were investigated: (a) fixation duration in milliseconds, (b) average number of fixations lasting between 200 and 300 ms, (c) saccade duration in milliseconds, i.e., 7–9 letter spaces, (d) proportion of left-to-right saccades, (e) proportion of saccade regressions, i.e., occurrences of re-reading, and proportion of vertical saccades, and (f) total time for reading three sets of words and three sentences. The aim was to understand which variable (a–f) would best discriminate between 6 years and 12 years old children with the diagnoses reading disability and attention deficit hyperactivity disorder (ADHD) compared to a control group of "normal" developed children. There were significant differences between the control group and the disability groups, who showed *atypical eye movements* for all variables apart from *saccade duration*. However, the results do not yet support the use of these eye movement variables to distinguish between the groups of participants with reading disorders and ADHD. The View Point Eye Tracker apparatus from Arrington Research[a]	Deans, O'Laughlin, Brubaker, Gay, and Krugay, and Krug (2010)

(*continued*)

Eye Tracking, Table 1 (continued)

Subject	Human eye behavior studied	Study summary	References
Linguistic research Brain physiology	Visual real gaze time, gaze fixation time. Search efficiency	An experimental study among people with and without dyslexics showed that both phonological and visual brain processes influence the eye movements, i.e., the *visual gaze and fixation time* when producing automatic naming. However, linear mixed effects analyses demonstrated that dyslexia readers need longer latencies, i.e., time used from observing words to response when performing the tasks	Jones, Obergón, Kelly, and Branigan (2008)
Linguistic research Brain physiology	Attention Glance behavior Eye behavior investigation	The direction of visual *attention* was investigated among people learning a foreign language. Less inaccurate initial word-referent mapping supports the learning	Fitneva and Chritiansen (2011)
Cognitive and behavioral psychology Oculomotor diagnosis in brain damage	Saccadic duration and direction	Ten people with developmental surface dyslexia, who read a short passage, showed an *altered pattern of eye movements with more frequent and smaller rightward saccades as well as longer fixation times*. The authors concluded that the cause is connected to the brain process and not to a deficit of the function of the oculomotor system	DeLuca, DiPace, Judica, Spinell, and Xoccolotti (1999)
Cognitive and behavioral psychology	Saccade performance	Saccadic eye tracking is an effective differential tool among people suffering from Parkinson's dementia disease and dementia with Lewy bodies. The sensitivity was 60% and specificity was 77–88% when reflexive saccade execution and complex saccade performance are used. Tobii X120 eye tracker, Tobii technology http://www.toibii.com and electro-oculargraph	Mosimann et al. (2005)
Cognitive and behavioral psychology Oculomotor rehabilitation	Search efficiency	The aim was to improve the reading profile among people with acquired brain damage suffering from error scanning or hemianoptic dyslexia. The training program during 8 weeks included single- and multiple-line-simulated reading and visional tracking, i.e., *eye movement fixation, saccade performance, and gaze pursuit*. Infrared eye movement technology[1]	Ciuffreda, Han, Kapoor, Ficarra, and Ficarra (2006)

Behavior medicine	Eye behavior investigation of attention. Saccadi c behavior	Smoking cues in movie clip affect smokers' attention behavior compared to nonsmokers. The smokers gaze more quickly, more often, and for longer time to the smoking-related cues	Lochbuehler, Voogd, Scholte, Engels, and Engels (2011)
Ophthalmology and vision science	Attention in saccadic search	*Attention* measured as *saccadic search* to attributes may be rendered in size, color, or orientation. The study result revealed that size, color, and orientation are not alike in dynamic attribute processing over time which has been the common conclusion drawn from earlier studies. Tobii 1750 eye tracker at 50 Hz sampling rate[1]	Yu (2010)
Assistive devices	Gaze eye pointing	Eye tracking systems are used to *control computers* by eye pointing and hence used as assistive devices among people with complex motor and language disabilities. MyTobii (P10)[1]	AbiltyNetGate (2011)
Market investigations	Search efficiency	The efficiency design of websites was compared using a three-layer hierarchical model for analyzing *eye-movement* data. Consumers switch frequently between attribute-based and product-based acquisition during moment-to-moment decision making of buying a product (e.g., a computer)	Shi, Wedel, and Pieters (2010)
Marketing/Advertising investigations	Search priority	Using eye tracking technology consumers buying behaviors in pub environments were measured in terms of *eye movement and gaze preferences*. The buying decision process among the consumers was reviled. The results showed that factors in the pub environment, like where the beer taps were placed, the impression of bartenders and various point-of-sale-advertising influenced the consumers' choice of beer	Press release from Carlsberg Sweden (a producer of beer) (2011)
Usability investigations Human factors ergonomic	Navigation usability	The usability, i.e., *eye-movement navigation and orientation* of five e-reading book devices was tested using an eye tracker. Among the results it was obvious that the mean duration of visual fixations differed significantly between the reading devices	Siegenthaler, Wurtz, and Groner (2010)

[a]Information used in the references

E

truck chauffeurs, captains, police officers, and air traffic controllers. Moreover, eye tracking provides unique methods to perform *marketing and media research*, for example, evaluate how users and consumers experience and perceive different media like websites and communication messages or make decisions about attractive products in shops and restaurants. The eye tracking technology is extensively used for *usability* studies, on websites, computer applications, games, and other human-made objects. Individually adapted eye tracker systems are used as assistive devices for people with complex motor and language disabilities, making them able to communicate, receive information, and play games by using eye pointing as demonstrated in video filmed cases (AbiltyNetGate, 2011). The recent technological development in the eye tracking field promises that eye movement will be the future way of controlling computers (Norrby, 2008; Wolverton, 2011) and other apparatus in home and work. Among these endless numbers of possible eye tracking applications some publications, emphasizing the various gaze parameters, are shown in Table 1.

Acknowledgment Dr. Ricardo Matos is acknowledged for the factual check of the entry.

Cross-References

- ▶ Cognition
- ▶ Social Marketing

References and Readings

AbiltyNetGate. (2011). *Global. assistive. technology. encyclopedia. eye pointing*. Retrieved May 26, 2011. from http://abilitynet.wetpaint.com/page/Eye+Pointing

Ciuffreda, K. J., Han, Y., Kapoor, N., & Ficarra, A. P. (2006). Oculomotor rehabilitation for reading in acquired brain injury. *NeuroRehabilitation, 21*(1), 9–21.

Deans, P., O'Laughlin, L., Brubaker, B., Gay, N., & Krug, D. (2010). Use of eye movement tracking in the differential diagnosis of attention deficit hyperactivity disorder (ADHD) and reading disability. *Psychology, 1*, 238–246.

DeLuca, M., DiPace, E., Judica, A., Spinell, D., & Xoccolotti, P. (1999). Eye movement patterns in linguistic and non-linguistic tasks in developmental surface dyslexia. *Neuropsychologia, 37*(12), 1407–1420.

Duchowski, A. T. (2003). *Eye tracking methodology theory and practice* (pp. 1–251). London: Springer.

Fitneva, S. A., & Chritiansen, M. H. (2011). Looking in the wrong direction correlates with more accurate word learning. *Cognitive Science, 25*(2), 367–380. Retrieved 2011/05/23 http://www.ncbi.nlm.nih.gov/pubmed?term=dyslexia%20eye%20traking.

Hristova, E., Gerganov, A., Georigieva, S., Todorova, E. (2010). Eye-movements of 7-years dyslexic children reading in regular orthography: Exploring word frequency and length effects. In: *Proceedings of the 32nd Annual Conference of Cognitive Science Society*. Portland, USA. August 11–14.

Jones, M. W., Obergón, M., Kelly, L. M., & Branigan, H. P. (2008). Elucidating the component processes involved in dyslexic and non-dyslexic reading fluency: An eye tracking study. *Cognition, 109*(3), 389–407.

Kano, F., & Tomonaga, M. (2010). Face scanning in chimpanzees and humans: Continuity and discontinuity. *Animal Behavior, 79*(1), 227–235.

Kochukkova, O., & Gredebäck, G. (2010). Preverbal infants anticipate that food will be brought to the mouth: An eye tracking study of manual feeding and flying spoons. *Child Development, 81*(6), 1729–1738. Retrieved 2011/05/27.

Komogortsev, O. V., Gobert, D. V., Jayarathna, S., Do Hyong Koh, & Gowda, S. (2010). Automated analyses of oculomotor fixation and saccadic behaviors. *IEEE Transactions on Biomedical Engineering*. July 26. (Epub ahead of print).

Lochbuehler, K., Voogd, H., Scholte, R. H. J., & Engels, R. (2011). Attentional bias in smokers: exposure to dynamic smoking cues in contemporary movies. *Journal of Psychopharmacology, 25*(4), 514–519.

Mosimann, U., Müri, R. M., Burn, D. J., Relblinger, J., O'Brien, J. T., & McKeith, I. G. (2005). Saccadic eye movement changes in Parkinson's disease dementia and dementia with Lewy bodies. *Brain a Journal of Neurology, 128*(6), 1267–1276.

Nielsen, J., & Pernice, K. (2009). *Eye tracking web usability*. Safari books on line. Retrieved May 26, 2011, from http://my.safaribooksonline.com/9780321549730

Norrby, A. (2008). *Toobii fick stora designpriset* (Tobii received the great design award). NyTeknik May Retrieved May 26, 2011, from http://www.nyteknik.se/nyheter/it_telekom/datorer/article241202.ece. (In Swedish)

Press release from Carlsberg Sweden (a producer of beer). (2011). *Sweden has investigated eye movements in people visiting bars*. Published 2011-04-26 12:26 Contact Jonas Ydén. Retrieved May 26, 2011, from

http://www.carlsbergsverige.se/Media/Nyheter/Sidor/CarlsbergSverigehartestat%C3%B6gonr%C3%B6relservidbardisken.aspx

Research Papers: *Tobii Eye Tracking Research*. Copyright © 2011 Tobii Technology, Sweden. Retrieved May 24, 2011 from http://www.tobii.com/en/analysis-and-research/global/library/research-papers/

Shi, S. W., Wedel, M., Pieters, F. G. M. (2010). *Information acquisition: An eye-tracking study of comparison websites*. Retrieved May 15, 2011, from http://gsgl.shufe.edu.cn/Article/UploadFiles/201011/20101130094412881.pdf

Siegenthaler, E., Wurtz, P., & Groner, R. (2010). Improving the usability of E-book readers. *International Journal of Usability Studies, 6*(1), 25–38.

SunshineCoast Daily. (2011). *Jeepers creepers, it's an eyeful*. Retrieved May 25, 2011, from http://www.sunshinecoastdaily.com.au/story/2011/05/09/jeepers-creepers-its-an-eyeful/

Tobii Eye Tracking Research, Diigo online library. (2011). *Research Papers*. Retrieved May 26, 2011, from http://www.tobii.com/en/analysis-and-research/global/library/research-papers/

Tobii eye tracking's list: *Tobii White papers*. (2011). Retrieved May 26, 2011, from http://www.diigo.com/list/tobiieyetracking/tobii-white-papers

Tobii eye-tracker integrated with ASA-Lab. *The real-time integration of eye-tracking and EEG*. Retrieved Oct 18, 2011, from http://www.tobii.com/ant-neuro.com/showcase/tobii-eyetracker

Wolverton, T. (2011). *San Jose Mercury News* (San Jose, CA). Retrieved March 26, 2011, from http://smartgrid.tmcnet.com/news/2011/03/23/5397145.htm

Yu, S. X. (2010). Feature transitions with saccadic search: size, color, and orientation are not alike. *Neural Information Processing Systems*, Vancouver, B.C, Canada. 6–9 Dec 2010. Retrieved May 17, 2011, from http://www.cs.bc.edu/~syu/publication/ftmc10.html

F

Failure

► Attribution Theory

Faith and Health

► Spirituality and Health

Faith Community Interventions

► Church-Based Interventions

Faith-Based Interventions

► Church-Based Interventions

Fall Risk Behavior

Lindy Clemson
Ageing, Work & Health Research Unit, Faculty of Health Sciences, University of Sydney, NSW, Lidcombe, Australia

Definition

Older people tend to define a fall as loss of balance and attribute it to external factors such as an obstacle or the weather, whereas health professionals relate it to intrinsic causes such as medications, medical reasons, or muscle strength. Although older people recognize inattention as important, neither group easily includes behavioral risks in their attribution.

The WHO definition of behavioral fall-risk factors includes those concerning human actions, emotions, beliefs, and daily choices. These are potentially modifiable and include, for example, sedentary behaviors, management of medications, inclusion of appropriate exercise in weekly routines, and better choices in safe shoe selection. Behaviors can be examined from their contribution to causing falls to the crucial part they play in reducing fall risk. For example, an individual's risk of falls is inexplicably related to the interaction between their mobility and balance capacity, their environmental demands and stressors, and their fall-risky lifestyle and behaviors.

Description

Fall-risk behaviors can be conceptualized as sequential events with a number of contributing factors which may be antecedents, present or consequential to the fall event. There has been some limited work in exploring risk behaviors surrounding the fall event, predictors of falls over time, personal characteristics, and socioeconomic and cultural influences. The consequences of falls can relate to the sequelae of injury, fear of falling, or reduced activity. Even for those who

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

experience slight injuries such as contusions there are frequently reported short-term physical function decreases and a loss of confidence. The earlier cohort studies on risk factors focused predominantly on intrinsic factors though several did identify reduced activity, leaving home less often, and in one cohort study, more than ten activities per week significantly increased fall risk. Older people who have fallen often attribute actions such as "hurrying," "carelessness," or "inattention" as causal fall behaviors.

A longitudinal study following fallers over an 8-year period has shown that depression and lower levels of morale are associated with increasing fall rates (Anstey et al., 2008). It may be that depression can contribute to motor or cognitive disturbances that can predispose people to falls. One intervention study (Salminen, Vahlberg, Salonoja, Aarnio, & Kivela, 2009) provided psychosocial group support for persons assessed with depression which lessened depression, enhanced involvement in the exercise components of the program, and resulted in a reduction of fall risk.

There are different cognitive profiles for those who have occasional falls, associated with subtle aging changes such as accuracy, planning, or inhibition of a response, compared to those who are having recurrent falls, which may be associated with broader deficits in processing speed, task-switching, and visual attention (Anstey, Wood, Kerr, Caldwell, & Lord, 2009). Few demographic characteristics are linked to falls though living alone is associated with a greater risk of multiple falls. Other profiling that highlights differences in risk behaviours has been identified in certain groups: those that are frailer compared to those who are very active; those in rural compared to city, particularly in terms of the type and extent of indoor/outdoor activities; and, differences in extreme weather conditions. Gendered responses to risk have been shown to vary, with women tending to blame themselves or others and men quicker to accept responsibility, and how family members respond which can vary from overprotective sons for women and younger female relatives who engage in negotiating actions for the men (Horton, 2007).

English-speaking people in Australia and Caucasians in the USA have higher falls injury admission rates than other groups. However, it is less clear whether this is a function of increased falls or is due to better access to health services. In countries where there are numerous immigrant groups with changing aging demographics, there is an increasing need to tailor falls prevention programs to meet particular linguistic and cultural requirements and differences in health-seeking behaviors and attitudes (Bradley & Harrison, 2007).

Fear of falling can be associated with falls but it has become evident that it is an independent phenomenon that can result in activity avoidance leading to social isolation, physical de-conditioning, and reduced quality of life whether or not falls occur. Delbaere, Close, Lord, and co-authors (2010a, b) have led important research defining how perceived risk of falling and "real" risk measured by validated physiological measures interact with psychological states. Four groups were identified: vigorous, anxious, stoic, and aware. The anxious group had a high-perceived fall risk compared to real risk and was related to depressive symptoms, neuroticism, and decreased executive functioning. The stoic group had attributes that protected them from falling, being positive, physically active, and participated in community life. This work suggests that measures of efficacy beliefs (e.g., *Assessment of Fear of Falling in Older Adults: The Falls Efficacy Scale-International* (*FES-I*), by S.A. Greenberg, http://consultgerirn.org/uploads/File/trythis/try_this_29.pdf) or fear of falling need to be considered in terms of other fall-risk factors and that better defining rational or irrational fears would be useful in falls prevention.

Michie et al. (2005) assert that in developing prevention guidelines, there needs to be better specificity in describing and defining the kinds of causal behaviors and to identify construct domains to help explain the underlying behavioral processes and therefore, the kinds of opportunities for change.

Two studies used recall and reenactment to explore themes and patterns associated with falls that occur at home (Connell & Wolf, 1997) and

those that occur in public places (Clemson, Manor, & Fitzgerald, 2003). Themes such as: or beliefs that a change in eyeglass prescription can cause deterioration of eyesight resulting in a reluctance to change; and low mobility self-efficacy affecting how the person safely negotiates the environment. Drawing on this work and conducting a review of studies that reported causes of falls, the *Falls Behavioural* (*FaB*) *Scale for older people* (Clemson, Cumming, & Heard, 2003) was developed to assess the kinds of subtle, day-to-day behaviors, both habitual and intentional, that offer an older person protection from falling during daily activity. Using factor analysis, dimensions within the scale were detected, which provided a profile of the kinds of adaptations people make or, alternatively, do not make, for example, *cognitive adaptations* (behaviors associated with reflection, intention, and planning, e.g., paying enhanced attention to changes in balance, level of alertness, etc., when trying a new medication) and *protective mobility* (protective mobility – negotiating the environment in a supportive or protective way, e.g., using defensive walking strategies such as heel toe walking and scanning ahead while walking). Clemson's and co-workers research has contributed to developing programs that incorporate evaluation of daily routines, the situational factors that shape these routines, and the kinds of adaptations the participants make. Programs need to incorporate techniques that support changing habits and maintaining them.

Habitual behaviors can be either risk taking or protective and are described as situational-guided goal-directed behaviors, and hence, behavioral responses are automatically elicited when a particular situation arises. Situational cues can be a specific place and a specific time, or other specific feature of the environment or pattern of interaction with the environment. There is evidence that habitual behaviors can be broken and new habits instituted in the same context by using planned *implementation intentions* (Holland, Aarts, & Langendam, 2006; Trope & Fishbach, 2004). These refer to a planned commitment to a behavioral response followed up with practiced and repeated actions in the same context. Thus, with conscious planning and repetition the new action is brought into active memory until it replaces the older action and becomes stable and enduring. The LiFE program, embedding balance and strength training in daily life activity, is an example of a program developed using these principles.

Knowing about their perception of falls risk and their fall experiences will assist in facilitating follow through of fall prevention strategies (Clemson, Cusick, & Fozzard, 1999). Older people tend to describe the fall in terms of its consequences and they do not easily make the link to "falls prevention" reporting this notion to be unfamiliar and puzzling. Recommendations center around ensuring that interventions are compatible with a positive identity, that they are tailored to the circumstances and values of the individual, and that validated methods are used to maintain longer-term adherence. If these are not addressed then older people are reluctant to participate (Yardley, 2007). There is also evidence on the importance of prevention strategies to include individual plans, to be contextually relevant and valued by the older person, and to recognize, in working with older people, the importance of self-identity and sense of control. The importance of reflecting on their falls and why they happened are directly related to engagement in adaptive strategies and hence are more likely to experience positive outcomes (Roe et al., 2008).

Two evidence-based programs tested in randomized trials that have been developed based on specific cognitive behavioral models are *Matter of Balance* (MoB) (Tennstedt et al., 1998), which reduced fear of falling and increased activity engagement, and the *Stepping On program* (Clemson et al., 2004), which reduced the rate of falls. The conceptual model of MoB recognizes self-efficacy, outcome expectations, and attributions as important influences on an older adults' sense of control over the course and consequences of aging. The program using cognitive restructuring exercises to address volitional attitudes and beliefs about falls before focusing on behaviors associated with the reduction of modifiable fear of falling risk factors such as activity avoidance. The conceptual model of *Stepping On*

draws on the process of decision-making operationalized into a framework to facilitate the adoption of behaviors to reduce fall risk and uses principles of enhancing self-efficacy, adult education, and story-telling as a technique to encourage reflective thinking, reframing, and problem-solving.

The outcome of a fall experience is complex and intricately related to personal, biological, and environmental factors. There needs to be a greater focus on understanding the behavioral and psychosocial effects of a fall. The aim for the older person is to understand and engage in protective behaviors and lifestyle choices that reduce risk thereby avoiding falls and remaining active and connected with community.

References and Readings

Anstey, K. J., Burns, R., von Sanden, C., Luszcz, M. A., Anstey, K. J., Burns, R., et al. (2008). Psychological well-being is an independent predictor of falling in an 8-year follow-up of older adults. *Journals of Gerontology Series B-Psychological Sciences and Social Sciences, 63*(4), P249–P257.

Anstey, K. J., Wood, J., Kerr, G., Caldwell, H., & Lord, S. R. (2009). Different cognitive profiles for single compared with recurrent fallers without dementia. *Neuropsychology, 23*(4), 500–508.

Bradley, C., & Harrison, J. E. (2007). *Fall-related hospitalisations among older people: Sociocultural and regional aspects*. Canberra: Australian Institute of Health and Welfare.

Clemson, L., Cumming, R. G., & Heard, R. (2003). The development of an assessment to evaluate behavioral factors associated with falling. *American Journal of Occupational Therapy, 57*(4), 380–388.

Clemson, L., Cumming, R. G., Kendig, H., Swann, M., Heard, R., & Taylor, K. (2004). The effectiveness of a community-based program for reducing the incidence of falls among the elderly: A randomized trial. *Journal of American Geriatrics Society, 52*(9), 1487–1494.

Clemson, L., Cusick, A., & Fozzard, C. (1999). Managing risk and exerting control: Determining follow through with falls prevention. *Disability and Rehabilitation, 13*(12), 531–541.

Clemson, L., Manor, D., & Fitzgerald, M. H. (2003). Behavioral factors contributing to older adults falling in public places. *Occupational Therapy Journal of Research: Occupational Participation Health, 23*(3), 107–117.

Connell, B., & Wolf, S. (1997). Environmental and behavioural circumstances associated with falls at home among healthy elderly individuals. *Archives of Physical and Medical Rehabilitation, 78*(2), 179–186.

Delbaere, K., Close, J. C. T., Brodaty, H., Sachdev, P., & Lord, S. R. (2010a). Determinants of disparities between perceived and physiological risk of falling among elderly people: Cohort study. *BMJ, 341*, c4165.

Delbaere, K., Close, J. C. T., Heim, J., Sachdev, P. S., Brodaty, H., Slavin, M. J., et al. (2010b). A multifactorial approach to understanding fall risk in older people. *Journal of American Geriatrics Society, 58*(9), 1679–1685.

Holland, R. W., Aarts, B., & Langendam, D. (2006). Breaking and creating habits on the working floor: A field-experiment on the power of implementation intentions. *Journal of Experimental Social Psychology, 42*, 776–783.

Horton, K. (2007). Gender and the risk of falling: A sociological approach (Research Support). *Journal of Advanced Nursing, 57*(1), 69–76. Sep;58(9): 1679–85.

Michie, S., Johnston, M., Abraham, C., Lawton, R., Parker, D., Walker, A., et al. (2005). Making psychological theory useful for implementing evidence based practice: A consensus approach. *Quality & Safety in Health Care, 14*(1), 26–33.

Roe, B., Howell, F., Riniotis, K., Beech, R., Crome, P., & Ong, B. N. (2008). Older people's experience of falls: Understanding, interpretation and autonomy. (Multicenter Study). *Journal of Advanced Nursing, 63*(6), 586–596.

Salminen, M. J., Vahlberg, T. J., Salonoja, M. T., Aarnio, P. T. T., & Kivela, S.-L. (2009). Effect of a risk-based multifactorial fall prevention program on the incidence of falls. *Journal of the American Geriatrics Society, 57*(4), 612–619.

Tennstedt, S., Howland, J., Lachman, M., Peterson, E., Kasten, L., & Jette, A. (1998). A randomized, controlled trial of a group intervention to reduce fear of falling and associated activity restriction in older adults. *Journal of Gerontology: Psychological Sciences, 53B*(6), P384–P392.

Trope, Y., & Fishbach, A. (2004). Going beyond the motivation given: Self-control and situational control over behavior. In R. Hassin, J. S. Uleman, & J. W. Bargh (Eds.), *The new unconscious* (pp. 537–565). New York: Oxford University Press.

Yardley, L., Beyer, N., Hauer, K., McKee, K., Ballinger, C., & Todd, C. (2007). Recommendations for promoting the engagement of older people in activities to prevent falls. *Quality & Safety in Health Care, 16*(3), 230–234.

False Negative

► False-Negative Error

False Positive

► False-Positive Error

False-Negative Error

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

False negative

Definition

In the context of medical diagnosis, a false-negative error occurs when an individual with a disease is "identified" by a diagnostic test as not having the disease.

In general science, if something is said to be true when in actuality it is false, the terms "type I error," "alpha error," or "false-positive error" are used. If something is said to be false when in actuality it is true, the terms "type II error," "beta error," or "false-negative error" are used (Jekel, Katz, Elmore, & Wild, 2007).

False-negative errors are meaningfully discussed along with false-positive errors.

Cross-References

► False-Positive Error

References and Readings

Jekel, J. F., Katz, D. L., Elmore, J. G., & Wild, D. M. G. (2007). *Epidemiology, biostatistics, and preventive medicine*. Philadelphia: Saunders/Elsevier.

Katz, D. L. (2001). *Clinical epidemiology and evidence-based medicine: Fundamental principles of clinical reasoning and research*. Thousand Oaks, CA: Sage.

False-Positive Error

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

False positive

Definition

In the context of medical diagnosis, a false-positive error occurs when an individual without a disease is "identified" by a diagnostic test as having the disease.

In general science, if something is said to be true when in actuality it is false, the terms type I error, alpha error, or false-positive error are used. If something is said to be false when in actuality it is true, the terms type II error, beta error, or false-negative error are used (Jekel, Katz, Elmore, & Wild, 2007).

When a diagnostic test is administered to a large group of individuals, it is very likely that, in truth, some will have the disease of interest and some will not. In the ideal scenario, all of those who have the disease will be correctly identified as having it, and all of those who do have the disease will be correctly identified as not having it. Unfortunately, such perfection is rare. As just noted, a false-positive error occurs when an individual without a disease is "identified" by a diagnostic test as having the disease. Another error that requires consideration is the false-negative error. In this case, an individual who does have the disease is incorrectly "identified" as not having the disease.

Associated terms are sensitivity and specificity (Katz, 2001). Sensitivity is the proportion of

those with the disease for whom the test result is positive. Specificity is the proportion of those without the disease for whom the test result is negative. Sensitivity and specificity are performance characteristics of every diagnostic test. To interpret the result of a test, and remain vigilant for false-positive and false-negative errors, these characteristics must be known along with the context in which the test is operating.

Cross-References

► False-Negative Error

References and Readings

Jekel, J. F., Katz, D. L., Elmore, J. G., & Wild, D. M. G. (2007). *Epidemiology, biostatistics, and preventive medicine*. Philadelphia: Saunders/Elsevier.

Katz, D. L. (2001). *Clinical epidemiology and evidence-based medicine: Fundamental principles of clinical reasoning and research*. Thousand Oaks, CA: Sage.

Family

► Family, Structure

Family Aggregation

Farrah Jacquez and Whitney Raglin
Department of Psychology, University of Cincinnati, Cincinnati, OH, USA

Synonyms

Family concordance; Parent-child concordance

Definition

Family aggregation of health behavior refers to the extent to which family members behave in the same or similar ways in areas that have consequences for health. In the literature, family aggregation often refers to the degree to which children exhibit the same behaviors as their parents. In review studies, significant evidence exists for the family aggregation of many health behaviors, including diet, physical activity, smoking, and alcohol and drug use (see Rossow & Rise, 1994). Despite the evidence of family aggregation, the findings within each health behavior are often inconsistent. Common methodological issues in the family aggregation literature are the inclusion of only one parent in studies and reliance on only one reporter to measure health behavior of both parents and children. Because family aggregation is heavily influenced by parental modeling, including only one parent in the research design does not provide the information necessary to detect the presence of aggregation or the mechanisms by which health behavior is transmitted from parent to child. Studies that rely on children to report on both their own and parent behaviors (and vice versa) tend to have inflated estimates of family aggregation due to mono-method bias. As a result of these methodological issues, family aggregation research has been somewhat inconsistent for certain health behaviors.

The childhood obesity problem in the United States has motivated intense scrutiny into the familial aggregation of obesity-related health behaviors. Research shows that parent/child concordance tends to be high for many diet- and eating-related constructs, including attitudes and perceptions about food, food preference, feeding styles, and eating behaviors (see Patrick & Nicklas, 2005). Parents influence their child's obesity-related health behavior not only through modeling but also through parental manipulation of the social and physical environment. Because parents purchase and provide food for the home, they largely dictate what and how much children are eating. Unlike tobacco, alcohol, or drugs, it is a parents' job to provide food for their children, and parents and children often eat together. As such, it is not surprising that the most consistent findings for parent/child concordance of health behaviors are in the areas of diet and eating. The

research findings for exercise and physical activity have been somewhat less consistent. Although many studies have not found parent/child concordance of physical activity, most of the work in this area has lumped the physical activity of mothers and fathers together (see Ferreira et al., 2006). When studies separate out father's and mother's physical activity, father's levels are highly positively correlated with those of their children while mother's levels are mostly unrelated.

There has been a large body of work devoted to examining the familial aggregation of tobacco use. Typically, familial aggregation of smoking is studied through genetic epidemiological studies (such as adult twin studies which have found genetic factors account for 50% of the variance attributed to regular tobacco use) and social risk factors (e.g., socioeconomic status, age, and gender). Despite the vast quantity of work in this area, there is little evidence that has been gained in support of the familial aggregation of smoking. Limitations in methodologies result in difficulties parceling out social and genetic influences in familial risk of smoking. For example, to date, there are no known family studies that examine the systematic development and maintenance of smoking within families. Nevertheless, in a recent review, Avenevoli and Merikangas (2003) found that in the 87 articles they reviewed, having a sibling who smoked was consistently found to be a predictor of current and lifetime smoking, while the evidence for parents was inconsistent.

Familial aggregation of alcohol use has been well established (McGue & McGue, 1994). Research shows that if child has a parent who drinks, he is three times more likely to drink and two times more likely to do drugs than a child with nondrinking parents. Results from twin and adoption studies suggest a significant genetic component to alcohol dependence. Although more of the research has focused on alcohol, family aggregation of drug use is also well established. Researchers estimate that relatives of individuals with drug disorders are 8 times more likely to use drugs than relatives of controls (Merikangas, 1998). Classic adoption studies also suggest a strong genetic component to substance abuse disorders (e.g., Cadoret, Troughton, O'Gorman, & Heywood, 1986), and twin studies have found the heritability of drug abuse to be even greater than that of alcoholism.

Cross-References

▶ Health Behaviors

References and Readings

Avenevoli, S., & Merikangas, K. R. (2003). Familial influences on adolescent smoking. *Addiction, 98S*, 1–20.

Cadoret, R. J., Troughton, E., O'Gorman, T. W., & Heywood, E. (1986). An adoption study of genetic and environmental factors in drug abuse. *Archives of General Psychiatry, 43*, 1131–1136.

Ferreira, I., van der Horst, L., Wendel-Vos, W., Kremers, S., van Lenthe, F. J., & Brug, J. (2006). Environmental correlates of physical activity in youth- review and update. *Obesity Reviews, 8*, 129–154.

McGue, M., & McGue, M. (1994). Genes, the environment, and the etiology of alcoholism. In R. A. Zucker, G. M. Boyd, & J. Howard (Eds.), *The development of alcohol problems: Exploring the biopsychosocial matrix. NIAA Research Monograph No. 26*. Rockville, MD: US Department of Health and Human Services, Public Health Service, National Institutes of Health, National Institute on Alcohol Abuse and Alcoholism.

Merikangas, K. R. (1998). Familial transmission of substance use disorders. *Archives of General Psychiatry, 55*, 973–979.

Patrick, H., & Nicklas, T. A. (2005). A review of family and social determinants of children's eating patterns and diet quality. *Journal of the American College of Nutrition, 24*, 83–92.

Rossow, I., & Rise, J. (1994). Concordance of parental and adolescent health behaviors. *Social Science & Medicine, 38*, 1299–1305.

Family Aid

▶ Family Assistance

Family and Medical Leave Act

Gabriela Reed
Psychiatry, Children's Medical Center, UT Southwestern Medical Center, Dallas, TX, USA

Synonyms

FMLA; Family and medical leave act of 1993, The

Definition

The Family and Medical Leave Act (FMLA) entitles eligible employees of covered employers to take unpaid leave for family and medical reasons with continuation of health insurance coverage under the same terms and conditions as if the employee had not taken leave.

A covered employer must grant an eligible employee up to a total of 12 workweeks of unpaid leave during any 12-month period for one or more of the following reasons:

- The birth of a child and to care for a newborn child within 1 year of birth
- The placement and care of an adopted or foster care child within 1 year of placement with the employee
- To care for the employee's spouse, child, or parent who has a serious illness
- A serious health condition that renders the employee unable to perform the essential functions of his or her job

A covered employer must also grant an eligible employee who is a spouse, child, parent, or next of kin of a current member of the Armed Forces with a serious injury or illness up to 26 workweeks of unpaid leave during a "single 12-month period" to care for the service member.

Eligibility

To be eligible for FMLA benefits, an employee must:

- Work for a covered employer
- Have worked for the employer for a total of 12 months
- Have worked at least 1,250 hours over the previous 12 months
- Work at a location in the United States or in any territory or possession of the United States where at least 50 employees are employed by the employer within 75 miles

Job Restoration

An employee's use of FMLA leave cannot result in the loss of any employment benefit that the employee earned or was entitled to before using FMLA leave, nor be counted against the employee under a "no fault" attendance policy. An employee must be restored to the employee's original job or to an equivalent job with equivalent pay, benefits, and other terms and conditions of employment upon return from FMLA leave.

References and Readings

U.S. Department of Labor Wage and Hour Division. (2010). *Fact sheet #28: The family and medical leave act of 1993*. Retrieved from http://www.dol.gov/whd/regs/compliance/whdfs28.pdf.

Family and Medical Leave Act of 1993, The

▶ Family and Medical Leave Act

Family Assistance

Gabriela Reed
Psychiatry, Children's Medical Center, UT Southwestern Medical Center, Dallas, TX, USA

Synonyms

Family aid; Office of family assistance

Definition

Family assistance refers to the provision of aid to families in need, usually with a focus on economic welfare and self-sufficiency.

The Office of Family Assistance

The Office of Family Assistance under the Administration for Children and Families is part of the United States Department of Health and Human Services. The Office of Family Assistance administers the Temporary Assistance for Needy Families (TANF) and Child Care and Development Fund (CCDF) programs.

Temporary Assistance for Needy Families (TANF)

TANF is designed to help needy families achieve self-sufficiency by providing assistance and work opportunities to needy families through grants of federal funds to states, territories, and tribes to allow for wide flexibility to develop and implement their own welfare programs.

The four major purposes of TANF are:

1. Aiding needy families so that children can be cared for in their own homes
2. Promoting job preparation, work, and marriage by reducing the dependency of needy parents
3. Preventing out-of-wedlock pregnancies
4. Encouraging the formation and maintenance of two-parent families

In February 2006, former President George W. Bush signed the Deficit Reduction Act of 2005 (DRA), which reauthorized the TANF program. The DRA reauthorization also included $150 million for discretionary grants to support programs designed to help couples form and sustain healthy marriages. Up to $50 million of this amount may be used for programs designed to encourage responsible fatherhood.

The Child Care and Development Fund (CCDF) Programs

The CCDF program enables low-income families to access child care, which in turn makes it possible for more parents to achieve economic self-sufficiency. The program also helps children succeed in school and life through affordable, quality early care and afterschool programs.

The Healthy Marriage Initiative is designed to strengthen families, foster safe and healthy relationships between married men and women, and promote the well-being of children.

The Responsible Fatherhood Program is designed to enable fathers to improve their relationships and reconnect with their children.

Other Forms of Economic Family Assistance

Other forms of economic family assistance are provided through Women, Infants, and Children (WIC), Low Income Home Energy Assistance Program (LIHEAP), and other emergency assistance programs available through government and nonprofit agencies.

Women, Infants, and Children (WIC)

WIC is administered by the Food and Nutrition Service as part of the USDA. WIC provides federal grants to States for supplemental foods, health-care referrals, and nutrition education for low-income pregnant, breastfeeding, and non-breastfeeding postpartum women, and to infants and children up to age 5 who are found to be at nutritional risk.

Low Income Home Energy Assistance Program (LIHEAP)

The mission of the Low Income Home Energy Assistance Program (LIHEAP) is to assist low-income households, particularly those with the lowest incomes that pay a high proportion of household income for home energy, primarily in meeting their immediate home energy needs.

Emergency financial assistance is usually temporary in nature.

Noneconomic Forms of Family Assistance

Noneconomic forms of family assistance include family counseling and intervention. Through family therapy, families or individuals within a family learn better ways to interact with each other and resolve conflicts. Family therapy is usually provided by clinical social workers or licensed therapists known as marriage and family therapists.

Cross-References

▶ Family Therapy

References and Readings

American Association for Marriage and Family Therapy. (n.d.). American Association for Marriage and Family Therapy. Retrieved from http://www.aamft.org

Food and Nutrition Service. (2005, November 2). Women, infants, and children. Retrieved from http://www.fns.usda.gov/wic

U.S. Department of Health and Human Services. (2012, January 6). TANF home. Retrieved from http://www.acf.hhs.gov/programs/ofa/tanf/index.html

U.S. Department of Health and Human Services. (2012, March 27). Office of family assistance. Retrieved from http://www.acf.hhs.gov/programs/ofa/index.html

U.S. Department of Health and Human Services. (2012, March 29). Low income home energy assistance program (LIHEAP). Retrieved from http://www.acf.hhs.gov/programs/ocs/liheap/#index.html

Family Caretaker

▶ Family, Caregiver

Family Concordance

▶ Family Aggregation

Family Medicine

▶ Family Practice/Medicine

Family Physician

▶ Primary Care Physicians
▶ Primary Care Providers

Family Planning

Jane Limmer[1] and Serina Floyd[2]
[1]Obstetrics and Gynecology, Duke Hospital, Durham, NC, USA
[2]Obstetrics and Gynecology, Duke Hospital, Raleigh, NC, USA

Synonyms

Birth control; Birth planning; Birth prevention; Contraception; Pregnancy spacing

Definition

Family planning is the practice of regulating the number and spacing of human births through the use of contraception and abortion. It allows couples and individuals to control the timing of their childbearing, and thereby to pursue educational and career goals, as well as to care for existing children and/or other family members, while limiting the possibility of pregnancy.

Description

Introduction

Family planning can be a controversial topic among different religious and cultural groups, and even at times within the medical community. Nonetheless, the World Health Organization, the American Medical Association, the American Congress of Obstetricians and Gynecologists, the American Medical Women's Association, the American Society for Reproductive Medicine, and the Society for Adolescent Medicine promote unbiased access to a wide range of family planning options as a fundamental component of comprehensive health care (Association of Reproductive Health Professionals, 2011).

Such an emphasis is placed on family planning for a variety of reasons. First, adequate

contraception decreases maternal mortality, both from high-risk pregnancies and unsafe abortions (World Health Organization, Department of Reproductive Health and Research, [WHO] 1995). Pregnancy prevention can be especially important in groups at high risk for complications, including very young women, older women, and women with certain coexisting medical conditions such as heart disease, kidney disease, certain cancers, and autoimmune diseases. Moreover, certain forms of contraception decrease the incidence of many sexually transmitted infections (STIs), the development of cancers of the female genital tract, and the prevalence of some common medical conditions. For example, hormonal contraceptives can protect against iron-deficiency anemia from menorrhagia, can prevent the growth and complications of ovarian cysts and uterine fibroids, and can decrease the risk of ovarian and endometrial cancer. In addition, barrier contraceptives decrease the transmission of STIs, including human immunodeficiency virus and human papillomavirus, which, in high-risk strains, can lead to cervical cancer (WHO, 1995). In addition, proper spacing of pregnancies improves infant and child health. An inter-pregnancy interval of less than 18 months has been associated with preterm labor and preterm delivery, as well as small-for-gestational-age infants, all of which increase neonatal morbidity and mortality (WHO). Limiting the overall number of children in a household also enables families to dedicate more resources to each individual child.

Contraception

Options and Use

In the United States, approximately 62% of all women of childbearing age are currently using a form of birth control (Guttmacher 2010a). There are multiple forms of birth control available in the USA, which vary by presence or absence of hormones, required frequency of administration, permanence, route of administration, as well as several other factors. One set of nonhormonal options that rely purely on planned human behavior includes abstinence, menstrual calendar-based method (avoiding intercourse on days 8–19 of the cycle, which are the woman's most fertile days), natural family planning (avoiding intercourse on days when a woman is most likely to have ovulated based on measurements of basal body temperature and cervical mucus), and withdrawal. Other nonhormonal options are barrier contraceptives, which must be used with each sexual encounter, including male and female condoms, diaphragms, sponges, and spermicides. Self-administered hormonal contraceptives include pills, which are taken daily, a transdermal patch, which is changed weekly, and a vaginal ring, which is changed monthly. Depo-Provera is an injectable progesterone which is dosed every 3 months. In addition, long-acting reversible contraceptives include intrauterine devices (or IUDs) and Implanon, a subcutaneous implant. Finally, permanent options for contraception include male and female sterilization (Trussell, 2007).

Among women in the USA, different age, racial, and socioeconomic groups tend to choose different birth control methods. In addition, the choice of a method often changes over the course of a woman's reproductive lifespan. For example, the birth control pill is the predominant method used by women younger than 30 years of age, whereas female sterilization is the predominant method used by women older than 30. Moreover, while white women most often select the pill, black and Hispanic women most often select female sterilization (Guttmacher, 2010a).

Women choose a birth control method based on several factors, both medical and personal. These frequently include risks and side effects of the method, cost, coexisting medical conditions, number of sexual partners, frequency of intercourse, number of desired children, and access to health care. Currently, apart from abstinence, the most efficacious contraceptive method is the Implanon device, with 0.05% of women experiencing an unintended pregnancy within the

first year of use. In comparison, with typical use of condoms, 15% of women per year will experience an unintended pregnancy (Trussell, 2007).

Methods of Birth Control among US Women, 2006–2008

Method	% of users	% of women with unintended pregnancy in first year of use – typical use –	% of women with unintended pregnancy in first year of use – perfect use –
Pill	28	8	0.3
Tubal sterilization	27.1	0.5	0.5
Male condom	16.1	15	2
Vasectomy	9.9	0.15	0.10
IUD	5.5	0.2–0.8	0.2–0.6
Withdrawal	5.2	27	4
Three-month injectable (Depo-Provera)	3.2	3	0.3
Vaginal ring (NuvaRing)	2.4	8	0.3
Periodic abstinence (calendar)	0.9	25	5
Other[a]	0.4	[b]	[b]
Periodic abstinence (natural family planning)	0.2	25	4
Diaphragm	[c]	16	6

Data in table taken from (1) Guttmacher 2010a. http://www.guttmacher.org/pubs/fb_contr_use.pdf, accessed February 7, 2011 and (2) Trussell, 2007

[a]Includes emergency contraception, female condom or vaginal pouch, foam, cervical cap, sponge, jelly or cream (without diaphragm), and other methods

[b]Efficacy figures vary widely depending on which of the aforementioned methods is used

[c] Figure does not meet standards of reliability or precision

Access to Contraception

According to data from the 2002 National Survey of Family Growth, 49% of all pregnancies in 2001 were unintended. Of these unintended pregnancies, 44% ended in births, 42% ended in abortions, and 14% ended in fetal losses. Unintended pregnancy rates are highest among women aged 20–24, women with low incomes, and women who are unmarried, of a minority (especially black) race and who have not completed high school (Finer and Henshaw, 2006).

In response to the high rate of untended pregnancy, especially among low-income women, public funding has been made available for contraceptive services and supplies. Medicare, state appropriations, and Title X of the Public Health Service Act provide such funding, with Medicare being the largest contributor (approximately 71%). In 2008, 17.4 million women were in need of these services, and 54% of these women received contraceptive care from a publicly funded family planning center. For many women, these centers are also the only source of primary medical care (Guttmacher 2010c).

Abortion

Definition

Abortion is the termination of a pregnancy and can be spontaneous or induced. A "spontaneous abortion," or what is more commonly known as a "miscarriage," is the natural loss of a pregnancy prior to 20 weeks' gestation. An "induced" abortion refers to a medical or surgical intervention performed with the intention to terminate a pregnancy (Kottke and Zieman, 2008).

There are several possible methods available for the termination of a pregnancy. The choice of method is largely based on the gestational age of the pregnancy, patient preference, and provider experience. Suction curettage, or the emptying of uterine contents by suction aspiration, is the most common method used up to 12 weeks' gestation. Medication abortion can also be performed up to 9 weeks' gestation, depending on the medication regimen used. These regimens consist of a combination of antiprogestins and prostaglandin analogues, and/or the use of methotrexate. In comparison, termination of pregnancy in the second trimester can be performed using dilation and evacuation (D&E) – dilation of the cervix with evacuation of uterine contents by suction, with or without extraction, at greater than 13 weeks' gestation – or induction of labor. D&E is performed more frequently than induction as it is a less

expensive and shorter procedure that does not require hospitalization (Kottke and Zieman, 2008, and Paul and Stewart 2007).

Epidemiology

An estimated four out of every ten unplanned pregnancies in the United States end in induced abortion. In 2000, a total of 1.31 million abortions were performed, but in 2005, this number decreased to 1.21 million. Approximately one-third of all American women will have had an abortion by the age of 45 years (Guttmacher 2010b). When asked to describe why they seek pregnancy terminations, women cite a variety of reasons: inability to afford a child (or another child); need to delay childbearing in order to devote time to work, school, or other family members; concerns about their own health or the health of the fetus; lack of access to contraception; relationship problems with the father of the pregnancy; and desire to end a pregnancy that resulted from rape or incest (Guttmacher Institute; Paul and Stewart 2007).

The demographics of women who have abortions are broad and include women of all ages, races, socioeconomic groups, and religions. Roughly half of all abortions are obtained by women aged 20–30, by women who have never been married, and by women who already have at least one child. Thirty-six percent of women seeking pregnancy termination are non-Hispanic white, 30% are non-Hispanic black, 25% are Hispanic, and 9% are of other races (Guttmacher Institude, May 2010). The rate of induced abortion is higher among low-income and black women, which many researchers attribute to lack of access to adequate contraception (Kottke and Zieman, 2008).

Health Impact

As stated by Speroff et al., "the legalization of abortion reduced maternal morbidity and mortality more than any single development since the advent of antibiotics to treat puerperal infections and blood banking to treat hemorrhage" (Speroff, Glass, & Kase, 1999). After abortion was legalized in 1973 through the *Roe v. Wade* Supreme Court case, the abortion-related mortality rate declined by 90%. Currently, the abortion-related mortality rate ranges from 1 in 1,000,000 for procedures performed prior to 8 weeks' gestation, to 8.9 per 100,000 for procedures performed after 21 weeks (Kottke and Zieman, 2008). In comparison, the maternal mortality rate for women who go on to deliver live infants in the United States was 13.3 per 100,000 live births in 2006 (U.S. Department of Health and Human Services, Health Resources and Services Administration, Maternal and Child Health Bureau, 2009). An overwhelming majority (greater than 85%) of all abortions occur within the first trimester of pregnancy (i.e., prior to 14 weeks' gestation). These early terminations pose the lowest health risks, both immediate and long-term, to women (Paul and Stewart 2007).

The long-term physical and mental sequelae of abortion have been studied extensively. These studies indicate that there is no increased risk of miscarriage, ectopic pregnancy, or infertility associated with first-trimester abortions. The outcomes of second-trimester procedures are not as well studied or understood. In addition, though the psychological impact of abortion on women has been widely debated, the vast majority of high-quality scientific evidence indicates that pregnancy termination does not pose mental health risks for most women. Two groups performed broad reviews of available scientific literature in 2008 – the American Psychological Association Task Force on Mental Health and Abortion and Johns Hopkins University – and both groups concluded that elective pregnancy termination is not associated with an increased risk of psychiatric sequelae (Charles, Polis, Srihara, & Blum, 2008 and Major et al., 2008). In fact, the best predictor of a woman's mental health after an abortion appears to be her mental health prior to the procedure.

Cross-References

- ▶ Condom Use
- ▶ Gender Role
- ▶ Health Care Access
- ▶ HIV Prevention

- ▶ Pregnancy
- ▶ Reproductive Health
- ▶ Sexual Risk Behavior
- ▶ Women's Health

References and Readings

Association of Reproductive Health Professionals. Position statement: Contraception. http://www.arhp.org/about-us/position-statements#11. February 06, 2011.

Charles, V. E., Polis, C. B., Srihara, S. K., & Blum, R. W. (2008). Abortion and long-term mental health outcomes: A systematic review of the evidence. *Contraception, 78*(6), 436–450.

Finer, L. B., & Henshaw, S. K. (2006). Disparities in rates of unintended pregnancy in the United States, 1994 and 2001. *Perspectives on Sexual and Reproductive Health, 38*(2), 90–96.

Guttmacher Institute, (2010). Facts on contraceptive use in the United States, *In Brief*, New York: Guttmacher Institute, 2010. http://www.guttmacher.org/pubs/fb_contr_use.pdf. Accessed February 7, 2011.

Guttmacher Institute, (2010). Facts on induced abortion in the United States, *In Brief*, New York: Guttmacher Institute, 2010. http://www.guttmacher.org/pubs/fb_induced_abortion.html. Accessed December 30, 2010.

Guttmacher Institute. (2010). Facts on publicly funded contraceptive services in the United States, *In Brief*, New York: Guttmacher Institute, 2010. http://www.guttmacher.org/pubs/fb_contraceptive_serv.html. Accessed December 30, 2010.

Kottke, M. J., & Zieman, M. (2008). Management of abortion. In J. A. Rock & H. W. Jones III (Eds.), *TeLinde's operative gynecology* (10th ed., pp. 776–797). Philadelphia: Lippincott Williams & Wilkins.

Major, B., Appelbaum, M., Beckman, L., Dutton, M.A., Russo, N.F., & West, C. (2008). Report of the task force on mental health and abortion. *American Psychological Association, task force on mental health and abortion*. Washington, DC. http://www.apa.org/pi/wpo/mental-health-abortion-report.pdf. Accessed February 6, 2011.

Paul, M., & Stewart, F. (2007). Abortion. In R. A. Hatcher (Ed.), *Contraceptive technology* (19th ed., pp. 637–672). Ardent Media: New York.

Speroff, L., Glass, R. H., & Kase, N. G. (1999). Family planning, sterilization, and abortion. In C. Gynecologic (Ed.), *Endocrinology and infertility* (6th ed., pp. 831–865). Baltimore: Lippincott Williams & Wilkins.

Trussell, J. (2007). Choosing a contraceptive: Efficacy, safety, and personal considerations. *In Contraceptive Technology*. Eds Hatcher RA, Trussell J, Nelson AL, Cates W, Stewart F, and Kowal D. New York: Ardent Media.

U.S. Department of Health and Human Services, Health Resources and Services Administration, Maternal and Child Health Bureau. (2009). Maternal mortality. *Women's health USA 2009*. Rockville, MA: U.S. Department of Health and Human Services, 2009. http://mchb.hrsa.gov/whusa09/hstat/mh/pages/237mm.html. Accessed February 06 2011.

World Health Organization, Department of Reproductive Health and Research. (1995). *Health Benefits of Family Planning*. December 31, 1995. WHO reference number: WHO/FHE/FPP/95.11. http://www.who.int/reproductivehealth/publications/family_planning/HRP_FHE_FPP_95_15/en/index.html. Accessed February 6 2011.

Family Practice

- ▶ Family Practice/Medicine

Family Practice/Medicine

Gabriela Reed
Psychiatry, Children's Medical Center, UT Southwestern Medical Center, Dallas, TX, USA

Synonyms

Family medicine; Family practice; General practice; Primary care

Definition

Family practice/medicine or primary care is the medical specialty that provides continuing and comprehensive health care for the individual and the family. It is the medical specialty that integrates the physical, clinical, and behavioral sciences. The scope of family medicine encompasses all organ systems, every disease entity and covers the life span.

Primary care is health care that is accessible, coordinated, comprehensive, and continuing. It is provided by physicians specifically trained for and skilled in comprehensive first-contact and

continuing care for persons with a diagnosed illness or those with an undiagnosed symptom, or health concern. In addition to diagnosis and treatment of acute and chronic illnesses, primary care includes health promotion and maintenance, disease prevention, and patient counseling/education in a variety of health-care settings, including private practice offices, the inpatient setting, critical care, and long-term care facilities, as well as in-home care. Primary care is performed by a personal physician, with consultation or referral to other health professionals as necessary.

Family practice/medicine encompasses the following functions:

1. It is first-contact care, serving as a point of entry for the patient into the health-care system.
2. It is highly personalized and assumes responsibility for individual follow-up.
3. It is comprehensive and includes continuity by virtue of caring for patients in sickness and in health.
4. It serves a coordinative function for the various health-care needs of the patient.
5. It assumes continuing responsibility for community health problems and concerns.

The term general practice is synonymous with the term family medicine in many countries. The Royal New Zealand College of General Practitioners emphasizes that a general practitioner provides care that is "anticipatory as well as responsive and is not limited by the age, sex, race, religion, or social circumstances of patients, nor by their physical or mental states." They argue that the family practice physician must be the patient's advocate; be caring, competent, and compassionate; and be willing to recognize limitations and refer when necessary (Richards, 1997).

Cross-References

- ▶ Chronic Disease Management
- ▶ Primary Care
- ▶ Primary Care Physicians
- ▶ Primary Care Providers

References and Readings

Rakel, R. (2007). The family physician. In R. Rakel (Ed.), *Textbook of family medicine* (7th ed.). Philadelphia: Saunders Elsevier.

Richards, J. G. (1997). *The nature of general practice: General practice in New Zealand*. Wellington, New Zealand: The Royal New Zealand College of General Practitioners.

Family Social Support

Elana Graber and Ryan M. Beveridge
Department of Psychology, University of Delaware, Newark, DE, USA

Synonyms

Social network; Social resources

Definition

Broadly defined, social support is information, clarification, aid, and emotional reassurance that an individual receives from others. Research has differentiated between perceived support (i.e., availability of support in one's environment) and received support (i.e., support one reports having received, often in response to a specific event). Perceived support, particularly in the early family environment, is associated with beneficial health outcomes, better (i.e., proactive) coping skills, and thus is associated with better health behaviors and outcomes. Perceived support is typically stable over time and is thought to develop from one's own early family structure and attachment through one's ability to develop and sustain close relationships with supportive others. As will be discussed further below, the benefits of received support are mixed and have been associated with both positive and negative health consequences.

Social support can also be further divided into practical and emotional support. Practical support includes giving advice, providing

information, or tangible assistance for another person (e.g., washing the dishes for a sick spouse or financial assistance from a family member). Emotional support can include physical affection and discussing one's thoughts and feelings showing warmth and nurturance to indicate the individual is valued and cared for. The degree to which practical and emotional support are helpful often depends on the particular stressor context in which they are occurring, including timing of support and phase of medical illness. Informational support may be needed to make medical decisions, and emotional support may help one cope with a medical diagnosis or treatment difficulties. Based on the matching hypothesis, what is important for individual outcomes is that the received support compliments the needs of the individual undergoing the stressor.

Description

As social beings, humans benefit from their connection to others through social support that they receive and provide, and in no greater context is this process observed than within the family system. Social support in a family context has been consistently linked to incidence of disease and recovery and coping responses to acute and chronic illness. The family context is one of the most common places individuals both seek and receive social support, and early life experiences within the family system shape one's ability to seek and benefit from social support in one's larger social network later in life. Children who develop secure attachments to their caretakers and perceive caretakers as available and attentive to their needs are better able to interpret and perceive supportive acts from others.

Negative Effects of Social Support

Interestingly, receiving social support from close others is not always beneficial. Indeed, patients who receive help from their family members may feel unable to reciprocate that support, creating a sense of inefficacy and bring awareness of one's lack of autonomy and control. Some research has found that received tangible support is even associated with higher mortality rates, while perceived support availability is associated with lower mortality rates.

Associations with Health Outcomes

Social support has been reliably linked with the development and progression of illness, for example, cardiovascular disease, diabetes, and lung disease, and has even been associated with lower risk for developing the common cold. The availability of support and the provision of "invisible support" (i.e., support that is provided but is not perceived) can have beneficial effects on health behavior and health outcomes. Specifically, perceived support can accelerate recovery from disease and improve mortality rates even after controlling for physical health status and demographic factors. Some of the mechanisms explaining the benefits of perceived support on health outcomes include greater proactive coping strategies, lower stress exposure, and healthier behavior choices in terms of diet and medical adherence. There are multiple theories on the utility of social support in alleviating the effects of stress. The stress-buffering hypothesis posits that social support is particularly important when individuals are undergoing large stressors, such as acute or chronic illness, but of relatively little import during periods of low stress. During periods of high stress, the availability of social support attenuates the negative effects of stress and allows the individual to cope more effectively. The direct effects hypothesis suggests that social support is beneficial during periods of high and low stress, making the family system and the general availability of support important at all times. As described above, the matching hypothesis of social support suggests help is likely most beneficial when the type of support provided matches the needs of the patient. A patient who desires emotional reassurance from their family but receives informational help is less likely to benefit from the social support and may even generate negative feelings of incompetence or dependency.

Support interventions have been shown to promote positive health outcomes. Creating supportive home environments early in life will likely place children on better health trajectories that will influence their susceptibility to disease as well as their ability to benefit from a social support network during acute or chronic illnesses.

Cross-References

- ▶ Family, Relationships
- ▶ Social Support

References and Readings

Berkman, L. F., Glass, T., Brissette, I., & Seeman, T. E. (2000). From social integration to health: Durkheim in the new millennium. *Social Science and Medicine, 51*, 843–857.

House, J. S., Landis, K. R., & Umberson, D. (1988). Social relationships and health. *Science, 241*, 540–545.

Uchino, B. N. (2009). Understanding the links between social support and physical health: A life-span perspective with emphasis on the separability of perceived and received support. *Perspective on Psychological Science, 4*, 236–255.

Family Stress

Ashley K. Randall[1] and Guy Bodenmann[2]
[1]Family Studies & Human Development, University of Arizona, Tucson, AZ, USA
[2]Department of Psychology, University of Zurich, Binzmuehlestrasse, Zurich, Switzerland

Synonyms

Dyadic stress; Marital stress; Relationship stress; Stress

Definition

Family stress can be defined as any stressor that concerns one or more members of the family (or the whole system) at a defined time, which impacts the emotional connection between family members, their mood, well-being, as well as the maintenance of the family relationship.

Description

While stress has been described as an individual phenomenon for many decades (e.g., Dohrenwend & Dohrenwend, 1974; Lazarus & Folkman, 1984; Selye, 1974), family stress has been conceptualized independently from individual stress theory.

Family stress theories first emerged in the 1930s and 1940s (Angell, 1936; Koos, 1946). The most influential family stress theory (ABC-X theory) was proposed by Hill (1958), and was further developed by Burr (1973) and McCubbin and Patterson (1983). The ABC-X theory includes three interacting variables: the event (A) that interacts with the family's resources (B), which allows the family to create their meaning and definition of the event (C). These three variables then produce the crisis (X). Although the ABC-X model is still used by some research groups and its utility is recognized by theorists, it is limited in different ways. First, the model explicitly deals with major stressors (critical life events) and crisis, and is not appropriate for understanding everyday stress processes in families. Second, the model claims to have a perspective on the whole family, but usually only partners/parents (or even more often only one partner/parent) are targeted while contributions of children are not considered.

Important theoretical additions of the ABC-X model were made by Burr (1973) and Burr and Klein (1994) by including the concepts of family vulnerability and regeneration. These authors focused more systematically on resources within the family and the use of different types of coping according to specific phases in the stress process. Subsequently, the complications the family faced in dealing with the stressor are addressed in the revised family stress model.

In the last two decades, marital research has provided new and stimulating input to the discussion of family stress. This line of research has led

to the development of theoretical models of dyadic stress and research activity (Bodenmann, 2005; Story & Bradbury, 2004, Randall & Bodenmann, 2009). Dyadic stress represents a distinct form of family stress, involving both partners directly or indirectly. According to Bodenmann (1997, 2005) dyadic stress is defined as a stressful event or encounter that always concerns both partners, either directly when both partners are confronted by the same stressful event, when there is stress within the relationship (e.g., disagreement with one's partner), or indirectly when the stress of one partner spills over to the close relationship and affects both partners. In both cases, dyadic stress *elicits* joint appraisals (in addition to individual appraisals) of the stressful situation that extends the primary and secondary appraisals in Lazarus' (1966) approach. These joint coping efforts, or cooperative use of common resources within the couples, are referred to as dyadic coping (Bodenmann, 2005).

Although the definition of dyadic stress has been used primarily in the focus of close relationships, it can be easily expanded to family systems. Specifically, this approach focuses on the following aspects of family stress (see Randall & Bodenmann (2009) for a review):

1. *Locus* of the stress
   (a) *External*: Stress that comes from outside the family, such as: (1) financial stress or (2) stress with regard to the extended family members, such as parents-in-law (grandparents) or other relatives. For example, if a grandparent is sick it may be the primary responsibility of their child to take care of them; subsequently, the mother or father may exhibit a stressful response and may be emotionally affected.
   (b) *Internal*: Stress that originates within the family. Examples would be conflicts and tensions between parents, parents and children, or siblings. For example, if parents have severe marital problems and if they often engage in disagreements, this may affect the family climate and the well-being of all family members.
2. *Intensity* of the stress
   (a) *Macro*: Stressors that can be common (e.g., critical life events). Examples would be (1) severe illness, (2) handicap, (3) unemployment, (4) death of a family member, or (5) important phases and changes, such as the birth of a child or an empty nest.
   (b) *Minor*: These are "everyday" stressors, such as (1) being late for an appointment or school, (2) having to get children to their extracurricular activities, or (3) stress at work, for example, both parents are working, one child is sick, and the family has to reorganize their work schedules to care for the child.
3. *Duration* of the stress
   (a) *Acute*. These stressors tend to be temporary and may be associated only with a single instance, for example, forgetting something at the grocery store or moving to a different house or city.
   (b) *Chronic*. These stressors are stable and can last a long time, for example, having a child that is ill or a partner that is unemployed.
4. *Affected person* of the stressor. Is it only one partner/parent, parents, one child or several children, or is the whole family affected by the stressful encounter? For example, the severe illness of a partner/parent affects the whole family system, whereas one daughter staying home sick may only affect her (the daughter) and perhaps the partner who is staying home with her.

Stress has been shown to have detrimental effects on health and well-being on all family members (see Randall & Bodenmann (2009) for a review). Recently, studies have shown the link between stress and behavioral medicine, specifically with respect to taking care of patients with dementia (Mitrani et al., 2006), and when the child is suffering from a chronic illness (Lewis & Vitulano, 2003). In addition, studies have shown increased family conflict when one child was diagnosed with Type 1 diabetes (Williams, Laffel, & Hood, 2009).

Families operate as a system and each member of the family is an interconnected entity that functions as a whole. Specifically, individuals cannot be understood without taking into account the family as a whole (Segrin & Flora, 2005). Thus, family stress impacts each member of the family and may have detrimental effects on their closeness, emotional connectedness, communication, and, ultimately, their well-being.

References and Readings

Angell, R. C. (1936). *The family encounters the depression*. New York: Charles Scribner's Sons.

Bodenmann, G. (1997). Dyadic coping - a systemic-transactional view of stress and coping among couples: Theory and empirical findings. *European Review of Applied Psychology*, *47*, 137–140.

Bodenmann, G. (2005). Dyadic coping and its significant for marital functioning. In T. Revenson, K. Kayser, & G. Bodenmann (Eds.), *Couples coping with stress: Emerging perspectives on dyadic coping* (pp. 33–50). Washington, DC: American Psychological Association.

Burr, W. R. (1973). *Theory construction and the sociology of the family*. New York: Wiley.

Burr, W. R., & Klein, S. R. (1994). *Reexamining family stress: New theory and research*. California: Sage.

Dohrenwend, S., & Dohrenwend, B. P. (1974). *Stressful life events: Their nature and effects*. New York: Wiley.

Hill, R. (1958). Generic features of families under stress. *Social Casework, 39*, 139–150.

Koos, E. L. (1946). *Families in trouble*. New York: Kings Crown Press.

Lazarus, R. S. (1966). *Psychological stress and the coping process*. New York: McGraw-Hill.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.

Lewis, M., & Vitulano, L. A. (2003). Biopsychosocial issues and risk factors in the family when the child has a chronic illness. *Child and Adolescent Psychiatric Clinics of North America, 12*, 389–399.

McCubbin, H. I., & Patterson, J. M. (1983). Family transitions: Adaptation to stress. In H. I. McCubbin & C. R. Figley (Eds.), *Stress and the family: Coping with normative transitions* (Vol. 2, pp. 5–25). New York: Brunner/Mazel.

Mitrani, V. B., Lewis, J. E., Feaster, D. J., Czaja, S. J., Eisdorfer, C., Schulz, R., et al. (2006). The role of family functioning in the stress process of dementia caregivers: A structural family framework. *The Gerontologist, 46*(1), 97–105.

Randall, A. K., & Bodenmann, G. (2009). The role of stress on close relationships and marital satisfaction. *Clinical Psychology Review, 29*, 105–115.

Segrin, C., & Flora, J. (2005). Theoretical perspectives on family communication: Family systems theory. In *Family communication* (pp. 28–33). Mahwah, NJ: Erlbaum.

Selye, H. (1974). *Stress without distress*. Philadelphia: J.B. Lippincott.

Story, L. B., & Bradbury, T. N. (2004). Understanding marriage and stress: Essential questions and challenges. *Clinical Psychology Review, 23*, 1139–1162.

Williams, L. B., Laffel, L. M. B., & Hood, K. K. (2009). Diabetes-specific family conflict and psychological distress in paediatric Type 1 diabetes. *Diabetic Medicine, 26*(9), 908–914.

F

Family Studies (Genetics)

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

Family studies are fundamental tools in the discipline of behavioral genetics (Turner, Cardon, & Hewitt, 1995) and can provide information of great interest in Behavioral Medicine. They permit assessments of degrees of familial resemblance, or aggregation, of physical, psychological, and behavioral characteristics.

Description

Pairs of siblings resemble each other more than do randomly chosen pairs of individuals, and children resemble their parents, on average, to a greater degree than they resemble randomly chosen adults. Such degrees of resemblance can be assessed in terms of correlation coefficients for continuous quantitative measurements, such as blood pressure and weight, and in terms of concordance rates for discretely defined characteristics, such as having or not having a specific disease state or psychiatric diagnosis.

In clinical studies, the proband is defined as the individual affected by a disease or condition of clinical concern that causes a family to be

included in a study. The probandwise concordance rate is then defined as the probability that a relative of a given type will also be affected. An important point to note is that in the absence of family resemblance, the concordance rate should be equal to the prevalence of the disease or condition. Therefore, for commonly occurring conditions (such as ever having smoked a cigarette), the baseline concordance rate might be as high as 80% in some populations. Family resemblance would then be indicated by concordance rates that were higher than this. In contrast, for conditions that are relatively infrequent, such as a psychiatric condition like schizophrenia with a 1% prevalence, a concordance rate as low as 10% might indicate substantial family resemblance (Hewitt & Turner, 1995).

It is typically found that there is a positive family resemblance for many characteristics, and the resemblance becomes more strongly positive as the degree of family relationship becomes closer. An important confounding factor, however (at least for the conventional nuclear family), is that the degree of genetic relationship is confounded with the degree of environmental or social relationship. Such families tend to eat the same food, for example, a factor that can influence blood pressure and weight. (Individuals who become married, and who perhaps ate relatively different diets, may in time come to eat a more similar diet, which can lead to various other characteristics becoming more similar.) Therefore, the mere observation of familial aggregation of a characteristic or condition of clinical concern is not sufficient to allow the inference of a genetic or environmental etiology. Instead, we need to study pairs of relatives in whom the degree of genetic relationship differs when the environmental resemblance is kept the same or, alternatively, pairs for whom the degree of environmental resemblance differs when the degree of genetic relationship is held constant.

The "natural experiment" of the birth of twins affords a good approximation to the first type of situation, and the adoption of children to be reared apart from their biological parents provides the second type. There are certainly other types of family relationships and study designs of relevance, for example, those involving half siblings and stepfamilies, but it is typically the case that both the statistical power and the conceptual clarity are greatest for research studies that start with a nucleus of either twins or families involved in adoption.

References and Readings

Hewitt, J. K., & Turner, J. R. (1995). Introduction. In J. R. Turner, L. R. Cardon, & J. K. Hewitt (Eds.), *Behavior genetic approaches in behavioral medicine*. New York: Plenum Press.

Turner, J. R., Cardon, L. R., & Hewitt, J. K. (Eds.). (1995). *Behavior genetic approaches in behavioral medicine*. New York: Plenum Press.

van Riper, M. V. (2010). Genomics and the family: Integrative frameworks. In K. P. Tercyak (Ed.), *Handbook of genomics and the family: Psychosocial context for children and adolescents* (pp. 109–139). New York: Springer.

Family Systems Theory

Neena Malik
Department of Pediatrics, Miller School of Medicine, University of Miami, Miami, FL, USA

Synonyms

Family therapy

Definition

Family systems theory is one of the major theories in behavioral and social sciences. The foundation of this theory is that all systems, human and mechanical alike, strive toward growth, development, and stability and that individual behavior cannot fully be understood without taking into account the context of the family system (Nichols, 2010a).

There are numerous models within family systems theory, and numerous associated therapeutic techniques designed to help families and individuals with relationship and mental health issues. In brief, Bowenian theory, named for Murray Bowen, focuses on intergenerational issues and family triangles. Strategic family therapy, pioneered by Jay Haley and others, focuses on understanding the function of symptoms and family communication patterns that relate to an individual patient's difficulties and creating strategies to change communication patterns.

Structural family systems theory and therapy, developed by Salvador Minuchin, is concerned with the hierarchical structures within families, positing mental health issues in individual family members when relationship structures are dysfunctional. Additional models include experiential, psychoanalytic, cognitive behavioral, solution-focused, narrative, and integrative approaches to both family systems theory and therapies (Doherty & McDaniel, 2010).

Cross-References

- ▶ Cognitive Behavior Therapy
- ▶ Mental Illness
- ▶ Therapy, Family and Marital

References and Readings

Doherty, W. J., & McDaniel, S. (2010). *Family therapy*. Washington, DC: APA.
Nichols, M. P. (2010a). *Family therapy: Concepts and methods*. Boston: Allyn & Bacon.
Nichols, M. P. (2010b). *Essentials of family therapy*. Boston: Allyn & Bacon.

Family Therapy

- ▶ Family Systems Theory
- ▶ Therapy, Family and Marital

Family Violence

Jason Jent
Department of Pediatrics, Mailman Center for Child Development, University of Miami, Miami, FL, USA

Synonyms

Domestic violence; Intimate partner violence

Definition

Family violence refers to any acts of violence that occur between family members, including but not limited to child maltreatment, intimate partner violence, and elder maltreatment (American Medical Association, 2011).

Description

Types of Family Violence

Child Maltreatment includes any act(s) of commission or omission by a caregiver that results in harm, potential harm, or threat of harm to a child (Leeb, Paulozzi, Melanson, Simon, & Arias, 2008). Child maltreatment is most broadly categorized as child abuse and child neglect. Child abuse consists of three different forms of acts of commission including physical abuse, sexual abuse, and psychological/emotional abuse. Acts of omission or neglect can also be characterized as family violence in specific instances where a caregiver knowingly fails to protect a child from maltreatment perpetrated by another caregiver or knowingly does not take appropriate measures to protect a child from being exposed to pervasive violence (e.g., intimate partner violence) or dangerous conditions (e.g., selling drugs out of the home). Both ▶ Child Abuse and ▶ Child Neglect are discussed in more detail in separate entries.

Intimate Partner Violence, a term that is often used interchangeably with domestic violence, includes acts of commission by a current or former partner or spouse that results in physical, sexual, or psychological harm (Saltzman, Fanslow, McMahon, & Shelley, 2002). Intimate partner violence occurs among heterosexual and same-sex couples and exists on a severity continuum. Intimate partner violence includes four types of behavior: physical violence, sexual violence, threats of physical or sexual violence, and psychological/emotional violence. In addition, stalking is often considered a type of intimate partner violence (Tjaden & Thoennes, 1998).

Elder Maltreatment includes any abuse and neglect of a person age 60 and older by a caregiver or another person in a relationship involving an expectation of trust (Center for Disease Control and Prevention, 2010). Forms of elder maltreatment include physical abuse, sexual abuse or abusive sexual contact, psychological or emotional abuse, neglect, abandonment, and financial abuse or exploitation.

Cross-References

- ▶ Abuse, Elder
- ▶ Child Abuse
- ▶ Child Neglect

References and Readings

American Medical Association (2011). *Violence prevention. In promoting healthy lifestyles*. Retrieved on July 20, 2011 from http://www.ama-assn.org/ama/pub/physician-resources/public-health/promoting-healthy-lifestyles/violence-prevention.shtml

Center for Disease Control and Prevention (2010). *Elder maltreatment: Definition*. Retrieved on July 20, 2011 from http://www.cdc.gov/ViolencePrevention/eldermaltreatment/definitions.html

Leeb, R. T., Paulozzi. L., Melanson, C., Simon, T., & Arias, I. (2008). *Child maltreatment surveillance: Uniform definitions for public health and recommended data elements, version 1.0*. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control. Retrieved on July 20, 2011 from http://www.cdc.gov/violenceprevention/pdf/CM_Surveillance-a.pdf

Saltzman, L. E., Fanslow, J. L., McMahon, P. M., & Shelley, G. A. (2002). *Intimate partner violence surveillance: uniform definitions and recommended data elements, version 1.0*. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control. Retrieved on July 20, 2011 from http://www.cdc.gov/ncipc/pub-res/ipv_surveillance/intimate.htm

Tjaden, P., & Thoennes, N. (1998). *Stalking in America: Findings from the national violence against women survey*. Washington, DC: Department of Justice (US). Publication No. NCJ 169592. Retrieved on July 20, 2011 from http://www.ncjrs.gov/pdffiles/169592.pdf

Family, Caregiver

Alyssa Parker
UTSW Health Systems, South western Medical Center, Dallas, TX, USA

Synonyms

Custodian; Family caretaker; Home health care

Definition

Family caregiving is not a new phenomenon and is considered a norm of the family based on family and societal obligations. According to a survey conducted in 2009 by the National Alliance for Caregiving, 65.7 million caregivers make up 29% of the United States adult population and 31% of all United States households. Of these 65.7 caregivers, 48.9 million care for adult recipients only, 3.9 million care for child recipients only, and 12.9 million care for both child and adult recipients. Family caregivers provide an estimated $375 billion worth of uncompensated care to loved ones annually, making them the backbone of long-term care. Caregivers are predominantly female (66%), are an average of 48 years old, and frequently take care of two or more people. The top two care recipient conditions are old age and Alzheimer's disease or dementia. Other conditions frequently mentioned include mental/emotional illness, cancer, heart

disease, and stroke (National Alliance for Caregiving, 2011).

On average, family caregivers spend 20.4 h per week providing care, though this number increases dramatically when the care recipient is living with the caregiver or when the recipient is under the age of 18. Caregiver time is predominantly spent helping loved ones complete activities of daily living. This may include helping the care recipient get in and out of beds or chairs, personal care tasks (getting dressed, assisting with bathing or showering, helping the recipient to and from the toilet, and helping deal with incontinence), and feeding. Additionally, caregivers are involved with instrumental activities of daily living, such as transportation, housework, grocery shopping, meal preparation, managing finances, and arranging or supervising outside services. Many caregivers also spend a significant amount of time advocating for the care recipient with care providers, government agencies, and performing medical therapies or treatments. Caregiving for a child with special needs requires a number of additional time-consuming support activities (NAC with AARP, 2009).

Prior to making the decision to become a family caregiver, it is imperative to make a realistic appraisal of the situation. Over half of all caregivers are married and more than 1/3 of caregiving families have children or grandchildren under the age of 18 living in the home (NAC with AARP, 2009). The entire family system is disrupted with the advent of caregiving, and many family members, including the care recipient, frequently experience significant adjustment and coping difficulties (Agosta & Melda, 1995). While many support services focus on the individual caregiver, the total family context is often overlooked. Caregivers who are employed must work to balance the competing demands of employment commitments and family responsibilities. The majority of caregivers who work outside of the home report having gone in late to work, having left early, or having taken time off during the day to deal with caregiving issues. One in five caregivers ultimately takes a leave of absence from work at some point. The Family Medical Leave Act (FMLA) was created in 1993 in order to help caregivers balance work and family responsibilities. This act provides certain employees with up to 12 weeks of unpaid, job-protected leave per year (United States Department of Labor, 2011). Despite changes in employment ability, it is important to note that caregiving can be an expensive endeavor. Not only do many caregivers decrease their work hours, there are a number of potential out-of-pocket expenses that must be addressed (NAC with AARP, 2009). Finally, studies of family caregiving have consistently demonstrated that a host of negative emotional and physical effects develop as a function of assuming responsibility for the care of a dependent family member. Vulnerability factors that may increase caregiver distress include the degree of care burden (both mental and physical), restricted activities, fear (not knowing what will come next), insecurity (feelings of loss of control over life or concerns regarding one's competency), loneliness (decreased partnership and less time to spend outside the home with others), facing death, and lack of support (from the patient, other family members, and health care providers) (Proot et al., 2003).

Despite the risks for distress, there are actions the caregiver can take to boost coping and decrease caregiver strain. It is beneficial for the family caregiver to receive caregiver training. By seeking out educational resources, the caregiver can help avoid serious injury to him/herself and the care recipient, as well as reduce the risk of recipient hospitalization for chronic sores or infections. Learning all one can about the care recipient's condition, its treatments, and the prognosis will help the caregiver and the caregiver's family have a better idea of what to expect in the future and how best one can help. Maintaining precise, up-to-date medical records and learning how to communicate with health care professionals allows caregivers to better advocate for their loved ones (NAC with AARP, 2009). Knowing how to ask for help, delegating duties, and getting friends and family involved in caregiving can also alleviate caregiver strain. Most importantly, however, the caregiver must learn how to manage his/her time and take care of

him/herself. Though time away from caregiving is often associated with fear or guilt, it is imperative that caregivers schedule time away from caregiving obligations. Recharging oneself ultimately makes for a better caregiver. Caregivers can regain control by setting limits about what they will and will not do, then voicing these boundaries to health care professionals. Finding satisfaction in the care one is providing may also decrease vulnerability to strain. Seeking good support, professional, instrumental, and emotional, is also a necessity for all caregivers. Professional support is associated with caregiver inclusion in education and decision-making by the care recipient's health care team. This decreases caregiver burden, fear, insecurity, and may provide realistic hope and facilitation of control. Instrumental support includes practical assistance in the daily care of the patient, which relieves care burden and facilitates the continuing of the caregiver's own activities. Finally, emotional support involves respect for the choices the caregiver must make, acknowledgment of the care they give, and provides someone to listen to the caregiver's concern. Emotional support generates satisfaction and may decrease fear, insecurity, and loneliness (Proot et al., 2003).

Cross-References

- ▶ Assisted Living
- ▶ Bereavement
- ▶ Care Recipients
- ▶ Caregiver/Caregiving and Stress
- ▶ Chronic Disease or Illness
- ▶ Daily Stress
- ▶ Dementia
- ▶ Disability
- ▶ Disease Burden
- ▶ Elderly
- ▶ End-of-Life Care
- ▶ Family Assistance
- ▶ Family Stress
- ▶ Grieving
- ▶ Home Health Care
- ▶ Medical Decision-Making
- ▶ Quality of Life
- ▶ Self-Care
- ▶ Stress, Caregiver

References and Readings

Agosta, J., & Melda, K. (1995). Supporting families who provide care at home for children with disabilities. *Exceptional Children, 62*(3), 271–282.

Caregiving. General. Retrieved from http://www.caregiving.org

Family and Medical Leave Act. Retrieved from http://www.dol.gov/dol/topicbenefits-leave/fmla.htm

Family caregiving. Retrieved from http://aarp.org/relationships/caregiving/

Family caregiving tips. Retrieved from http://www.familycaregiving101.org/

NAC in Collaboration with AARP. (2009). *Caregiving in the US: Executive summary*. Retrieved from http://www.caregiving.org/pdf/research/CaregivingUSAllAgesExecSum.pdf

National Alliance for Caregiving. (2011). *Research*. Retrieved from http://www.caregiving.org/

Proot, I. M., Abu-Saad, H. H., Crebolder, H. F., Golsteen, M., Luker, K. A., & Widdershoven, G. A. (2003). Vulnerability of family caregivers in terminal palliative care at home; balancing between burden and capacity. *Scandinavian Journal of Caring Sciences, 17*(2), 113–121.

United States Department of Labor. (2011). *Leave Benefits: Family & Medical Leave*. Retrieved from http://www.dol.gov/dol/topic/benefits-leave/fmla.htm

Family, Income

Jenny T. Wang[1] and Sarah J. Newman[2]
[1]Department of Medical Psychology, Duke University, Durham, NC, USA
[2]Duke University, Durham, NC, USA

Synonyms

Household income

Definition

Family income is a measurement of economic position of individuals who are considered to be part of one familial unit. Income is broadly

inclusive of wages, pensions, investments, governmental assistance or benefits, rent earnings, and any other source of finances. In sociodemographic and epidemiological research, family income is often used interchangeably with household income. However, household income denotes all individuals who are living in the same home, regardless of whether they are blood relatives, legally-bound, or neither. On the contrary, family income typically refers to individuals who are related by blood or by law, especially in societies in which there is a large emphasis on nuclear family compositions (e.g., father, mother, and minor children) (see ► Family, Structure). Consequently, household income and family income are not always equivalent.

Description

There is significant evidence to suggest that income is associated with health outcomes. The income and health gradient demonstrates that, in general, higher income is associated with greater health (see ► Health Disparities). Some plausible mechanisms for this relationship may include the influences of income on access to health care services or education, and health promoting assets and environments, such as grocery stores to obtain fresh food or having places to exercise. Income distribution of neighborhoods or environments can vary greatly, which can result in differential consequences on health.

There is little consensus as to what constitutes family income and how best to measure it, but most researchers agree that a combination of household and neighborhood levels of income should be taken into consideration. At the most basic level, family income can be measured directly by surveying or interviewing members of a family unit to learn how much income they each generate. It is important to consider that family income is determined in part by the number of employed individuals in a household as well as by the number of family members dependent on that income. As such, income should be measured to include all sources (e.g., wages, investments, and benefits), denoting disposable income, and be weighted to different family compositions such that larger families are considered to have lower disposable income given the greater number of people dependent on that income (Galobardes, Lynch, & Davey Smith, 2007). Measurement of family income is becoming more complex as family structures have changed dramatically in recent years and the definition of a "family unit" continues to evolve.

Family income can vary considerably across one's lifespan as individuals may change jobs or lose employment several times during their working life. Education and occupations of family members can provide a more stable estimate of family socioeconomic status, and can be used as proxies for estimating income level. Education and occupations should be obtained for all household members to estimate family income thoroughly, especially in the context of immigration and intergenerational differences in educational opportunities. A higher educational level can reflect knowledge-related assets as well as lifestyle choices, and can be predictive of better jobs, higher income, and safer housing. Occupation is another proxy for income as it links educational experiences with actual income earned (Shavers, 2007). When using educational level or occupation as proxies for income, it is important to consider that occupational status may change during one's lifetime with new jobs or careers and may more readily capture income variability. However, educational level, once attained, tends to remain the same throughout a lifetime and is a more static proxy for income. Of note, the income associated with an educational level or occupation is not always equivalent across race and gender (Shavers, 2007). For example, women and minorities tend to earn less than white male counterparts; therefore, utilizing educational level or occupation as proxies for income may actually overestimate income levels for women and minorities compared to white males of similar educational background or occupational standing (Krieger, Williams, & Moss, 1997).

There may be situations in which individuals or families cannot be asked directly about their economic status, such as when conducting retrospective reviews of health data or when looking

at regional differences in health outcomes. In these circumstances, neighborhood levels of income can be examined as proxies for family-level income. For instance, census tract variables help to estimate median family income and household income levels based on large geographical units such as zip code areas. A combination of variables can be used to estimate neighborhood socioeconomic status, such as percentage of homeownership, percentage of a population with health insurance, or percentage of single-parent households. Neighborhood income is suspected to be closely related to health outcomes because it may reflect environmental factors that impact health, such as neighborhood safety, parks and places to exercise, social services, and access to healthy foods, as well as social capital – the social resources available to families and individuals. One should be cautious when interpreting results from neighborhood-level income estimates; while the estimates can serve as proxies for family-level income, they may not reflect the economic realities of each family.

The significant impact of income on health outcomes points to the importance of accounting for economic factors in behavioral medicine research. While there is no current consensus on how best to measure family income, researchers agree that multiple measurements of individual, family, and neighborhood levels can provide a more comprehensive understanding of family income factors for health research.

Cross-References

- ▶ Racial Inequality in Economic and Social Well-being
- ▶ Social Class

References and Readings

Auerbach, J. A., & Krimgold, B. K. (Eds.). (2001). *Income, socioeconomic status, and health: Exploring the relationships*. Washington, DC: National Policy Association.

Galobardes, B., Lynch, J., & Davey Smith, G. (2007). Measuring socioeconomic position in health research. *British Medical Bulletin, 81*, 21–37.

Krieger, N., Williams, D. R., & Moss, N. E. (1997). Measuring social class in US public health research: Concepts, methodologies, and guidelines (Review). *Annual Review of Public Health, 18*, 341–378.

Shavers, V. (2007). Measurement of socioeconomic status in health disparities research. *Journal of the national medical association, 99*(9), 1013–1023.

Family, Relationships

Ryan M. Beveridge and Elana Graber
Department of Psychology, University of Delaware, Newark, DE, USA

Synonyms

Interpersonal relationships; Social network

Definition

Family relationships have long been a focus of researchers interested in exploring how the social context relates to health. Researchers have shown that differences in family relationships are associated with the development of acute and chronic illnesses, as well as to how individuals and their families adjust to ongoing health problems. Several explanations for the family's impact on health and disease have been proposed. These explanations include the following: the family's role in buffering or exacerbating the physiological stress responses of individuals, the modeling of appropriate health behaviors, and the social control that family members may have on one another's health behaviors. Furthermore, because of the central role most families play in the development and maintenance of stress responses, as well as health behaviors and beliefs, psychosocial interventions are often implemented at the family level.

Description

Research has consistently indicated that the nature of parent-child and spousal relationships associates with the etiology of health, illness, and disease. For example, stable and supportive family relationships are related to long-term health outcomes. Individuals who perceive that their parents were warm and supportive during their childhood are less likely to develop coronary artery disease, hypertension, and alcoholism, than individuals who report less support from their families. Furthermore, being married and having children in the home is related to positive health outcomes such as lower mortality rates, especially for men. Conversely, parent-child relationships characterized as interpersonally hostile, critical, overly controlling, and highly conflictual relate to several health problems in adulthood including lung disease, cardiovascular disease, some types of cancer, diabetes, and drug and alcohol abuse. Furthermore, marital relationships characterized by high levels of conflict as well as interpersonally hostile behaviors are associated with the development of myriad diseases, including cardiovascular disease, which is the leading cause of death in the United States. Indeed, spouses who appraise their partner as collaborative (versus controlling or uninvolved) and who are interpersonally warm and encourage autonomy are less likely to develop coronary artery disease. Marital conflict can also confer negative outcomes on children, including mental health, social, and physiological functioning. Children from families characterized by hostility show increased physiological reactivity when exposed to couples arguing in the laboratory compared with children from less conflictual and hostile families.

In addition to research indicating a link between family relationships and the etiology of health and disease, strong findings indicate that families play a role in how well individuals and families adjust to living with an illness. Specifically, different types of family involvement in coping with chronic illnesses are an important aspect of understanding physical and psychosocial adjustment to illness. Research supports the notion that chronic illnesses are experienced within a social context and that the family environment is significantly impacted by living with a family member's illness. Oftentimes, individuals and families must learn to adjust their diet, exercise practices, daily routines, and other health behaviors in order to successfully manage the illness. Therefore, understanding how family members relate to one another to manage illness demands has been a prominent focus of interest for researchers and clinicians. For example, individuals with type 1 diabetes and cardiovascular disease who appraise their families as being involved in their illness by brainstorming, negotiating, engaging in problem solving, working as a team, and providing helpful suggestions or advice have better psychosocial and physical outcomes than those who perceive their family members as uninvolved or overly controlling. In addition, treatment adherence is generally greater when family members are collaboratively involved in the everyday stressors associated with chronic disease management. Furthermore, when family members provide emotional support, informational support, or tangible support (e.g., money, food, and supplies), individuals, and family members, often have more positive outcomes.

However, family involvement is not always associated with positive outcomes for individuals with illnesses or their families. Specifically, when family members perceive others in their family as being intrusive or causing feelings of dependency, this type of family involvement is associated with poorer treatment adherence, physical outcomes, and higher levels of family conflict. Furthermore, family members may underestimate the physical and psychological resources that an individual with an illness may have, leading to overcompensation and a lack of self-efficacy in the individual with the illness. Additionally, family members providing support may become distressed over providing support and higher levels of depression may be seen in caregivers (although some research indicates that providing support can be beneficial to family members as well). Therefore, understanding the family context in which coping with a chronic illness occurs provides insight into how well individuals, and family members, adjust to living with a disease.

Several researchers, drawing from psychophysiological research, have found interesting mechanisms by which family relationships may associate with the etiology of health and disease, as well as how individuals adjust to living with a chronic illness. First, researchers have posited that parent-child relationships provide a context in which an individual's stress system may be affected for better or worse across the lifespan. Specifically, children who are chronically faced with familial stress may develop increased sympathetic reactivity to stress and exaggerated cortisol and catecholamine responses. Indeed, research suggests that the neuroendocrine stress response system may become deregulated under chronically stressful conditions, creating the potential for diseases such as cardiovascular disease, among others. Furthermore, chronic stress occurring in the context of hostile, critical family environments may dampen killer T-cell activity, and relate to other markers of compromised immune system functioning. This process can relate to the development of chronic health problems, as well as more acute difficulties when coping with a chronic disease. Specifically, family relationship conflict can have direct and indirect effects on immune system functioning. For example, proinflammatory cytokine secretion can be affected by family stress via central nervous system and endocrine system activation as well as indirectly through relationships with emotional distress, depression, anxiety, and poor health behaviors.

Family relationships also provide a key context in which health behaviors, beliefs, and habits form, all of which can have profound associations with health outcomes. Research has shown that family social norms are an important predictor of health behaviors such as proper diet, treatment adherence, smoking, drug and alcohol abuse, and breastfeeding. Family members may provide a model that individuals within the family utilize to form health habits that influence their overall well-being. Some health behaviors, such as healthy diets, are highly related to family norms and are quite stable by early adolescence, indicating the need to intervene early within the family context. Social control, wherein family members' beliefs about health influence individuals health behaviors such as following treatment regiments, going to the doctor, or exercising more regularly, is also an important predictor of health. Furthermore, in some cultures (e.g., Asian, Latino, or Black), individuals may engage in healthy behaviors for the good of the family. Therefore, family relationships can have an important impact on health through both direct and indirect processes.

As a result of the myriad studies that show the importance of family relationships for health, clinical interventions that focus on the family have been successfully developed. Typically, family interventions within health settings include an emphasis on the modeling and education of healthy behaviors. Furthermore, families are provided strategies on how to integrate healthy behaviors into the structure of families' daily routines. Additionally, family members are taught effective problem-solving skills that provide strategies to solve daily hassles, as well as major difficulties that may arise when coping with an illness. Also, families are taught effective communication skills, such as listening to one another, reflecting what others have said, praising appropriate health behaviors, and negotiating when there are different opinions of how to manage health-related decisions. In summary, family functioning and relationships have become an important focus of intervention in health settings.

Cross-References

▶ Family Social Support
▶ Family Systems Theory

References and Readings

Heffner, K. L., Kiecolt-Glaser, J. K., Loving, T. J., Glaser, R., & Malarkey, W. B. (2004). Spousal support satisfaction as a modifier of physiological responses to marital conflict in younger and older couples. *Journal of Behavioral Medicine, 27*, 233–254.

House, J. S., Landis, K. R., & Umberson, D. (1988). Social relationships and health. *Science, 241*, 540–545.

Uchino, B. N. (2009). Understanding the links between social support and physical health: A life-span perspective with emphasis on the separability of perceived and received support. *Perspective on Psychological Science, 4*, 236–255.

Family, Structure

Jenny T. Wang
Department of Medical Psychology,
Duke University, Durham, NC, USA

Synonyms

Composition; Family

Definition

Family structure reflects the organization of individuals who may be related by blood or legally bound (i.e., marriage, adoption) that are considered of the same relational unit.

Description

The most basic family structure within Western culture comprises of the "nuclear" family, which includes father, mother, and any children under the age of 18. Information about family structure is often asked of parents or children involved in biopsychosocial research and often documented retrospectively or at the time the information is assessed. However, it can be argued that family stability and family structure are fluid and evolving influences on health, socioeconomic resources, and family dynamics and is more accurately assessed longitudinally.

In health research, family structure is often dichotomized into "single-parent households" and "two-parent households." Data regarding parental structure is of importance as single-parent households have been associated with lower income (see ▶ Family, Income) and poorer health outcomes. However, the definition of family structure is becoming increasingly complex and extends beyond the traditional dichotomy of single versus two-parent households.

For example, single parenthood can include numerous possibilities such as a single biological, adoptive, foster, or stepparent parent due to life transitions including death of a parent, remarriage, divorce, adoption, and advances in assisted reproductive technology (ART, see ▶ Infertility and Assisted Reproduction: Psychosocial Aspects) allowing single adults to conceive without an identified partner. In the most simplistic form, two-parent households may comprise of intact biological parents or stepparents who are legally defined as being married and of the same family unit. However, children are increasingly born to unmarried partners or those who are cohabiting. These families do not conform to legal definitions of marriage in the traditional sense. Furthermore, some unmarried partners may share the same residence while others do not, maintaining residences with children and previous partners. Consequently, the measurement of family structure can be ambiguous and influenced by the context in which the family relationships are defined.

Furthermore, sibling relationships can be highly variable in current-day family structures as stepfamilies and blended families often result from the dissolution of marriages or changes in cohabitation. Biological, half, and stepsiblings can influence family cohesiveness and stability, which can either ameliorate or exacerbate the difficulties of family structure changes. The ages of children in families as well as their developmental and social needs (e.g., children with disabilities) can increase the financial and emotional strain in recently combined families.

In some non-Western cultures, extended family members such as grandparents, aunts, uncles, and cousins are considered part of the core family unit given emphasis on interdependency and collectivism. Collecting information about family members beyond the "nuclear" family may be particularly important in some cultures given the additional social resources that extended family members can provide. For example, assistance from grandparents, in particular grandmothers in certain cultures may be critical to understanding how families and youth cope with chronic illness or other health demands. Extended family members can provide practical support (e.g., providing transportation to doctor appointments, reminding one to take medications, etc.) as well as emotional support to deal with health-related stressors.

Cross-References

- ▶ Social Capital and Health
- ▶ Social Factors

References and Readings

Barrett, A. E., & Turner, R. J. (2005). Family structure and mental health: The mediating effects of socioeconomic status, family process, and social stress. *Journal of Health and Social Behavior, 46*(2), 156–169.

Bramlett, M. D., & Blumberg, S. J. (2007). Family structure and children's physical and mental health. *Health Affairs (Millwood), 26*(2), 549–558.

Brown, S. L., & Manning, W. L. (2009). Family boundary ambiguity and the measurement of family structure: The significance of cohabitation. *Demography 46*(1), 85–101. *Project MUSE*. Web. January 21, 2011. http://muse.jhu.edu/

Carlson, M. J. (2006). Family structure, father involvement, and adolescent behavioral outcomes. *Journal of Marriage and Family, 68*(1), 137–154.

Dawson, D. A. (1991). Family structure and children's health: United States. *Vital and Health Statistics, 10*(178), 1–47.0020.

Lee, G. R. (1982). *Family structure and interaction: A comparative analysis* (2nd ed. rev.). Minneapolis: University of Minnesota Press.

Montgomery, L. E., Kiely, J. L., & Pappas, G. (1996). The effects of poverty, race, and family structure on US children's health: Data from the NHIS, 1978 through 1980 and 1989 through 1991. *American Journal of Public Health, 86*(10), 1401–1405.

Thompson, S. J., Auslander, W. F., & White, N. H. (2001). Comparison of single-mother and two-parent families on metabolic control of children with diabetes. *Diabetes Care, 24*(2), 234–238.

Fasting Glucose

Adriana Carrillo and Carley Gomez-Meade
Department of Pediatrics, Miller School of Medicine, University of Miami,
Miami, FL, USA

Synonyms

Fasting sugar

Definition

Fasting blood glucose refers to blood glucose concentrations after an overnight fast. Normal fasting blood glucose is $\leq$100 mg/dL. Impaired fasting glucose is an intermediate stage between normal glucose homeostasis and overt diabetes. Impaired fasting glucose is between 100 and 125 mg/dL. Fasting blood glucose greater than 125 mg/dL indicates diabetes. Diabetes signifies a failure of glucose regulation. Normal fasting blood glucose is maintained by multiple mechanisms, as described below (Gardner & Shoback, 2007; Kronenber et al., 2008).

Mechanisms that prevent hypoglycemia by raising blood glucose during fasting begin when glucose falls below 85 mg/dL. Initially, pancreatic beta cells sense glucose levels and suppress insulin secretion. Decreasing insulin levels decreased glucose utilization by insulin-sensitive tissue such as skeletal muscle. Lower insulin levels also increase hepatic glucose production. Endogenous glucose production mainly via glycogenolysis is also stimulated by increased glucagon levels. Glucagon is secreted from the pancreatic alpha cells in response to decreasing blood glucose. Glucagon levels begin to rise when blood glucose is less than 70 mg/dL. Glucagon increases hepatic glucose production within minutes via gluconeogenesis and to a lesser extent gluconeogenesis. Epinephrine can also acutely increase blood glucose when blood glucose decrease below 70 mg/dL. Adrenal chromaffin cells in the adrenal medulla secrete epinephrine in response to declining blood glucose. Epinephrine increases blood glucose by stimulating hepatic glucose production and decreasing glucose utilization. Epinephrine acts directly to increase hepatic glycogenolysis and enhance glucagon secretion by activating β_2-adrenergic stimulation. Gluconeogenesis is increased by epinephrine through mobilization of precursors and fatty acids. Insulin secretion is limited by α_2-adrenergic stimulation of epinephrine. Epinephrine decreases glucose utilization in insulin-sensitive tissue through β-adrenergic stimulation. Glucose utilization and endogenous glucose production can also be augmented by

long-term elevation in growth hormone and cortisol. Acute increases in growth hormone have insulin-like effects, but after several hours increase hepatic gluconeogenesis and decrease glucose uptake. Increased cortisol levels for 2–3 h increase hepatic gluconeogenesis and decrease peripheral glucose utilization. After 24–48 h, gluconeogenesis becomes the only source of glucose production. Lipolysis and ketogenesis provide alternative substrate for brain metabolism. Suppression of insulin secretion and secretion of counterregulatory hormones increase endogenous glucose production and decrease glucose utilization during fasting (Sperling, 2008).

Cross-References

► Blood Glucose

References and Readings

Gardner, D. G., & Shoback, D. (2007). *Greenspan's basic and clinical endocrinology* (8th ed.). New York: McGraw-Hill.
Kronenber, H., Melmed, S., Polonsky, K., & Larsen, P. R. (2008). *Williams textbook of endocrinology* (11th ed.). Philadelphia: Saunders Elsevier.
Sperling, M. (2008). *Pediatric endocrinology* (3rd ed.). Philadelphia: Saunders Elsevier.

Fasting Insulin

Adriana Carrillo and Carley Gomez-Meade
Department of Pediatrics, Miller School of Medicine, University of Miami,
Miami, FL, USA

Synonyms

Insulin sensitivity

Definition

Fasting insulin levels are primarily used to assess insulin sensitivity. Impaired insulin sensitivity precedes glucose intolerance in the development of type 2 diabetes. Elevated fasting insulin is a compensatory mechanism to prevent glucose intolerance and diabetes. Insulin resistance is defined as the impaired ability to promote peripheral glucose disposal and suppress hepatic glucose production.

The primary site of glucose disposal is skeletal muscle. The gold standard for measuring skeletal muscle insulin sensitivity is the hyperinsulinemic-euglycemic insulin clamp. This is an invasive and time-consuming test that involves infusion of a fixed rate of insulin and a variable rate of glucose to maintain normoglycemia; therefore, alternatives have been proposed. Calculations of fasting insulin and glucose can be used to measure insulin resistance. The most common are fasting glucose to insulin ratio, homeostasis model assessment of insulin resistance (HOMA), and quantitative insulin sensitivity check index (QUICKI). The HOMA and QUICKI models are mathematical estimates of beta cell function and insulin resistance (Gardner & Shoback, 2007; Lifshitz, 2007; Wallace, Levy, & Matthews, 2004).

Fasting insulin levels can also be evaluated during hypoglycemia. An insulin level greater than 2 μU/mL when serum blood glucose is less than 50 mg/dL suggests hyperinsulinism (Gardner & Shoback, 2007).

Cross-References

► Hyperinsulinemia
► Insulin
► Insulin Resistance

References and Readings

Gardner, D. G., & Shoback, D. (2007). *Greenspan's basic and clinical endocrinology* (8th ed.). New York: The McGraw-Hill.
Lifshitz, F. (2007). *Pediatric endocrinology* (5th ed.). New York: Informa Healthcare.
Wallace, T. M., Levy, J. C., & Matthews, D. R. (2004). Use and abuse of HOMA modeling. *Diabetes Care, 27*(6), 1487–1495.

Fasting Sugar

▶ Fasting Glucose

Fat Absorption

Manjunath Harlapur[1] and Daichi Shimbo[2]
[1]Center of Behavioral Cardiovascular Health, Division of General Medicine, Columbia University, New York, NY, USA
[2]Center for Behavioral Cardiovascular Health, Columbia University, New York, NY, USA

Synonyms

Dietary lipids absorption

Definition

The absorption of the fat begins in the intestine with the help of several enzymes which is closely regulated by local hormones.

Description

More than 90% of dietary fat is in the form of triacylglycerol (TAG). The remainder of the fat is in the form of cholesterol, cholesteryl esters, phospholipids, and free fatty acids, which are unesterified. The mechanism of fat absorption includes degradation by the local enzymes, mediated by the hormones in the gastrointestinal system.

The digestion of the lipids begins in the stomach, when the food content is mixed with salivary lipase produced in the mouth. The gastric lipase is produced by the stomach and salivary lipase, which are resistant to gastric acidity, helps the breakdown of short- and medium-chain TAG molecules. Once the lipid-rich food reaches the duodenum, which is the first part of small intestine, the process of emulsification begins with the addition of bile salts and mechanical peristalsis. With the emulsification, the surface area of the lipid molecules is increased and also prevents their coalescing with other lipid molecules. Eventually, pancreatic enzymes play a major role in the absorption of dietary lipids. TAG is initially a larger molecule when it enters the intestine, and the pancreatic lipase removes the fatty acids from TAG to make it a smaller molecule, which is then taken up by intestinal villi.

Dietary cholesteryl esters in the form of cholesterol are in the nonesterified (free) form. Cholesterol esterase, a pancreatic enzyme, degrades cholesteryl esters into free fatty acids and cholesterol. Phospholipids in the food are degraded by the phospholipase, another pancreatic enzyme.

The lipid digestion is controlled by two important hormones in the intestine. Cholecystokinin (CCK) is a local hormone produced by jejunum and duodenum after the partially digested fat-rich food. CCK contracts the gall bladder to release bile and also act on the cells of pancreas to produce the pancreatic digestive enzymes. Bile fluid is produced from the liver and stored in the gall bladder, and it is rich in bile salts, phospholipids, and free cholesterol. Bile salts help in the emulsification process as mentioned before. Secretin, another small peptide hormone produced by the intestinal cells, act on the liver and pancreas to produce the watery solution rich in bicarbonates and this helps to alkalinize the gastric acidity when the food enters the duodenum. This helps to maintain the optimum pH for the action of all the above-described enzymes.

Free fatty acids, free cholesterol, and 2-monoacylglycerol are the final products of lipid digestion in the intestine. These end products along with bile salts and fat-soluble vitamins form mixed micelles. The micelles are disk-shaped clusters with water-soluble components located outside and water insoluble components located inside their surface. These mixed micelles are absorbed from the brush border of intestinal mucosal cells. Short- and medium-chain length fatty acids are directly absorbed without the assistance of micelles.

After the absorption of these lipids in the intestinal cells, the longer-chain fatty acids are further taken up by the endoplasmic reticulum of intestinal cells where the synthesis of complex lipids

takes place. The longer fatty acids are activated by the enzyme, fatty acyl Co A synthetase, and are transformed into TAG with the help of TAG synthase. The small and medium-chain fatty acids are directly released into the portal circulation to the liver after binding to albumin.

Absorbed lipid components either go to liver through the portal vein or directly in to systemic circulation to the rest of the body through the lymphatic system.

Cross-References

- ▶ Lipid Metabolism
- ▶ Plasma Lipid

References and Readings

Harvey, R. A., & Ferrier, D. R. (2008). Cholesterol and steroid metabolism. In R. A. Harvey (Ed.), *Lippincott's illustrated reviews biochemistry* (pp. 173–180). Philadelphia: Wolters Kluwer/ Lippincott Williams and Wilkins.

Fat Mass

- ▶ Body Composition

Fat Metabolism

- ▶ Lipid Metabolism

Fat, Dietary Intake

Tavis S. Campbell, Jillian A. Johnson and Kristin A. Zernicke
Department of Psychology, University of Calgary, Calgary, AB, Canada

Synonyms

Monounsaturated fats; Polyunsaturated fats; Saturated fats; Trans fats

Definition

Dietary fat is broken down into glycerol and fatty acids in the stomach and intestine when ingested. The glycerol and fatty acids are then altered in a process called emulsification in order for fats to be held in the digestive fluids long enough to be digested. Once digested, the fat is transported by the body's cells via the bloodstream and lymphatic system. If the fat is not required immediately, glycerol can be converted into glucose and either used for energy, stored as glycogen (short term) or stored as ▶ body fat (long term).

Given that fats are higher in calories than carbohydrates or protein, all dietary fats should be ingested with moderation. The American Heart Association recommends that approximately 20–35% of daily ▶ caloric intake should come from fats. Depending on height, weight, and activity level, recommended intake ranges from 45 to 75 g of fat a day for women and 60 to 105 g of fat a day for men. The amount of fat recommended for children and adolescents depends on height, weight, gender, and activity level.

Unlike carbohydrates and proteins that have one major function, dietary fat has a number of important roles in the body. These include forming the structure of cell membranes, helping absorb vitamins, lubricating joints, providing insulation for nerves (myelin sheath), supporting strong bones, and supporting a strong immune system.

Description

Fats are constructed from a combination of carbon and hydrogen atoms that are chemically bonded together. The structure of this chemical bond determines the type of dietary fat. There are four main types of dietary fat: saturated, monounsaturated, polyunsaturated, and ▶ trans fats.

▶ *Saturated fats (saturated fatty acids) (SAFAs):* All carbon atoms are bonded to hydrogen atoms in the chemical structure of a saturated fat. These fats have the highest

melting point of all the natural fats and therefore remain solid at room temperature. Saturated fats increase levels of both HDL and LDL ▶ cholesterol; therefore, moderate consumption is recommended for healthy individuals. These fats are found primarily in animal products such as fatty meats, full-fat dairy products, butter, lard, coconut oil, and palm oil.

▶ *Monounsaturated fats (monounsaturated fatty acids) (MUFAs):* In the chemical structure, monounsaturated fats contain one double bond between carbon atoms. Thus, the carbon atoms are bonded to hydrogen atoms everywhere but at the double carbon bond and are therefore only saturated with hydrogen atoms at this single point. Monounsaturated fats have a lower melting point than saturated fats and a higher melting point than polyunsaturated fats. Unsaturated fats increase HDL ▶ cholesterol levels while reducing LDL ▶ cholesterol levels and are therefore highly recommended for consumption. Ingesting foods high in MUFAs may also benefit ▶ insulin levels and blood sugar control. Foods rich in MUFAs include avocados, nuts, and olive oil.

▶ *Polyunsaturated fats (polyunsaturated fatty acids) (PUFAs):* In the chemical structure of polyunsaturated fats, there are two or more double bonds between carbon atoms. Thus, they are not fully saturated with hydrogen atoms at two or more points in the structure. Polyunsaturated fats have the lowest melting point of all dietary fats and remain liquid at low temperatures. The two main types of polyunsaturated fats are ▶ omega-3 fatty acids and omega-6 fatty acids. Omega-3s are found in coldwater oily fish, such as salmon, whereas omega-6s are found in vegetable oils. Both are ▶ essential fatty acids, meaning they cannot be produced by the body and must be acquired from PUFA-rich foods or ▶ dietary supplement. Unsaturated fats increase HDL ▶ cholesterol levels while reducing LDL ▶ cholesterol levels and are therefore highly recommended for consumption. Omega-3 s appear to decrease the risk of ▶ coronary artery disease and may also protect against blood clotting, reducing the risk of stroke, and lowering triglycerides.

▶ *Trans fats (trans-isomer fatty acids) (TFAs):* Trans fats involve adding hydrogen atoms to a fat that was originally unsaturated. These fats are created naturally when a hydrogen bond on an unsaturated fat gets twisted. However, the vast majority of trans fats are man-made in a process called hydrogenation. Man-made trans fats, called industrial or synthetic trans fats, are found in processed foods such as partially hydrogenated margarine, many commercially baked products, and deep-fried foods. TFAs have a high melting point and remain solid at room temperature, making them easier to cook with and less likely to spoil compared to naturally occurring oils. Synthetic trans fats increase unhealthy LDL ▶ cholesterol and lower healthy HDL ▶ cholesterol, increasing risk for ▶ cardiovascular disease and therefore should be avoided. Synthetic trans fats are believed to have no health benefits.

Research indicates long-term consumption of a high-fat diet contributes to increased mortality and morbidity. Consumption of a high-fat diet is a contributing factor to the development of obesity. ▶ Obesity and excessive body weight are associated with various diseases, such as ▶ cardiovascular disease, diabetes mellitus type 2, certain types of cancers, ▶ obstructive sleep apnea, osteoarthritis, and ▶ metabolic syndrome (a combination of disorders including diabetes mellitus type 2, high blood pressure, high blood cholesterol, and high triglyceride levels). As a specific example, a diet high in fat may contribute to the development of ▶ atherosclerosis by activating elevations in blood pressure, which can lead to further risk of other cardiovascular events. While a high-fat diet contributes to negative health outcomes, a change in diet including an increase in the consumption of certain fats, like ▶ omega-3 fatty acids, in combination with a reduction in the consumption of saturated fats can have protective and therapeutic health benefits. These benefits may include a reduction in overall ▶ cholesterol levels and blood pressure, reduced risk for chronic illness, as well as improvements in mood. It is generally recommended daily intake of dietary fat should be limited to 30% of daily ▶ caloric

intake, with the majority of these calories coming from monounsaturated fats or polyunsaturated fatty acids.

Cross-References

- ▶ Caloric Intake
- ▶ Cholesterol
- ▶ Fat: Saturated, Unsaturated
- ▶ Omega-3 Fatty Acids
- ▶ Trans Fatty Acids

References and Readings

DeMeester, F., Zibadi, S., & Watson, R. R. (Eds.). (2010). *Modern dietary fat intakes in disease promotion*. New York: Humana Press.

Heart and Stroke Foundation (2010, January). *Dietary fats, oils and cholesterol*. Retrieved April 8, 2011 from http://www.heartandstroke.com/site/c.ikIQLcMWJtE/b.3484237/k.D734/Healthy_living__Dietary_fats_oils_and__cholesterol.htm

Panel on Dietary Reference Intakes for Macronutrients, Subcommittees on Upper Reference Levels of Nutrients and Interpretation and Uses of Dietary Reference Intakes, & Standing Committee on the Scientific Evaluation of Dietary Reference Intakes (Eds.). (2005). Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (macronutrients). Washington, DC: The National Academies Press.

Taubes, G. (2001). Nutrition: The soft science of dietary fat. *Science, 291*, 2536–2545.

U.S. Department of Agriculture & U.S. Department of Health and Human Services. (2010). *Dietary guidelines for Americans 2010* (7th ed.). Washington, DC: U.S. Government Printing Office.

Fat: Saturated, Unsaturated

Kelly Flannery
School of Nursing, University of Maryland Baltimore, Baltimore, MD, USA

Synonyms

Monounsaturated fatty acids; Oils; Polyunsaturated fatty acids; Saturated fatty acids; Solid fats; Trans-fatty acids

Definition

Fat is an energy source derived from food. There are four types of dietary fat: saturated, trans, monounsaturated, and polyunsaturated fat (U.S. Department of Agriculture & U.S. Department of Health and Human Services, 2010).

Description

Fat is an essential dietary element because it supplies calories, helps insulate the body, aids in the absorption of fat-soluble vitamins (i.e., A, D, E, K) and keeps hair and skin healthy. Dietary fat also provides the body with essential fatty acids that aid in controlling inflammation, blood clotting, and developing the brain (U.S. National Library of Medicine & National Institutes of Health, 2011a).

Types of Fats

Based on their composition, the four fats are grouped into two subgroups: saturated fatty acids (saturated fat) and unsaturated fatty acids (which includes trans, monounsaturated, and polyunsaturated fat). However, trans fat is structurally different and not healthy like the other unsaturated fats, so for clarity it will be helpful to use the groups unhealthy (i.e., saturated and trans fat) and healthy fats (monounsaturated and polyunsaturated fat) (Mayo Clinic, 2011; U.S. Department of Agriculture & U.S. Department of Health and Human Services, 2010; U.S. National Library of Medicine & National Institutes of Health, 2011b). Sometimes, unhealthy fats are called solid fats and healthy fats are called oils (U.S. Department of Agriculture & U.S. Department of Health and Human Services, 2010).

Unhealthy Fats

Saturated fats. Saturated fats are grouped under the unhealthy fat category because they can raise

total cholesterol and LDLs (low-density lipoproteins – which are also called "bad" cholesterol). Diets high in saturated fat can result in occluded or narrowed arteries and increased risk for cardiovascular disease (Mayo Clinic, 2011; U.S. Department of Agriculture & U.S. Department of Health & Human Services, 2010; U.S. National Library of Medicine & National Institutes of Health, 2011a). Some examples of saturated fat include: ice cream, butter, cheese, whole milk, coconut oil, palm oil, and most animal fats (U.S. Department of Agriculture & U.S. Department of Health and Human Services, 2010; U.S. National Library of Medicine & National Institutes of Health, 2011a). The body makes enough saturated fat; therefore, it is not a dietary requirement (U.S. Department of Agriculture & U.S. Department of Health and Human Services, 2010).

Trans fats. Trans fats are unhealthy because they can raise LDL (bad) cholesterol and lower HDLs (high-density lipoproteins – which are also called "good" cholesterol). Excessive trans fat consumption can lead to increased risk for cardiovascular disease (Mayo Clinic, 2011; U.S. National Library of Medicine & National Institutes of Health, 2011a). Some examples of trans fat include: processed foods, fried foods, and commercially baked foods (U.S. National Library of Medicine & National Institutes of Health, 2011a). Trans fats are also not essential dietary fats (U.S. Department of Agriculture & U.S. Department of Health and Human Services, 2010). Sometimes trans fats are also called partially hydrogenated oils (Centers for Disease Control and Prevention [CDC], 2010).

Healthy Fats

Monounsaturated fats. Monounsaturated fats are grouped under healthy fats because replacing unhealthy fats (i.e., saturated and trans fats) with monounsaturated fats can lower LDL (bad) cholesterol (U.S. National Library of Medicine & National Institutes of Health, 2011a). Monounsaturated fats also provide essential fatty acids that are not produced by the body; these essential fatty acids are needed for physiological and structural functions (Centers for Disease Control and Prevention [CDC], 2011; U.S. Department of Agriculture & U.S. Department of Health and Human Services, 2010). Some examples of monounsaturated fat include: canola oil, olive oil, and avocados (CDC, 2011).

Polyunsaturated fats. Polyunsaturated fats are grouped under healthy fats because replacing unhealthy fats (i.e., saturated and trans fats) with polyunsaturated fats can lower LDL (bad) cholesterol (U.S. National Library of Medicine & National Institutes of Health, 2011a). Polyunsaturated fats also provide essential fatty acids that are not produced by the body; these essential fatty acids are needed for physiological and structural functions (CDC, 2011; U.S. Department of Agriculture & U.S. Department of Health and Human Services, 2010). Additionally, there are two subgroups of polyunsaturated fats, omega 6 polyunsaturated fats, and omega 3 polyunsaturated fats. Some examples of omega 6 polyunsaturated fats include safflower oil and corn oil whereas some examples of omega 3 polyunsaturated fats include flaxseed, canola oil, walnuts, trout, and salmon (CDC, 2011).

Recommendations

Generally, it is advised that most fat intake should come from healthy sources of fat (i.e., monounsaturated and polyunsaturated fat) and unhealthy sources of fat (i.e., saturated and trans fat) should be limited. Total fat intake should range from 30% to 40% of calories for children 1–3 years of age, 25–35% of calories for those 4–18 years of age and 20–35% of calories for adults over the age of 19 (U.S. Department of Agriculture & U.S. Department of Health and Human Services, 2010). The recommendations suggest healthy Americans over the age of 2 consume less than 1% of daily calories from trans fat, less than 7% of daily calories from saturated fat, and replace remaining fat calories with healthy fats (American Heart Association, 2012).

Further, the recommendations suggest limiting unhealthy fat and replacing it with healthy fat.

However, regardless of the type of fat (i.e., healthy or unhealthy) fat intake should be limited because dietary fat provides 9 calories per gram which is more than double the amount of calories other nutrients provide (e.g., carbohydrates provide 4 calories per gram) (U.S. Department of Agriculture & U.S. Department of Health and Human Services, 2010; U.S. National Library of Medicine & National Institutes of Health, 2011b). Therefore, eating a diet of more fat than recommended (healthy or unhealthy) can lead to overweight or obesity and its associated risk factors (U.S. National Library of Medicine & National Institutes of Health, 2011b). Most Americans consume more total fat and more unhealthy fat than recommended and less healthy fat than recommended (U.S. Department of Agriculture & U.S. Department of Health & Human Services, 2010).

Cross-References

- ▶ Essential Fatty Acids
- ▶ Fat Absorption
- ▶ Fat, Dietary Intake
- ▶ Hyperlipidemia
- ▶ Omega-3 Fatty Acids

References and Readings

American Heart Association. (2012). Know your fats. Retrieved March 22, 2012, from http://www.heart.org/HEARTORG/Conditions/Cholesterol/Prevention-TreatmentofHighCholesterol/Know-Your-Fats_UCM_305628_Article.jsp#.T2oNgvWLGeJ

Centers for Disease Control and Prevention. (2010). *Nutrition for everyone: Trans fat*. Retrieved April 15, 2011, from http://www.cdc.gov/nutrition/everyone/basics/fat/transfat.html

Centers for Disease Control and Prevention. (2011). *Nutrition for everyone: Polyunsaturated fats and monounsaturated fats*. Retrieved April 15, 2011, from http://www.cdc.gov/nutrition/everyone/basics/fat/unsaturatedfat.html

Mayo Clinic. (2011). *Dietary fats: Know which types to choose*. Retrieved April 16, 2011, from http://www.mayoclinic.com/health/fat/NU00262

U.S. Department of Agriculture & U.S. Department of Health and Human Services. (2010). *Dietary guidelines for Americans 2010* (7th ed.). Washington, DC: U.S. Government Printing Office.

U.S. National Library of Medicine, & National Institutes of Health. (2011a). *Dietary fats explained*. Retrieved April 15, 2011, from http://www.nlm.nih.gov/medlineplus/ency/patientinstructions/000104.htm

U.S. National Library of Medicine, & National Institutes of Health. (2011b). *Fat*. Retrieved April 15, 2011, from http://www.nlm.nih.gov/medlineplus/ency/article/002468.htm

F

Fatalism

Karla Espinosa de los Monteros
Clinical Psychology, SDSU/UCSD Joint Doctoral Program in Clinical Psychology, San Diego, CA, USA

Definition

Fatalism refers to the general belief that events, such as the actions and occurrences that form an individual life, are determined by fate, and, thus, beyond the capacity of human beings to control. When applied to health, fatalism can be conceptualized as the belief that the development and course of health problems is beyond an individual's personal control (Straughan & Seow, 1998). Research on the relationship between fatalism and health has generally focused on fatalistic beliefs about specific diseases, the most commonly studied being cancer. Powe and Johnson (1995) defined cancer fatalism as a situational manifestation of fatalism where an individual feels powerless in the face of cancer and views its diagnosis as a struggle against insurmountable odds.

Description

The shift from ▶ acute disease to chronic disease as the major cause of morbidity and mortality in developed countries has highlighted the importance of lifestyle factors in the prevention of

disease. This paradigm shift has fostered efforts to understand how ▶ cognitive factors, such as fatalism, influence an individual's decision to adopt health-promoting behaviors. Fatalism's influence on behavior may stem from its impact on an individual's perceived ▶ self-efficacy to control life events, the outcomes attributed to a behavior, and overall motivation to change, maintain, or adopt behaviors (Freeman, 1989; Powe & Johnson, 1995; Straughan & Seow, 1998). For example, an asymptomatic individual who believes that cancer is unavoidable regardless of personal action is likely to perceive few benefits to cancer screening, particularly in light of the material losses (e.g., time, money) and aversive experiences (e.g., discomfort, anxiety) associated with the behavior. Indeed, cancer fatalism has been associated with the underutilization of cancer screening services, delay of care, smoking, physical inactivity, and poor dietary practices (Espinosa de los Monteros & Gallo, 2010; Niederdeppe & Levy, 2007; Powe & Finnie, 2003). Research focusing on fatalistic beliefs about diseases other than cancer is more limited, but preliminary evidence suggests that the construct may also be associated with high-risk sexual behavior and diabetes management.

Cultural differences in the endorsement of fatalistic beliefs about health and illness have been reported. For example, in the United States, cancer fatalism is more common in African American and Hispanic American populations than in non-Hispanic Whites (Abraido-Lanza et al., 2007; Powe & Finnie, 2003). While this pattern may in part be attributed to variation in the dominant worldviews of different cultural groups, generally, fatalistic beliefs about health and illness are most prominent in older adults and less educated populations, as well as in groups that have historically experienced significant social disadvantages (Abraido-Lanza et al., 2007; Davison et al., 1992; Powe & Johnson, 1995). Given these differences, fatalism has at times been studied as a means to understand the factors contributing to socioeconomic, racial, and ethnic ▶ disparities in health behavior, and many studies have called for the development of culturally sensitive interventions to address fatalistic perceptions about health within high-risk groups.

However, theories on the development and maintenance of fatalistic beliefs in regards to health and illness stress the importance of considering certain points. First, care should be taken in interpreting fatalistic beliefs as irrational before considering the social and physical barriers to health that are faced by certain populations. For example, poverty, ▶ discrimination, and limited access to health-promoting resources such as health education and medical care represent tangible barriers to disease prevention and treatment (Freeman, 1989). Therefore, for certain individuals, fatalistic beliefs about health and illness may be grounded on realistic appraisals of individual control and may more accurately represent a balance between the almost universally valued goal of good health, and the recognition that some barriers to health may not be overcome through personal effort (Davison et al., 1992).

Second, while all or none categories are often used to describe the nature of fatalistic beliefs – i.e., individuals are either fatalistic or they are not – empirical evidence shows that people rarely embrace either extreme (Davison et al., 1992). Thus, a more accurate conceptualization of fatalistic beliefs about health and illness is that they fall within a spectrum ranging from high to low, and where an individual falls within that spectrum will likely depend on the disease or behavior in question, as well as the context in which fatalism is assessed.

Finally, while fatalistic beliefs about health and illness may be more prominent in certain populations, they are not exclusive to any one group. As Davison et al. (1992) pointed out, as long as people continue to witness health outcomes that are inconsistent with their current biomedical understanding of disease, there will

always be a place for the notion of fate to help people make sense of what cannot be easily explained.

Cross-References

- ▶ Acute Disease
- ▶ Discrimination
- ▶ Disparities
- ▶ Self-efficacy

References and Readings

Abraido-Lanza, A. F., Viladrich, A., Florez, K. R., Cespedes, A., Aguirre, A. N., & De La Cruz, A. A. (2007). Fatalismo reconsidered: A cautionary note for health-related research and practice with Latino populations. *Ethnicity & Disease, 17*, 153–158.

Davison, C., Frankel, S., & Smith, G. D. (1992). The limits of lifestyle: Re-assessing 'fatalism' in the popular culture of illness prevention. *Social Science & Medicine, 34*(6), 675–685.

Espinosa de los Monteros, K., & Gallo, L. C. (2010). Fatalism, Latinas, and cancer screening: A systematic review of the literature. *International Journal of Behavioral Medicine*. doi:10.1007/s12529-010-9119-4.

Freeman, H. (1989). Cancer and the socioeconomically disadvantaged. *CA A Cancer Journal for Clinicians, 39*, 266–288.

Niederdeppe, J., & Levy, A. G. (2007). Fatalistic beliefs about cancer prevention and three prevention behaviors. *Cancer Epidemiology, Biomarkers, & Prevention, 16*, 998–1003.

Powe, P. D., & Finnie, R. (2003). Cancer fatalism: The state of the science. *Cancer Nursing, 26*(6), 454–465.

Powe, B. D., & Johnson, A. (1995). Fatalism as a barrier to cancer screening among African-Americans: Philosophical perspectives. *Journal of Religion & Health, 34*, 119–125.

Straughan, P. T., & Seow, A. (1998). Fatalism reconceptualized: A concept to predict health screening behavior. *Journal of Gender, Culture, & Health, 3*(2), 85–100.

Fatality

- ▶ Mortality

Fat-Free Mass

- ▶ Body Composition

Fatigue

Fred Friedberg
Psychiatry and Behavioral Sciences, Stony Brook University Medical Center, Stony Brook, NY, USA

Synonyms

Chronic fatigue; Chronic fatigue syndrome; Energy; Exhaustion; Illness fatigue; Self-management; Tiredness; Treatment of fatigue

Definition

Fatigue is a subjective sense of tiredness or exhaustion. Although "fatigue" is a general term intended to encompass both tiredness and exhaustion, it can also refer to the midrange intensities between (milder) tiredness and (more severe) exhaustion. Tiredness is a normal time-limited response to sustained physical, mental, or emotional effort. More persistent states of fatigue and exhaustion may arise from behavioral and environmental stressors, or be a symptom of physical or psychological disorders. A number of physiologic mechanisms for fatigue have been proposed, and some correlations have been identified, but clear biologic markers have yet to be established.

Description

The nearly ubiquitous experience of tiredness in daily life may devalue the symptom of fatigue as

a potential concern to health professionals. As such, the complaint of fatigue is often regarded as non-serious by physicians, but is considered one of the most important symptoms by patients.

Self-report fatigue severity in the population is normally distributed with pathological fatigue represented at higher levels on this quantitative continuum. However, fatigue may also be qualitatively and biologically different depending on its origins (e.g., disease, occupational). For instance, mental and physical fatigue are empirically distinguishable constructs. Persistent fatigue may impact physical and cognitive functioning as well as emotional well-being and quality of life.

Useful distinctions can be made on a severity dimension of tiredness, fatigue, and exhaustion. Similar to Selye's general adaptation syndrome (alarm, resistance, exhaustion), individuals with normal tiredness experience loss of energy in proportion to the amount of energy expended, whereas individuals with (persistent) fatigue experience loss of energy sooner than expected and out of proportion to the amount of energy expended. Finally, individuals with ongoing exhaustion experience sudden and unpredictable losses of energy, often without any identifiable energy expenditure.

These three states of fatigue are also linked to increasing cognitive difficulties, reduced sleep quality, and lessened ability to engage in social interaction. The relative places of tiredness, fatigue, and exhaustion in the adaptation process have implications for the types of interventions that are most appropriate. For example, mild exercise, which might be appropriate for someone experiencing fatigue, might be an additional stressor (and therefore harmful) for a person experiencing exhaustion. This piece will focus on persistent fatigue with respect to diagnosis, lifestyle factors, illness conditions, medication side effects, and treatment.

Diagnosis

Persistent fatigue is a common symptom in health care and is usually not due to an identifiable disease. Definitive physical conditions are found in less than 1/10. In those people who have a clear diagnosis, musculoskeletal and psychological problems are the most common. If a person with fatigue decides to seek medical advice, the overall goal is to identify and/or rule out any treatable conditions. This is done by considering the person's medical and psychosocial history and other symptoms that may be present, conducting standard laboratory tests, and evaluating the qualities of the fatigue itself.

Lifestyle Factors

Behavioral and psychosocial factors linked to persistent fatigue include physical inactivity, overwork, poor ▶ sleep, affective distress, and poor diet/overweight.

Physical inactivity. In modern sedentary societies, occupational, social, and leisure activities typically involve little physical effort. This often indicates a lack of exercise or physically active hobbies. Persistent fatigue may be generated by such physical inactivity. In addition, general inactivity, both physical and mental, may trigger boredom and apathy which can further increase fatigue.

Overwork. In work-focused cultures, near-continuous engagement in goal-directed mental and intellectual activities (often in combination with mild to moderate sleep deprivation) may result in persistent fatigue.

Sleep difficulties. Disrupted ▶ sleep, a significant contributor to fatigue, is related to sleep quality, amount of sleep, the hours set aside for sleep, and the number of times that a person awakens during the night.

Affective distress. Emotions including anxiety, discouragement, and depressed mood may be accompanied by the symptom of fatigue.

Poor diet/overweight. Western diets – typically high calorie, high sugar, and high fat – are linked to overweight and obesity. Self-reported fatigue is associated with higher body mass index and a higher waist circumference.

Illness-Related Fatigue

Chronic illnesses both psychiatric and medical often feature the symptom of persistent fatigue. Psychiatric conditions that commonly exhibit fatigue are clinical depression and generalized anxiety disorder. Medical conditions linked to significant fatigue include anemia, low thyroid, diabetes, cardiovascular disease, arthritis, HIV/AIDS, autoimmune diseases, cancer, fibromyalgia, and traumatic brain injury. Sleep disorders such as ongoing ► insomnia, ► obstructive sleep apnea, and narcolepsy exhibit fatigue as well.

In cancer patients, fatigue has emerged as one of the most prevalent, troubling, and undertreated of all symptoms. In addition, subjective reports of fatigue after activities in the elderly have been found to be a strong independent predictor of functional decline, disability, and death.

Finally, chronic fatigue syndrome (CFS) is an illness defined by medically unexplained fatigue of 6 months or more plus 4/8 secondary symptoms (e.g., post-exertional fatigue, muscle and joint pain symptoms, flu-like symptoms) and significant impairments in physical and role functioning. The fatigue in CFS is only partially alleviated by rest and is qualitatively different from ordinary tiredness. In addition, exercise that was easily tolerated before illness onset may worsen fatigue-related symptoms. Biomedical and behavioral factors have been found in CFS, but no definitive etiology or pathophysiology has been identified.

Drugs and Medication Side Effects

The use of alcohol, ► caffeine, or illegal drugs, such as cocaine or narcotics, especially with regular use or abuse may result in persistent fatigue. Fatigue may also be a side effect of certain medications, e.g., antihistamines, sleeping pills, and lithium salts; blood pressure medicines such as beta-blockers, which can induce exercise intolerance; and many cancer treatments, particularly chemotherapy and radiotherapy.

Treatment of Fatigue

Given the current limitations of medicine in treating fatigue, self-management is an essential clinical issue because sufferers have options ranging from doing nothing to actively seeking help. Early intervention (e.g., individuals with persistent fatigue of less than 12 months) is most likely to be beneficial. However, if the patient has already reached a stabilized level of persistent fatigue, more powerful interventions may be necessary to restart the self-management process.

Non-pharmacological treatment options include patient education about fatigue and its relation to stress and lifestyle. Diary-keeping to track activities, stressors, and sleep patterns and their relation to fatigue and energy are important first steps in designing a self-management program.

Self-management. Self-management techniques for unexplained persistent fatigue, CFS, and illness fatigue in general have shown efficacy in various combinations that include pacing of activities, low-level exercise, low-effort pleasant experiences, sleep improvement techniques, and cognitive coping skills to reduce both illness catastrophizing and over-focusing on symptoms. It should be noted that low-level exercise, typically walking or stretching, is initially prescribed as a stress reduction activity rather than a physical fitness program. Relaxation techniques are particularly helpful for affective distress linked to fatigue. In general, the goal of an effective self-management program is to learn to balance activity and rest in order to avoid the extremes of too little or too much activity. This approach to self-management will lessen fatigue, but probably not eliminate it.

Medications. Medical practice includes corticosteroids as a short-term therapeutic option for relief of fatigue in palliative care. Given limited evidence, no specific drug can be recommended for the treatment of persistent fatigue, although amantadine in multiple sclerosis and methylphenidate in cancer patients have shown promise. Selective serotonin reuptake inhibitors, such as fluoxetine, paroxetine, or sertraline, may improve energy in some patients with comorbid depression. Modafinil, a wakefulness-promoting agent

that is pharmacologically different from other stimulants, has not been adequately tested as a treatment for fatigue.

Alternative treatments. Regarding alternative treatments, acupuncture and several types of meditative practice show the most promise for future scientific investigation. Likewise, magnesium, L-carnitine, and *S*-adenosylmethionine are non-pharmacological supplements with potential in further research. Chinese medicinal herbs for persistent fatigue and CFS lack evidence from controlled studies.

Summary

Fatigue is a commonly experienced symptom that is associated with inactivity, overwork, affective distress, physical and psychiatric illnesses, and drug side effects. In contrast to normal tiredness, the persistent state of fatigue can impact physical and role functioning and quality of life. Medical treatment of fatigue is not well-established. By comparison, self-management of debilitating fatigue through behavioral interventions such as pacing, low-level exercise, and exposure to low-effort pleasant activities can lead to reduced fatigue, increased functioning, and improved quality of life.

Cross-References

- ▶ Cardiovascular Disease
- ▶ Diabetes
- ▶ HIV Infection
- ▶ Sleep

References and Readings

DeLuca, J. (Ed.). (2005). *Fatigue as a window to the brain*. Cambridge, MA: Massachusetts Institute of Technology.

Friedberg, F. (2010). Chronic fatigue syndrome, fibromyalgia, and related illnesses: A clinical model of assessment and intervention. *Journal of Clinical Psychology, 6*, 641–665.

Olsen, K. (2007). A new way of thinking about fatigue: A reconceptualization. *Oncology Nursing Forum, 34*(1), 93–99.

Porter, N. S., Jason, L. A., Boulton, A., Bothne, N., & Coleman, B. (2010). Alternative medical interventions used in the treatment and management of myalgic encephalomyelitis/chronic fatigue syndrome and fibromyalgia. *Journal of Alternative & Complementary Medicine, 16*(3), 235–249.

Schor, J. (1992). *The overworked American: The unexpected decline in leisure*. New York: Basic Books.

Taylor, R., Fennell, P., & Jason, L. A. (Eds.). (2003). *Handbook of chronic fatigue syndrome*. New York: Wiley.

Fatty Acids, Free

Manjunath Harlapur[1] and Daichi Shimbo[2]
[1]Center of Behavioral Cardiovascular Health, Division of General Medicine, Columbia University, New York, NY, USA
[2]Center for Behavioral Cardiovascular Health, Columbia University, New York, NY, USA

Synonyms

Fatty acids-unesterified

Definition

Free fatty acids are unesterified, long-chain carboxylic acids. After esterification, fatty acids are found in the complex molecules like triacylglycerols. Usually, low levels of free fatty acids are seen in all tissues, but the substantial increase in the plasma is seen during fasting state. Free fatty acids are transported by albumin. Free fatty acids are oxidized mainly in the liver and muscle cells to provide energy. Fatty acids are also structural components of membrane lipids such as glycolipids and phospholipids. Fatty acids are also the precursor of prostaglandins. After esterification, fatty acids are stored as triacylglycerol in the adipose tissue, and this serves as a major reservoir of energy during fasting.

Fatty acids are called as "unsaturated" if they have at least one double bond in their chemical structure and "saturated" if they have none. Humans can only produce few unsaturated fatty acids in the body, and remaining fatty acids are

obtained from the dietary intake, and these are called essential fatty acids. Two important essential fatty acids are linolenic and linoleic acids.

Omega numbering system is used for unsaturated fatty acids, and it depicts the position of double bond relative to the end of chain. Dietary intake of omega-3 fatty acids (e.g., linolenic acid) in the diet is associated with a reduced risk of cardiovascular disease events.

Cross-References

- ▶ Coronary Artery Disease
- ▶ Lipid Metabolism
- ▶ Plasma Lipid

References and Readings

Harvey, R. A. PhD, & Ferrier, D. R. (2008). *Lippincott's illustrated reviews: Biochemistry (chap. 16)* (Lippincott's illustrated reviews series 4th ed., pp. 181–200). Philadelphia: Wolters Kluwer/ Lippincott Williams & Wilkins.

Fatty Acids-Unesterified

- ▶ Fatty Acids, Free

Fear

- ▶ Anxiety

Fear and Fear Avoidance

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

Fear is an unpleasant negative emotion usually rising due to or in association with a specific source or stimulus. Fear avoidance would take place by a behavior which reduces the probability of exposure to the fear-eliciting stimulus or situation. A negative reinforcement process takes place where the lack of elicited fear reinforces the behavior which reduced its probability – normally the avoidance. This is often seen, for example, in the context of chronic pain, where patients fear a situation that in the past increased their pain (e.g., going for a walk). Remaining at home and avoiding activity (disability) is negatively reinforced by the lack of further increases in pain from walking outside. This process could then increase the patient's disability levels. This process is at the core of the fear-avoidance model of pain (Leeuw et al., 2007; Vlaeyen & Linton, 2000). Additional variables that are thought to play roles in this model include cognitive variables such as catastrophizing (severely negative appraisals about anticipated pain), psychophysiological (e.g., elevated muscle reactivity, high sympathetic arousal), and emotional (subsequent fear and anxiety). Numerous elements of the model have been validated. Indeed, one study found in a large sample of back pain patients that baseline fear avoidance predicted high rates of long-term sick leave (Boersma & Linton, 2006). However, one review of nine prospective studies in low back pain patients found that fear and fear avoidance were not consistently predictive of pain. There was some evidence that such fears do play a role in predicting future pains when pain has become consistent (Pincus, Vogel, Burton, Santos, & Field, 2006). It is possible that disability may be more influenced by fear avoidance than pain. Importantly, the clinical application of the fear-avoidance model was tested in a small-scale study with chronic back pain patients. All patients received either first in vivo exposure to individualized fear-eliciting movements (to reduce fear avoidance) or to general graded physical activity, or the reversed order. Only during in vivo exposure to fear-eliciting movements were there reductions in pain-related fears and in catastrophizing (Vlaeyen et al., 2002). This model could be applicable to other health contexts – patients may avoid attending oncology clinics where they

receive chemotherapy which elicits nausea and vomiting, at the possibly grave price of not treating adequately their cancer. Fear and fear avoidance are also of much relevance to anxiety disorders. Thus, fear avoidance is a prevalent problem in medical settings, and its treatment could possibly result in improved health outcomes. Treating fear avoidance could be done by behavioral or cognitive-behavioral methods.

Cross-References

- ▶ Anxiety Disorder
- ▶ Pain Management/Control
- ▶ Pain-Related Fear

References and Readings

Boersma, K., & Linton, S. J. (2006). Expectancy, fear and pain in the prediction of chronic pain and disability: a prospective analysis. *Eur J Pain, 10*, 551–557.

Leeuw, M., Goossens, M. E., Linton, S. J., Crombez, G., Boersma, K., & Vlaeyen, J. W. (2007). The fear-avoidance model of musculoskeletal pain: Current state of scientific evidence. *Journal of Behavioral Medicine, 30*, 77–94.

Pincus, T., Vogel, S., Burton, A. K., Santos, R., & Field, A. P. (2006). Fear avoidance and prognosis in back pain: A brssystematic review and synthesis of current evidence. *Arthritis and Rheumatism, 54*, 3999–4010.

Vlaeyen, J. W. S., & Linton, S. J. (2000). Fear-avoidance and its consequences in chronic musculoskeletal pain: A state of the art. *Pain, 85*, 317–332.

Vlaeyen, J. W., de Jong, J., Geilen, M., Heuts, P. H., & van Breukelen, G. (2002). The treatment of fear of movement/(re)injury in chronic low back pain: further evidence on the effectiveness of exposure in vivo. *Clin J Pain, 18*, 251–261.

Fear of Death

- ▶ Death Anxiety

Fear of Hospitals

- ▶ Hospital Anxiety

Feasibility Study

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

A feasibility study is undertaken to determine whether there is a sufficiently high (acceptable) likelihood that a research study being considered can be successfully executed.

Description

When planning a large and complex research study (particularly an experimental study such as a randomized clinical trial of a behavioral intervention), it is wise to conduct a feasibility study once the study protocol has reached a relatively final stage of development. At that point, the researchers have a good idea of the number of subjects they will need to participate in the trial (the sample size) and many other methodological requirements. The question then becomes: Is there an acceptably high likelihood that it is actually feasible to conduct the trial? Phrased in another manner, the question is: Can the trial be executed as currently laid out in the protocol? A feasibility study is undertaken to answer this question. If the answer is "no," the researchers can consider making changes to the protocol before its finalization to improve the likelihood that the trial is capable of providing a meaningful answer to the research question being asked.

If the research team has done a previous (smaller) trial, they will have information on investigational sites used, principal investigators, and subject recruitment rates. This will help to answer the following questions:

- Where were the investigational sites used in the previous trial(s) located?
- How easy was it to recruit and retain the required number of subjects for the previous

trial, and did ease of recruitment vary across geographic locations within the country? (For some large trials, where investigational sites were located in more than one country, and potentially more than one continent, the answer to this question becomes more complex.)

- How similar is the study design on this occasion? We know that the study size (size of the subject sample) is going to be larger, but are there any other factors that might impact subject recruitment and retention? Such possibilities include more extensive measurement schedules. In cases where more blood samples are to be taken, or more invasive assessment procedures are to be used (all of which information will be in the study's informed consent form), it is possible that the subject recruitment and retention rates could be impacted.

An additional way of collecting relevant feasibility information is to create a survey that is sent with the draft study protocol to behavioral medicine specialists at various clinical and medical centers, i.e., potential investigational sites, at which the trial may be conducted. Such a survey asks a series of questions targeted at understanding if the specialists would be able to recruit certain subjects into the trial, and the timeline by which enrollment will be completed.

When the research team receives back the completed surveys, they need to consider the feedback carefully. It is widely acknowledged that physicians and clinicians interested in participating in clinical trials often inflate (unconsciously or consciously) the number of subjects they say they can recruit. They may also make overly ambitious statements about the suitability of their facilities and their abilities to operationally execute any particularly complicated aspects of the protocol. While such rose-tinted self-appraisals may initially make the physician's site look attractive for inclusion in the trial, subsequent site underperformance has a cascade of unfortunate consequences. Overall subject recruitment is negatively impacted, impacting completion of the trial. From the patients' perspective, this could mean that it takes a (much) longer period of time before a new intervention is available to them.

Cross-References

▶ Informed Consent
▶ Recruitment of Research Participants

Feeling

▶ Affect
▶ Mood

Feeling State

▶ Affect
▶ Mood

Feminine Role

▶ Gender Role

FHS

▶ Framingham Heart Study

Fibrinogen

Leah Rosenberg
Department of Medicine, School of Medicine, Duke University, Durham, NC, USA

Synonyms

Cardiovascular risk factors; Dimeric glycoprotein; Platelet plug

Definition

Fibrinogen is a glycoprotein that plays a critical role in blood clotting and has been also identified as a novel risk factor for acute coronary syndromes and strokes. Fibrinogen is produced by the liver and upregulated in the setting of physiologic stress. It is used clinically in the identification of disseminated intravascular coagulopathy, a serious dysregulation of blood clotting as well as diagnosis of rare bleeding disorders such as congenital hypofibrinogenemia.

A key component in the clotting cascade, fibrinogen functions in creating the platelet plug that creates hemostasis in an injured vessel. Fibrinogen is converted to fibrin via thrombin, a serine protease. Fibrin then undergoes further protein-based cross-linkages to create the plug for the vessel wall. Glycoprotein IIb/IIIa is a receptor for fibrinogen found on platelets that functions in platelet aggregation. Glycoprotein IIb/IIIa is a target for several drugs used to avoid thrombosis in cardiovascular disorders (Maron, Ridker, Grundy, & Pearson, 2010).

Fibrinogen's relevance in behavioral medicine centers on its potential importance as a marker of the stress reaction. The relationship of fibrinogen to other known inflammatory mediators such as IL-6 suggests a cascade of endogenous procoagulant proteins that may be associated with situations of psychosocial stress.

Cross-References

► Coagulation of Blood
► Fibrinolysis

References and Readings

Gomella, L. G., & Haist, S. A. (2007). *Clinician's pocket reference. Fibrinogen* (11th ed.). London: McGraw Hill.

Maron, D.J., Ridker, P.M., Grundy, S.M., & Pearson, T.A. (2010) Chapter 51. Preventive strategies for coronary heart disease (Chapter). Fuster, V., O'Rourke, R.A., Walsh, R.A., Poole-Wilson, P., (Eds.) King, S.B., Roberts, R., Nash, I.S., & Prystowsky, E.N., Assoc. (Eds.), *Hurst's the heart*(12 ed)

Fibrinolysis

Leah Rosenberg
Department of Medicine, School of Medicine
Duke University, Durham, NC, USA

Definition

Fibrinolysis is the process of dissolving the protein known as fibrin, after an injured vessel has healed and no longer requires a plug to achieve hemostasis. It is a complex cascade of enzymatic processes tightly orchestrated by a shifting balance of bloodstream procoagulant and anticoagulant factors (Boyle & Jaffe, 2010). Intravenous fibrinolytic agents have been utilized in the treatment of myocardial infarction. As an alternative to invasive procedures such as primary coronary interventions (PCI), medical approaches such as fibrinolysis are appropriate for certain patients who may not access to primary PCI.

Cross-References

► Coagulation of Blood

References and Readings

Boyle, A. J., & Jaffe, A. S. (2010). Acute myocardial infarction (chap. 5). In M. H. Crawford (Ed.), *Current diagnosis & treatment: Cardiology* (3rd ed.). New York: McGraw Hill Medical.

Fibromyalgia

Alexandre Morizio and Simon Bacon
Department of Exercise Science, Concordia University, Montreal Behavioral Medicine Centre, Montreal, QC, Canada

Synonyms

Fibromyalgia syndrome; Fibrositis

Definition

Fibromyalgia is a chronic rheumatoid disorder/syndrome characterized by widespread pain in the body. It affects 2–4% of the American population and is disproportionally higher in women compared to men (ratio 9:1). While it was once thought to be a psychosomatic problem (this belief was only recently dispelled), fibromyalgia is presently thought to be a condition associated with the body's central neuromodulators. The American College of Rheumatology classification criteria for fibromyalgia (released in 1990) include a history of widespread pain in the body for at least three months and pain from digital palpation in 11 of 18 tender points.

Description

According to the 1990 American College of Rheumatology (ACR) classification criteria for fibromyalgia, a patient can be diagnosed with fibromyalgia if the patient has a history of widespread pain in the body as well as in the axial skeleton (neck or back) for at least three months and must have pain from digital palpation in 11 of 18 tender points, these points being located bilaterally at the suboccipital muscle attachments on the occiput, at the anterior aspects of intertransverse spaces between the C5 to C7 vertebrae, at the midpoint of the upper border of the trapezius muscle, at the supraspinatus muscle near the medial border of the spine of the scapula, at the level of the second rib at the costochondral junctions, slightly distal to the lateral epicondyles, in the upper outer quadrants of the buttocks, at the greater trochanters, and at the medial aspect of the knee at a level slightly proximal to the joint line. More recently (2010), the ACR proposed a new set of diagnostic criteria which require patients to fulfill the following three criteria: widespread pain index (WPI) $\geq$7 and symptom severity (SS) scale score $\geq$5 or WPI 3–6 and SS scale score $\geq$9. Symptoms have been present at a similar level for at least 3 months, and the patient does not have a disorder that would otherwise explain the pain. However, it should be noted that these criteria have not yet been validated with independent samples.

One of the major problems in recognizing and categorizing fibromyalgia is that it presents in a similar fashion to other muscle pain disorders, fails to present specific features, and presently has no clinical diagnostic test. Pain in fibromyalgia is described as throbbing, stabbing, or burning and as such may be confused with arthritis; however, unlike arthritis, fibromyalgia does not have associated joint or inflammation damage. Fibromyalgia pain may also cause paresthesia and mimic nerve root compression, despite no evident change in the peripheral nervous system. Further complicating the issue, patients with fibromyalgia also have normal results in blood tests and diagnostic imaging exams such as computed tomography (CT) scans, magnetic resonance imaging (MRI), and electromyography (EMG). Due to these difficulties, fibromyalgia has earned the nickname of the "invisible disability" of chronic pain syndromes.

The causes of fibromyalgia remain unknown, but it has been reported that the onset of fibromyalgia has a tendency to follow a physical or psychological traumatic event. Presently, central sensitization is considered one of the more likely causes for fibromyalgia and is supported by the demonstration of altered pain processing pathways via functional MRI (fMRI) and by the presence of allodynia (pain from usually nonpainful stimuli) in patients with fibromyalgia. Other models for the pathogenesis of fibromyalgia have also been developed; one such model suggests that fibromyalgia results from stress and abnormalities in the hypothalamic-pituitary-adrenal (HPA) axis. Though the underlying pathophysiology remains to be outlined in great detail, research has generated some consistent findings. One such finding is the elevated level of the substance P neuromodulator in the cerebrospinal fluid (CSF) of patients with fibromyalgia. Substance P is known to participate in nociception and is thought to augment an individual's sensitivity to pain. In addition, research has shown that serotonin inhibits the release of substance P by afferent spinal cord neurons and that in patients with fibromyalgia, levels of

serum serotonin and CSF serotonin metabolites were reduced.

In addition to widespread pain and allodynia, fibromyalgia often presents with a variety of other symptoms, the most common of these being chronic fatigue, nonrestorative sleep, or sleep disturbances, the latter of which is thought to be problematic and contributing to pain perception by preventing the secretion of growth hormone (which helps to fix muscular microtears) in the third and fourth stages of sleep. Fibromyalgia also frequently presents with irritable bowel syndrome, migraines, and decreased cognitive functioning. For example, one study found that cognitive function in individuals with fibromyalgia was comparable to control participants who were 20 years older (Glass, 2008). In addition, a large portion of patients with fibromyalgia have been found to have associated psychological conditions, such as anxiety or depression. It would seem that the combination of comorbid psychological issues and fibromyalgia worsen the pain perception in fibromyalgia.

While there is presently no cure for fibromyalgia, many treatments have been shown to be effective in the management of fibromyalgia-related pain, sleep disturbances, and psychological conditions. The US Food and Drug Administration (FDA) has approved two drugs to date for the treatment of fibromyalgia, these being pregabalin and duloxetine (a serotonin-norepinephrine reuptake inhibitor). In addition, many studies have shown that tricyclic antidepressants such as amitriptyline have been associated with improvements in a number of fibromyalgia-related outcome measures. In general, a multidisciplinary approach, which may include interventions such as patient education, cognitive-behavioral therapy, and exercise, is also indicated for the management of fibromyalgia.

Cross-References

► Pain

References and Readings

Burkhardt, C., Goldenberg, D. L., Crofford, L. J., et al. (2005). *Guideline for the management of fibromyalgia syndrome pain in adults and children*. APS clinical practice guidelines series, No. 4, 2005.

Dell, D. D. (2007). Getting the point about fibromyalgia. *Nursing, 37*(2), 61–64.

Glass, J. M. (2008). Fibromyalgia and cognition. *The Journal of Clinical Psychiatry, 69*(Suppl 2), 20–24.

Goldenberg, D. L., Burckhardt, C., & Crofford, L. (2004). Management of fibromyalgia syndrome. *Journal of the American Medical Association, 292*(19), 2388–2395.

Häuser, W., Bernardy, K., Üçeyler, N., & Sommer, C. (2009). Treatment of fibromyalgia syndrome with antidepressants: A meta-analysis. *Journal of the American Medical Association, 301*(2), 198–209.

Lucas, H. J., Brauch, C. M., Settas, L., & Theoharides, T. C. (2006). Fibromyalgia–new concepts of pathogenesis and treatment. *International Journal of Immunopathology and Pharmacology, 19*(1), 5–10.

Millea, P. J., & Holloway, R. L. (2000). Treating fibromyalgia. *American Family Physician, 62*(7), 1575–1582, 1587.

Staud, R. (2007). Treatment of fibromyalgia and its symptoms. *Expert Opinion on Pharmacotherapy, 8*(11), 1629–1642.

U.S. Food and Drug Administration. (2008). *Living with fibromyalgia, drugs approved to manage pain* (PDF Document). Retrieved from the FDA's Consumer Health Information Web site: http://www.fda.gov/downloads/ForConsumers/ConsumerUpdates/ucm107805.pdf. Accessed April 16, 2012.

Wolfe, F., Clauw, D. J., Fitzcharles, M.-A., Goldenberg, D. L., Katz, R. S., Mease, P., & Yunus, M. B. (2010). The American college of rheumatology preliminary diagnostic criteria for fibromyalgia and measurement of symptom severity. *Arthritis Care and Research, 62*(5), 600–610.

Fibromyalgia Syndrome

► Fibromyalgia

Fibrositis

► Fibromyalgia

Fish Oil

► Omega-3 Fatty Acids

Fissure

► Brain, Cortex

Fitness Test

► Maximal Exercise Stress Test

Five-Factor Model of Personality

Michael S. Chmielewski[1] and
Theresa A. Morgan[2]
[1]Department of Psychology, University of Toronto, Toronto, ON, Canada
[2]Alpert Medical School of Brown University, Department of Psychiatry, Brown University, Providence, RI, USA

Synonyms

Big five, The

Definition

The five-factor model (also referred to as "The Big Five") is the most widely used and empirically supported model of normal personality traits. It consists of five main traits: Neuroticism, Extraversion, Openness (to experience), Agreeableness, and Conscientiousness.

Description

The five-factor model (FFM; Digman, 1990), or the "Big Five" (Goldberg, 1993), consists of five broad trait dimensions of personality. These traits represent stable individual differences (an individual may be high or low on a trait as compared to others) in the thoughts people have, the feelings they experience, and their behaviors. The FFM includes Neuroticism, Extraversion, Openness, Agreeableness, and Conscientiousness. Neuroticism is the tendency to experience negative emotions (e.g., sadness, anxiety, and anger) and to have negative thoughts (e.g., worry, self-doubt). In general, Neuroticism represents the predisposition to experience psychological distress. It has been linked to the negative affectivity/negative emotionality dimension from other trait models (Digman, 1990; Goldberg, 1993; John, Naumann, & Soto, 2008; McCrae & Costa, 2008). In contrast, Extraversion is the tendency to be sociable, energetic, assertive, lively, and to experience positive emotions (e.g., happiness), and have positive thoughts (e.g., optimism) (Digman, 1990; Goldberg, 1993; John et al., 2008; McCrae & Costa, 2008). It has been linked to the positive affectivity component of other trait models. Although it was originally thought that Neuroticism and Extraversion were strongly related, they are actually quite independent from one another.

Openness consists of intellectual curiosity, creativity, aesthetic sensitivity, and having nondogmatic attitudes (Digman, 1990; Goldberg, 1993; John et al., 2008; McCrae & Costa, 2008). Agreeableness can be defined as how well one gets along with others. It includes being prosocial, altruistic, trusting, warm, and sympathetic (Digman, 1990; Goldberg, 1993; John et al.; McCrae & Costa, 2008). Finally, conscientiousness encompasses being responsible, dependable, disciplined, and organized. In addition, Conscientiousness represents a disciplined striving after goals and a strict adherence to principles (John et al.).

The five factors of the FFM were independently discovered by several different researchers; all utilizing slightly different methods (see Digman, 1990). The FFM was initially identified in structural investigations of the human language in the mid-1900s. In the mid-1980s, McCrae and Costa (2008) documented that the FFM could be found in psychologically developed self-report questionnaires as well. Moreover, they documented that the FFM subsumed the vast majority of competing personality trait models (McCrae & Costa, 2008). Since that time, the FFM has become most widely

utilized and empirically supported model of personality (John et al., 2008; McCrae & Costa, 2008). The FFM is often referred to as "universal" model of personality as it replicates across gender, language, and culture (McCrae & Costa, 2008; John et al.). In addition, there is substantial self-other agreement on all five of the FFM traits (John et al.; McCrae & Costa, 2008). Furthermore, FFM traits are moderately heritable, with heritability estimates of approximately 50% for each of the five traits (McCrae & Costa, 2008). Moreover, the traits of the FFM have been linked to childhood temperament.

The FFM demonstrates impressive stability over time even across intervals of several years and that stability increases as individuals grow older (McCrae & Costa, 2008; Roberts & DelVecchio, 2000). However, the FFM is less stable over longer periods of time (e.g., 20 years vs. 3 years), indicating that people can and do change on their trait levels, given sufficient time and motivation (Roberts & DelVecchio, 2000). This suggests that environmental factors, life experiences, and gene by environment interactions can and do play a role in the development of FFM traits, although there remains disagreement on this point (McCrae & Costa, 2008; Roberts & DelVecchio, 2000). Women consistently report higher Neuroticism and Agreeableness, and men often report higher Extraversion and Conscientiousness (McCrae & Costa, 2008). There is also evidence of a maturation effect: on average, levels of Agreeableness and Conscientiousness typically increase with age, whereas Neuroticism and Openness tend to decrease.

Taken together, research consistently underscores the import of the FFM for such life outcomes as (but not limited to) relationship quality, adaptation to life, psychopathology, functional impairment, occupational success, happiness, health, and even mortality (McCrae & Costa, 2008). At high levels, Neuroticism has been linked to negative life outcomes including most psychological disorders, medical illness, and negative social experiences (e.g., interpersonal conflict and other life stressors). In general, high Extraversion is a protective factor against many negative life outcomes (e.g., psychological disorders). High Openness has been linked to political liberalism and intelligence. Finally, high Agreeableness is linked with having more positive social experiences with friends, family, and colleagues.

Cross-References

▶ Personality

References and Readings

Digman, J. M. (1990). Personality structure: Emergence of the five-factor model. *Annual Review of Psychology, 41*, 417–440.

Goldberg, L. R. (1993). The structure of phenotypic personality traits. *American Psychologist, 48*, 26–34.

John, O. P., Naumann, L. P., & Soto, C. J. (2008). Paradigm shift to the integrative big-five trait taxonomy: History, measurement, and conceptual issues. In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of personality: Theory and research* (pp. 114–158). New York: Guilford Press.

McCrae, R. R., & Costa, P. T., Jr. (2008). Empirical and theoretical status of the five-factor model of personality traits. In G. J. Boyle, G. Matthews, & D. H. Saklofske (Eds.), *The SAGE handbook of personality theory and assessment* (Personality theories and models, Vol. 1, pp. 273–294). Thousand Oaks, CA: Sage.

Roberts, B. W., & DelVecchio, W. F. (2000). The rank-order consistency of personality traits from childhood to old age: A quantitative review of longitudinal studies. *Psychological Bulletin, 126*, 2–25.

Flight-or-Fight Response

▶ Neuroendocrine Activation

Flourishing

▶ Perceived Benefits

Fluid Pill

► Diuretic

Fluoxetine

► Selective Serotonin Reuptake Inhibitors (SSRIs)

Fluvoxamine

► Selective Serotonin Reuptake Inhibitors (SSRIs)

FMLA

► Family and Medical Leave Act

Focus Groups

Amber Daigre
University of Miami, Miami, FL, USA

Synonyms

Group interview

Definition

Focus groups are group interviews that facilitate focused communication among research participants and generate qualitative data about specific populations. Focus groups range in size from very small (e.g., four people) to as many as 12 participants in a larger group (Krueger, 1988). Whatever the size, groups should be small enough so that all members can share insight, while still remaining large enough to facilitate diverse ideas. Groups should also be composed of people who are not familiar with each other.

Focus groups have several uses, which include gathering information for questionnaire development, assessing community needs, testing new programs, and discovering customer preferences. These types of groups are also widely used to examine people's experiences and concerns with health services. Focus groups may be exploratory to generate ideas, or they can be used to pilot test new materials and interventions.

Focus groups are helpful to researchers wishing to understand the population with whom research will be conducted. For example, the group format allows researchers to observe different forms of communication (e.g., anecdotes, jokes) that are used in the population of interest. Other forms of data collection (e.g., questionnaire self-report) are not well suited to measure these subtleties of communication.

Within behavioral medicine research, focus groups have been used extensively. Data have been gathered regarding quality of life, psychosocial issues, health beliefs, and preferences for various types of interventions among diverse patient populations. Focus groups have been especially useful in gaining understanding of these issues within minority populations. By looking within minority groups, researchers can gather information about shared experiences as well as important within-group differences (Kitzinger, 1995). A number of focus groups are generally conducted until the point of saturation is reached, i.e., the point when there are no longer new ideas being generated. Data generated from focus groups is analyzed qualitatively to identify themes, and this is typically done with the use of qualitative data analysis programs.

In addition to the benefits of focus group research, there are several limitations that researchers should be aware of. At times, the group setting may inhibit some members from participating and sharing their opinions. Additionally, participant confidentiality is compromised by the presence of other research

subjects (Kitzinger, 1995). However, these concerns may be mitigated by the skill of the focus group facilitator.

References and Readings

Kitzinger, J. (1995). Introducing focus groups. *British Medical Journal, 311*, 299–302.
Krueger, R. A. (1988). *Focus groups: A practical guide for applied research*. Newbury Park, NJ: Sage.

Folk Health Beliefs

▶ Cultural Factors

Follow-up Study

Lynda H. Powell and Imke Janssen
Department of Preventive Medicine, Rush University Medical Center, Chicago, IL, USA

Synonyms

Cohort study; Incidence study; Longitudinal study; Prospective study

Definition

A follow-up study, more commonly called a cohort study, has three basic components: exposure, time, and outcome. A group of individuals (the cohort) is assembled and then assessed at baseline on the *exposure* of interest (a risk or protective factor), other risk factors known to influence the outcome of interest, and the prevalence of the outcome of interest. The cohort is followed over *time* to determine development of the *outcome*. To determine incidence of the outcome, subjects who have the outcome at baseline are excluded. Then subjects with and those without the exposure are compared, controlling for extraneous confounders.

Description

Follow-up studies have various names derived from either study group (cohort studies), the timing of observation (follow-up, prospective, or longitudinal studies), or the disease outcome (incidence studies). They begin with the assembly of a cohort which is any group of individuals ranging from the very diverse (e.g., a general population) to a defined geographical area (e.g., Framingham), a defined occupation (e.g., nurses), a defined high-risk subgroup (e.g., homosexual men), or a group defined by logistical ease of follow-up (e.g., health insurance beneficiaries).

Exposure. In contrast to randomized controlled trials (RCTs), exposure to the putative causal agent is not under the researcher's control but simply based upon history. Therefore, the exposure (i.e., risk/protective factor) could simply be correlated with a true risk factor and result in the problem of confounding. A confounder is a third variable that is correlated with both the exposure and outcome and is not part of the causal path. For example, education could confound the relationship between depression and heart disease because it is correlated both with depression and with heart disease and is not part of the causal path by which depression links to heart disease. Thus, an assumed relationship between depression and heart disease could actually be a true relationship between education and heart disease. To guard against confounding, baseline assessment should not only include the exposure of interest but also potential demographic, medical, occupational, and psychosocial confounding factors. Since exposure status can change over time, many follow-up studies feature repeated assessments of exposure status and confounders beyond baseline. These updated changes are handled in various ways in statistical analyses.

Time. One criterion for making causal claims is temporality; that is, the risk/protective factor precedes the disease. The strength of these studies is that they permit an inference about this temporal relationship. For incidence studies, any subject with prevalent disease is excluded from the cohort at baseline. The non-diseased subjects

are then assessed for exposure and followed over time until disease occurs, they are lost or withdraw, or the study ends. Since exposure assessment predated the observed occurrence of disease, it is possible to argue that the exposure predated the outcome. For example, to make the inference that depression *predicts* heart disease, depression (1) must be more common among those who subsequently develop heart disease and (2) heart disease (the outcome) must not have been present before the onset of depression (the risk factor). It is crucial to maximize complete follow-up of the original cohort. Losses and withdrawals should not exceed 10% or the true incidence of the outcome in the cohort will be underrepresented. Comparisons of baseline characteristics between those who were lost/withdrew and those who completed determine whether missingness was random or nonrandom. If missingness was nonrandom, comparisons of risk in exposed vs. unexposed may be biased (over- or underestimated). For example, if more depressed than non-depressed subjects withdraw, any increase in events in the depressed will be weakened and the study could underestimate the importance of depression as a risk factor for the outcome.

Outcome. Outcomes can include diseases, preclinical diseases, risk factors, or other health-related events and can be categorical or continuous. Periodic follow-ups of the cohort permit a rigorous assessment of the outcome. This assessment must be conducted by individuals who are blinded to the exposure assessment to prevent bias. Outcome incidence and/or pattern over time is then compared, respectively, between those who do and those who do not have the exposure of interest.

Statistical Analyses

Risk associated with an exposure is handled in several ways. The (average) incidence rate of a disease over a specified time interval equals the number of new cases during the interval divided by the total amount of time at risk for the disease accumulated by the entire population over the same interval and is reported as the number of events per person-years. To compare the incidence of disease between two groups (exposed and not exposed), data are usually summarized in a 2×2 table and compared with a χ^2 *test*. The *relative risk* is calculated as the ratio of the proportion of exposed who develop the disease to the proportion of the unexposed developing it. The *odds ratio* (OR) is similarly defined as the ratio of the odds of developing the disease. If either the disease is rare or the time interval is short, the relative hazard is approximately equal to the relative risk and the odds ratio. This odds ratio, transformed by natural logarithm, is the outcome in *logistic regression* models. *Confidence intervals* for these log odds can be derived from normal theory and then transformed back to the original scale to yield confidence intervals for the odds.

With long follow-up time intervals, there is often interest in examining the pattern of events over time. In *time-to-event analysis*, the hazard function (or its integral, the survival function) presents events as a function of time. Under the assumption of *proportional hazards*, that is, that the hazard rates in the exposed and the unexposed groups are the same over time, the hazard can be modeled as a function of covariates (*Cox regression model*). If the proportional hazards assumption does not hold, then parametric survival curves (e.g., accelerated failure time (AFT) models) may provide a better fit. These models allow for *time-varying covariates* where exposure status is updated periodically.

When the outcome is continuous, growth curve models (linear regression in its simplest form) are the preferred method of analysis, modeling the trajectory of the outcome over time. This approach allows separating aging effects (i.e., changes over time within individuals) from cohort effects (i.e., differences between subjects at baseline). These models allow, for example, for different patterns over time between treatment groups.

All of the models just discussed belong to a class of generalized mixed models. Potential covariates such as demographic, medical, occupational, and psychosocial confounding factors are added as a linear function in these *multivariable analyses* (Hedeker & Gibbons,

2006). Time-varying covariates including the change in risk factors can be included in these models.

Missing data in follow-up studies can cause serious problems. Therefore, every effort should be made to avoid missing values and to document the reason for missingness for the unavoidable cases, for example, deaths. The usual assumption is that data are missing at random. If that is not true, that is, if depressed patients are more likely to drop out of the study than not depressed patients, the missing process can be modeled separately and incorporated into the analysis (Little & Rubin, 2002).

In summary, the key strength of follow-up studies is their ability to establish the timing and directionality of exposure and outcome events. Bias in ascertainment of exposure is impossible because neither subject nor observer is aware of future outcome status. Bias in ascertainment of outcome is minimized if outcomes assessors are kept blinded. These studies are more difficult and costly than a cross-sectional study, but the gain in inferential strength and minimization of bias is worth the effort.

The key weakness of these studies is the difficulty in making causal claims since temporality does not imply causality. The problem of confounding can never be completely eliminated for there will always be unknown, and thus unmeasured, confounders that could be the true causal agent. The art of these studies is in a careful review of the literature to determine predictors of the outcome, with a particular eye to those predictors that are also related to the exposure, and in ensuring that this full array of predictors is included in baseline assessment. A common problem in the use of convenience follow-up studies (e.g., the Framingham Study, the Nurses' Health Study) is that they often do not have all potential covariates of interest in their assessment batteries.

Although these studies cannot make conclusive claims for a causal relationship between exposure and outcome, their ability to disentangle temporality can strengthen the chain of evidence by which causal claims can be made. They provide valuable support, or nonsupport, for justifying a rigorous RCT which is the strongest basis for claiming that an exposure causes a disease.

To increase the quality of the reporting of observational studies, several prominent medical researchers issued the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement.

Cross-References

- ▶ Cohort Study
- ▶ Health Promotion and Disease Prevention
- ▶ Kuopio Ischemic Heart Disease Risk Factor Study
- ▶ Mini-Finland Health Survey

References and Readings

Hedeker, D. R., & Gibbons, R. D. (2006). *Longitudinal data analysis*. Hoboken, NJ: Wiley-Interscience.

Jewell, N. P. (2004). *Statistics for epidemiology*. New York: Chapman and Hall.

Little, R. J. A., & Rubin, D. B. (2002). *Statistical analysis with missing data* (2nd ed.). Hoboken, NJ: Wiley.

Morton, R. F., Hebel, J. R., & McCarter, R. J. (2001). *A study guide to epidemiology and biostatistics* (5th ed.). Gaithersburg, MD: Aspen.

Szklo, M., & Nieto, F. J. (2000). *Epidemiology. Beyond the basics*. Gaithersburg, MD: Aspen.

von Elm, E., Altman, D. G., Egger, M., Pocock, S. J., Gøtzsche, P. C., Vandenbroucke, J. P., et al. (2008). Strengthening the reporting of observational studies in epidemiology (STROBE) statement: Guidelines for reporting observational studies. *Journal of Clinical Epidemiology, 61*(4), 344–349.

Food Control

- ▶ Food Safety

Food Pyramid

- ▶ Healthy Eating

Food Safety

Tereza Killianova
Free University of Brussels (VUB), Jette, Belgium

Synonyms

Food control

Definition

Food safety refers to all efforts done to monitor and overcome temporary or long-lasting hazards that may make food have adverse effect to the health of the consumer. Food hazards include microbiological hazards, pesticide residues, misuse of food additives, chemical contaminants, including biological toxins, adulteration, as well as genetically modified organisms, allergens, veterinary drug residues, and growth-promoting hormones used in the production of animals. The food hazards can be present along the entire food chain; therefore, it is important that all sectors in the chain operate in an integrated way, and food control systems address all stages of this chain to guarantee food safety. This monitoring from primary producer through consumer is often described as the farm-to-table continuum. However, it is difficult and expensive to test for food hazards and quality loss at each point in the food chain. Especially in many developing countries, the resources are limited, food control laboratories are frequently poorly equipped, there is no suitably trained analytical staff, and the management is poor. This leads to an inadequate food control infrastructure. Therefore a well-structured, preventive approach which controls processes is the preferred method for improving food safety and quality. Factors, such as improper agricultural practices, poor hygiene at all stages of the food chain, lack of preventive controls in food processing and preparation operations, misuse of chemicals, contaminated raw materials, ingredients and water, inadequate or improper storage, which contribute to potential hazards in foods, should be taken into account (Food and Agriculture Organization/World Health Organization [FAO/WHO], 2003).

Food safety is an increasingly important public health issue, both in developing and industrialized countries. The emergence of food-borne illnesses is influenced by factors such as large genetic variability of microorganisms, environmental factors, human actions, and behavior (e.g., traveling), urbanization, raw food production, new technologies, human risk factors such as age, illness, and others (Hall, 1997).

The food-borne illnesses, when focusing on the microbiological hazards, are caused by bacterial agents (e.g., Salmonella or *E. coli*), viral agents, or parasites. The microbiological safety of food is a dynamic situation influenced to a great extent by multiple factors contributing to changing trends in food-borne illnesses. Examples of these factors are rapid population growth, an increasingly global market in vegetables, fruit, meat, and ethnic foods, and changing eating habits, such as the consumption of raw or lightly cooked food, climate change, and others. However, the list of factors influencing the prevalence of food-borne diseases is long and their relative importance is largely unknown (Newell et al., 2010). From a behavior medicine perspective, one may examine, for example, how certain psychological traits such as risk-taking or low conscientiousness affect the prevalence of food hazards in a food-producing company, of importance for prevention.

Cross-References

- ▶ Health Behaviors
- ▶ Preventive Care

References and Readings

Food and Agriculture Organization/World Health Organization. (2003). *Assuring food safety and quality: Guidelines for strengthening national food control systems*. FAO Food and Nutrition Paper 76. Rome: Author. Retrieved July 12, 2011, from http://www.fao.org/docrep/006/Y8705E/Y8705E00.HTM

Hall, L. (1997). Foodborne illness: Implications for the future. *Emerging Infectious Diseases, 3*(4), 555–559.
Newell, D. G., et al. (2010). Food-borne diseases: The challenges of 20 years ago still persist while new ones continue to emerge. *International Journal of Food Microbiology, 139*, S3–S15.

Food Supplement

▶ Nutritional Supplements

Foot Care

▶ Diabetes Foot Care

Forgiveness

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

Forgiveness refers to the purposeful decision by a victim of wrongdoing to relinquish anger and the desire to punish an offender responsible for inflicting harm (e.g., Enright & The Human Development Study Group, 1991). Forgiveness may go beyond mere indifference, to express goodwill toward the offender. This concept is commonly pertinent to daily social interactions, and it constitutes a basic social process which maintains interactions and relationships. Various scales exist for assessing forgiveness. For example, the Heartland Forgiveness Scale (Thompson et al., 2005) is an 18-item scale assessing forgiveness of oneself, others, and situations. These three subscales demonstrate the importance of this concept to one's personal and interpersonal lives. Investigators have examined the psychosocial correlates or determinants of forgiveness. For example, perceptions of severity of the offense, the intentions of the offender, sincerity of apology, and empathy have been investigated as factors possibly affecting forgiveness (Kearns & Fincham, 2004). In the context of traumatic events, difficulty forgiving others is significantly correlated with depression and posttraumatic stress symptoms (Witvliet, Phipps, Feldman, & Beckham, 2004), implicating a possible relationship and resemblance with the term "rumination." Interventions aimed at inducing forgiveness have resulted in reduced anxiety, depression, and anger in various populations (reviewed by Kearns & Fincham, 2004). In a groundbreaking experimental study, vanOyen, Ludwig, and Vander Laan (2001) asked people to imagine a real person who they either held grudges toward or they had an empathic perspective and imagined themselves forgiving. Compared to baseline levels, the forgiving group evidenced significantly lower psychological (aversive emotions) and physiological (heart rate, skin conductance, etc.) changes, while the first group evidenced worsening of those psychophysiological responses. These studies together point at forgiveness as an important variable in social interactions, which has short- and possibly long-term health implications, since forgiveness can be in question for very long periods of time in people's lives.

Cross-References

▶ Anger Management
▶ Interpersonal Relationships

References and Readings

Enright, R., & The Human Development Study Group. (1991). The moral development of forgiveness. In W. Kurtines & J. Gewirtz (Eds.), *Handbook of moral behavior and development* (pp. 123–152). Hillsdale, NJ: Erlbaum.
Kearns, J. N., & Fincham, F. D. (2004). A prototype analysis of forgiveness. *Personality and Social Psychology Bulletin, 30*, 838–855.
Thompson, L. Y., Snyder, C. R., Hoffman, L., Michael, S. T., Rasmussen, H. N., Billings, L. S., Heinze, L., Neufeld, J. E., Shorey, H. S., Roberts, J. C., & Roberts,

D. E. (2005). Dispositional forgiveness of self, others, and situations. *Journal of Personality, 73*, 313–359.
vanOyen, W. C., Ludwig, T. E., & Vander Laan, K. L. (2001). Granting forgiveness or harboring grudges: Implications for emotion, physiology, and health. *Psychological Science, 12*, 117–23.
Witvliet, C. V., Phipps, K. A., Feldman, M. E., & Beckham, J. C. (2004). Posttraumatic mental and physical health correlates of forgiveness and religious coping in military veterans. *Journal of Traumatic Stress, 17*, 269–73.

Former Smokers

▶ Ex-Smokers

FOS

▶ Framingham Offspring Study

Frailty Assessment

▶ Activities of Daily Living (ADL)

Framingham Heart Study

Andrew J. Wawrzyniak
School of Nursing & Health Studies, University of Miami, Coral Gables, FL, USA

Synonyms

FHS

Definition

The Framingham Heart Study is a longitudinal cohort study that began in 1948 with 5,209 participants to examine how lifestyle choices and psychosocial elements impact cardiovascular health. It had helped coin the term "risk factors" in identifying clusters of variables, such as poor diet, lack of physical activity, and negative psychological factors, that contribute to cardiovascular disease.

Description

In 1948, the Framingham Heart Study ventured to better understand cardiovascular disease (CVD) in that little was known about the etiology of heart disease and stroke despite their epidemic proportions due to their steadily increasing rates since the 1900s. A joint project of the National Heart, Lung, and Blood Institute (NHLBI) and Boston University, the Framingham Heart Study sought to longitudinally track a large cohort of participants who initially had no outward CVD symptoms, heart attack, or stroke. Initially, 5,209 participants aged 30–62 were recruited from Framingham, Massachusetts; this was one of the first studies to administer extensive physical exams accompanied by lifestyle interviews over multiple assessments. Participants have returned for assessments every 2 years since the study's inception.

The Framingham Heart Study helped mainstream the concept of preventive medicine in the context of stopping CVD before it starts by promoting health behaviors beneficial to future cardiovascular health. Framingham helped dispel past theories of CVD progression that were heavily reliant on diastolic blood pressure (Kannel, 1995a, 1995b). Importantly, the Framingham Heart Study established the concept of clusters of risk factors rather than one single factor detrimental to CVD (Kannel, 2000); the study is the origin of the term "risk factor."

The Framingham Heart Study has been instrumental in identifying now commonly known primary risk factors of CVD such as increased blood pressure, cholesterol, smoking, obesity, diabetes, and lack of physical activity over long-term follow-up observations (Kannel, D'Agostino, & Cobb, 1996). Additionally, the study also acknowledged secondary risk factors of CVD such as blood triglycerides and HDL

cholesterol along with demographic factors such as age and gender (Kannel & Eaker, 1986); more recently, increased plasma homocysteine has been identified as a risk factor for CVD (Sundström & Vasan, 2005).

Importantly, the Framingham Heart Study's work has published research that specifically gives weights to individual risk factors as predictors of CVD progression through regression models. Risk of CVD can be calculated based on variables such as age, gender, blood pressure, smoking status, parental CVD history, blood markers such as cholesterol and triglycerides, and BMI. Collectively, this equation has been termed the Framingham Risk Score that estimates the 10-year risk of CVD; NHLBI provides an online calculator at http://hp2010.nhlbihin.net/atpiii/calculator.asp.

The Framingham Heart Study helped establish diabetes mellitus type 2 as a risk factor for CVD (Fox, 2010; Kengne, Turnbull, & MacMahon, 2010). In that type 2 diabetes is primarily due to lifestyle choices, this finding helped shape health psychology in addressing prevention of diabetes through weight control and diet.

Specific to behavioral medicine, the Framingham Heart Study has reported increased incidence of CVD in those with greater negative psychosocial factors, such as greater depression, anxiety, perceived stress, anger, hostility, and social isolation. Importantly, the Framingham Heart Study's psychosocial findings controlled for other variables that may have influenced the relationship; in other words, links between psychosocial factors and CVD outcomes were independent of the common risk factors of CVD worsening attributable to demographics and lifestyle.

Additionally, the Framingham Heart Study was one of the first studies to help define Type A personality. Type A behavior is generally defined by high levels of daily stress, emotional lability, tension, anger, and ambitiousness (Haynes, Levine, Scotch, Feinleib, & Kannel, 1978). Framingham established one of the early links between Type A personality and CHD prevalence in women and higher incidence of myocardial infarction (MI) in men (Haynes, Feinleib, Levine, Scotch, & Kannel, 1978) after controlling for age, blood pressure, smoking status, and cholesterol.

Examining longitudinal outcomes with respect to psychosocial factors, 20-year incidence of MI or coronary death in 749 females free of initial coronary disease was predicted by greater tension, anxiety, and loneliness after controlling for age, systolic blood pressure, total/HDL cholesterol Ratio, diabetic status, smoking status, and BMI (Eaker, Pinsky, & Castelli, 1992).

While these psychosocial factors alone did not necessarily account for a majority of the variance in the CVD health outcomes in deference to lifestyle factors such as health behaviors, psychosocial factors have been shown to be an effective complementary treatment target for supplementing lifestyle changes and medication regimens.

Among the other studies that have stemmed from the original study, the Framingham Heart Study has expanded to examine the children and grandchildren of the initial cohort using the same techniques of longitudinally assessing health and lifestyle; this study is aptly named the Framingham Offspring Study. Future directions for the Framingham Heart Study include examination of genetic factors in CVD outcomes using participants' cell lines (Govindaraju et al., 2008).

Cross-References

- ▶ Aging
- ▶ Alcohol Consumption
- ▶ Anxiety and Heart Disease
- ▶ Body Fat
- ▶ Body Mass Index
- ▶ Carbohydrates
- ▶ Cardiac Events
- ▶ Cardiovascular Disease
- ▶ Cardiovascular Risk Factors
- ▶ Cholesterol
- ▶ Community Sample
- ▶ Coronary Heart Disease
- ▶ Demographics

- Diabetes
- Diastolic Blood Pressure (DBP)
- Eating Behavior
- Endothelial Function
- Epidemiology
- Fasting Glucose
- Fasting Insulin
- Fat, Dietary Intake
- Fibrinogen
- Framingham Offspring Study
- Gender Differences
- Gene Expression
- Genetic Polymorphisms
- Genetics
- Ghrelin
- Glucose: Levels, Control, Intolerance, and Metabolism
- Health Behaviors
- Health Psychology
- Healthy Eating
- Heart Disease and Emotions: Anger, Anxiety, Depression
- Heart Disease and Stress
- Heart Disease and Type A Behavior
- Homocysteine
- Hyperlipidemia
- Hypertension
- Inflammation
- Insulin Resistance (IR) Syndrome
- Intima-Media Thickness (IMT)
- Leptin
- Lifestyle
- Lipoprotein
- Longitudinal Research
- Low Glycemic Index
- Magnetic Resonance Imaging (MRI)
- Marriage and Health
- Metabolic Syndrome
- Multiple Risk Factors
- National Heart, Lung, and Blood Institute
- Nutrition
- Obesity
- Overweight
- Perceived Stress
- Personality
- Prospective Cohort Study
- Psychosocial Factors
- Psychosocial Predictors
- Psychosocial Variables
- Smoking Behavior
- Social Support
- Socioeconomic Status (SES)
- Stress
- Stressor
- Systolic Blood Pressure (SBP)
- Trait Anger
- Trait Anxiety
- Triglyceride
- Type 2 Diabetes Mellitus
- Type A Behavior
- Work-Related Stress

References and Readings

10-year CVD risk calculator: Risk assessment tool for estimating your 10-year risk of having a heart attack (n.d.). Retrieved 18 Mar 2012 from http://hp2010.nhlbihin.net/atpiii/calculator.asp

Eaker, E. D., Pinsky, J., & Castelli, W. P. (1992). Myocardial infarction and coronary death among women: Psychosocial predictors from a 20-year follow-up of women in the Framingham study. *American Journal of Epidemiology, 135*(8), 854–864.

Fox, C. S. (2010). Cardiovascular disease risk factors, type 2 diabetes mellitus, and the Framingham heart study. *Trends in Cardiovascular Medicine, 20*(3), 90–95.

Govindaraju, D. R., Cupples, L. A., Kannel, W. B., O'Donnell, C. J., Atwood, L. D., D'Agostino, R. B., Sr., et al. (2008). Genetics of the Framingham heart study population. *Advances in Genetics, 62*, 33–65.

Haynes, S. G., Feinleib, M., Levine, S., Scotch, N., & Kannel, W. B. (1978). The relationship of psychosocial factors to coronary heart disease in the Framingham study. II. Prevalence of coronary heart disease. *American Journal of Epidemiology, 107*(5), 384–402.

Haynes, S. G., Levine, S., Scotch, N., Feinleib, M., & Kannel, W. B. (1978). The relationship of psychosocial factors to coronary heart disease in the Framingham study. I. Methods and risk factors. *American Journal of Epidemiology, 107*(5), 362–383.

Kannel, W. B. (1995a). Framingham study insights into hypertensive risk of cardiovascular disease. *Hypertension Research, 18*(3), 181–196.

Kannel, W. B. (1995b). Clinical misconceptions dispelled by epidemiological research. *Circulation, 92*(11), 3350–3360.

Kannel, W. B. (2000). Fifty years of Framingham study contributions to understanding hypertension. *Journal of Human Hypertension, 14*(2), 83–90.

Kannel, W. B., D'Agostino, R. B., & Cobb, J. L. (1996). Effect of weight on cardiovascular disease. *American Journal of Clinical Nutrition, 63*(3 Suppl), 419S–422S.

Kannel, W. B., & Eaker, E. D. (1986). Psychosocial and other features of coronary heart disease: Insights from the Framingham study. *American Heart Journal, 112*(5), 1066–1073.

Kengne, A. P., Turnbull, F., & MacMahon, S. (2010). The Framingham study, diabetes mellitus and cardiovascular disease: Turning back the clock. *Progress in Cardiovascular Diseases, 53*(1), 45–51.

Sundström, J., & Vasan, R. S. (2005). Homocysteine and heart failure: A review of investigations from the Framingham heart study. *Clinical Chemistry and Lab Medicine, 43*(10), 987–992.

Framingham Offspring Study

Andrew J. Wawrzyniak
School of Nursing & Health Studies, University of Miami, Coral Gables, FL, USA

Synonyms

FOS

Definition

In 1971, the Framingham Offspring Study recruited 5,124 of the original Framingham Heart Study's participants' children and their spouses; this new generation were similarly biennially assessed on physiological and psychological measure. The Framingham Offspring Study sought to epidemiologically study CVD in younger adults (Feinleib, Kannel, Garrison, McNamara, & Castelli, 1975).

The Framingham Offspring Study included more physiological variables than its parent study, such as specific dietary measures, neuropsychological measures to assess cognition and reading performance, and MRI to directly measure brain volume. The introduction of cognitive function measures lead to the finding that cardiovascular risk factors and the presence of CVD mediated the association between left ventricular mass and cognition (Elias et al., 2007).

Additional blood marker risk factors of CVD were identified; one of the first reports on high-density lipoprotein (HDL) and low-density lipo-protein (LDL) cholesterol levels in relation to CVD was from the Framingham Offspring Study (Wilson et al., 1980). Other risk factors identified as increasing CVD risk included increased adiponectin (Ai et al., 2011), lipoproteins, and serum albumin and bilirubin. Greater levels of extracellular matrix turnover, specifically matrix metalloproteinase (MMP)-9 and tissue inhibitors of MMPs (TIMPs), were associated with greater internal carotid artery stenosis in the Framingham Offspring Study (Romero et al., 2008). Higher von Willebrand factor (vWF) was found to be a risk factor for CVD in participants with type 2 diabetes (Frankel et al., 2008).

Socioeconomic factors were also recorded. Researchers found that longitudinally, education impacted health: mean systolic blood pressure over 30 years was higher in participants with less than 12 years of education compared to those with more than 17 years of education (Loucks, Abrahamowicz, Xiao, & Lynch, 2011). Additionally, life course socioeconomic status influenced type 2 diabetic status in women and was primarily associated with participants' education levels and occupations (Smith et al., 2011).

From a health psychology perspective, the Framingham Offspring Study helped establish guidelines for healthy eating behavior by suggesting an increase in whole grain food consumption and a decrease in dietary glycemic index to reduce the risk of metabolic syndrome and insulin resistance (McKeown, Meigs, Liu, Wilson, & Jacques, 2002; McKeown et al., 2004).

Psychosocially, anger and hostility were predictive of atrial fibrillation development over a 10-year follow-up in males after controlling for age, diabetes, hypertension, history of myocardial infarction, history of congestive heart failure, and valvular heart disease (Eaker, Sullivan, Kelly-Hayes, D'Agostino, & Benjamin, 2004); trait anger related to total mortality in men. Taken together, the Framingham Offspring Study has expanded the knowledge of the mechanisms of CVD progression through repeated assessments of extensive physiological, behavioral, and psychological factors.

Cross-References

- Aging
- Alcohol Consumption
- Anxiety and Heart Disease
- Atrial Fibrillation
- Body Fat
- Body Mass Index
- Brain, Imaging
- Carbohydrates
- Cardiac Events
- Cardiovascular Disease
- Cardiovascular Risk Factors
- Cholesterol
- Cognitive Function
- Community Sample
- Coronary Heart Disease
- Demographics
- Diabetes
- Diastolic Blood Pressure (DBP)
- Eating Behavior
- Endothelial Function
- Epidemiology
- Fasting Glucose
- Fasting Insulin
- Fat, Dietary Intake
- Fibrinogen
- Framingham Heart Study
- Gender Differences
- Gene Expression
- Genetic Polymorphisms
- Genetics
- Ghrelin
- Glucose: Levels, Control, Intolerance, and Metabolism
- Health Behaviors
- Health Psychology
- Healthy Eating
- Heart Disease and Stress
- Heart Disease and Type A Behavior
- Homocysteine
- Hyperlipidemia
- Hypertension
- Inflammation
- Insulin Resistance (IR) syndrome
- Intima-Media Thickness (IMT)
- Leptin
- Lifestyle
- Lipoprotein
- Longitudinal Research
- Low Glycemic Index
- Magnetic Resonance Imaging (MRI)
- Marriage and Health
- Metabolic Syndrome
- Multiple Risk Factors
- National Heart, Lung, and Blood Institute
- Nutrition
- Obesity
- Overweight
- Perceived Stress
- Personality
- Prospective Cohort Study
- Psychosocial Factors
- Psychosocial Predictors
- Psychosocial Variables
- Smoking Behavior
- Social Support
- Socioeconomic Status (SES)
- Stress
- Stressor
- Systolic Blood Pressure (SBP)
- Trait Anger
- Trait Anxiety
- Triglyceride
- Type 2 Diabetes Mellitus
- Type A Behavior
- Work-Related Stress

References and Readings

Ai, M., Otokozawa, S., Asztalos, B. F., White, C. C., Cupples, L. A., Nakajima, K., et al. (2011). Adiponectin: an independent risk factor for coronary heart disease in men in the Framingham offspring study. *Atherosclerosis, 217*(2), 543–548.

Eaker, E. D., Sullivan, L. M., Kelly-Hayes, M., D'Agostino, R. B., Sr., & Benjamin, E. J. (2004). Anger and hostility predict the development of atrial fibrillation in men in the Framingham offspring study. *Circulation, 109*(10), 1267–1271.

Elias, M. F., Sullivan, L. M., Elias, P. K., D'Agostino, R. B., Sr., Wolf, P. A., Seshadri, S., et al. (2007). Left ventricular mass, blood pressure, and lowered cognitive performance in the Framingham offspring. *Hypertension, 49*(3), 439–445.

Feinleib, M., Kannel, W. B., Garrison, R. J., McNamara, P. M., & Castelli, W. P. (1975). The Framingham offspring study. Design and preliminary data. *Preventive Medicine, 4*(4), 518–525.

Frankel, D. S., Meigs, J. B., Massaro, J. M., Wilson, P. W., O'Donnell, C. J., D'Agostino, R. B., et al. (2008). Von Willebrand factor, type 2 diabetes mellitus, and risk of cardiovascular disease: The Framingham offspring study. *Circulation, 118*(24), 2533–2539.

Loucks, E. B., Abrahamowicz, M., Xiao, Y., & Lynch, J. W. (2011). Associations of education with 30 year life course blood pressure trajectories: Framingham offspring study. *BMC Public Health, 11*, 139.

McKeown, N. M., Meigs, J. B., Liu, S., Saltzman, E., Wilson, P. W., & Jacques, P. F. (2004). Carbohydrate nutrition, insulin resistance, and the prevalence of the metabolic syndrome in the Framingham offspring cohort. *Diabetes Care, 27*(2), 538–546.

McKeown, N. M., Meigs, J. B., Liu, S., Wilson, P. W., & Jacques, P. F. (2002). Whole-grain intake is favorably associated with metabolic risk factors for type 2 diabetes and cardiovascular disease in the Framingham offspring study. *American Journal of Clinical Nutrition, 76*(2), 390–398.

Romero, J. R., Vasan, R. S., Beiser, A. S., Polak, J. F., Benjamin, E. J., Wolf, P. A., et al. (2008). Association of carotid artery atherosclerosis with circulating biomarkers of extracellular matrix remodeling: The Framingham offspring study. *Journal of Stroke and Cerebrovascular Disease, 17*(6), 412–417.

Smith, B. T., Lynch, J. W., Fox, C. S., Harper, S., Abrahamowicz, M., Almeida, N. D., et al. (2011). Life-course socioeconomic position and type 2 diabetes mellitus: The Framingham offspring study. *American Journal of Epidemiology, 173*(4), 438–447.

Wilson, P. W., Garrison, R. J., Castelli, W. P., Feinleib, M., McNamara, P. M., & Kannel, W. B. (1980). Prevalence of coronary heart disease in the Framingham offspring study: Role of lipoprotein cholesterols. *The American Journal of Cardiology, 46*(4), 649–654.

Fraternal Twins

► Dizygotic Twins

Free-Radical Theory of Aging

Carrie Brintz
Department of Psychology, University of Miami, Coral Gables, FL, USA

Definition

The free-radical theory of aging was formally proposed by Denham Harman in 1956 and postulates that the inborn process of aging is caused by cumulative oxidative damage to cells by free radicals produced during aerobic respiration. Free radicals are atoms or molecules with single unpaired electrons. They are unstable and highly reactive, as they attack nearby molecules in order to steal their electrons and gain stability, causing radical chain reactions to occur. Free radicals are generated in vivo primarily within mitochondria during mitochondrial electron transport as well as by other physiological processes. Harman later extended the free-radical theory of aging to incorporate the role of mitochondria in the generation of free radicals and other reactive oxygen species. The theory proposes that the rate of oxidative damage to mitochondrial DNA primarily determines life span.

While free-radical reactions are implicated in the normal aging process, free-radical damage may occur in varying patterns across individuals, modulated by genetic and environmental factors, and in some individuals may be implicated in a number of disorders. These so-called free-radical diseases include cancer, atherosclerosis, Alzheimer's disease, essential hypertension, the immune deficiency of age, and a number of other disorders. The process of aging by free-radical damage may be slowed by a calorie-restricted diet that includes essential nutrients and antioxidants derived from dietary fruits and vegetables. It is theorized that the prevention or slowing of certain "free-radical" diseases such as cancer may be achieved through dietary intervention with antioxidant supplementation, although evidence supporting this hypothesis in humans is mixed.

Cross-References

► Aging

References and Readings

Beckman, K. B., & Ames, B. N. (1998). The free radical theory of aging matures. *Physiological Reviews, 78*, 547–581.

Harman, D. (1956). Aging: A theory based on free radical and radiation chemistry. *Journal of Gerontology, 2*, 298–300.
Harman, D. (1984). Free radical theory of aging: the "free radical" diseases. *Age, 7*, 111–131.
Shringarpure, R., & Davies, K. J. A. (2009). Free radicals and oxidative stress in aging. In V. L. Bengtson, D. Gans, N. M. Putney, & M. Silverstein (Eds.), *Handbook of theories of aging* (pp. 229–243). New York: Springer Publishing Company.

Frequency Analysis

► Quantitative EEG Including the Five Common Bandwidths (Delta, Theta, Alpha, Sigma, and Beta)

Frontal

► Brain, Cortex

Functional Capacity Assessment

► Functional Versus Vocational Assessment

Functional Capacity, Disability, and Status

M. Di Katie Sebastiano
Kinesiology, University of Waterloo, Waterloo, ON, Canada

Synonyms

Functional testing

Definition

Functional capacity refers to one's ability to perform the activities and tasks necessary to live independently. These tasks change throughout the life span; children, adults, and the elderly each have their own unique set of activities necessary to maintain their independence. Age, however, is not the only factor that can determine functional capacity or status: specific disease or injury states each bring their own physical limitations. The interactions among age and injury or disease state determine one's functional capacity.

Description

There are two types of tasks that are usually evaluated to determine functional capacity: activities of daily living (ADL) and instrumental activities of daily living (IADL). ADL refer to activities that are involved in basic human survival such as mobility, eating, using the washroom, dressing, and grooming (Besdine, 1988). The inability to perform these tasks severely inhibits one's ability to live independently and maintain health. IADL tend to refer to activities that are necessary to live independently, but disabilities in these areas do not result in serious health implications. IADL can include housekeeping, cooking, shopping, banking, driving, or using public transportation (Besdine, 1988). A functional disability or impairment is defined as the decreased ability to meet one's own needs in either or both of these areas.

Through the life span, the tasks that determine one's functional capacity change to fit the requirements of daily life. As children, activities such as participation in family life and chores, learning at school, and the ability to participate in extracurricular activities can be considered an assessment of functional capacity along with the basic ADL and IADL. In adulthood, functional capacity often describes the ability to perform work-related tasks. Following injury, assessment of functional capacity commonly determines a person's ability to return to the work force or the injured person's ability to participate in modified duties. In elderly populations, functional capacity generally refers to an individual's ability to meet their own survival needs, which can be determined using ADL and IADL assessments.

Since functional capacity is specific to an individual's ability to perform a given task, there is a broad spectrum of tools that are available for

functional assessment. The original assessments of functional capacity examined hip fracture patients during their rehabilitation. Patients were classified as either independent or dependent based on six daily activities of daily living including bathing, dressing, using the washroom, transferring in and out of beds or chairs, continence, and eating (Katz et al., 1963). The current assessment of functional capacity or functional disability in the elderly often occurs through large-scale interdisciplinary assessment of ADL and IADL such as the Geriatric Functional Assessment (GFA) (Besdine, 1988). Functional capacity can also be assessed through cardiovascular fitness tests, frequently used in populations with compromised aerobic function, such as cardiac patients (Clini & Crisafulli, 2009). These tests may use maximal oxygen consumption measurements or VO_{2max} assessments, commonly performed on the bike or treadmill. Indirect measures of exercise capacity can also be used to predict VO_{2max} such as the 6-min walk test, which estimates maximal oxygen consumption without the use of expensive equipment (Rostangno & Gensini, 2008). In the workplace, job-specific functional assessments can also be used to determine if an individual is capable of performing work-related tasks.

The ability to improve functional disability is dependent on the cause of the functional impairment. Following functional disability resulting from injury or a specific disease, rehabilitation may aid an individual to return to his/her functional capacity from prediagnosis. If functional disability results from disabling chronic illness or simply through aging, it may not be possible for the individual to regain his/her ability to live independently. However, for aged individuals or those with disabling chronic disease, rehabilitation may allow individuals to maintain certain aspects of independence. Functional capacity is specific to each individual and the daily tasks that a person performs. The determinants of functional capacity change over the life span and vary depending on disease and injury status. The appropriate measures of functional capacity must address an individual's unique needs and daily tasks.

References and Readings

Applegate, W. B., Blass, J. P., & Williams, T. E. (1990). Instruments for the functional assessment of older patients. *The New England Journal of Medicine, 322*(17), 1132–1148.

Bergner, M., & Rothman, M. L. (1987). Health status measures: An overview and guide for selection. *Annual Reviews of Public Health, 8*, 191–210.

Besdine, R. E. (1988). Functional assessment as a model for clinical evaluation of geriatric patients. *Public Health Reports, 103*(5), 530–536.

Clini, E. M., & Crisafulli, E. (2009). Exercise capacity as a pulmonary rehabilitation outcome. *Respiration, 77*(2), 121–128.

Katz, S. (1983). Assessing self-maintenance; activities of daily living, mobility, and instrumental activities of daily living. *Journal of American Geriatrics Society, 31*(12), 721–727.

Katz, S., Ford, A. B., Moskowitz, R. W., & Jaffee, M. W. (1963). Studies of illness in the aged. The index of ADL: A standardized measure of biological and psychosocial function. *Journal of the American Medical Association, 185*, 94.

Katz, S., & Stroud, M. W. (1989). Functional assessment in geriatrics: A review of progress and directions. *Journal of American Geriatrics Society, 37*(3), 267–271.

Rostangno, C., & Gensini, G. F. (2008). Six minute walk test: A simple and useful tool to evaluate functional capacity in patients with heart failure. *Internal and Emergency Medicine, 3*(3), 205–212.

Functional Health

▶ Physical Fitness

Functional Magnetic Resonance Imaging (fMRI)

Yoshiya Moriguchi
Department of Psychophysiology, National Institute of Mental Health, National Center of Neurology and Psychiatry, Kodaira, Tokyo, Japan

Definition

Functional magnetic resonance imaging (fMRI) is a technique for measuring neural activity, by

detecting the hemodynamic changes in blood oxygenation and blood flow in response to neural activity, based on blood-oxygenation-level-dependent (BOLD) effect.

Description

Blood-oxygen-level dependent (BOLD) effect is the MRI contrast originated from blood deoxyhemoglobin in the tissue, first discovered by Ogawa, Lee, Kay, and Tank (1990) (Ogawa, Lee, Nayak, & Glynn, 1990; Ogawa et al., 1992). This method depends on the differential susceptibility between deoxyhemoglobin and oxyhemoglobin. Hemoglobin is diamagnetic when oxygenated but paramagnetic when deoxygenated (=deoxyhemoglobin). Therefore, magnetic resonance (MR) signal of blood is slightly different depending on the level of oxygenation. Higher BOLD signal intensities arise from increases in the concentration of oxygenated hemoglobin since the blood magnetic susceptibility more closely matches the tissue magnetic susceptibility. Since deoxyhemoglobin is paramagnetic, it alters the T2*-weighted magnetic resonance image signal to decrease. This deoxyhemoglobin is referred to as an endogenous contrast-enhancing agent and serves as the source of the signal for fMRI. With MRI sequence parameters sensitive to the changes in this differential magnetic susceptibility, changes in BOLD contrast can be assessed. Here, when a brain region is more active, more oxygen is consumed and blood flow increases to the activated region to meet the increased oxygen demand. Actually, increases in cerebral blood flow to the local vasculature that accompanies neural activity in the brain far overtake changes in oxygen consumption, which will lead to relative decrease of deoxyhemoglobin and increased BOLD signal.

The relationship between oxygenation change with increased activity and change of the BOLD signal is in fact a little more complex. There is a momentary decrease in blood oxygenation immediately after neural activity increases, known as the "initial dip" in the hemodynamic response. This is followed by the blood flow increases, not just to a level where oxygen demand is met, but overcompensating for the increased demand. This means that blood oxygenation actually increases following neural activation. The blood flow peaks after around 5–6 s and then falls back to baseline, often accompanied by a poststimulus undershoot.

The fMRI technique has been increasingly used to produce activation maps showing which parts of the brain are involved in a particular mental process or state. While lying in the MRI scanner, a subject experiences some mental states or does some task (e.g., visual stimuli on a screen, response to some cue in a certain manner). Meanwhile, the MRI scanner tracks the signal throughout the brain or some specific part of interest. In brain areas, the BOLD signal is changing as the stimulus or task condition is varying. The hemodynamic change is measured by BOLD contrast in a "voxel" (a volume pixel; a small unit consisting of three-dimensional part of the brain image). The activity in a voxel is defined as how closely the time course of BOLD signal from that voxel matches the hypothesized time course. If the signals from a certain voxel match and correlate the hypothesized time course, this voxel is given a high statistical value, that is, a high activation score. These statistics in voxels can then be translated into a statistical brain map that shows the extent of "activation."

Compared with earlier neuroimaging techniques like positron emission tomography (PET), the advantages to fMRI as a technique to image brain activity related to a specific task or sensory process are the following: (1) fMRI does not require injections of radioactive isotopes, (2) the total scan time required is shorter, (3) the spatial resolution of the obtained functional image is higher (typically several mm, although high resolutions less than 1 mm are now possible technically), and (4) the temporal resolution of time course data is much higher than PET (highest resolution is ~0.5 s; typically ~1–4 s is used) so that it enables to analyze finer hemodynamic changes accompanied with short event-related neural events (called event-related design).

On the other hand, fMRI has disadvantages versus PET: (1) fMRI is loud, and its noise is

over ~90 dB, (2) quite sensitive to movement artifact because fMRI uses sequential excitation method by radio-frequent pulses across multislices of the brain, (3) fMRI causes signal distortion and loss because of susceptibility artifact in orbitofrontal and inferior/medial temporal areas which are important for emotional processing or social cognition, and (4) behavioral and physiological measurement inside the scanner is hard without specially designed MRI-compatible hardware because of a very strong static magnetic field or rapidly changing gradient magnetic field.

Nowadays, researchers started to combine the imaging techniques to determine brain activity and concurrent measurement of various physiological indexes. For example, measurements of the autonomic nervous system, such as pulse/skin conductance/electromyogram, are used in the scanner to detect dynamic associations of the bodily states with neural states. EEG has been also used simultaneously with fMRI, to utilize the advantages of the high spatial resolution of MRI and high temporal resolution of EEG. The transcranial magnetic stimulation method (TMS) is also used with fMRI as a noninvasive method to temporarily suppress the neuronal activity in a local region by electric stimulation by a coil outside the head.

Recently, a movement has arisen that attempts to understand the function of the brain both comprehensively and integratively as a network: This evolved from the point of view that the various sites of the brain and the wide variety of information from the body dynamically form a complicated web of consciousness, recognition, and feelings in a mutually influenced manner. For this purpose, connectivity between different brain regions has been assessed using correlation or multivariate analyses. Furthermore, there is a new direction of neuroimaging studies that includes a wider context than that of specific studies focusing only on a certain disease or nervous system. For example, a new field called "social neuroscience" is spreading rapidly in which neuroscience has begun to be applied to probing highly advanced cognitive functions, such as what kind of role a neuronal system plays in human social interactions.

A unique technique called "real-time fMRI" (rtfMRI) has been recently used in some neuroimaging studies (deCharms, 2007). This method resembles the "biofeedback" therapy that has conventionally been used in clinical settings to therapeutically give patients feedback on their distal physiological signs. The rtfMRI can be called "neurofeedback" in which fMRI simultaneously feeds back to the subjects the regional neural activity of the brain so that they can learn to directly control activation of localized regions by themselves – self-control. The rtfMRI has possibilities for use in rehabilitation through training by noninvasive and non-pharmacological means. A trial has just started into the clinical applications for chronic pain/drug dependency/depression and the ability to support psychotherapy.

Despite the great advantages of fMRI, there are some "pitfalls" to use this kind of neuroimaging technique like fMRI (Bennett & Miller, 2011; Logothetis, 2002). The BOLD response can be affected by a variety of factors, including drugs/substances, age, brain pathology, local differences in neurovascular coupling, attention, amount of carbon dioxide in the blood, etc. The images produced must be interpreted carefully, since correlation does not imply causality, and brain processes are complex and often nonlocalized. Statistical methods must be used carefully because they can produce false positives (Vul, Harris, Winkielman, & Pashler, 2009). One team of researchers studying reactions to pictures of human emotional expressions reported a few activated voxels in the brain of a dead salmon when no correction for multiple comparisons was applied, illustrating the need for rigorous statistical analyses. The BOLD signal is only an indirect measure of neural activity and is, therefore, susceptible to influence by non-neural changes in the body. This also means that it is difficult to interpret positive and negative BOLD responses. BOLD signals are most strongly associated with the input to a given area rather than with the output (Lauritzen, 2005). It is therefore possible (although unlikely) that a BOLD signal could be present in a given area even if there is no single unit activity. fMRI has poor temporal

resolution. The BOLD response peaks approximately 5–6 s after neuronal firing begins in an area. This means that it is hard to distinguish BOLD responses to different events which occur within a shorter time window. fMRI has often been used to show activation localized to specific regions, thus minimizing the distributed nature of processing in neural networks. Several recent multivariate statistical techniques work around this issue by characterizing interactions between "active" regions found via traditional univariate techniques.

The attractions of fMRI have made it a popular tool for imaging normal brain function – especially for psychologists. fMRI is also being applied in clinical and commercial settings. At the moment, there are no clinical applications immediately available, but in the near future, the progress of neuroscience shows promise for its clinical utility.

Cross-References

- ▶ Brain, Imaging
- ▶ Magnetic Resonance Imaging (MRI)
- ▶ Neuroimaging

References and Readings

Bennett, C. M., & Miller, M. B. (2011). How relia are the results from functional magnetic resonance imaging? *Annals of the New York Academy of Sciences, 1191*, 133–155.

deCharms, R. C. (2007). Reading and controlling human brain activation using real-time functional magnetic resonance imaging. *Trends in Cognitive Sciences, 11*(11), 473–481.

Friston, K. J., Frith, C. D., Dolan, R. J., Price, C. J., Zeki, S., Ashburner, J. T., et al. (Eds.). (2004). *Human brain function* (2nd ed.). San Diego: Academic Press.

Lauritzen, M. (2005). Reading vascular changes in brain imaging: Is dendritic calcium the key? *Nature Reviews Neuroscience, 6*(1), 77–85.

Logothetis, N. K. (2002). The neural basis of the blood-oxygen-level-dependent functional magnetic resonance imaging signal. *Philosophical Transactions of the Royal Society B: Biological Sciences, 357*(1424), 1003–1037.

Ogawa, S., Lee, T. M., Kay, A. R., & Tank, D. W. (1990). Brain magnetic resonance imaging with contrast dependent on blood oxygenation. *The Proceedings of the National Academy of Sciences of the United States of America, 87*(24), 9868–9872.

Ogawa, S., Lee, T. M., Nayak, A. S., & Glynn, P. (1990). Oxygenation-sensitive contrast in magnetic resonance image of rodent brain at high magnetic fields. *Magnetic Resonance in Medicine, 14*(1), 68–78.

Ogawa, S., Tank, D. W., Menon, R., Ellermann, J. M., Kim, S. G., Merkle, H., et al. (1992). Intrinsic signal changes accompanying sensory stimulation: Functional brain mapping with magnetic resonance imaging. *The Proceedings of the National Academy of Sciences of the United States of America, 89*(13), 5951–5955.

Vul, E., Harris, C., Winkielman, P., & Pashler, H. (2009). Puzzlingly high correlations in fMRI studies of emotion, personality, and social cognition. *Perspectives on Psychological Science, 4*(3), 274–290.

Functional Somatic Symptoms

- ▶ Functional Somatic Syndromes
- ▶ Somatoform Disorders

Functional Somatic Syndromes

Tetusya Ando
Department of Psychosomatic Research, National Institute of Mental Health, National Center of Neurology and Psychiatry, Kodaira-shi, Tokyo, Japan

Synonyms

Functional somatic symptoms; Medically unexplained symptoms; Somatoform disorders

Definition

Several related syndromes that are characterized more by symptoms, suffering, and disability than by disease-specific, demonstrable abnormalities of structure or function (Barsky and Borus, 1999).

Symptoms that cannot be explained in terms of a conventionally defined medical disease (Wessely, Nimnuan, & Sharpe, 1999).

Description

Concern about symptoms is a major reason for patients to seek medical help. Many of the somatic symptoms, such as pain of different location (back, head, muscles or joints, abdomen, chest), fatigue, dizziness, edema, dyspnea, insomnia, and numbness, often remain unexplained by identifiable disease even after extensive medical assessment. Terms such as somatization, somatoform disorders, abnormal illness behavior, functional symptom, and medically unexplained symptom (MUS) have been used to describe these symptoms. The term functional symptom assumes only a disturbance in body function but no psychogenesis of the symptom.

Concept of functional somatic syndromes (FSS) was first proposed by Barsky A.J. and others in 1999. They defined FSS as several related syndromes that are characterized more by symptoms, suffering, and disability than by structural or functional abnormality which include specific somatic syndromes such as multiple chemical sensitivity, the sick building syndrome, repetition stress injury, the side effects of silicone breast implants, the Gulf War syndrome, chronic whiplash, the chronic fatigue syndrome, the irritable bowel syndrome, and fibromyalgia. Patients with FSS have self-diagnosis, and their symptoms are often refractory to reassurance, explanation, and palliative treatments of symptoms. Although individual functional FSS may present with some organ-specific symptoms (e.g., gastrointestinal symptoms in irritable bowel syndrome) and may differ in its lead symptoms, the various FSS have similar symptoms that are diffuse, nonspecific and ambiguous, and very prevalent in healthy, non-patient populations. Symptoms common to the FSS include fatigue, weakness, sleep difficulties, headache, muscle aches and joint pain, problems with memory, attention, and concentration, nausea and other gastrointestinal symptoms, anxiety, depression, irritability, palpitations, shortness of breath, dizziness, sore throat, and dry mouth.

The concept was soon followed by Wessely S and others who postulated that the existence of specific somatic syndromes is largely an artifact of medical specialization on the basis of literature review because they found considerable overlap and similarities in definition, diagnostic criteria, symptoms and non-symptom characteristics, and response to treatment between individual syndromes as described later. This evoked debates regarding commonality and individuality of the specific syndromes and advantages and disadvantages of the concept in understanding and treatment of these illnesses.

Though an objective criterion of general FSS does not exist, epidemiological studies of individual specific syndromes have indicated that FSS are very common in all countries and cultures. For example, the prevalence of irritable bowel syndrome, chronic fatigue syndrome, and fibromyalgia are 10–20%, 0.01–0.3%, and 1–6%, respectively.

The syndromes are strongly associated with emotional distress and disorders such as anxiety and depression, and sufferers are often severely disabled. Costs to patients and to medical resources are substantial with repeated investigation and treatment.

Each single or specific functional syndrome is signified by current lead symptoms or implied cause. But overlap in case definitions of specific syndromes has been suggested. Patients with one functional syndrome often meet diagnostic criteria for other syndromes, for example, temporomandibular joint disorders and nonspecific facial pain, fibromyalgia and tension headache, and non-cardiac chest pain and hyperventilation syndrome are reported to be frequent combinations. Functional somatic symptoms are generally more common in women than in men.

Although, the causes of functional symptoms and syndromes are not fully understood, biological, psychological, interpersonal, and

health-care factors are considered to be all important. Dualistic, single factor view such as whether symptoms are psychological or physical will be unhelpful.

The symptoms of FSS are exacerbated by psychosocial stress and strongly associated with psychological distress, anxiety, and depression. History of childhood maltreatment and abuse has been reported to be frequent in FSS as they have been in psychiatric diseases.

Difficulties in doctor-patient relationship are quite usual. Because symptoms are not explained even after extensive medical assessment and conventional medical therapies are fairly ineffective, physicians are frustrated and patients are dissatisfied. Raising fear of disease, performing unnecessary investigations and treatments, and encouraging disability are adverse effects of medical consultation. Denying the reality of patients' symptoms may damage the doctor-patient relationship. Those iatrogenic components are important in the maintenance of FSS.

As biological mechanisms, altered functioning or abnormality of central nervous system, especially serotonergic system and neuroendocrine system, and immunological disturbances have been implicated, in addition to peripheral functional abnormalities in specific organ systems.

Barsky A.J. recommended medical management of FSS in six steps: (1) ruling out the presence of diagnosable medical disease, (2) searching for psychiatric disorders, (3) building a collaborative alliance with the patient, (4) making restoration of function the goal of treatment, (5) providing limited reassurance, and (6) prescribing cognitive behavioral therapy for patients who have not responded to the aforementioned five steps.

Peripheral or organ-oriented pharmacotherapy primarily aimed at peripheral physiological processes (e.g., bowel function, muscle tension, inflammation, pain, etc.) is also applied. Antidepressants (tricyclic antidepressants and SSRI) are often effective whether or not patient is depressed.

Cross-References

- ▶ Antidepressant Medications
- ▶ Fatigue
- ▶ Psychosomatic Disorder
- ▶ Somatoform Disorders

References and Readings

Barsky, A. J., & Borus, J. F. (1999). Functional somatic syndromes. *Annals of Internal Medicine, 130*, 910–921.

Henningsen, P., Zipfel, S., & Herzog, W. (2007). Management of functional somatic syndromes. *The Lancet, 369*, 946–955.

Wessely, S., Nimnuan, C., & Sharpe, M. (1999). Functional somatic syndromes: One or many? *The Lancet, 354*, 936–939.

Functional Testing

▶ Functional Capacity, Disability, and Status

Functional Versus Vocational Assessment

Ingrid Söderback
Department of Public Health and Caring Science, Uppsala University, Uppsala, Sweden

Synonyms

Aptitude testing; Assessment of functions; Assessments of work functions; Capacity assessment; Functional Capacity Assessment; Interest testing; Vocational evaluation; Vocational testing

Definition

Function is an individual's use of his/her best capabilities for performing daily activities and

work and for participating in social contexts and present environment. The concepts of functioning and disability are closely related. These make ends of a continuum and are viewed as a complex interaction between the health condition of the individual and the contextual factors of the environment as well as personal factors (WHO, 2011).

Work is the effort people expose themselves to with the aim to produce products or render services that are valuable to other people. *Vocational* is related to a specific job task, a work, or a profession. A work or a job task requires specific *work demands* to be fulfilled with acceptable quality and quantity (Perry, 2007). One of the following methods is chosen to stipulate the work demands, i.e., to perform a job-task analysis for a specific work: (a) the worker's depiction of the job circle, (b) description of a situational work-place assessment documented by video-films and a valid schema for observation (Söderback, 2011; Stein, Söderback, Cutler, & Larsen, 2006), (c) use of the Revised Handbook of Analyzing Jobs (RHAJ) including 60 criterion-referenced factors (U.S. Department of Labor, 1991), and (d) use of the computerized Occupational Information Network Numerical Index (O*NET) (Information Technology & U.S. Department of Labor, 2011; The Work Suite, 2011), as based on the Dictionary of Occupational Titles (DOT) (Information Technology & U.S. Department of Labor, 2011; U.S. Rehab. A division of the VGM Group, 2011). DOT classifies thousands of works into nine occupational categories. Using DOT the demands that a work requires on a worker are stated in the *worker qualification profile* including the following: (a) work performed (the work functions, the work fields, machines, tools, equipments, and work aids (MTEWA)), (b) materials, products, subject matter services (MPSMS), and (c) the *worker characteristics needed to perform the work*. These are stated as follows: (c^1) General Educational Development (GED) including reasoning, mathematical, and language development; (c^2) aptitudes, which is physical or mental competency, knowledge, understanding, or atti-64 tude to do a certain work at a certain level. This competence is innate or acquired, or learned and continuously developed; (c^3) physical activity demands, which means rating of strength and movement, i.e., standing, walking, sitting, carrying, pushing, pulling, lifting, raising; (c^4) temperaments, i.e., personal traits that are required by the worker in specific job-worker situations (U.S. Rehab. A division of the VGM Group, 2011); (c^5) Specific Vocational Preparation (SVP), which is the amount of time required by a typical worker to learn the techniques, acquire information, and develop the facility needed for average performance in a specific job-worker situation; (c^6) worker interests are stated in the Guide to Occupational Exploration (GOE); or the Holland codes, which classify occupations as realistic, i.e., outdoors and hands-on; investigative, i.e., scientific; artistic, i.e., creative; social, i.e., counseling and teaching; enterprising, i.e., management and sales; and conventional, i.e., clerical (Holland, 1985). The results of GED, physical demands, aptitudes, and temperament assessments reveal a person's capability level, but the results of worker interests and attitude inventories reveal which job or occupation a person wants.

Assessment is the process by which an individual is evaluated using specific, reliable, and valid instruments, tools, and procedures. In this process, data is gathered, hypothesis formulated, and decisions and recommendations for future measures stated (Stein et al., 2006; Söderback, 2009).

Description

Investigation: *Functional assessments* investigate a person's level of his/her body functions (mental, cognitive, sensory, speech, and neuromusculoskeletal), activities and participation (learning and applying knowledge, general tasks and demands, communication, mobility, self-care, domestic life, interpersonal interactions and relationships, major life community, and social and civic life areas), behavior (ability, competence, and performance) in interaction with the present environment, health, quality of life, and personality characteristics (ICF, 2011),

sometimes related to specific diagnoses like rheumatoid arthritis, multiple sclerosis, dementia, etc. The results are referred to *standardized norms*, i.e., a person's result is compared with the results of people with similar prerequisites.

For example, a *functional capacity evaluation* (*FCE*) investigates a person's *general* capability to perform a range of physical activities that might be required to be able to successfully fulfill simulated work tasks without any relation to a specific job (Australian Government CRS & Department of Human Services, 2011; Gibson, 2009). These results are referred to *standardized norms*.

Vocational assessment investigates a person's potential to perform a *specific work* and his/her *work* functioning with emphasis on health and wellness at work. It is a tool for finding the optimal match between the demands of a specific work and the person's current work capacity, skills, and experiences. The result is *criterion-referenced*, determining how well the work demands of a specific work produced with a normal performance of a product or service *correspond to* the individual worker's physical, psychological, intellectual capacity and skills, and actual social and environmental circumstances. A vocational assessment should focus on identifying the person's abilities and strengths (Australian Comprehensive Psychological Assessment Centre, 2011; Matthews, 2010; Michaels, 2011; Rich, 2011). An example is the Criterion-Referenced-Multidimensional Model for Vocational Assessment (CMVA), including investigation of (a) the demands on a specific work and (b) the worker's contribution for finishing the job (personal characteristics, job performances, personal prerequisites, and psychosocial work environment), which is valid for people with long-term musculoskeletal pain (Soderback, Schult, Jacobs, 2000; Söderback, 2011).

Aims: *Functional assessments* are used for determining a person's level of body function/dysfunction and how these levels influence his/her performances of personal-home-work activities and participation in the actual environment. Moreover, it is used to estimate a person's perception of his/her health and well-being and for description of personal characteristics. Functional assessments are aimed to determine facts useful for supporting a plan for guiding an objective intervention and for preventing ill-health. It is worth to underline that a *FCE* should *not* be used as the only tool for determining a person's capacity for work.

Vocational assessments are used for determining an appropriate occupational match between the job and the worker. The assessment results determine the following: (a) the worker's readiness for work, and if so, to set a vocational goal and identify a range of suitable employment options, either connected to the ordinary or to a new job; (b) the worker's ability to continuously perform a specific job; (c) a suitable occupational rehabilitation program; (d) amount of health insurance for employment and supporting allowance claims; and (e) used for preventing the worker's long sick leave.

Target groups: *Functional assessment* may be used for people who need response to what is intended to be investigated. For example, it may concern people of all ages, in risk for or living with chronic diseases and/or impairments, disabilities, and restricted participation in social life including those who are outside the labor market.

Vocational assessment is used for career decisions and decisions about return-to-work (Ahlers et al., 2003; Lombard, 1994; Perry, 2007). *Career decision assessments* are used in healthy adolescents, young adults, and unemployed people who need advices with their careers and for young adults who need support for their school-to-work transition (Nel & van der Westhuyzen, 2009). The examinations concern which occupations best fit a person's interests, abilities, and personality.

Return-to-work is used in the injured or disabled people who need rehabilitation, i.e., vocational therapy before return-to-work (ICD-9CM: 9385) (MDGuidelines, 2011). The examinations concern the following: (a) *readiness for work* (i.e., medical and health status and prognoses, a realistic job goal, ability for transporting, manage money, understand the job requirements,

appropriate work habits) (O'Neill & Wolf, 2010) and need for technical aids and adaptation of work environment; (b) *worker meets the work demands*, i.e., if the level of a person's medical and health status showing incapacity, adaptability, ability, work skills, and personality matches a certain job criteria as defined by a job-task analysis essential to successfully fulfill job requirements; and (c) *effects of recruitment and job placement*. Here the train-and-place model is commonly used, i.e., the person participates in a rehabilitative process of vocational assessment, work training in work shops or work hardening, and placement to a job on a supported employment sometimes assisted with a job coach.

Content of the Assessments: Functional and vocational assessments are conducted using a great variation of standardized or non-standardized tools and techniques. Examples are self-assessment inventories, standardized psychometric paper and pencil tests, checklists, structured interviews and observations. In addition, conducting a vocational assessment includes criterion-referenced tests, work samples (e.g., VALPAR component work sample (VALPAR International Cooperation, 2011)), ERGOS® II™ Work Simulator System (Rich, 2011), BTE work simulator (BTE Technologies, 2011), performance-based tasks, examination of records of work experiences and educational certificates, situational assessments of work tasks, and a vocational exploration used. There are enormous number of tools for functional and vocational assessments and the preferable used tool differs according to a country's culture and social system. However, the ultimate demands on actual assessment must be that its psychometric functions are scientifically proved for its validity and reliability.

Providers of *functional assessments* may be professionals working in health or school or social services that have required knowledge in psychometric theories and are certified for the specific assessment to be conducted. However, standardized tests are mainly conducted by a psychologist. Providers of *vocational assessments,* aimed for a career decision, should be conducted by authorities like *vocational consultants*, or *licensed professional counselors*. However, *vocational assessments*, aimed for return-to-work, are often conducted in cooperation between a *vocational rehabilitation consultant* and a *multidisciplinary team*, including specialists from various professional fields, such as occupational therapy, rehabilitation medicine or psychiatry, psychology, social work, schools, and employers. The constitution of these teams depends on the client's disability.

The requirements for working as vocational consultants, vocational rehabilitation consultants, or licensed professional counselor (Career Org, 2011; wiseGEEK, 2011) vary extensively around the world. In Australia (e.g., The University of Sidney, 2010), Hong Kong (e.g., Vocational Training Council (2010)), and the USA (e.g., Lebednik, 2010), these practitioners should have obtained a state license and have master degree in rehabilitation counseling or rehabilitation services (4-year) or doctorate degree in counseling or psychology. In EU, a report on standards of Training for Vocational Rehabilitation Services (2011) has recently been published. However, most employment officers in Sweden have a bachelor degree in behavioral science, social work, occupational therapy, etc. (The Swedish Public Employment Service, 2011), but a specific master degree in vocational counseling cannot yet be obtained and therefore not required.

Current Best Practices and Evidence

The current best practices for conducting functional assessments and FCAs follow the manual of the assessment tool and for vocational assessments the guiding principles stated (e.g., by Smith et al., 2011). However, improvement of the assessments' evidence and effectiveness is required by use of repeated quality assurance measures. Here the goals and objectives should be directly linked to employment outcomes (Ahlers et al., 2003).

Vocational assessments include a great variation of normative and criterion-referenced assessment instruments and tools. This mix is ordinarily a part of vocational rehabilitation (VR) services.

Therefore, the clinical evidence of vocational assessment is so far sparsely documented. Available information might be sought as an embedded part among random controlled studies of VR services. Pruett, Swett, Chan, Rosenthal, and Lee (2008) stated that VR services have "empirical evidence to support the efficacy, clinical utility, and cost effectiveness in returning people with disabilities to competitive employment" (p. 9), if conducted by graduated rehabilitation counselors. However, random controlled studies are strongly requested as results of vocational assessments influence decisions about people's private economy and quality of life.

Summary

Functional assessments differ from vocational assessments in terms of aims, target groups, content, the way of conducting the assessments, and requirements of the providers, and should, therefore, be used with appropriate quality.

Cross-References

- ▶ Job Demand/Control/Strain
- ▶ Job Demands

References and Readings

Ahlers, M,, Annis, J., AshleyGary Cusick, J., Derwart, B., Fried, J., Glisson, C., Lannucci-Waller, J-T., Johnson, L., Langton, A., Leconte, P., O'Brien, M., Power, P. W., Sligar, S. R., Smith, E., Thomas, S. (2003). *A New Paradigm for Vocational Evaluation: Empowering the VR Consumer through Vocational Information*. 30th Institute of rehabilitation issues. Retrieved from http://www.rcep6.org/iri/30th/iri30.pdf

Australian Comprehensive Psychological Assessment Centre. (2011). *Vocational assessment*. [Fact sheet]. Retrieved from http://comprehensivepsychology.com.au/vocational_assessment.htm

Australian Government CRS Australia, Department of Human Services. (2011). *List of services. Functional capacity evaluation (FCE)*. http://www.crsaustralia.gov.au/list_of_our_services.htm#voc_axmt

BTE Technologies. (2011). *BTE work simulator*. [Fact sheet]. Retrieved from http://www.nvortho.com/pdfs/bte.pdf

Career Org. (2010) *Occupation Profile for Rehabilitation Counselors* [Fact sheet]. Retrieved from http://occupations.careers.org/21-1015.00/rehabilitation-counselors

EU: CEDEFOP European Centre for the Development of Vocational Training. (2011). *European Training Thesaurus – ETT*. Retrieved from http://libserver.cedefop.europa.eu/ett/en/download/tr2025.htm

EU: Education and Culture DG Lifelong learning program. (2011). Training for Vocational Rehabilitation Services (TRAVORS). *Welcome to the TRAVORS project!* [Project information]. Retrieved from http://www.travors.eu

Gibson, L. (2009). Functional capacity evaluation: An integrated approach to assessing work activity limitations. In I. Söderback (Ed.), *International handbook of occupational therapy interventions*. New York: Springer Science+Media.

Hansen, K. (2011). *Online career assessments: Helpful tools of self-discovery*. Quintessentional Careers™. [Fact sheet]. Retrieved from http://www.quintcareers.com/online_career_assessments.html

Holland, J. (1985). *Making vocational choices*. (2nd ed.) Odessa, FL.: Psychological Assessment Resources. Retrieved from Rogue Community College. (2011). [Fact sheet] Retrieved from http://www.roguecc.edu/counseling/hollandcodes/about.asp

Information Technology Associates, U.S. Department of Labor. (2011). *Network Numerical Index (O*NET)* [Fact sheet]. Retrieved from http://www.occupationalinfo.org/onet/)

Information Technology Associates, U.S. Department of Labor. (2011). *Dictionary of Occupational Titles (DOT)*[Fact sheet]. Retrieved from http://www.occupationalinfo.org/contents.html

Lebednik, C. (2010). eHow Contributor: *How to get a certification in vocational testing*. Bureau of Labor Statistics, U.S. Administration [Information sheet]. Retrieved from http://www.ehow.com/how_7529197_certification-vocational-testing.html.

Lombard, R. C., (1994). *Vocational assessment practices:* What Works. Office of Special Populations Brief 6: 2. Retrieved from http://vocserve.berkeley.edu/briefs/Brief62.html

Matthews, A. (2010). *Vocational assessment testing*. [Fact sheet]. Retrieved from Vocational Assessment Testing | eHow.com http://www.ehow.com/facts_7479649_vocational-assessment-testing.html#ixzz1BUC6L0SX

MDGuidelines (2011). *Medical Disability Advisor > Vocational Therapy* [Fact sheet]. Retrieved from http://www.mdguidelines.com/vocational-therapy

Michaels, R. D., (2011). *Vocational assessment/evaluation: why do it*. [Fact sheet]. Retrieved from http://www.michaels-assoc.com/why.htm

Nel, L., & van der Westhuyzen, C. (2009). Conducting transitional strategies that support children with special needs in assuming adult roles. In I. Söderback (Ed.), *International handbook of occupational therapy interventions*. New York: Springer.

O'Neill, K., & Wolf, T. J. (2010). Development and pilot-testing of a work readiness assessment battery. *Work, 36*(4), 423–430.

Perry, D. A. (Eds.). (2007). *A resource guide on disability for employers in Asia and the Pacific*. International Labour Office (ILO). Regional office for Asia and the Pacific. Employability. Retrieved from http://www.ilo.org/wcmsp5/groups/public/—asia/—ro-bangkok/documents/publication/wcms_bk_pb_98_en.pdf

Pruett, S. R., Swett, E. A., Chan, F., Rosenthal, R. A., & Lee, G. K. (2008) Empirical evidence supporting the effectiveness of vocational rehabilitation. *Journal of Rehabilitation*, April–June, http://findarticles.com/p/articles/mi_m0825/is_2_74/ai_n27966257/pg_9/?tag=content;col1

Rich, J. (2011). *A guide to psychological testing and assessment*. *Vocational assessment* [Fact sheet]. Retrieved from http://www.psychologicaltesting.com/vocation.htm. Simwork systems (2011). *ERGOS® II™ Work Simulator System*. [Fact sheet]. Retrieved from http://www.simwork.com/products/ergos/ergos.htm

Smith, F., Lombard, R., Neubert, D., Leconte, P., Rothernbacher, C., & Sitlington, P. (2011). *Position paper of the interdisciplinary council on vocational evaluation and assessment*. Retrieved from http://www.vecap.org/images/uploads/docs/Interdisciplinary_Council.pdf

Söderback, I. (2009). Basic elements for conducting evidence-based occupational therapy. In I. Söderback (Ed.), *International handbook of occupational therapy interventions*. New York: Springer.

Söderback, I. (2011). *Assessing disabilities and illness on capacity for work and employment*. Retrieved from [Power Point Slides] http://www.soederback.se/assessing_disabilities/index.html

Soderback, I., Schult, M.-L., & Jacobs, K. (2000). A criterion-referenced multidimensional job-related model prediction capability to perform occupations among persons with chronic pain. *Work A Journal of Prevention, Assessment and Rehabilitation, 15*(1), 25–39.

Stein, F., Söderback, I., Cutler, B., & Larsen, B. (2006). *Occupational therapy and ergonomics. Applying ergonomic principles to everyday occupation in the home and at work*. London: Whurr.

The Swedish Public Employment Service. (2011). *Vi som arbetar här (We who are working here)* In Swedish [Information sheet]. Retrieved from http://www.arbetsformedlingen.se/Om-oss/Jobba-hos-oss.html

The University of Sidney. (2010). *Graduate diploma in rehabilitation counselling/Master of rehabilitation counselling. Course 23, Rehabilitation counseling*. [Fact sheet]. Retrieved from http://sydney.edu.au/handbooks/health_sci/23_rehab.shtml

The Work Suite. (2011). *O*NET versus DOT*. [Lecture] Retrieved from http://www.theworksuite.com/id13.html

U.S. Rehab. A division of the VGM Group. (2011). *Lecture three*. [Lecture]. Retrieved from http://www.uscrehab.com/RHAB%20712/Occanly%20Lecture%20Three.htm

VALPAR International Cooperation. (2011). *VALPAR component work sample* (2011). [Fact sheet]. Retrieved from http://www.valparint.com/index.htm

Vocational Training Council. (2010). *Vocational assessment services and program*. Hong Kong [Fact sheet]. Retrieved from, http://www.vtc.edu.hk/vtc/web/template/text.jsp?fldr_id=1512

WHO. (2011). *International Classification of of Functioning, Disability and Health (ICF)*. The Browser. Retrieved from http://apps.who.int/classifications/icfbrowser/

wiseGEEK. (2010). *What is a vocational rehabilitation consultant*. [Fact sheet]. Retrieved from http://www.wisegeek.com/what-is-a-vocational-rehabilitation-consultant.htm Also see: ILO Vocational Rehabilitation and Employment (Disabled Persons) Convention (No. 159) and Recommendation (No. 168); United Nations Convention on the Rights of Persons with Disabilities.

U.S. Department of Labor. (1991). *The Revised Handbook for Analyzing Jobs* (Career Reference Books). New York: JIST Works.

G

Galvanic Skin Response

► Electrodermal Activity (EDA)

Gamma-Aminobutyric Acid (GABA)

Elizabeth Galik
School of Nursing, University of Maryland, Baltimore, MD, USA

Definition

Gamma-aminobutyric acid (GABA) is the primary inhibitory neurotransmitter in the central nervous system of humans and other mammals. GABA is a highly polar and flexible molecule that is formed from glutamate in enzymatic reaction that causes its release into the synapse where it is inactivated by reuptake into glia cells (Chebib & Johnston, 1999). GABA transmission within the central nervous system modulates noradrenergic, dopaminergic, and serotonergic neurons which ultimately influences behavior and mood (Brambilla, Perez, Barale, Schettini, & Soares, 2003; Emrich, von Zerssen, Kissling, Moller, & Windorfer, 1980). A deficiency of GABA has been associated with a variety of neuropsychiatric disorders, including mood disorders, anxiety, panic, addiction, and schizophrenia, and neurological disorders such as Alzheimer's disease, Parkinson's disease, and Huntington's chorea. More recently, GABA has been studied as a mediating factor in the transmission and perception of pain (Enna & McCarson, 2006). GABA is also directly associated with the regulation of muscle tone. Pharmacologic agents that act as agonists of GABA receptors result in relaxation and sedation and have anticonvulsant properties.

References and Readings

Brambilla, P., Perez, J., Barale, F., Schettini, G., & Soares, J. C. (2003). GABAergic dysfunction in mood disorders. *Molecular Psychiatry, 8*, 721–737.

Chebib, M., & Johnston, G. A. (1999). The "ABC" of GABA receptors: A brief review. *Clinical and Experimental Pharmacology and Physiology, 26*(11), 937–940.

Emrich, H. M., von Zerssen, D., Kissling, W., Moller, H. J., & Windorfer, A. (1980). The GABA-hypothesis of affective disorders. *Archiv für Psychiatrie und Nervenkrankheiten, 229*, 1–16.

Enna, S. J., & McCarson, K. E. (2006). The role of GABA in the mediation and perception of pain. *Advanced Pharmacology, 54*, 1–27.

Gastric Ulcers and Stress

Shin Fukudo and Yukari Tanaka
Department of Behavioral Medicine, School of Medicine, Tohoku University Graduate, Aoba-ku, Sendai, Japan

Synonyms

Peptic ulcer

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

Definition

A gastric ulcer is a disease of gastric mucosal damage, caused by impaired mucosal defense and/or increasing gastric acid. Gastric acid consisting of hydrochloric acid and pepsin helps to digest intragastric contents, but they may also damage the gastric wall.

Description

Signs and Symptoms

The symptoms of gastric ulcer are the following: piercing or burning pain in the upper abdomen, poor appetite, nausea, vomiting, loss of weight, and feeling tired and weak. Tarry stool is the alarm sign of a bleeding, which suggests damage of the blood vessels in the submucosal or muscular layers of the gastroduodenal wall.

Causes

A type of bacteria called *Helicobacter pylori* (*H. pylori*) is responsible for most peptic ulcers. *H. pylori* is a common gastric pathogen and often begins to infect in childhood. Most people do not show any symptoms by *H. pylori* infection, but it sometimes causes chronic gastritis, peptic ulcer, dyspepsia, gastric adenocarcinoma, and B-cell mucosa-associated lymphoid tissue (MALT) lymphoma. *H. pylori* is motile and attaches to gastric mucosa through specific adhesion mechanisms. *H. pylori* urease, which produces ammonia on the gastric mucosa, increases the local pH and protects itself from acid. It may produce damage to the mucous coating and local inflammation. Another cause is the long-term use of nonsteroidal anti-inflammatory drugs (NSAIDs). NSAIDs can cause damage to the gastric and duodenal mucosa via some mechanisms, including block cyclooxygenase (COX)-1 and COX-2 inhibition. These two enzymes produce prostaglandins, substances that help to maintain blood flow and facilitate the repair of injury in the stomach. Cigarette smoking does not cause ulcers by itself but can make them worsen and delayed the healing.

Stress

Stress may not cause ulcers but can make them worsen. Patients with posttraumatic stress disorder (PTSD) showed higher prevalence of gastric ulcer than those without trauma. After the Hanshin-Awaji earthquake in Japan, the number of patients with gastric ulcer increased more than the previous year. This study also reported that *H. pylori* infection was a strong predisposing factor of the development of peptic ulcer. Stress sometimes induces psychological and physiological disorder and also closely relates to the central sympathetic activity. Spinal cord transection rat showed hypovolemia and higher prevalence of gastric ulcer. Brain angiotensin II AT1 receptors, which response the stress-induced hormone, relate to the stress-induced ischemia and inflammation in the gastric mucosa.

Diagnosis

First, taking a detailed history of symptoms and risk factors including medications, smoking, and drinking habits and if anyone in the patient's family has had ulcers. Physician will check of the patient's abdomen and chest as well as a rectal exam to look for any sign of bleeding. An abdominal X-ray is to rule out perforation (check free air under the diaphragm), and a blood test is to assess anemia. If a patient complains of sudden upper abdominal pain, it is important to rule out acute coronary syndrome by electrocardiogram. The diagnosis is confirmed by upper gastrointestinal (GI) endoscopy or upper GI series. If peptic ulcer is detected, biopsy of ulcer edge is recommended to rule out malignancy and check *H. pylori*. Noninvasive tests to detect *H. pylori* in a patient's blood, breath, or stool are also available.

Treatment

If acute bleeding is suspected, emergent gastrointestinal endoscopy or, in some cases, surgery is needed. Medication that reduces gastric acid secretion includes proton-pump inhibitors (PPIs) and histamine H2 receptor blockers (H2 blockers). PPIs cannot kill *H. pylori* but some study reported that it helps to eradicate *H. pylori* infection. *H. pylori* testing, therefore, should be done before PPIs medication, or after stopping

PPIs for a month at least. Patients with confirmed *H. pylori* infection should receive 1-week triple therapy consisting of PPIs and the antibiotics clarithromycin and amoxicillin, which can cure 80–90% of patients with peptic ulcer. After *H. pylori* eradication is completed, patients still have a higher incidence of gastric carcinoma than uninfected people. Patients with peptic ulcer therefore should be followed for a long time.

Cross-References

- ▶ Cigarette Smoking Behavior
- ▶ Stress

References and Readings

Davidson, J. R., Hughes, D., Blazer, D. G., & George, L. K. (1991). Post-traumatic stress disorder in the community: An epidemiological study. *Psychological Medicine, 21*, 713–721.

Kusters, J. G., van Vliet, A. H., & Kuipers, E. J. (2006). Pathogenesis of *Helicobacter pylori* infection. *Clinical Microbiology Reviews, 19*, 449–490.

Matsushima, Y., Aoyama, N., Fukuda, H., Kinoshita, Y., Todo, A., Himeno, S., et al. (1999). Gastric ulcer formation after the Hanshin-Awaji earthquake: A case study of *Helicobacter pylori* infection and stress-induced gastric ulcers. *Helicobacter, 4*, 94–99.

Mayer, E. A. (2000). The neurobiology of stress and gastrointestinal disease. *Gut, 47*, 861–869.

Saavedra, J. M., Ando, H., Armando, I., Baiardi, G., Bregonzio, C., Juorio, A., et al. (2005). Anti-stress and anti-anxiety effects of centrally acting angiotensin II AT1 receptor antagonists. *Regulatory Peptides, 128*, 227–238.

Strain, G. M., & Waldrop, R. D. (2005). Temperature and vascular volume effects on gastric ulcerogenesis after cord transection. *Digestive Diseases and Sciences, 50*, 2037–2042.

Gastrin-Releasing Peptide (GRP)

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

Gastrin releasing peptide (GRP) is a peptide with multiple roles which primarily regulates and stimulates secretion of gastric acid (Schubert, 2008). GRP also has other biological roles. For example, in the respiratory system, it causes bronchoconstriction on one hand and vasodilatation on the other hand. Indeed, GRP plays a role in pulmonary diseases leading to asthma, and its blockade serves as a therapeutic target in such conditions (Zhou, Potts, Cuttitta, Foster, & Sunday, 2011). GRP also has a role in several cancers. In neuroblastomas, for example, it acts as a tumor growth factor. The blockade of GRP augments the effects of chemotherapy (Paul, Gillory, Kang, Qiao, & Chung, 2011). Furthermore, GRP also plays a role in the circadian rhythm and in stress. GRP appears to mediate in part the stress response. In rats given corticosterone, higher GRP levels were seen in the amygdala and the medial prefrontal cortex during stress compared to control animals exposed to stress alone (Merali, Anisman, James, Kent, & Schulkin, 2008). Thus, it is possible that GRP alters brain activity upon exposure to chronic stress and to its neuroendocrine concomitants. Finally, GRP and its receptor are distributed in several brain regions and play a role in psychiatric and neurodegenerative disorders (Roesler, Henriques, & Schwartsmann, 2006). For these reasons, it has been suggested that GRP may serve as a therapeutic target in several health conditions (Roesler et al., 2006). Due to the complex and vast roles of GRP in psychological and biological processes and outcomes, it appears that this peptide deserves much attention in the field of behavior medicine as it may partly mediate effects of stress on somatic systems and health conditions.

Cross-References

- ▶ Asthma and Stress
- ▶ Cortisol
- ▶ Hypothalamic-Pituitary-Adrenal Axis

References and Readings

Merali, Z., Anisman, H., James, J. S., Kent, P., & Schulkin, J. (2008). Effects of corticosterone on

corticotrophin-releasing hormone and gastrin-releasing peptide release in response to an aversive stimulus in two regions of the forebrain (central nucleus of the amygdala and prefrontal cortex). *European Journal of Neuroscience, 28*, 165–172.
Paul, P., Gillory, L. A., Kang, J., Qiao, J., & Chung, D. H. (2011). Targeting gastrin-releasing peptide as a new approach to treat aggressive refractory neuroblastomas. *Surgery, 149*, 425–432.
Roesler, R., Henriques, J. A., & Schwartsmann, G. (2006). Gastrin-releasing peptide receptor as a molecular target for psychiatric and neurological disorders. *CNS & Neurological Disorders Drug Targets, 5*, 197–204.
Schubert, M. L. (2008). Gastric secretion. *Current Opinion in Gastroenterology, 24*, 659–664.
Zhou, S., Potts, E. N., Cuttitta, F., Foster, W. M., & Sunday, M. E. (2011). Gastrin-releasing peptide blockade as a broad-spectrum anti-inflammatory therapy for asthma. *Proceedings of the National Academy of Sciences of the United States of America, 108*, 2100–2105.

Gastrointestinal Disorders

▶ Gastric Ulcers and Stress

Gate Control Theory of Pain

Tavis S. Campbell, Jillian A. Johnson and Kristin A. Zernicke
Department of Psychology,
University of Calgary, Calgary, AB, Canada

Synonyms

Pain; Pain perception; Pain sensitivity

Definition

The gate control theory (GCT) of ▶ pain was introduced in 1965 by Ronald Melzack and Patrick Wall. It was the first theory to introduce the concept that pain experience is not simply the result of a linear process that begins with the stimulation of pain pathways in the peripheral nervous system and ends with the experience of pain in the central nervous system. Rather, neural impulses that potentially signal pain from the peripheral nervous system are subject to a number of modulations in the spinal cord by a "gatelike" mechanism in the dorsal horn before the experience of pain is transmitted to the central nervous system (Melzack & Wall, 1965). It also proposes that the gate mechanism is modulated by emotions, cognitive state, and past experiences. While this theory is based on physiology, it explains both sensory and psychological aspects of pain perception.

Description

The gate control theory of ▶ pain was first introduced by Canadian psychologist Ronald Melzack and British physician Patrick Wall in the 1965 *Science* article titled "Pain Mechanisms: A New Theory." The theory proposed that physical pain is not a direct result of activation of pain receptor neurons, but rather its perception is impacted by the interaction between different neurons. It proposes the existence of neural structures in the spinal cord and brainstem that modulate the experience of pain. These structures function like a gate, swinging open to increase the flow of transmission from nerve fibers or swinging shut to decrease the flow. With the gate open, signals arriving in the spinal cord stimulate sensory neurons which relay the signals upward to reach the brain and trigger pain. With the gate closed, signals are blocked from reaching the brain, and no pain is felt.

Gate Mechanism

According to the gate control theory of pain, three main types of nerve fibers are involved in the process of pain perception: A fibers, C fibers, and the "gate" interneurons. The diameters of these fibers vary in size. A-beta fibers have a large diameter and are myelinated, resulting in quick transmission of impulses. C fibers are smaller in diameter and are not myelinated, resulting in the slower transmission of impulses. A-delta fibers, another form of A fiber, are also small in diameter and have a function similar to that of C fibers.

The gate through which the pain pathways send signals to the nervous system is located in the dorsal horns of the spinal cord. The dorsal horns are composed of several layers, called laminae. Two of these layers make up the substantia gelatinosa, the hypothesized location of the gate mechanism. Both the small-diameter A-delta and C fibers and the large-diameter A-beta fibers travel through the substantia gelatinosa. The interneurons, located in the substantia gelatinosa, are the hypothetical gating mechanisms.

Activity of the large-diameter A-beta fibers produces an initial burst of activity in the spinal cord, followed by an inhibitory response. If the interneurons of the substantia gelatinosa are stimulated by activity in the large A-beta fibers, the interneurons produce an inhibitory response and do not allow pain sensations to be relayed up to the brain. Therefore, when the interneurons are stimulated by large fiber activity, the gate closes and no pain is experienced. Activity of the small-diameter A-delta and C fibers produces prolonged activity in the spinal cord. This type of activity promotes sensitivity and subsequently increases sensitivity to pain. If the interneurons are inhibited by the action of the small-diameter C fibers or A-delta fibers, or if they are not stimulated at all, the interneurons allow pain sensations to be sent up the brain. Thus, if the interneurons receive activity from small-diameter fibers, the gate remains open and results in the experience of pain.

In short, when the gate is open, impulses flow through the spinal cord toward the brain, neural messages reach the brain, and pain is experienced. When the gate is closed, impulses are inhibited from ascending through the spinal cord, messages do not reach the brain, and pain is not experienced. Therefore, the status of the gate depends on the balance of activity between the larger A-beta fibers and the smaller A-delta fibers and C fibers. This arrangement of neurons provides a physiological basis for the modulation of incoming sensory impulses.

Influence of the Brain on Pain

Although the gate may be closed by neural activity in the spinal cord, it may also be controlled by messages that descend from the brain. Melzack and Wall proposed the concept of a central control trigger consisting of nerve impulses that descend from the brain and influence the gating mechanism. They hypothesized that this system consists of large neurons that conduct impulses rapidly. These impulses from the brain affect the opening and closing of the gate in the spinal cord and are affected by cognitive processes. That is, the experience of pain is influenced by beliefs and prior experience. According to the gate control theory then, pain has not only sensory components but also motivational and emotional components. The theory explains the influence of cognitive aspects of pain and allows for learning and experience to affect how pain is experienced. ▶ Anxiety, ▶ worry, and ▶ depression, can increase pain by affecting the central control trigger, thus opening the gate. Distraction, ▶ relaxation, and ▶ positive emotions can cause the gate to close, thereby decreasing pain. The gate control theory is not specific about how these experiences affect pain but helps in the understanding that the sensation of pain can be dampened or aggravated by cognitions. For example, the theory helps explain how some people are able to withstand a large amount of pain through sheer willpower.

This theory provided a new way of thinking about pain and pain management and paved the way for current definitions of pain (e.g., International Association for the Study of Pain (IASP) pain terminology).

Impact and Critique

Prior to the gate control theory, pain was thought to be a direct response to a stimulus. Pain theories could not explain how two different people exposed to the same painful stimulus may have different reactions, nor did it explain phenomena such as phantom limb pain, defined as the sensation of pain in a limb that was previously amputated or removed. Melzack and Wall's theory was the first to suggest that psychological factors such as past experiences, attention, and emotion may have an impact on pain responses and perception. In addition, by highlighting the role of spinal and brain mechanisms on pain perception, the gate

control theory triggered an explosive advance in pain research and therapy. Today, it is considered the most influential theory of pain. It forced the medical and biological sciences to accept the brain as an active system that can modulate, filter, and select inputs. It has inspired several clinical techniques for controlling pain, including transcutaneous nerve stimulation (TENS), that involves the artificial stimulation of the large pain fiber system.

The gate control theory represents an important advance on previous simple response theories of pain. It introduced a role for psychology and described a multidimensional process rather than a simple linear one. However, the theory has received several criticisms. Although there is evidence illustrating the mechanisms to increase and decrease pain perception, the location of the gate itself is unknown. Another critique of the theory is that although the input from the site of physical injury may be moderated and mediated by experience and psychological factors, the model still assumes an organic basis for pain. This integration of physiological and psychological factors can explain individual variability and phantom limb pain to an extent, but because the model still assumes some organic basis for all pain, it is still foundationally based upon the flawed stimulus response process. Finally, the gate control theory attempts to depart from traditional dualist models of health by integrating the mind and body. Today, however, the mind and body are still seen as separate processes, although there is an attempt at some integration. The model suggests that physical processes are influenced by psychological processes, yet the two processes remain distinct.

Despite these criticisms, the gate control theory of pain is still the inspiration for the dominant theory of pain today, the ▶ biopsychosocial model of pain, and has stood the test of time remarkably well for a theory that triggered much research on relatively previously understudied health issue.

Cross-References

▶ Pain

References and Readings

Melzack, R. (1973). *The puzzle of pain*. Harmondsworth, England: Penguin Education.
Melzack, R. (1993). Pain: Past, present and future. *Canadian Journal of Experimental Psychology, 47*(4), 615–629.
Melzack, R., & Wall, P. D. (1962). On the nature of cutaneous sensory mechanisms. *Brain, 85*, 331–356.
Melzack, R., & Wall, P. D. (1965). Pain mechanisms: A new theory. *Science, 150*, 971–979.
Melzack, R., & Wall, P. D. (1988). *The challenge of pain*. New York: Basic Books.

Gay Men's Health Crisis

Jennifer Pellowski
Department of Psychology, University of Connecticut, Storrs, CT, USA

Definition

The Gay Men's Health Crisis (GMHC) is a volunteer-based, nonprofit organization that provides health, employment, and legal services to people living with HIV/AIDS in New York City. GMHC is an advocate for both public and private research concentrating on HIV/AIDS care and prevention and provides information about such issues as safer sex, testing and substance use through their community awareness campaigns (Gay Men's Health Crisis [GMHC], n.d.).

Description

GMHC was founded in 1981 by six men, including playwright and activist Larry Kramer, who were all concerned about the lack of information and services available to people living with a mysterious disease which is now known as HIV/AIDS. These men aimed to spread the word about "gay cancer" as well as raise money to fund research (Kayal, 1993). Among the first volunteers was Roger McFarlane who played an integral part in the establishment of the organization, particularly with the creation of the first

AIDS information hotline from his own home phone which evolved into the GMHC helpline. McFarlane became the first executive director of GMHC in 1982 (Hevesi, 2009).

One of the first major goals of the Gay Men's Health Crisis was to raise money to support research concerning HIV/AIDS. Within the first year, it raised $50,000 (Kayal, 1993). Currently, GMHC is an advocate for both public and private research, working with a yearly budget of nearly $20 million. In addition to conducting its own research, GMHC publishes several quarterly magazines/journals free of charge focused on health research and policy issues written in a widely accessible manner.

Since its conception, GMHC has been a major force in the dissemination of information regarding HIV and sexual health to the lesbian, gay, bisexual, and transexual (LGBT) community in New York City. It also works to educate people in the larger community, and despite its name, this organization serves people of all sexual orientations and genders (Reinfeld, 1993). In addition to sexual health information, it also provides a wide range of services to People Living with HIV/AIDS (PLWHA) and those at risk of contracting the virus (GMHC, n.d.).

One of their most extensive services is their multidisciplinary mental health clinic. It provides both short- and long-term counseling for individuals, families, and couples. Additionally, it conducts topic-based support groups focused on issues ranging from depression and anxiety to newly diagnosed individuals to living well with HIV (GMHC, n.d.).

GMHC also provides its clients with various services aimed at helping people living with HIV/AIDS like a healthy, normal life as well as reaching out to those who may be a risk for contraction the disease. These services include a hot meal program, nutritional information, an HIV and sexually transmitted infection (STI) testing clinic, legal support, and many different types of career services (GMHC, n.d.).

In addition to support for community members, GMHC is also an advocate for city, state, and federal policies concerning LGBT health care, youth services, and testing. The Gay Men's Health Crisis is integral to not only the LGBT community but to the New York City community at large. This organization works to bridge the gap between public health, government, health-care services, and the community. This, coupled with its support of HIV research, makes GMHC a key organization for the acquisition and dissemination of sexual health knowledge which is a key goal of the field of behavioral medicine.

Cross-References

- ▶ HIV Infection
- ▶ HIV Prevention
- ▶ Sexual Orientation
- ▶ Sexual Risk Behavior

References and Readings

Gay Men's Health Crisis. (n.d.). About us. Retrieved February 8, 2011, from www.gmhc.org

Hevesi, D. (2009, May 18). Rodger McFarlane, who led AIDS-related groups, dies at 54. *The New York Times*, p. A23.

Kayal, P. M. (1993). *Bearing witness: Gay men's health crisis and the politics of AIDS*. Boulder: Westview Press.

Reinfeld, R. M. (1993). The gay men's health crisis: A model for community based intervention. In J. P. Van Vugt (Ed.), *AIDS prevention and services: Community based research* (pp. 179–198). Westport, CT: Bergin & Garvey.

Gaze Tracking

- ▶ Eye Tracking

GDS

- ▶ Geriatric Depression Scale

GDS-15

- ▶ Geriatric Depression Scale

GDS-4

► Geriatric Depression Scale

Gender

► Gender Role

Gender Differences

Luis I. García and Jason W. Mitchell
Center for AIDS Intervention Research, Medical College of Wisconsin, Milwaukee, WI, USA

Definition

Gender refers to the identity humans tend to develop around their biological sex. While gender may be related to biology, gender identity is significantly influenced by culture and social learning. Gender differences refer to the observed differences in behavior displayed by females and males (or another gender). Specifically, differences in behavior can be observed between genders because society imposes a different set of appropriate activities and behaviors for females and males.

It is important to make a distinction between the biological differences between sexes (sexual dimorphism) and the way in which their behavior differs (gender differences). This difference is exemplified by the relationship between testosterone and aggression. For example, the serum level of testosterone in humans is sexually dimorphic; males tend to have higher blood levels of testosterone than females (Torjesen & Sandnes, 2004). It has also been found that higher levels of testosterone are associated with more aggression (Mehta & Beer, 2009). However, we refer to higher rates of aggression in males than in females as a gender difference because testosterone (biology) is not the only factor influencing aggression (behavior).

It is important to note that because gender is a social construct, the roles of each gender vary across culture as each society has a particular prescription of appropriate behavior for females and males. Although not without some controversy, Margaret Mead in the book *Temperament in Three Primitive Societies* (1963) discussed the influence of culture over aggression in three societies, one in which both females and males were gentle, one in which both females and males were aggressive, and one in which females were dominant and males were more emotionally dependent. The author's findings highlight the influence of socialization on the expression of aggression. Similarly, Bandura (1977) found that both girls and boys were able to learn and subsequently express aggressive behavior by observing others (a model) behaving aggressively, supporting the idea that individuals can be socialized to express more or less aggressiveness.

Many gender differences between males and females have been found. However, very frequently these differences are very small and the variation within genders tends to be much larger than the variation between genders. For example, it has been found that girls tend to do better in verbal tasks and boys in abstract problem solving skills such as those required in math (Hyde & Linn, 1988; Linn & Hyde, 1989). However, while the difference between boys and girls on either task is very small, big differences in verbal skills can be found among girls and in abstract problem solving among boys.

The literature offers many examples of the ways in which females and males are socialized differently and the influence of socialization is so profound that it seems to start even before birth. For example, Smith (2005) observed a series of changes in the way she related to her fetus during pregnancy once she learned it was a boy. For example, she observed that her voice became lower and firm and she stopped "nibbing clockwise" her belly when talking to the fetus and started patting it. She also noticed

her language use accommodated the "prescribed stereotypes" for males (e.g., referring to the fetus as "strong"). After birth, socialization seems to continue throughout the person's life in the form of rewards when gender-appropriate behavior is exhibited and punishment when gender-inappropriate behavior is chosen.

References and Readings

Bandura, A. (1977). *Social learning theory*. Englewood Cliffs, NY: Prentice-Hall.
Hyde, J. S., & Linn, M. C. (1988). Gender differences in verbal ability: A meta-analysis. *Psychological Bulletin, 104*, 53–69.
Linn, M. C., & Hyde, J. S. (1989). Gender, mathematics, and science. *Educational Researcher, 18*, 17–27.
Mead, M. (1963). *Sex and temperament: In three primitive societies*. New York: Harper Collins.
Mehta, P. H., & Beer, J. (2009). Neural mechanisms of the testosterone-aggression relation: The role of orbitofrontal cortex. *Journal of Cognitive Neuroscience, 22*(10), 2357–2368.
Smith, K. (2005). Pre-birth gender talk: A case study in prenatal socialization. *Women and Language, 28*(1), 49–54.
Torjesen, P. A., & Sandnes, L. (2004). Serum testosterone in women as measured by an automated immunoassay and a RIA. *Clinical Chemistry, 50*(3), 678–679.

Gender Norms

▶ Gender Role

Gender Role

Jennifer Toller Erausquin
Chronic Disease and Injury Prevention, NC Division of Public Health, Durham, NC, USA

Synonyms

Feminine role; Gender; Gender norms; Masculine role

Definition

Gender roles are the socially constructed patterns of behavior ascribed to men and to women. Gender roles encompass concepts about masculinity and femininity, and determine what behavior is expected or appropriate for men and women in a given situation. Gender roles are a social (rather than biological) definition of what it means to be a man or a woman. They may differ by society, culture, socioeconomic class, age, and period in history.

Gender roles are created and reinforced through social institutions such as family and intimate relationships, schools, religious institutions, industry and private enterprise, legal and medical systems, and the media. Not only do gender roles result in differential expectations of behavior, they also assign differential prestige to masculine and feminine behavior. To the extent that they reflect inequalities between men and women in access to resources or control, gender roles may contribute to unequal power relations between men and women (Connell, 1987; Sen, George & Ostlin, 2002).

Gender roles are of importance to behavioral medicine because they can affect an individual's health-related decisions and behaviors. Gender roles are considered particularly important in sexual health, as they can affect sexual practices, selection of partners, experience of pleasure, as well as contraceptive use and reproduction. *Masculine gender roles* typically define maleness through independence, aggressiveness, and exploration. Men may be expected to initiate sex early in life, have multiple sexual partners, and maintain control over their sexual partners. Masculine gender roles may therefore condone or encourage risky sexual behaviors (such as sex without a condom, anonymous sex, and sex with sex workers). Furthermore, in many societies, men are expected to be knowledgeable and experienced in sexual matters. *Feminine gender roles*, on the other hand, often dictate that "good" women lack sexual knowledge or experience, particularly prior to marriage. Feminine gender roles also frequently

assume that women have more control over their sexual desires than men. They may include the expectation that women act as "gatekeepers," limiting sexual access. In contrast to the masculine role of initiator of sexual activity, traditional feminine gender roles confine women to a reactive or passive role. Taken together, these gender roles may encourage behaviors that place both men and women at risk for unplanned pregnancy, sexually transmitted infections, and HIV.

The gender role perspective, while a useful framework for understanding social influences on behavior for men and women, is not without criticism. First, there is some debate regarding how deterministic or fluid gender roles are. Although awareness of and conformity to gender roles is influenced by an individual's childhood upbringing and ongoing experiences, several authors argue that individuals can be active agents in creating or challenging masculine or feminine gender roles. Second, public health scholars have questioned the utility of examining gender roles, as opposed to the more pervasive ways gender is interwoven into the structure of social institutions. As Sen et al. note, "...[E]xclusive or excessive emphasis on roles leads to a focus on behavior change at the individual level, rather than on policy change at the societal level" (2002, p. 6).

Cross-References

- ▶ Gender Differences
- ▶ Norms
- ▶ Sex Differences

References and Readings

Connell, R. W. (1987). *Gender and power: Society, the person and sexual politics*. Stanford: Stanford University Press.

Parker, R., Barbosa, R. M., & Aggleton, P. (2000). *Framing the sexual subject: The politics of gender, sexuality, and power*. Berkeley, CA: University of California Press.

Risman, B. J. (1998). Gender as structure. In *Gender Vertigo*. New Haven, CT: Yale University Press.

Sen, G., George, A., & Ostlin, P. (2002). *Engendering international health: The challenge of equity*. Cambridge, MA: MIT Press.

Wingood, G. M., & DiClemente, R. J. (2002). The theory of gender and power: A social structural theory for guiding the design and implementation of public health interventions to reduce women's risk of HIV. In R. J. DiClemente, R. A. Crosby, & M. C. Kegler (Eds.), *Emerging theories in health promotion practice and research: Strategies for enhancing public health* (pp. 313–347). San Francisco: Jossey-Bass.

Gene

Rany M. Salem[1] and Laura Rodriguez-Murillo[2]
[1]Broad Institute, Cambridge, MA, USA
[2]Department of Psychiatry, Columbia University Medical Center, New York, NY, USA

Synonyms

Locus

Definition

The gene is the fundamental unit of heredity, passing genetic information from parents to offspring. It is an ordered DNA sequence, which encode (a locus on a chromosome) that is involved in producing a protein. It is therefore an ordered sequence of nucleotide bases, which encode the necessary information to produce a specific functional product.

A gene includes regions that precede and follow the coding regions (the nucleotide bases that create a protein). In the mid-1970s, it was generally accepted that genes existed as continuous segments within a DNA molecule. This view changed radically with the discovery in 1977 that in higher organisms (eukaryotic cells), an individual gene can comprise several DNA (exons) segments separated by chunks of noncoding DNA (introns) (see Roberts, 1993). After the DNA sequence within a gene gets transcribed into messenger ribonucleic acid

(mRNA), an elegant process called splicing removes the introns and connects the exons to form the mRNA sequence that will get ultimately translated into a sequence of amino acids. These amino acid sequences make proteins, which are therefore made from the genetic instructions encoded in the DNA molecule.

The human genome contains approximately 20,000–25,000 genes (Human Genome Sequencing Consortium, 2004), a remarkably lower estimate than the figure of 100,000 genes that was commonly used before the Human Genome Project was finished. Human DNA contains approximately three billion base pairs, but the 20,000–25,000 genes comprise only a small percentage (on the order of 2–5%) of these base pairs. Taylor and Bristow (2006) commented as follows: "The "genes" themselves are only a modest part of the whole genome. It is clear that some of the "non-translated" DNA is required for genes to function normally, but the function of large portions of our DNA remains enigmatic. Equally remarkable, perhaps, is that humans do not use all of their genes at any one time, so far less than 25,000 genes are utilized on a day-to-day basis." As noted earlier, necessary genes are transcribed by being translated in to mRNA intermediates, which are subsequently translated into functional proteins.

Finding genes is not an easy task. Watson (2006) commented that "protein-coding regions are but strings of As, Ts, Gs, and Cs embedded among all the other As, Ts, Gs, and Cs of the genome – they do not stand out in any obvious way." Additionally, as noted already, the base pairs that comprise a gene are not arranged in an uninterrupted linear sequence. A typical human gene has eight introns that lie between the exon coding sections. For example, the gene dystrophin is spread across approximately 2.4 million base pairs, but only less than 1% of the total base pairs encode the actual protein. The other 99% are 79 introns located throughout the coding region. Given these difficulties in identifying human genes, knowledge of other genomes has proved remarkably helpful. Comparison between genomes leverages the fact that functional regions are preferentially conserved between specifies and can be used to identify novel genes. Thus, "looking for similarity in sequence between the human and mouse data is therefore an effective way of identifying functional areas, like genes" (Watson, 2006) (See also Table 1).

Gene, Table 1 Approximate genomic data for humans and several other species of interest (Adapted from Palladino (2006))

Organism and date genomic data obtained	Size of genome (base pairs)	Number of genes	Percentage of genes shared with humans
Human (2004)	3 billion	20,000–25,000	(100%)
Dog (2003)	6 billion	18,000	75%
Fruit fly (2000)	165 million	14,000	50%
Mouse (2002)	2.5 billion	30,000	80%
Rat (2004)	2.75 billion	22,000	80%

Cross-References

- Chromosomes
- DNA
- Genetics
- Genomics
- Human Genome Project
- Locus
- Locus (Genetics)
- Proteomics

References and Readings

Human Genome Sequencing Consortium. (2004). Finishing the euchromatic sequence of the human genome. *Nature, 431*(7011), 931–945. 10.1038/nature03001.

Palladino, M. A. (2006). *Understanding the human genome project* (2nd ed.). San Francisco: Pearson/Benjamin Cummings.

Richards, R. J. (1993). An amazing distortion in DNA induced by a methyltransferase. Lecture given upon receipt of the 1997 Nobel Prize for Medicine.

Watson, J. D. (2006). *DNA: The secret of life*. New York: Knopf.

Gene Expression

Ornit Chiba-Falek
Duke University Medical Center, Durham, NC, USA

Synonyms

Expression pattern; Gene regulation; Regulation of expression

Definition

The process by which information from a gene is used to synthesize a functional gene product. These products are often proteins, but some gene code to functional (noncoding) RNAs, such as rRNA, tRNA, and miRNA. The process of gene expression is used by all known organisms – eukaryotes, prokaryotes, and viruses – to generate the macromolecular machinery for life. Gene expression process may include several steps of regulation – transcription, RNA splicing, RNA stability, translation, and posttranslational modification of a protein – that determine the expressed product/s from each gene and control the timing, location (cell type), and amount of gene expression. Gene expression is tightly modulated and therefore serves the basis for cellular differentiation and morphogenesis, the versatility and adaptability of any organism, and evolutionary change.

In genetics, gene expression is the most fundamental level at which genotype gives rise to the phenotype. The genetic code is "interpreted" by gene expression, and the properties of the expression products give rise to the organism's phenotypes, such as appearance, behavior traits, and diseases.

A number of human diseases are known to result from the disruption of gene expression, including cancer, neurological diseases, etc. Thus, research of gene regulation is of high relevance to human health.

Cross-References

- ▶ Gene
- ▶ Genetics
- ▶ Genotype
- ▶ RNA

References and Readings

Barash, Y., Calarco, J. A., Gao, W., Pan, Q., Wang, X., Shai, O., et al. (2010). Deciphering the splicing code. *Nature, 456*, 53–59.

Barrett, T., et al. (2010). NCBI GEO: Archive for functional genomics data sets–10 years on. *Nucleic Acids Research, 39*(Database issue), D1005–D1010.

Birney, E., et al. (2007). Identification and analysis of functional elements in 1% of the human genome by the ENCODE pilot project. *Nature, 447*(7146), 799–816.

Emilsson, V., Thorleifsson, G., Zhang, B., Leonardson, A. S., Zink, F., et al. (2008). Genetics of gene expression and its effect on disease. *Nature, 452*, 423–428.

Gene Methylation

- ▶ Methylation

Gene Regulation

- ▶ Gene Expression

Gene-Environment Interaction

Rong Jiang
Department of Psychiatry and Behavioral Sciences, Duke University, Durham, NC, USA

Synonyms

GxE

Definition

Gene-environment interaction refers to the fact that the effects of genes on a disease often depend on the environment or that the effect of environment depends on the genotype (Dick, 2011).

In genetic studies of interest in Behavioral Medicine, gene-environment interaction is often used to describe the effect of genes modified by environmental exposure, including behavioral, nutritional, infectious, chemical, and physical factors, or any other nongenetic factors. It has been increasingly accepted that most common diseases involves not only genetic and environmental causes but also interactions between the two, which may account for a significant proportion of the heritabilities of a complex disease (Ober & Vercelli, 2011). The study of gene-environment interaction has faced some challenges, such as the environmental measurement errors or lack of true full range of environments (Dick, 2011), the need for large sample size (Hunter, 2005), the study methodologies, and the underlying biological mechanisms of gene-environment interaction.

Cross-References

- ▶ Complex Traits
- ▶ Gene
- ▶ Genetic Polymorphisms
- ▶ Genotype
- ▶ Heritability
- ▶ Phenotype

References and Readings

Dick, D. M. (2011). Gene-environment interaction in psychological traits and disorders. *Annual Review of Clinical Psychology, 7*, 383–409.

Hunter, D. J. (2005). Gene-environment interactions in human diseases. *Nature Reviews Genetics, 6*(4), 287–298.

Ober, C., & Vercelli, D. (2011). Gene-environment interactions in human disease: Nuisance or opportunity? *Trends in Genetics, 27*(3), 107–115.

Gene-Gene Interaction

Rong Jiang
Department of Psychiatry and Behavioral Sciences, Duke University, Durham, NC, USA

Synonyms

Epistasis; GxG

Definition

In genetics, gene-gene interaction (epistasis) is the effect of one gene on a disease modified by another gene or several other genes. Biological epistasis, i.e., the gene-gene interaction has biological basis, is in contrast to statistical epistasis that describes deviation from additivity in a linear statistical model (Gilbert-Diamond & Moore, 2011). Epistasis can be contrasted with dominance, which is an interaction between alleles at the same gene locus. Gene-gene interaction is a common component of genetic architecture of human complex diseases; however, it is difficult to detect. The multilocus genotype combinations for gene-gene interaction increase exponentially and require larger sample size as well as more computation burden. The commonly used linear models have limited ability to detect nonlinear patterns of gene-gene interaction. Multifactor dimensionality reduction (MDR) has been developed to detect gene-gene interaction as a nonparametric method by pooling genotypes from multiple SNPs without assuming genetic model (Moore & Williams, 2009). Another alternative to linear models is combinatorial partitioning method (CPM) (Nelson et al., 2001). The biological interpretation of gene-gene interaction identified from a statistical model may be the most important and most difficult to understand the disease etiology.

Cross-References

- ▶ Complex Traits
- ▶ Gene

- ▶ Genotype
- ▶ Locus
- ▶ Single Nucleotide Polymorphism (SNP)

References and Readings

Gilbert-Diamond, D., & Moore, J. H. (2011). Analysis of gene-gene interactions. *Current protocols in human genetics*, 70:1.14.1–1.14.12 © 2011 by John Wiley & Sons, Inc. doi:10.1002/0471142905.hg0114s70.

Moore, J. H., & Williams, S. M. (2009). Epistasis and its implications for personal genetics. *The American Journal of Human Genetics, 85*(3), 309–320.

Nelson, M. R., Kardia, S. L., Ferrell, R. E., & Sing, C. F. (2001). A combinatorial partitioning method to identify multilocus genotypic partitions that predict quantitative trait variation. *Genome Research, 11*(3), 458–470.

General Adaptation Syndrome

Tavis S. Campbell, Jillian A. Johnson and Kristin A. Zernicke
Department of Psychology, University of Calgary, Calgary, AB, Canada

Synonyms

Responses to stress; Stress reactivity

Definition

The general adaptation syndrome (GAS) is a theory of stress responding proposed by ▶ Hans Selye. It refers to the nonspecific, generalized responses of the body in response to stress and provides a framework for the link between stress and chronic illness (Selye, 1956). This syndrome is divided into three stages: alarm reaction, resistance, and exhaustion.

Description

▶ Hans Selye (1907–1982), known as "the father" of the stress field, was a Hungarian endocrinologist who emigrated to Montreal, Canada, in 1932. He pioneered research on the biological effects of exposure to "noxious agents," or stress, subsequently developing the concept of the general adaptation syndrome.

Development

▶ Selye first wrote about the general adaptation syndrome in the British journal *Nature* in 1936 when he was an assistant at McGill University's Biochemistry Department in Montreal. In an experiment designed to discover a new hormone, he injected laboratory rats with ovarian extracts in hopes of uncovering changes in the organism that could not be caused by any previously known ▶ sex hormones. He found that following injection of the extract, the adrenal cortex of the rats became enlarged; the thymus, spleen, and lymph nodes all showed signs of deterioration; and deep bleeding ulcers were formed in the stomach and duodenum which eventually lead to death. Interestingly, ▶ Selye discovered that each of these symptoms could be increased or decreased in severity by adjusting the amount of extract injected into the animals. To ▶ Selye, these symptoms appeared to be the workings of a previously unknown hormone. However, later experiments with placental, pituitary, kidney, spleen, and numerous other organ extracts, all resulted in the expression of the same symptoms, causing ▶ Selye to reject the idea that these symptoms were being produced by a specific substance.

Further animal experiments conducted by ▶ Selye with rats demonstrated that if the animals were damaged by acute nonspecific noxious agents (e.g., cold exposure, surgical injury, excessive exercise, and injection of toxic drugs), a typical syndrome appeared, with symptoms that were independent of the type of noxious agent, representing instead a general response to the stimulus. ▶ Selye noted that this syndrome developed in three stages. The first stage began 6–48 h after the noxious agent was administered and involved several key changes in physiological functioning, including a rapid decrease in the size of the thymus, spleen, lymph glands, and liver, disappearance of fat tissue, and a drop in body temperature. The second stage began 48 h

after the initial administration of the noxious agent and was characterized by an overall decrease in general parasympathetic activity, including a cease in general body growth, a deterioration of the gonads, discontinued milk production in lactating animals, and an increase in general sympathetic activity, including enlarged adrenal glands and hyperplasia of the thyroid. Upon continued treatment with the noxious agent, the animals would build up resistance such that by the end of the second stage, the appearance and function of the organs returned to normal. However, with further administration, after a period of approximately 1–3 months and depending on the severity and dose of the noxious agent, the animals eventually lost their ability to resist, and physical symptoms, similar to those seen in the first stage, began to reappear. This apparent exhaustion of the ability to resist was labeled as the third stage.

▶ Selye compared his findings in the laboratory to clinical experiences with humans. Similar to animals, ▶ Selye noticed that physical and emotional stress in humans induced a specific, predictable pattern of health outcomes that, if left untreated, would lead to infection, illness, disease, and eventually death. He noted that the recommended treatments for almost all of these complaints were those that were useful to patients suffering from almost any illness, including rest, changes in diet, and temperature regulation. Given that there was only one recommended treatment for such a wide range of generalized complaints, ▶ Selye thought that there may be a mechanism in the body whose response to external noxious agents was general. He proposed that certain changes take place within the nervous and endocrine systems within the body during stress that can disrupt normal physiological mechanisms, triggering disease or illness. This specific pattern of changes is now known as the general adaptation syndrome, which occurs in three generalized stages (Selye, 1956).

General Adaptation Syndrome Stages

Stage 1, the *alarm reaction*, occurs when the body's defenses against a stressor are mobilized through activation of the sympathetic nervous system. This reaction is known to activate body systems involved in the fight-or-flight response. ▶ Epinephrine (▶ adrenaline) is released, heart rate and blood pressure increase, respiration becomes faster, blood is diverted away from the internal organs toward the skeletal muscles, sweat glands increase production, and gastrointestinal system activity is suppressed. These physiological reactions were believed by ▶ Selye to be adaptive for acute emergency situations. However, many modern stress situations involve prolonged exposure to stress and typically do not require an alarm response. The magnitude of the alarm reaction may also depend on the degree to which the event is perceived as a threat.

If a stressful situation persists, the body's reaction will progress to stage 2, which ▶ Selye called the *resistance* stage. In this phase, physiological arousal remains high, although not as high as in the alarm reaction stage. The body attempts to adapt to the emergency by replenishing adrenal hormones. The duration of this stage depends on the severity of the stressor and the adaptive capacity of the organism. If the organism successfully adapts, the resistance stage will continue for a longer period of time. During this stage, an organism may appear unaffected, but physiologically, the body's internal functioning is active. Continued stress will lead to a stress-induced neurological changes and a breakdown of the hormonal system, leading to conditions know as the "diseases of adaptation," which Selye defined to include ▶ peptic ulcer, ▶ hypertension, hyperthyroidism, and immune deficiencies. At this point, there is a decrease in the organism's ability to cope with everyday events and hassles, possibly leading to behavior change (e.g., irritability, impatience, and increasing vulnerability to health problems).

If the stressful event persists to the point where resistance is no longer possible, the body enters the final stage of the general adaptation syndrome which ▶ Selye called *exhaustion*. At this point, the body's energy reserves are depleted. This stage is characterized by activation of the ▶ parasympathetic division of the ▶ autonomic nervous system (ANS). Under normal circumstances, activation of this division helps keep the body functioning in a balanced state. However, in the exhaustion stage, ▶ parasympathetic functioning is

at an abnormally low level, causing an organism to become exhausted. If stress persists, the "diseases of adaptation" are present and physical deterioration or even death may occur.

Impact and Critique

► Selye's breakthrough ideas about stress helped build an entirely new medical field based on the study of biological stress and its effects on the body. His research has inspired numerous researchers and continues to make contributions to this day by providing a theoretical framework for connecting stress to illness and leading to the study of methods to help the body effectively deal with life's chronic demands.

The concept of the general adaptation syndrome aids in the understanding of how stress may be linked with an abundant source of health problems. Of specific interest is the role of the ► Hypothalamic-pituitary-adrenal axis activity in response to stress. Early life stress has been linked with malfunctions in the normal cycle and functioning of the HPA axis. Instead of reducing the production of ► hormones once the stress is removed or ended, the cycle may be ongoing, with the ► hypothalamus continuing to signal the adrenals to produce ► cortisol. Eventually, high ► cortisol levels may lead to a suppression of the immune system through increased production of interleukin-6. This increased production may lead to the exhaustion of the stress mechanism, resulting in fatigue and depression, apparent in research findings that suggest that stress and depression have a negative effect on the immune system. As a result of the prolonged attempts to resist the stressor, the body may eventually lose its ability to resist all together. This person may then be at a higher risk to contract a disease related to immune deficiency, such as an infection.

Prolonged stress may also lead to blockages in the arteries by fat and ► cholesterol released by the body as part of the stress response, possibly contributing to a heart attack or stroke. The body's reactions to stress may also manifest itself into a number of other illnesses such as ► depression, sleep disorders, ► hypertension, ulcers, and ► asthma.

The general adaptation syndrome theory has not gone unchallenged, however, by evidence that different stressors or emotional states may elicit autonomic specificity or unique patterning during experimental manipulation. Nevertheless, the theory of general adaptation syndrome is a cornerstone of ► behavioral medicine because it led to the study of the effects of stress and hormones on brain function, including research investigating the biological functioning of ► glucocorticoids.

References and Readings

Selye, H. (1936). A syndrome produced by nocuous agents. *Nature, 138*, 32.
Selye, H. (1956). *The stress of life*. New York: McGraw-Hill.
Selye, H. (1974). *Stress without distress*. Philadelphia, PA: J.B. Lippincott.
Selye, H. (1976). *Stress in health and disease*. Reading, MA: Butterworths.
Selye, H. (1982). History and present status of the stress concept. In L. Goldberger & S. Breznitz (Eds.), *Handbook of stress: Theoretical and clinical aspects* (pp. 7–17). New York: Free Press.
Szabo, S. (1985). The creative and productive life of Hans Selye: A review of his major scientific discoveries. *Experientia, 41*, 564–567.

General Internist

► Primary Care Physicians
► Primary Care Providers

General Population

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

The general population is the entire population of individuals with a characteristic of interest, such as a particular disease or condition of clinical concern. It is differentiated from the subject sample chosen from that population for a particular study.

Description

The general population of interest in a particular case will differ from other general populations defined in other ways. For example, if a researcher is interested in testing a new behavioral intervention to lower blood pressure, the general population would be all individuals with high blood pressure (hypertension). In other cases, it may be all individuals with type 2 diabetes mellitus. It can also be defined by previous experiences, that is, individuals who have ever smoked, regardless of whether they currently smoke.

The reason for differentiating the term "general population" from the subject sample is that a subject sample chosen for a study is (virtually) always smaller than the general population. Since there are tens of millions of individuals in the United States with hypertension, for example, an intervention cannot be tested on all individuals in the general population of individuals with hypertension. A subject sample must be chosen. The key challenge here is one of generalizability. Interest does not actually lie with the treatment's therapeutic benefit for the particular individuals taking part in the study, but rather with how the treatment is likely to work for many more individuals comprising the general population. Therefore, great methodological care is required to ensure (to the greatest degree possible) that the subject sample is representative of the general population.

There are also statistical techniques that allow extrapolation of results from the subject sample to the general population. A useful parameter here is the confidence interval associated with a treatment effect, that is, the degree of therapeutic benefit offered by a treatment. Imagine that a behavioral intervention intended to lower blood pressure (let us just use systolic blood pressure in this example) is tested on 100 subjects, and that the average decrease in SBP is 8 millimeters of mercury (mmHg). The research question becomes: To what extent can this result be generalized to the general population on the basis of this one clinical study? Statistical methodology allows us to place a confidence interval around the treatment effect obtained, which is referred to as a point estimate, since it is an estimate of the "truth" in the general population based on the data collected here. Confidence intervals can be created for any percentage greater than zero and less than 100%, but a common and useful one is the 95% confidence interval. Imagine that this was done for the data from our hypothetical example, and the 95% confidence interval placed around the point estimate of 8 mmHg had a lower limit of 4 mmHg and an upper limit of 12 mmHg (in such cases, the limits will always lie symmetrically around the point estimate). These confidence intervals allow us to make the following statement with regard to the general population:

The data obtained from this single trial are compatible with a treatment effect in the general population as small as 4 mmHg and as large as 12 mmHg, and our best estimate is 8.00 mmHg.

In more formal statistical language, the 95% confidence interval (the interval from the lower limit to the upper limit) is a range of values that is likely to cover, with 95% confidence, the true but unknown general population treatment effect.

Cross-References

▶ Generalizability

General Practice

▶ Family Practice/Medicine

Generalizability

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

It is noted in the entry entitled "▶ Demographics" that when reporting a research study it is necessary to provide a summary of the relevant demographic characteristics of the subjects who

participated in the study. The fundamental goal of an individual research study is to provide information that allows a well-reasoned indication of how the general population would respond to the intervention of interest. If all subjects are younger than 30 years of age, it would be unreasonable to claim that the results of a study provided useful information regarding how individuals older than 60 years of age might respond.

Clinicians constantly face the challenge of assessing, on the basis of research evidence collected on subject samples participating in clinical research studies, how best to treat their individual patients. As a research scientist and a physician, Katz (2001) captured the issues here succinctly and eloquently:

> The inapplicability of some evidence to some patients is self-evident. Studies of prostate cancer are irrelevant to our female patients; studies of cervical cancer are irrelevant to our male patients. Yet beyond the obvious exclusions is a vast sea of gray. If our patient is older than, younger than, sicker than, healthier than, ethnically different from, taller, shorter, simply different from the subjects of a study, do the results pertain?

It is reasonable to acknowledge that the more closely the nature of a study's subject sample reflects the general population to whom one wishes to generalize the information gained from the study, the more likely it is that the evidence can indeed be generalized in a clinically informative manner. However, it must always be realized that the practice of medicine, including behavioral medicine, also requires the clinician to include knowledge of and reasoning about each individual patient. This is discussed further in the entry titled "► Clinical decision-making."

Cross-References

► Clinical Decision-Making

References and Readings

Katz, D. L. (2001). *Clinical epidemiology and evidence-based medicine: Fundamental principles of clinical reasoning and research*. Thousand Oaks, CA: Sage.

Genetic Consultation

► Genetic Counseling

Genetic Counseling

Bonnie S. LeRoy
Department of Genetics Cell Biology and Development, University of Minnesota, Minneapolis, MN, USA

Synonyms

Genetic consultation

Definition

"Genetic counseling is the process of helping people understand and adapt to the medical, psychological and familial implications of genetic contributions to disease. This process integrates the following:

Interpretation of family and medical histories to assess the chance of disease occurrence or recurrence.

Education about inheritance, testing, management, prevention, resources and research.

Counseling to promote informed choices and adaptation to the risk or condition" (Resta et al., 2006, p. 77).

This definition of genetic counseling was adopted by the National Society of Genetic Counselors in 2006, and it remains the standing definition of the service. The definition expresses the basic nature of the service which is grounded in the science of genetics as well as related sciences and the importance of competence in psychosocial counseling skills on the part of the practitioner. Genetic counseling as a formal, free-standing health-care profession in the United States began in 1969 with the development of the first graduate program at Sarah Lawrence College (Stern, 2009). The service, as an

integral part of the health-care field, is in various stages of formal development in many countries around the world (Transnational Alliance for Genetic Counseling, 2011).

Currently, genetic counseling services are provided to patients and families with genetic concerns that cover a broad range of medical conditions (Hampel et al., 2009). Genetic counseling has become an integral clinical service in the perinatal medicine, oncology, neurology, cardiology, pediatrics, public health, and many more health-care arenas (National Society of Genetic Counselors, 2011).

Assessing and addressing the psychosocial needs of patients and families are essential components of genetic counseling (Fine et al., 1996). The relationship between the counselor and the patient is critical to effective genetic counseling where the major goals of a better informed and empowered patient who is able to use genetic information to make important life and health-care decisions are met (McCarthy Veach et al., 2007).

Cross-References

- ▶ Cardiology
- ▶ Genetic Counselor
- ▶ Public Health

References and Readings

Fine, B. A., Baker, D. L., Fiddler, M. B., & ABGC Consensus Development Consortium (1996). Practice-based competencies for accreditation of and training in graduate programs in genetic counseling. *Journal of Genetic Counseling, 5*, 113–121.

Hampel, H., Grubs, R. E., Walton, C. S., Nguyen, E., Breidenback, D. H., Nettles, S. The American Board of Genetic Counseling 2008 Practice Analysis Advisory Committee including Callanan, N., Corliss, M., Fox, S., Hiraki, S., Ku, L., Neufeld-Kaiser, W., Riley, B., Taylor, J., & Weik, L. (2009). Genetic counseling practice analysis. *Journal of Genetic Counseling, 18*, 205–216.

McCarthy Veach, P., Bartels, D. M., & LeRoy, B. S. (2007). Coming full circle: A reciprocal-engagement model of genetic counseling practice. *Journal of Genetic Counseling, 16*, 713–728.

National Society of Genetic Counselors. (www.nsgc.org). Accesed 2011.

Resta, R., Bowles Biesecker, B., Bennett, R. L., Blum, S., Estabrooks Hahn, S., Strecker, M. N., et al. (2006). A new definition of genetic counseling: national society of genetic counselors' task force report. *Journal of Genetic Counseling, 15*, 77–83.

Stern, A. M. (2009). A quiet revolution: The birth of the genetic counselor at Sarah Lawrence College, 1969. *Journal of Genetic Counseling, 18*, 1–11.

Transnational Alliance for Genetic Counseling. http://tagc.med.sc.edu/index.asp. Accesed 2011.

Genetic Counselor

▶ Genetic Counseling

Genetic Material

▶ DNA

Genetic Polymorphisms

▶ Polymorphism

Genetic Testing, Psychological Implications

Julianne O'Daniel
Illumina, Inc, San Diego, CA, USA

Synonyms

Psychosocial implications

Definition

Genetic testing, psychological implications refers to the potential cognitive and emotional consequences of undergoing genetic testing and learning the test result.

Description

Psychological Impact of Genetic Testing

The potential psychological impact of genetic testing can be affected by numerous factors including the purpose of testing and the test result as well as the individual's expectations, perception of the disease in question, and coping style. In general, however, studies appear to indicate that although genetic testing may have an immediate negative effect for individuals receiving "bad news," the long-term psychological impact is often negligible or even slightly positive (Broadstock, Michie, & Marteau, 2000; Cameron & Muller, 2009).

Pretest counseling is an important step to minimize the potential for negative effects. This discussion should include a review of the purpose of testing, potential results, and medical consequences of results as well as patient expectations and plans for dealing with the results. Some genetic tests require an informed consent from the individual. This document may also help guide the pretest counseling discussion.

Purpose of Testing

The purpose of testing can have a significant impact on how the individual responds to the genetic result. Currently, genetic tests can be used for numerous scenarios such as:

- Identifying an underlying diagnosis
- Determining carrier status
- Predicting future disease (e.g., Huntington disease, BRCA 1/2)
- Guiding therapeutic management (e.g., pharmacogenetic testing)
- Estimating risk for common disease

Diagnostic testing in a symptomatic individual may bring a sense of relief or closure to know the genetic cause. In pediatric cases, determining the genetic cause may ease parent guilt about whether they did anything to cause the condition or raise parent guilt if the condition was inherited.

Predictive genetic testing is one of the most well-studied scenarios as the tested individual may live healthy and asymptomatic for many years with the knowledge of their molecular diagnosis and impending condition. Although the potential for negative psychological reactions and reduced quality of life is significant, research has generally demonstrated tested individuals cope well regardless of the test result (Fanos et al., 2011; Lammens et al., 2010; Mariotti et al., 2010). These studies may be positively biased, however, as those who choose not to be tested have been under studied.

Genetic testing to learn one's estimated lifetime risk for common disease utilizes knowledge gleaned from genetic association studies and should be considered within the context of other biometric and family history indicators of risk. Studies have not demonstrated a significant psychological impact from this type of testing (O'Daniel, Haga, & Willard, 2010).

Individual Perceptions, Expectations and Coping

An individual's prior perceptions of a disease or condition can significantly affect not only the information they expect to learn from a test but also what they believe the result means and how they respond to it. Preexisting perceptions may be informed by lived experiences especially in the case of a positive family history, by societal views and/or perceived stigma, and by a personal assessment of resources to deal with the condition. The relationship between these dynamic factors in regards to genetic testing has been described by several models including the Health Belief Model, Model of Stress and Coping, the Common Sense Model of self-regulation, and the Theory of Planned Behavior (Gooding, Organista, Burack, & Biesecker, 2006; Marteau & Weinman, 2006).

Cross-References

▶ Family Studies (Genetics)
▶ Gene

▶ Genetics
▶ Genome-Wide Association Study (GWAS)

References and Readings

Broadstock, M., Michie, S., & Marteau, T. (2000). Psychological consequences of predictive genetic testing: A systematic review. *European Journal of Human Genetics, 8*(10), 731–738.

Cameron, L. D., & Muller, C. (2009). Psychosocial aspects of genetic testing. *Current Opinion in Psychiatry, 22*(2), 218–223.

Elger, B. S. (2010). Ethical, legal, and social issues in the genetic testing of minors. In K. P. Tercyak (Ed.), *Handbook of genomics and the family: Psychosocial context for children and adolescents* (pp. 485–521). New York: Springer.

Fanos, J. H., Gronka, S., Wuu, J., Stanislaw, C., Andersen, P. M., & Benatar, M. (2011). Impact of presymptomatic genetic testing for familial amyotrophic lateral sclerosis. *Genetics in Medicine, 13*(4), 342–348.

Gooding, H. C., Organista, K., Burack, J., & Biesecker, B. B. (2006). Genetic susceptibility testing from a stress and coping perspective. *Social Science & Medicine, 62*(8), 1880–1890.

Hadley, D. W., Letocha Ersig, A. D., & Holohan Quattrocchi, M. K. (2010). Guidelines and policies on genetic testing in children and families. In K. P. Tercyak (Ed.), *Handbook of genomics and the family: Psychosocial context for children and adolescents* (pp. 523–557). New York: Springer.

Lammens, C. R., Aaronson, N. K., Wagner, A., Sijmons, R. H., Ausems, M. G., Vriends, A. H., et al. (2010). Genetic testing in Li-Fraumeni syndrome: Uptake and psychosocial consequences. *Journal of Clinical Oncology, 28*(18), 3008–3014.

Mariotti, C., Ferruta, A., Gellera, C., Nespolo, C., Fancellu, R., Genitrini, S., et al. (2010). Predictive genetic tests in neurodegenerative disorders: A methodological approach integrating psychological counseling for at-risk individuals and referring clinicians. *European Neurology, 64*(1), 33–41.

Marteau, T. M., & Weinman, J. (2006). Self-regulation and the behavioural response to DNA risk information: A theoretical analysis and framework for future research. *Social Science & Medicine, 62*(6), 1360–1368.

O'Daniel, J. M., Haga, S. B., & Willard, H. F. (2010). Considerations for the impact of personal genome information: A study of genomic profiling among genetics and genomics professionals. *Journal of Genetic Counseling, 19*(4), 387–401.

Genetics

Jeanette McCarthy[1] and J. Rick Turner[2]
[1]Community and Family Medicine, Duke University Medical Center, Durham, NC, USA
[2]Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

Genetics focuses on heredity and its biological basis (genes). It is the science that examines how traits are passed from one generation to the next.

Various subfields can be identified. Robinson (2010) provided a simple but useful classification system, including the following:

1. Classical, or Mendelian, genetics, which describes how traits, physical or psychological characteristics, are passed from one generation to the next. The name Mendelian refers to Gregor Mendel, who had conducted scientific studies of the inheritance of traits in plants as far back as the 1860s, even though the significance of his work was not appreciated and acknowledged until the early twentieth century (see Edelson, 1999). The name transmission genetics also conveys a similar meaning, focusing on how traits are transmitted from one generation to another.
2. Molecular genetics, which focuses on the physiochemical structure of DNA, ribonucleic acid (RNA), and proteins. Classical genetic studies, e.g., twin studies, can be conducted without any knowledge of molecular genetics: It is not necessary to know the biological basis of inheritance of traits to determine that there is a genetic influence in the inheritance of the trait. DNA is discussed in more detail shortly.
3. Population genetics, which looks at the genetic composition of large groups of individuals. It can be defined as the field studying the genetic diversity of a subset of a particular species. It searches for patterns that help

identify and discuss the genetic signature of a particular group. This includes behavioral components of the genetic signature. Population genetics also provides insights into how the collective genetic diversity of a population influences the health of the individuals within the population, providing a direct link to its importance in Health Psychology and Behavioral Medicine.

4. Quantitative genetics, which employs sophisticated mathematical and statistical models to examine the statistical relationships between genes and the traits they code for, or encode. Quantitative genetics is interested in estimating how much variation in a given trait is due to genetic inheritance and how much is due to the environment (and interactions between genes and environmental influences). Heritability is an assessment of how much influence is due to genetic makeup.

The general familiarity with the acronym DNA (deoxyribonucleic acid) is such that the definition is almost never provided when using the acronym. Every time we watch a contemporary detective show on television we are almost waiting to hear about the DNA evidence that will indicate or refute a suspect's guilt. Nonetheless, the field of genetics existed well before the structure of DNA was proposed and published in 1953 by Francis Crick and Jim Watson (Watson & Crick, 1953) and supportive evidence published in separate papers in the same issue of *Nature* by Rosalind Franklin and Maurice Wilkins (Franklin & Gosling, 1953; Wilkins, Stokes & Wilson, 1953). The word "proposed" is deliberately used since Watson and Crick used the following words at the start of their paper: "We wish to suggest a structure for the salt of deoxyribose nucleic acid (D.N.A.)." They concluded their paper with one of the most beautifully understated sentences in scientific literature: "It has not escaped our notice that the specific pairing we have postulated [specific base pairs bonding to each other – see the ▶ DNA entry] immediately suggests a possible copying mechanism for the genetic material."

It was several years before definitive evidence was collected and published. In more recent years, molecular genetic knowledge has proved extremely helpful in many fields within Medicine, Behavioral Medicine, and Pharmaceutical Medicine.

Cross-References

- ▶ DNA
- ▶ Gene
- ▶ Human Genome Project

References and Readings

Britannica. (2009) *The Britannica guide to genetics* (Introduction by Steve Jones). Philadelphia: Running Press Book.

de Geus, E. (2010). Quantitative genetics in behavioral medicine. In A. Steptoe (Ed.), *Handbook of behavioral medicine: Methods and applications* (pp. 399–422). New York: Springer.

Edelson, E. (1999). *Gregor Mendel and the roots of genetics*. New York: Oxford University Press.

Emde, R. N., & Hewitt, J. K. (Eds.). (2001). *Infancy to early childhood: Genetic and environmental influences on developmental change*. Oxford: Oxford University Press.

Franklin, R., & Gosling, R. G. (1953). Molecular configuration in sodium thymonucleate. *Nature, 171*, 740–741.

Manuck, S. B., & McCaffery, J. M. (2010). Genetics of stress: Gene-stress correlation and interaction. In A. Steptoe (Ed.), *Handbook of behavioral medicine: Methods and applications* (pp. 455–478). New York: Springer.

Markon, K. E. (2010). Psychological genetics: Understanding the nature of psychological differences through etiology. In K. P. Tercyak (Ed.), *Handbook of genomics and the family: Psychosocial context for children and adolescents* (pp. 33–55). New York: Springer.

Petrill, S. A., Plomin, R., DeFries, J. C., & Hewitt, J. K. (Eds.). (2003). *Nature, nurture, and the transition to early adolescence*. Oxford: Oxford University Press.

Plomin, R., DeFries, J. C., McClearn, G. E., & Rutter, M. (Eds.). (1997). *Behavioral Genetics* (3rd ed.). New York: W.H. Freeman and Company.

Robinson, T. R. (2010). *Genetics for dummies* (2nd ed.). Hoboken, NJ: Wiley.

Vimaleswaran, K. S., & Loos, R. J. F. (2010). Genetics of obesity and diabetes. In A. Steptoe (Ed.), *Handbook of behavioral medicine: Methods and applications* (pp. 499–521). New York: Springer.

Watson, J. D. (2006). *DNA: The secret of life*. New York: Alfred A. Knopf.

Watson, J. D., & Crick, F. H. C. (1953). A structure for deoxyribose nucleic acid. *Nature, 171*, 737–738.

Wilkins, M. H. F., Stokes, A. R., & Wilson, H. R. (1953). Molecular structure of deoxypentose nucleic acids. *Nature, 171*, 738–740.

Genital Blisters, Sores, or Lesions

▶ Genital Herpes

Genital Herpes

Deidre Pereira[1] and Timothy S. Sannes[2]
[1]Department of Clinical and Health Psychology, University of Florida, College of Public Health and Health Professions, Gainesville, FL, USA
[2]Department of Clinical and Health Psychology, College of Clinical Health and Health Professions, University of Florida, Gainesville, FL, USA

Synonyms

Genital blisters, sores, or lesions; Genital herpes infection; Herpes simplex virus (HSV) infection; HSV-1; HSV-2

Definition

Genital herpes is one of the most common sexually transmitted infections (STIs) in the United States. Genital herpes is caused by infection with herpes simplex virus 1 (HSV-1) and herpes simplex virus 2 (HSV-2), with the latter classification being the most common cause. About 1 out of every 6 individuals between the ages of 14 and 49 years old have genital herpes, many of whom are unaware that they are carrying the virus because they have not experienced any physical manifestations (Centers for Disease Control, 2011).

Transmission of genital herpes from one person to another occurs through direct contact with an infected cutaneous or mucosal area, which may include areas with active herpes lesions as well as areas in which herpes lesions are not visible. Once a first or "primary" exposure to HSV occurs, physical manifestations are typically evident within a week and may include painful and pruritic (itchy) lesions on or around the genital area, swollen inguinal lymph nodes, flu-like symptoms, and fever. These lesions typically heal within 2–4 weeks (National Institute of Allergy and Infectious Disease, 2011). Following this acute phase of infection, the virus becomes latent within the dorsal root ganglia of the spinal cord. An infected individual carries the virus for life; however, recurrent or "secondary" outbreaks are typically less severe and less frequent and primarily involve localized lesions.

An infected individual may reduce the risk of transmitting genital herpes to a sexual partner through proper condom use. However, it is important to note that infected individuals may "shed" virus before herpes lesions become visible or in between herpes outbreaks, meaning that sexual contact with proper condom use may still result in the transmission of genital herpes. As a result, individuals infected with HSV are recommended to abstain from sexual activity when symptoms of herpes are present (Centers for Disease Control).

Health care providers typically diagnose genital herpes by inspecting the infected area and taking a swab of the lesion to look for the presence of HSV. Additionally, blood samples may be collected to assess for the presence of HSV-1 or HSV-2 antibodies in the blood in between herpes outbreaks (U. S. Preventive Services Task Force, 2011).

Currently, there is no cure for genital herpes. However, outbreaks can be managed with antiviral medications. These antiviral medications are often effective for controlling the duration of an outbreak and the pain associated with herpes lesions. There is also some evidence that antiviral therapy may reduce the risk for genital herpes recurrence (National Institute of Allergy and Infectious Disease).

People with genital herpes may be at increased risk for contracting human immunodeficiency virus (HIV), the virus that causes AIDS, from an HIV-infected sexual partner. Furthermore, individuals who are immunosuppressed, including individuals living with HIV and those undergoing chemotherapy for cancer, may experience serious physical illness if infected with HSV, because their immune systems are less able to mount a proper immune response to an acute infection (National Institute of Allergy and Infectious Disease).

Finally, pregnant women with genital herpes may be at risk for transmitting HSV to a newborn during a vaginal delivery. Newborns may experience serious or fatal illness following exposure to HSV. As such, mothers with active genital herpes will usually deliver their babies via Cesarean section (U.S. Preventive Services Task Force).

References and Readings

Center for Disease Control and Prevention. (2011). *Genital herpes – CDC fact sheet*. Retrieved March 25, 2011, from http://www.cdc.gov/std/herpes/stdfact-herpes.htm

National Institute of Allergy and Infectious Disease, Department of Health and Human Services, National Institutes of Health. (2011). *Genital herpes*. Retrieved February 26, 2011, from http://www.niaid.nih.gov/topics/genitalHerpes/Pages/default.aspx

U.S. Preventive Services Task Force. (2011, March). *Screening for genital herpes: Recommendation statement* (AHRQ Publication No. 05-0573-A). Retrieved May 26, 2011, from http://www.uspreventiveservicestaskforce.org/uspstf05/herpes/herpesrs.htm

Genital Herpes Infection

▶ Genital Herpes

Genome-Wide Association Study (GWAS)

Matthew A. Simonson
Institute for Behavioural Genetics, Boulder, CO, USA

Synonyms

GWA study; Whole-genome association study (WGAS)

Definition

Genome-wide association studies are designed to identify points of common variation in DNA that are associated with particular traits, including diseases and responses to medication (Wang, Barratt, Clayton, & Todd, 2005). By examining the genetic variants associated with traits related to health and disease, it is hoped that a better understanding of the etiology of physical and mental disorders, as well as responses to treatment, will be gained (Carlson, Eberle, Kruglyak, & Nickerson, 2004).

Individual differences between people in traits, such as personality, eye color, and height, are all highly influenced by genetic variation (Yang et al., 2010). The development of rare medical conditions, such as hemophilia and muscular dystrophy, is also influenced by genetic variation, while the same is true for more common forms of illness, such as heart disease, cancer, and obesity (Iles, 2008). Understanding how our genetic architecture influences the development of disease is a very high priority for current medical science. One major ambition of the GWAS approach is leading to the development of better treatments that target illness with increased precision and reduced risks (Carlson et al., 2004).

DNA is a molecule that contains the genetic instructions that regulate cellular activity and ultimately plays a large part in the development of traits in living organisms (Watson & Crick, 1953). The order of nucleotide bases in an organism's DNA determines how genetic instructions are executed, through the direct coding of proteins or through regulatory functions. Genetic variation is caused by differences in DNA sequence between individuals; these variants are referred to as *alleles* (Keller, Howrigan, & Simonson, 2011). When a difference in DNA sequence occurs at a single base position, it is called a single-nucleotide polymorphism, or a SNP (den Dunnen & Antonarakis, 2000). Most of the time, SNPs have no biological effect; however, sometimes a single-nucleotide alteration can change the function of a gene, or the regulation of genes, and have an effect on cellular functioning (Wang & Moult, 2001).

Approximately 10 million common SNPs exist in the human genome (Gabriel et al., 2002). Recent research has demonstrated that the 10 million variants cluster into groups where the states of SNPs are correlated with each other

(haplotypes) (International HapMap C, 2003). By carefully sampling the most informative SNPs from these haplotypes, much of the information on common genetic variants can be ascertained. Using DNA microarrays, the allelic state of hundreds of thousands of highly informative SNPs can be determined rapidly and at a low cost (Oliphant, Barker, Stuelpnagel, & Chee, 2002).

In a genome-wide association study, the association between alleles (states of SNPs on a microarray) and a phenotype of interest is assessed. When a trait is dichotomous (affected or not), the genomes of two groups of people are compared. Subjects with some trait of interest (cases) are compared to people without this trait (controls). When a trait is continuous, such as height, associations between the state of SNPs and the degree of a continuous trait are examined. By examining which alleles are associated with the phenotype (or degree of phenotype), genetic differences between individuals can be identified (Hirschhorn & Daly, 2005). GWAS is a hypothesis-free method of analysis, in the sense that no prior candidate allele is investigated for association with a phenotype; instead, the entire genome is scanned for significant associations (Kitsios & Zintzaras, 2009).

Several factors can influence the validity of GWAS results and must be controlled for through methods of data cleaning. Some of these include:

Admixture/Ancestry. Spurious associations can arise when performing a GWAS on a sample composed of subjects from different ancestral populations. Part of the sample that shares common ancestry could have higher rates of the phenotype being investigated for nongenetic (e.g., cultural) reasons, resulting in alleles indicative of ancestry being associated with the trait in question rather than true risk alleles. By controlling for genetic ancestry, false associations can be avoided (Hirschhorn & Daly, 2005).

Data Artifacts. Due to the large number of SNPs examined on a microarray, technical artifacts are likely to occur at some SNPs and can result in false associations. Several methods of data cleaning and study design exist for detecting and controlling for the effects of technical artifacts that exist in SNP data (Williams & Haines, 2011).

Multiple Testing. The aim of multiple testing correction procedures used in a standard GWAS is to simultaneously maximize the likelihood of detecting a true association, if one exists, while at the same time minimizing false associations that are due to capitalizing on chance (Moskvina & Schmidt, 2008).

Given the standard alpha level of 0.05, one expects to get an average of 5 false associations for every 100 independent tests performed on data where no true association exists. For genome-wide association studies, this fact presents a major problem for two reasons. The first is due to the excess of Type I errors (false positives) that will occur due to the very large number of tests for association between SNPs and disease that are being performed. The second problem is that the simple correction method commonly used to remove multiple testing bias from repeated independent tests, the Bonferroni correction, leads to an excess of Type II errors (failed detection of true associations) when applied to GWAS data, because regions of the genome are not entirely independent. Accepted correction values based on replication of results and permutation procedures have been developed to overcome these problems (Johnson et al., 2010).

Overall, GWAS has been very successful at identifying regions associated with disorders and phenotypes. However, the majority of these associated variants have only small effect sizes individually in terms of risk contribution (Clarke & Cooper, 2010). With increased sample size, GWAS will likely provide further insight into the human genome. As sample sizes increase, the power to detect small effects increases. For example, a recent GWAS involving approximately 180,000 subjects was able to explain approximately 10% of the genetic variance in height while simultaneously identifying each significant locus that contributes to this estimate (Lango Allen et al., 2010).

Cross-References

- ▶ Admixture
- ▶ Allele
- ▶ DNA
- ▶ Phenotype

References and Readings

Bouchard, J. (2004). Genetic Influence on Human Psychological Traits A Survey. *Current Directions in Psychological Science, 13*(4), 148–151.

Carlson, C. S., Eberle, M. A., Kruglyak, L., & Nickerson, D. A. (2004). Mapping complex disease loci in whole-genome association studies. *Nature, 429*(6990), 446–452.

Clarke, A. J., & Cooper, D. N. (2010). GWAS: Heritability missing in action? *European Journal of Human Genetics, 18*(8), 859–861.

den Dunnen, J. T., & Antonarakis, S. E. (2000). Mutation nomenclature extensions and suggestions to describe complex mutations: a discussion. *Human Mutation, 15*(1), 7–12.

Gabriel, S. B., Schaffner, S. F., Nguyen, H., Moore, J. M., Roy, J., Blumenstiel, B., et al. (2002). The structure of haplotype blocks in the human genome. *Science, 296*(5576), 2225–2229.

Hirschhorn, J. N., & Daly, M. J. (2005). Genome-wide association studies for common diseases and complex traits. *Nature Reviews Genetics, 6*(2), 95–108.

Iles, M. M. (2008). What can genome-wide association studies tell us about the genetics of common disease? *PLoS Genetics, 4*(2), e33.

International HapMap C. (2003). The international hapmap project. *Nature, 426*(6968), 789–796.

Johnson, R. C., Nelson, G. W., Troyer, J. L., Lautenberger, J. A., Kessing, B. D., Winkler, C. A., et al. (2010). Accounting for multiple comparisons in a genome-wide association study (GWAS). *BMC Genomics, 11*, 724.

Keller, M. C., Howrigan, D. P., & Simonson, M. A. (2011). Theory and methods in evolutionary behavioral genetics. In D. M. Buss & P. H. Hawley (Eds.), *The evolution of personality and individual differences*. New York: Oxford University Press.

Kitsios, G. D., & Zintzaras, E. (2009). Genome-wide association studies: Hypothesis-"free" or "engaged"? *Translational Research, 154*(4), 161–164.

Lango Allen, H., Estrada, K., Lettre, G., Berndt, S. I., Weedon, M. N., Rivadeneira, F., et al. (2010). Hundreds of variants clustered in genomic loci and biological pathways affect human height. *Nature, 467*(7317), 832–838.

Moskvina, V., & Schmidt, K. M. (2008). On multiple-testing correction in genome-wide association studies. *Genetic Epidemiology, 32*(6), 567–573.

Oliphant, A., Barker, D.L., Stuelpnagel, J.R., Chee, M.S. (2002). BeadArray technology: Enabling an accurate, cost-effective approach to high-throughput genotyping. *Biotechniques* (Suppl. 56–58), 60–61

Wang, W. Y., Barratt, B. J., Clayton, D. G., & Todd, J. A. (2005). Genome-wide association studies: Theoretical and practical concerns. *Nature Reviews Genetics, 6*(2), 109–118.

Wang, Z., & Moult, J. (2001). SNPs, protein structure, and disease. *Human Mutation, 17*(4), 263–270.

Watson, J. D., & Crick, F. H. (1953). Molecular structure of nucleic acids; a structure for deoxyribose nucleic acid. *Nature, 171*(4356), 737–738.

Williams, S. M., & Haines, J. L. (2011). Correcting away the hidden heritability. *Annals of Human Genetics, 75*(3), 348–350.

Yang, J., Benyamin, B., McEvoy, B. P., Gordon, S., Henders, A. K., Nyholt, D. R., et al. (2010). Common SNPs explain a large proportion of the heritability for human height. *Nature Genetics, 42*(7), 565–569.

Genomics

Jeanette McCarthy[1] and J. Rick Turner[2]
[1]Community and Family Medicine, Duke University Medical Center, Durham, NC, USA
[2]Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

The discipline of genomics is described slightly differently by different authorities. Brown (2009) defines it as the use of high-throughput molecular biology technologies to study large numbers of genes and gene products all at once in whole cells, whole tissues, or whole organisms. The Britannica Guide to Genetics (2009) describes it as the study of the structure, function, and evolutionary comparison of whole genomes, where the genome refers to the complete genetic complement of an organism.

The discipline of genetics is itself a relatively young science and is concerned with how traits are passed from one generation to the next. Since the advent of genomics more recently, the term transmission genetics is commonly used to represent what had previously been simply called genetics. The mathematics of transmission genetics were first described by Mendel in 1866 (see Edelson, 1999), approximately 150 years ago. In contrast, while the word genome appeared relatively early in the twentieth century, genomics as a new form of experimental biology is a recent phenomenon. Raw biological information, such as the sequence of nucleotide base pairs in a DNA molecule, is itself complex, and the

discipline of bioinformatics is useful. The next step is to integrate all of this information and to address questions about what is happening in very complex systems when tens of thousands of different genes are interacting simultaneously (Brown, 2009). An understanding of genomes and genomic technologies builds upon the knowledge of transmission genetics (how hereditary information is transmitted from one generation to the next) and molecular biology (how genes function to control biochemical processes within the cell).

Cross-References

- ▶ Genetics
- ▶ Human Genome Project

References and Readings

Brown, S. M. (2009). *Essentials of medical genomics* (2nd ed.). Hoboken, NJ: Wiley-Blackwell.

Dale, J. W., von Schantz, M., & Plant, N. (2012). *From genes to genomes: Concepts and applications of DNA technology* (3rd ed.). Hoboken, NJ: Wiley-Blackwell.

Edelson, E. (1999). *Gregor Mendel and the roots of genetics*. New York: Oxford University Press.

Koehly, L. M., & McBride, C. M. (2010). Genomic risk information for common health conditions: Maximizing kinship-based health; promotion. In K. P. Tercyak (Ed.), *Handbook of genomics and the family: Psychosocial context for children and adolescents* (pp. 407–433). New York: Springer.

Miller, G. E., & Cole, S. W. (2010). Functional genomic approaches in behavioral medicine. In A. Steptoe (Ed.), *Handbook of behavioral medicine: Methods and applications* (pp. 443–453). New York: Springer.

Nolte, I. M., McCaffery, J. M., & Sneider, H. (2010). Candidate gene and genome-wide association studies in behavioral medicine. In A. Steptoe (Ed.), *Handbook of behavioral medicine: Methods and applications* (pp. 423–441). New York: Springer.

O'Neill, S. C. (2010). Public health genomics. In K. P. Tercyak (Ed.), *Handbook of genomics and the family: Psychosocial context for children and adolescents* (pp. 577–593). New York: Springer.

Primrose, S. B., & Twyman, R. M. (2004). *Genomics: Applications in human biology*. Hoboken, NJ: John Wiley & Sons.

Venter, J. C. (2007). *A life decoded: My genome, my life*. New York: Penguin.

Genotype

Laura Rodriguez-Murillo[1] and Rany M. Salem[2]
[1]Department of Psychiatry, Columbia University Medical Center, New York, NY, USA
[2]Broad Institute, Cambridge, MA, USA

Definition

The genotype of an individual refers to their specific genetic constitution. It can apply to the entire genetic constitution of an individual or to the combination of alleles at a specific locus.

All individuals carry two homologous copies of their genome, one copy inherited from their father and one copy from their mother. If the gene or position contains variation, the variants are called alleles. Alleles for each gene contain differences in their nucleotide sequence and may codify proteins with different properties. If the allele inherited from the mother is different from the allele inherited from the father, then the individual is termed heterozygous for that trait or gene. If these two alleles are identical, then that person is termed homozygous for that trait or gene. The genotype for one gene describes its allelic composition.

In diploid organisms, a maximum of two alleles are present for each gene or trait. For instance, in Huntington's disease, an affected individual's genotype consists of at least one allele of the gene that carries a mutation, i.e., a change in the DNA sequence, that leads to a defective protein, called Huntingtin (Walker, 2007). However, in general, more than two alleles can exist depending on the type of variation of interest.

Cross-References

- ▶ Allele
- ▶ DNA
- ▶ Gene
- ▶ Heterozygous
- ▶ Homozygous
- ▶ Locus

References and Readings

Lewis, R. (2005). *Human genetics. Concepts and applications* (7th ed.). Boston: McGraw-Hill Science/Engineering/Math.

Strachan, T., & Read, A. P. (2003). *Human molecular genetics* (3rd ed.). London/New York: Garland Science/Taylor & Francis Group.

Walker, F. O. (2007). Huntington's disease. *Seminars in Neurology, 27*(02), 143–150.

Geographic Information System (GIS) Technology

Phil Jones
School of Geography, Earth & Environmental Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Computer cartography; Spatial analysis

Definition

Geographic information systems (GIS) are a means of visualizing and analyzing spatial data, usually in the form of maps (Longley, Goodchild, Maguire, & Rhind, 2005).

Description

An early example of a GIS was John Snow's work mapping cases of cholera in nineteenth-century London, revealing outbreaks clustered around a contaminated water pump. Modern GIS can be used to analyze a range of material from scanned maps and aerial photographs to topographic models and boundary data. Different forms of quantitative and qualitative data can thus be connected to points and regions within geographic space. The powerful analytical tools offered by desktop systems such as ArcGIS and MapInfo are used by professionals in a wide variety of fields, including urban planning, resource management, epidemiology, surveying, and the military. Even at a quite simple level, the outputs of GIS analysis can be striking. An example might be a choropleth map, where different regions are shaded according to characteristics – such as the level of social deprivation – to reveal areas where these characteristics are concentrated. GIS treats datasets as *layers*, allowing them to be compared by placing one on top of another. Thus, clusters of patients suffering from heart disease in areas with high levels of social deprivation can be examined against public transport corridors to determine the accessibility of specialist treatment centers. In a more advanced vein, it is possible to test whether there is a statistically significant relationship between incidents of respiratory problems and people living downwind of an incinerator or to model different flooding scenarios and weigh the costs of improving flood defenses against likely property damage.

Increasingly, GIS has migrated onto the Web through services such as Yahoo! Maps, Open Street Map, and Google Earth, meaning that many people use GIS daily without even realizing it. While these services lack the advanced analytical capacity of proprietary desktop systems, many allow users to visualize their own data as overlays onto the basic maps provided. This might, for example, be tracks of jogging routes recorded by an individual using their own global positioning system (GPS) device or photos linked to the location where they were taken. As GPS is increasingly embedded in portable devices, especially smart phones, a number of lightweight mobile GIS applications are becoming available. These systems move beyond basic navigation functions to allow collection of field data on a mobile device, tagged to spatial location using GPS. This online mapping revolution enables many more people to use GIS, and researchers are increasingly interested in *crowdsourced* data (Crampton, 2009). Here, nonspecialists gather field data of different kinds and post them to a central database. This has a number of applications, for example, in disaster management, where it can be difficult for survey teams to cover

a sufficiently wide area to accurately determine where best to intervene and thus can benefit from local eyes-on-the-ground. This, of course, raises questions about the accuracy and reliability of the data gathered, but offers tremendously interesting possibilities for research and other applications.

References and Readings

Crampton, J. W. (2009). Cartography: Maps 2.0. *Progress in Human Geography, 33*(1), 91–100.
Longley, P., Goodchild, M., Maguire, D., & Rhind, D. (2005). *Geographic information systems and science*. Chichester: Wiley.
Wood, D., & Fels, J. (1993). *The power of maps*. London: Routledge.

Geriatric Depression Scale

Ivan Molton
Department of Rehabilitation Medicine, University of Washington, Seattle, WA, USA

Synonyms

GDS; GDS-15; GDS-4

Definition

The Geriatric Depression Scale (GDS) is a self-report instrument designed to assess depressive symptoms in older adults. The GDS was first developed as a 30-item measure by Jerome Yesavage and colleagues at Stanford University (Yesavage, Brink, Rose, & Lum, 1983), in response to concerns that available depression inventories contained many items that overlapped with common aging processes (including dementia, sleep disturbance, and gastrointestinal symptoms). The scale was therefore designed to avoid somatic symptoms (such as psychomotor retardation or pain) as well as questions that the authors believed would create defensiveness in older persons (such as those assessing sexual interest or suicidality). To simplify responding, the authors chose a yes/no scale for each item. In contrast to measures based around criteria for major depression taken from the Diagnostic and Statistical Manual of Mental Disorders, the GDS was not originally intended to be a diagnostic or screening measure, although it is frequently used as such in clinical settings. A 15-item short form of this measure and a 4/5 item ultra-short form have also been developed (the GDS-15 and GDS-4, respectively) (Mitchell, Bird, Rizzo, & Meader, 2010; Sheikh & Yesavage, 1986). Although subscales are sometimes used in research efforts, the scale is generally treated as unidimensional, with the total number of "yes" responses used as the outcome.

The GDS has generally performed well in psychometric and clinical testing. In a recent meta-analysis of 25 studies conducted in medical settings and nursing homes, Mitchell et al. (2010) reported overall sensitivity of the GDS-30 as 81.9% when compared to a structured clinical interview for major depression, with a specificity of 77.7%. Similar results were demonstrated in tests of the GDS-15 (sensitivity = 84.3%; specificity = 73.8%). Although only a handful of studies have examined the psychometric properties of the GSD-4/5, early results are promising, with reported sensitivity of 92.5% and specificity of 77.2% (Mitchell et al., 2010).

Despite the fact that the GDS is one of the only scales to be developed specifically to assess depression in older persons, most studies comparing its performance to "gold standard" measures (such as the Patient Health Questionnaire, the Center for Epidemiologic Studies Depression Scale, or the Beck Depression Inventory II) show it to be equally effective in terms of diagnosing major depression (Laprise & Vezina, 1998; Watson, Zimmerman, Cohen, & Dominik, 2009). However, there is some evidence that the GDS may outperform other standardized measures of depression in older adults for whom there is a significant overlay of medical symptoms (Low & Hubley, 2007; Thompson et al., 2011). It has also been suggested in the literature

that some clinicians prefer the GDS over other measures because they find specific questions to be more clinically relevant to their older patients.

The usefulness of the GDS in cognitive impaired older adults is equivocal. Several prospective studies comparing GDS scores to clinician ratings in older adults with cognitive impairment have demonstrated no (Laprise & Vezina, 1998) or very small differences (Burke, Miller, Rubin, Morris, & Berg, 1988) in sensitivity, reliability, and validity. Other studies have demonstrated that although the GDS is unaffected in those with mild cognitive impairment, it performs poorly in severely affected Alzheimer's patients (Debruyne et al., 2009).

The GDS has now been translated from English into a number of other languages, including Spanish, Korean, Japanese, Cantonese, Portuguese, and Arabic.

Cross-References

- ▶ Beck Depression Inventory (BDI)
- ▶ Depression: Measurement

References and Readings

Burke, W. J., Miller, J. P., Rubin, E., Morris, J. C., & Berg, L. (1988). Reliability of the Washington University clinical dementia rating (CDR). *Archives of Neurology, 45*, 31–32.

Debruyne, H., Van Buggenout, M., Le Bastard, N., Aries, M., Audenaert, K., De Deyn, P. P., et al. (2009). Is the geriatric depression scale a reliable screening tool for depressive symptoms in elderly patients with cognitive impairment? *International Journal of Geriatric Psychiatry, 24*, 556–562.

Laprise, R., & Vezina, J. (1998). Diagnostic performance of the geriatric depression scale and the beck depression inventory with nursing home residents. *Canadian Journal on Aging, 17*, 401–413.

Low, G. D., & Hubley, A. M. (2007). Screening for depression after cardiac events using the beck depression inventory-II and the geriatric depression scale. *Social Indicators Research, 82*, 527–548.

Mitchell, A. J., Bird, V., Rizzo, M., & Meader, N. (2010). Which version of the geriatric depression scale is most useful in medical settings and nursing homes? Diagnostic validity meta-analysis. *The American Journal of Geriatric Psychiatry, 18*, 1066–1077.

Sheikh, J. I., & Yesavage, J. A. (1986). Geriatric depression scale (GDS) recent evidence and development of a shorter version. In T. L. Brink (Ed.), *Clinical gerontology: A guide to assessment and intervention* (pp. 165–173). New York: Haworth Press.

Thompson, A., Liu, H., Hays, R. D., Katon, W. J., Rausch, R., Diaz, N., et al. (2011). Diagnostic accuracy and agreement across three depression assessment measures for Parkinson's disease. *Parkinsonism & Related Disorders, 17*, 40–45.

Watson, L. C., Zimmerman, S., Cohen, L. W., & Dominik, R. (2009). Practical depression screening in residential care/assisted living: Five methods compared with gold standard diagnoses. *The American Journal of Geriatric Psychiatry, 17*, 556–564.

Yesavage, J. A., Brink, T. L., Rose, T. L., & Lum, O. (1983). Development and validation of a geriatric depression screening scale: A preliminary report. *Journal of Psychiatric Research, 17*, 37–49.

Geriatric Medicine

Barbara Resnick
School of Nursing, University of Maryland, Baltimore, MD, USA

Synonyms

Care of older adults

Definition

By definition, geriatrics is a subspecialty of medicine that focuses on proving health care to older adults, generally considered to be those 55 years of age and older. The goal of geriatric care is to focus on health promotion and disease prevention and optimize quality of life versus length of life. Moreover, much of the focus is on use of behavioral interventions to accomplish these outcomes whenever possible.

Geriatric medicine is interdisciplinary, and to be provided at the highest level, it requires input from nursing, social work, physical therapy, occupation therapy, speech therapy, psychology, nutrition, and pharmacy, among others. The focus of care tends to be more syndrome than disease driven and addresses such things as

functional performance, falls, urinary incontinence, frailty, congestive heart failure, and dementia, among others.

Description

Given the central importance of prevention and disease/syndrome management in care of older adults, knowledge of behavioral medicine is critical to the training and practice of those in geriatric medicine. At the same time, the behavioral interventions and approaches used for those who provide care to older adults are different than those used with younger individuals. For example, Stage of Change focused interventions may not be as effective for smoking cessation among older adults compared to those who are younger and motivational interviewing may not be useful for those who are older with cognitive impairment.

The special knowledge and skill set of those who practice geriatric medicine is reflected by the ability to identify disease and the atypical presentation of disease among these individuals. For example, symptoms of an infection in older adults often are vague and nonspecific, and the only indication of a problem may be acute delirium or a fall. Pneumonia, for example, may or may not present with fever, but will often present as dehydration, confusion, or a fall.

The management of disease among older adults is also different and central to geriatric medicine. For example, older adults require specific attention to medications and are at particularly risk to complications from polypharmacy. Those with expertise in geriatric medicine need to decipher the need for and appropriate use of medications for multiple medical disorders and counsel older adults about the safety and efficacy of using over-the-counter medications and herbals. The challenges of frailty, complex comorbidity, different patterns of disease presentation, slower response to treatment, and requirements for social support call for special medical skills.

Those with a knowledge of geriatric medicine understand that the presentations of illness among older adults is often nonspecific and thus any presenting problem may be indicative of an acute medical problem. Geriatricians also address and in fact focus on clinical problems such as falls, immobility, incontinence, and confusion as well as adverse drug reactions. Geriatrics tend to manage a broad range of illnesses, acute and chronic, such as stroke, heart disease, infections, diabetes, delirium, and the dementias.

At its core, geriatric medicine requires comprehensive assessment of older adults. This involves working closely with other members of the interdisciplinary team such as nurses, therapists, pharmacists, dietitians, social workers, and many other health professionals. There is an increased focus and need for interdisciplinary teamwork as the number of older adults increases and those with expertise in geriatric medicine decrease.

Those that practice geriatric medicine do so out of a true dedication to care of older adults and consider it an honor to interact with these individuals. The American Geriatrics Society provides some insight about the wonderful benefit of this type of work on their webpage. Examples of comments made by those include the following: “Often, when you work with older people you stumble into a moment of drama when you’re listening to them tell a story. It’s fine to read history in books, but to talk with someone who’s lived it is precious. That’s one of the joys of geriatric practice;” and “Perhaps the most satisfying aspect of geriatrics for me is the opportunity to meet the people who are the history of our nation.”

United States medical school graduates (USMDs) are choosing specialties other than geriatrics due in part to compensation given for the years of additional training. This will have a major impact on the availability of physicians with expertise in care of older adults. It is anticipated that by 2050 there will be 1.6 geriatricians/10,000 people. To address the increased need for more individuals with knowledge of geriatric medicine, educational programs across all disciplines are attempting to integrate geriatric coursework into programs and

there is a trend toward interdisciplinary educational endeavors to further facilitate training in geriatric medicine.

Cross-References

► Gerontology

References and Readings

American Geriatrics Society. Accessed October, 2011, from http://www.americangeriatrics.org/
Geriatrics for Specialists. Accessed September, 2011, from http://specialists.americangeriatrics.org/
Institute of Medicine Report. *Retooling for an Aging America*. Accessed September, 2011, from http://www.iom.edu/Reports/2008/Retooling-for-an-Aging-America-Building-the-Health-Care-Workforce.aspx

Geriatrics

► Gerontology

Gerontology

Barbara Resnick
School of Nursing, University of Maryland, Baltimore, MD, USA

Synonyms

Geriatrics; Medical specialty

Definition

Gerontology stems from the Greek words "geron" which means "old man" and "logy" which means "study of." Gerontology is the study of the social, psychological, and biological aspects of aging. Gerontology is often confused with the term geriatrics, which addresses the medical aspects of aging with a focus on disease and disease management.

Gerontology is the study or management of aging-related issues and may include healthy aspects of aging as well as age-related disease. It also includes the study of aging within society and the impact that this has on society. Information learned through the work of gerontologists can be applied to policies and programs and to the development of systems and models of care within communities.

A critically important aspect of gerontology is the interdisciplinary aspect of this work. Gerontology includes and requires input from medicine, psychological, sociology, physiology, etc. The complexity of aging-related issues requires this interdisciplinary approach.

The field of gerontology has grown since the first early gerontologists back in the 1940s. Interdisciplinary gerontology organizations were developed such as the Gerontological Society of America in 1945 and the first academic research center devoted exclusively to the study of aging, the Ethel Percy Andrus Gerontology Center at the University of Southern California likewise was established around this time. Approximately 20 years later in 1967, the nation's first master's degree programs in gerontology were initiated with grants provided by the U.S. Administration on Aging. In 1975 the University of Southern California's Leonard Davis School of Gerontology became the country's first school of gerontology within a university and, shortly thereafter, offered the first Ph.D. degree in gerontology. Since that time, a number of other universities have formed departments or schools of gerontology or aging studies and established interdisciplinary gerontology PhD programs.

The increased interest in gerontology is in response to the rapidly growing number of older adults in the United States. Moreover, it is anticipated that the number of individuals over the age of 85 will increase from 5.3 million to 21 million by 2050 (http://www.answers.com/topic/gerontology#ixzz1bTu2NTaA, Aging Stats: http://www.agingstats.gov/agingstatsdotnet/Main_Site/Data/2008_Documents/Population.aspx). To

best respond and manage the physical and psychosocial health needs of this rapidly growing group of individuals, there is a critical need for more individuals to consider gerontology as a profession whether this be from a clinical perspective or research. The opportunities within gerontology are prevalent within academic settings, in service areas such as retirement housing, assisted living, exercise programs, health care, adult education, travel, and entertainment. Gerontology has helped expand our scientific knowledge base related to aging issues for the older adults and the aging community and the need for knowledge explosion in this area is great.

Cross-References

▶ Geriatric Medicine

References and Readings

Bengtson, V. L., & Schair, K. W. (1999). *Handbook of theories of aging*. New York: Springer Publishing.
Binstock, R. H., & George, L. K. (2001). *Handbook of aging and the social sciences*. San Diego, CA: Academic Press.
Birren, J. E. (1964). *The psychology of aging*. Englewood Cliffs, NJ: Prentice Hall.

Gestation

Pearl Ghaemmaghami and Ulrike Ehlert
Department of Psychology, University of Zurich, Binzmuehlestrasse, Zurich, Switzerland

Synonyms

Gravidity; Pregnancy

Definition

Gestation is the period of time during which the fetus develops in the uterus of the mother. It begins with conception and ends with birth.

The word originates from the Latin verb *gestare*, meaning to bear. When a mother is carrying more than one fetus, as is the case with twins or triplets, the term "multiple gestation" or "multifetal gestation" is used.

Description

Gestational age is the age of an embryo, fetus, or newborn measured from the time of conception. Knowing the gestational age is essential in monitoring fetal development and determining the treatment of complications during pregnancy.

Embryologists measure the beginning of gestation from ovulation. This typically occurs within 1 or 2 h before conception. However, because most women do not know the exact time of ovulation, it has become practice to calculate the beginning of gestation from the first day of the last menstrual cycle which normally takes place approximately 2 weeks prior to ovulation.

In the past, different methods of measuring the beginning of gestation led to confusion and inconsistent usage of terminology. For this reason, the Committee on Fetus and Newborn (COFN) of the American Academy of Pediatrics (AAP) suggested determining gestational age from the first day of the last menstrual cycle. This method has proven reliable in estimating a normal duration of gestation and adds up to approximately 40 weeks or 280 days. In cases of pregnancy achieved by assisted reproductive techniques (where the precise date of conception can be determined), the length of gestation adds up to 38 weeks or 266 days. In such cases, 2 weeks are added to the conceptional age in order to monitor fetal development and growth outcome in a standardized fashion and to be able to compare results across studies.

The gestational period can be clustered into three equal phases that are approximately of 3 calendar months duration or 13–14 weeks long. Depending on the method of calculation, the first trimester ranges from week 1 to the completion of week 13 (1–14, respectively), the second trimester from week 14 to week 26 (15–28), and the

third trimester extends from week 27 to week 39 (29–42). These three trimesters can be regarded as reference intervals in the progression of gestation and fetal development. When dealing with eventual obstetric problems, the more precise method of calculating gestational age uses completed weeks and days, for example, 28 + 2 weeks of gestation, for 28 completed weeks and 2 days.

The embryonic period is defined as beginning 3 weeks after ovulation and fertilization and extends to week 8 after fertilization or to week 10 after the last menstrual cycle. It is followed by the fetal period which ends at birth.

A Biopsychosocial Approach to Pregnancy

Pregnancy-Induced Alterations of the Endocrine and the Autonomic Nervous System (ANS)

Every female organ system goes through remarkable anatomical and functional adaptation during gestation. The fact that these drastic alterations return to the pre-pregnant state after delivery and lactation is equally fascinating.

The Maternal Hypothalamus-Pituitary-Adrenal (HPA) Axis

The maternal neuroendocrine system undergoes significant changes as well. The placenta becomes an additional source of hormone production. From week 8 to week 10 of gestation onward, it induces a drastic rise of maternal plasma corticotropin-releasing hormone (CRH) which leads to a 1,000-fold higher level than in the nonpregnant state. It is furthermore assumed that placental CRH stimulates the HPA axis and causes an increase in maternal adrenocorticotropin (ACTH) and cortisol levels. Therefore, pregnancy has also been described as a state of hypercortisolism.

In the nonpregnant state, cortisol regulates the HPA axis activity through a negative feedback mechanism. By contrast, during gestation cortisol has a stimulating effect on the CRH production in the placenta. However, the exponential rise of CRH does not lead to an overstimulation of the maternal HPA axis, because CRH is bound to the CRH-binding protein (CRH-BP) thereby reducing its bioactivity. While maternal plasma concentrations continue to rise with progressing gestation, approximately 30 days before birth CRH-BP levels suddenly drop by about 50%. The result is an abundance of free bioactive CRH and a cascade of reactions is triggered leading to the onset of labor and delivery. Findings have linked higher CRH levels in early gestation with a higher risk for preterm delivery. When a certain level of CRH is reached during pregnancy, the onset of parturition (i.e., the process of giving birth) is activated. It has therefore been suggested that CRH functions as a placental clock.

Although gestation induces profound changes of the maternal HPA axis, the circadian rhythm is nevertheless maintained during pregnancy.

After delivery, the placenta is expelled from the body and maternal cortisol levels return to normal. The state of hypercortisolism is followed by a temporary suppression of the CRH secretion from the hypothalamus.

The Interaction Between the Maternal HPA Axis, the Fetal HPA Axis, and the Placenta

In early gestation, a negative feedback mechanism regulates the HPA axis of the fetus. Later, however, placental CRH enters into the fetal circulation through the umbilical vein and begins stimulating the fetal HPA axis. In addition, maternal cortisol is to a certain degree additionally able to cross the placenta and stimulate the fetal HPA axis. One third of fetal cortisol levels are ascribed to maternal levels. Fetal cortisol finds its way through the umbilical arteries back into the placenta and stimulates the production of placental CRH. Thus, by the end of gestation a positive feedback loop is established. The resulting massive increase of cortisol in the fetus is necessary for the development and maturation of fetal organs, such as

the brain and the lungs. An overexposure to glucocorticoids can, however, have detrimental effects on the fetus.

The ANS During Gestation

Maternal blood volume begins to increase during the first trimester. At 32–34 weeks of gestation, it can reach levels of up to 45% above the pre-pregnant values. This expansion in blood volume, also called hypervolemia, results from an increase in plasma and red blood cell volume. It serves the functions of meeting the metabolic requirements of the enlarged uterus, providing enough nutrients to the placenta and fetus, enabling blood flow back to the heart when the mother is in a supine or erect position, and protecting her from excessive blood loss at delivery.

During gestation, the resting heart rate is increased by approximately 10 beats per minute and from week 5 there is an increase in cardiac output. The increase in cardiac output is caused by this heart rate acceleration and a decrease in systemic vascular resistance. The latter originates from a vasodilatory effect of progesterone and prostaglandins. Correspondingly, arterial blood pressure gradually falls. After reaching a nadir at 24–26 weeks, these levels begin to rise again until delivery.

In late gestation, the uterus pushes against the pelvic veins and the inferior vena cava aggravating venous return from the lower body. As a result, heart rate, cardiac output, and blood pressure are influenced by the body position.

Conflicting findings have been reported concerning epinephrine and norepinephrine that range from no changes to decreased or even increased levels during gestation. The diurnal rhythm of epinephrine and norepinephrine, however, seems to be preserved.

With regard to heart rate variability (HRV), the high frequency component reflecting the parasympathetic branch of the ANS seems to be decreased in comparison to nonpregnant women, while the low frequency component representing the sympathetic branch seems to show no differences. Findings indicate no alterations in the balance between the sympathetic and parasympathetic branches during gestation.

Stress Reactivity

Maternal stress and anxiety during gestation is related to dysregulated physiological stress systems as well as pregnancy complications. In order to better understand this association, it is essential to examine the acute maternal stress response of the HPA axis and the ANS under controllable circumstances. In studies using standardized laboratory stressors (the cold pressor test, the mental arithmetic test, or the Stroop color-word test) comparing nonpregnant and pregnant women, the physiological stress reactivity of pregnant women appears to be increasingly attenuated with progressing gestation. It has therefore been assumed that pregnant women become less sensitive to the impact of stress with advancing gestation. Similar results have been found in studies using the Trier Social Stress Test and the cortisol awakening response, the latter of which measures the stress response in a more natural but nevertheless standardized setting.

Emerging findings indicate that psychological factors can alter the stress response of the HPA axis and ANS during pregnancy, providing new insight on how maternal prenatal stress may affect the course of gestation, fetal development, and maternal and child well-being after birth.

Medical Complications During Gestation

Women suffering from obstetric complications often experience anxiety about their own well-being and that of their unborn child. These women can benefit greatly from psychological health treatment.

Following are a few common medical complications during gestation:

- Fetal growth restriction is defined as fetal weight that is at or below the 10th percentile

for gestational age. Intrauterine growth restriction (IUGR) is an often-used synonym.
- Gestational diabetes describes a form of diabetes (high blood sugar) that begins (or is diagnosed for the first time) during gestation. The high blood sugar usually returns to normal levels after delivery.
- Gestational hypertension occurs when the maternal blood pressure rises rather than falls during the second (or third) trimester of gestation. In some cases, it may be a sign of beginning preeclampsia.
- Preeclampsia is characterized by (a) an increased blood pressure, (b) edema, and (c) an excess of proteins in the urine. It can develop after the 20th week of gestation.
- The HELLP syndrome is thought to be a severe manifestation of preeclampsia. The acronym stands for hemolysis (H; breakdown of red blood cells), elevated liver enzymes (EL), and low platelet count (LP). It is a syndrome with a series of symptoms such as headaches, nausea and vomiting, abdominal pain, increased blood pressure, and visual problems.
- Hyperemesis gravidarum is severe, persistent, and uncontrollable nausea and vomiting during gestation that may lead to weight loss, electrolyte disturbance, and dehydration. This condition is not to be confused with morning sickness which occurs in many pregnant women.
- Spontaneous abortion (miscarriage) is defined as the involuntary loss of the fetus before 20 weeks of gestation. Over 80% of spontaneous abortions occur during the first 13 weeks of gestation. Many women do not realize that they are pregnant when this occurs. Both mother and father often experience feelings of bereavement and grief. Psychological interventions can help here as well.
- Preterm birth defines a birth before 37 weeks of gestation. Studies have associated pregnancy anxiety, prenatal depression, and chronic stress with preterm birth and low birth weight (which is defined as birth weight of 2,500 g or less).

Psychosocial and Behavioral Factors Influencing Gestation

Psychosocial and behavioral factors can have a strong influence on the course of gestation and have been associated with pregnancy complications and birth outcome. While the detrimental influence of poor health behavior such as smoking, alcohol and drug abuse, malnutrition, inadequate weight gain, and physical strain are well established, the negative impact of psychosocial stress has recently received increased attention.

Psychological Maladaptation During Gestation

Although pregnancy is ideally expected to be a happy experience in life, adapting to the physical, hormonal, psychological, social, and socioeconomic changes can be challenging. Gestation is, therefore, also a vulnerable phase that can be sufficiently stressful to cause psychological maladaptation. A Swedish study found that every fourth pregnant woman expressed considerable fear of childbirth. Other studies have reported no heightened or even a decreased risk of mental problems in pregnant women compared to nonpregnant women. All in all, the prevalence rates show inconsistent results depending on the country or region in which the study was conducted, the measuring instruments used, whether psychiatric disorders or psychological symptoms were examined, or how the investigated time period was defined (during pregnancy only, postpartum only, or both combined), among other factors. For example, a report of the World Health Organization (WHO) and the United Nations Population Fund (UNFPA) in 2008 states that the prevalence rates of mental disorders during gestation tend to be twice as high in low- and middle-income countries as in high-income countries.

Several studies have called the attention to maternal suicide as the leading cause of death during gestation and the postpartum period with

significant higher rates compared to nonpregnant and nonpuerperal women.

Despite the unresolved question whether prevalence rates of psychiatric disorders are higher during gestation or not, there is general consensus that the rates worldwide are high and that mental health problems during gestation appear to be greatly under-identified and untreated.

Examples of possible risk factors discussed in the literature for psychological maladaptation and psychiatric disorders during gestation are adolescent or unwanted pregnancy, an unsupportive marital or partner relationship, previous stillbirth, or repeated miscarriages. Also, women belonging to an ethnic minority seem to be at an increased risk of experiencing a depressive disorder during the prenatal or postpartum period. Further risk factors for psychological maladaptation include trauma, domestic violence, multiple stressful life events, chronic stress, poverty, and lack of financial resources.

Psychiatric disorders during gestation are associated with adverse birth outcome and poor maternal and infant mental health in the postpartum period.

Prevention of Psychiatric Disorders During Gestation

The fact that psychiatric disorders often remain unidentified and untreated indicates that much needs to be done in the field of prevention. A vital measure would be the correct and early identification of pregnant woman at risk and improving access to psychological health treatment. The WHO and the UNFPA suggest including prenatal mental health care in general antenatal care.

Some studies on the impact of partner support and social support have found a buffering effect of these factors on anxiety and depressive symptoms during gestation. Further psychosocial resources such as self-efficacy and the experience of daily uplifts seem to attenuate the psychological as well as physiological response to acute stress in pregnant women. In one study, a simple intervention instructing pregnant women to avoid stress and increase relaxation in everyday life caused a decrease in the perception of stress, negative affect, and morning cortisol levels.

However, broad systematic studies on the impact of psychological treatment still seem to be scarce, calling for further research in this field.

Strengthening coping strategies in the pregnant woman and enhancing resilience through behavioral medicine interventions may greatly reduce maternal stress and prevent related psychiatric disorders and protect the mother and infant from their detrimental effects.

Cross-References

- ▶ ACTH
- ▶ Autonomic Nervous System (ANS)
- ▶ Birth Weight
- ▶ Cardiac Output
- ▶ Circadian Rhythm
- ▶ Corticotropin-Releasing Hormone (CRH)
- ▶ Cortisol
- ▶ Epinephrine
- ▶ Heart Rate
- ▶ Heart Rate Variability
- ▶ Hypothalamic-Pituitary-Adrenal Axis
- ▶ In Vitro Fertilization
- ▶ Norepinephrine/Noradrenaline
- ▶ Postpartum Depression
- ▶ Pregnancy Outcomes: Psychosocial Aspect
- ▶ Stroop Color-Word Test
- ▶ Trier Social Stress Test

References and Readings

Andersson, L., Sundström-Poromaa, I., Bixo, M., Wulff, M., Bondestam, K., & Åström, M. (2003). Point prevalence of psychiatric disorders during the second trimester of pregnancy: A population-based study. *American Journal of Obstetrics and Gynecology, 189*, 148–154.

Cunningham, F. G., Leveno, K. J. L., Bloom, S. L., Hauth, J. C., Rouse, D. J., & Spong, C. Y. (2010). *Williams obstetrics* (23rd ed.). New York: McGraw-Hill.

De Weerth, C., & Buitelaar, J. K. (2005). Physiological stress reactivity in human pregnancy: A review. *Neuroscience and Biobehavioral Reviews, 29*, 295–312.

Dennis, C. L., Ross, L. E., & Grigoriadis, S. (2007). Psychosocial and psychological interventions for treating antenatal depression. *Cochrane Database of Systematic Reviews*(3). doi:10.1002/14651858.CD006309.pub2. CD006309.

Dunkel Schetter, C. (2011). Psychological science on pregnancy: Stress processes, biopsychosocial models, and emerging research issues. *Annual Review of Psychology, 62*, 531–558.

Engle, W. A., & American Academy of Pediatrics Committee on Fetus and Newborn. (2004). Age terminology during the perinatal period. *Pediatrics, 114*, 1362–1364.

Entringer, S., Buss, C., Shirtcliff, E. A., Cammack, A. L., Yim, I. S., Chicz-DeMet, A., et al. (2010). Attenuation of maternal psychophysiological stress responses and the maternal cortisol awakening response over the course of human pregnancy. *Stress, 13*(3), 258–268.

Klinkenberg, A. V., Nater, U. M., Nierop, A., Bratsikas, A., Zimmermann, R., & Ehlert, U. (2009). Heart rate variability changes in pregnant and non-pregnant women during standardized psychosocial stress. *Acta Obstetricia et Gynecologica Scandinavica, 88*, 77–82.

Mastorakos, G., & Ilias, I. (2003). Maternal and fetal hypothalamic-pituitary-adrenal axes during pregnancy and postpartum. *Annals of the New York Academy of Science, 997*, 136–149.

McLean, M., & Smith, R. (1999). Corticotropin-releasing hormone in human pregnancy and parturition. *Trends in Endocrinology and Metabolism, 10*, 174–178.

Metzler-Brody, S. (2011). New insights into perinatal depression: Pathogenesis and treatment during pregnancy and postpartum. *Dialogues in Clinical Neuroscience, 13*, 89–100.

Monk, C., Myers, M. M., Sloan, R. P., Ellman, L. M., & Fifer, W. P. (2003). Effects of women's stress-elicited physiological activity and chronic anxiety on fetal heart rate. *Journal of Developmental and Behavioral Pediatrics, 24*(1), 32–38.

Mulder, E. J. H., Robles de Medina, P. G., Huizink, A. C., Van den Bergh, B. R. H., Buitelaar, J. K., & Visser, G. H. A. (2002). Prenatal maternal stress: Effects on pregnancy and the (unborn) child. *Early Human Development, 70*, 3–14.

Nierop, A., Bratsikas, A., Klinkenberg, A., Nater, U. M., Zimmermann, R., & Ehlert, U. (2006). Prolonged salivary cortisol recovery in second-trimester pregnant women and attenuated salivary alpha-amylase responses to psychosocial stress in human pregnancy. *The Journal of Clinical Endocrinology and Metabolism, 91*, 1329–1335.

Nierop, A., Bratsikas, A., Zimmermann, R., & Ehlert, U. (2006). Are stress-induced cortisol changes during pregnancy associated with postpartum depressive symptoms? *Psychosomatic Medicine, 68*, 931–937.

Nierop, A., Wirtz, P. H., Bratsikas, A., Zimmermann, R., & Ehlert, U. (2008). Stress-buffering effects of psychosocial resources on physiological and psychological stress response in pregnant women. *Biological Psychology, 78*, 261–268.

Urizar, G. G., Jr., Milazzo, M., Le, H. N., Delucchi, K., Sotelo, R., & Munoz, R. F. (2004). Impact of stress reduction instructions on stress and cortisol levels during pregnancy. *Biological Psychology, 67*, 275–282.

Vesga-López, O., Blanco, C., Keyes, K., Olfson, M., Grant, B. F., & Hasin, D. S. (2008). Psychiatric disorders in pregnant and postpartum women in the United States. *Archives of General Psychiatry, 65*, 805–815.

World Health Organization and the United Nations Population Fund. (2008). *Maternal mental health and child health and development in low and middle income countries*. Geneva: Switzerland.

Gestational Carrier

▶ In Vitro Fertilization, Assisted Reproductive Technology

Ghrelin

Yoshiyuki Takimoto
Department of Stress Science and Psychosomatic Medicine, Graduate School of Medicine, The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Definition

Ghrelin is a 28-amino acid peptide that has the particularity to be octanoylated in the serine in position 3. Ghrelin is a hormone produced mainly by the endocrine X/A-like cells of the stomach submucosa that stimulates hunger. Ghrelin is an endogenous orexigenic peptide and considered the counterpart of the satiety hormone, for example, leptin. Plasma ghrelin is regulated by several factors. A key regulator of plasma ghrelin level is food intake. Plasma ghrelin levels increase under starvation and decrease after meals in response to an increase of glycemia. Ghrelin stimulates appetite by acting on the hypothalamic arcuate nucleus. Ghrelin activates the nucleus of tractus solitaries communicating with ARC through afferent vagal stimulus in the stomach. Moreover, ghrelin modulates gastric acid secretion and motility. Ghrelin

is also secreted in the hypothalamic arcuate nucleus, where it dose-dependently stimulates growth hormone secretion.

Cross-References

► Leptin

References and Readings

De Vriese, C., & Delporte, C. (2008). Ghrelin: A new peptide regulating growth hormone release and food intake. *The International Journal of Biochemistry & Cell Biology, 40*(8), 1420–1424.

Inui, A., Asakawa, A., Bowers, C. Y., Mantovani, G., Laviano, A., Meguid, M. M., et al. (2004). Ghrelin, appetite, and gastric motility: The emerging role of the stomach as an endocrine organ. *The FASEB Journal, 18*(3), 439–456.

Kojima, M., & Kangawa, K. (2010). Ghrelin: More than endogenous growth hormone secretagogue. *Annals of the New York Academy of Sciences, 1200*, 140–148.

Girth

► Waist Circumference (WC)

Glucocorticoids

Mustafa al'Absi
University of Minnesota Medical School, University of Minnesota, 235 School of Medicine, Duluth, MN, USA

Synonyms

Corticosteroids; Cortisol; Cortisone

Definition

Glucocorticoids are a group of hormones that share steroidal structure and affect every tissue in the body due to the wide presence of their receptors. As indicated by their name, glucocorticoids have a significant role in regulating the metabolism of glucose.

Description

Glucocorticoids are released from the adrenal cortex in response to a cascade of hormonal events initiated by the release of corticotrophin-releasing factor (CRF) from the paraventricular nucleus (PVN). CRF reaches the median eminence of the hypothalamus through the portal circulation where it then activates the release of adrenocorticotropic hormone (ACTH) into the systemic circulation from the corticotrope cells of the anterior pituitary. Upon reaching the cortical part of the adrenal gland, ACTH stimulates the synthesis and release of glucocorticoids – most notably cortisol – in humans.

Glucocorticoids are derived from a common precursor, cholesterol, formed from circulating lipoprotein. The formation of cortisol takes place in the intermediate zone of the adrenal cortex, an area called Zona fasciculata. About 95% of cortisol is usually bound to corticosteroid-binding globulin (CBG), and the remaining free cortisol enters cells to affect their metabolic activity. The liver is the main site for cortisol metabolism, and free cortisol is excreted in urine. The daily secretory rate of cortisol is 20–30 mg/24 h, and its highest level is obtained in the morning (10–20 μg/dl) and lowest level (2–5 μg/dl) is reached in the early evening hours.

Glucocorticoid Receptors. There are two types of receptors, mineralocorticoid receptor (MR) and glucocorticoid receptor (GR). MR has a high affinity for cortisol and is found primarily in the limbic system. When occupied, MR serves as the major receptor regulator of normal activity of the hypothalamic-pituitary-adrenocortical (HPA) axis. GR is more widespread and has a lower affinity for cortisol. GR becomes occupied when there are higher levels of circulating cortisol. When cortisol concentrations are high, such as during diurnal peaks or under conditions of stress, this receptor may be up to 60%

occupied. Because of the wide distribution and different levels of sensitivity of both types of receptors, a wide range of peripheral and central nervous system functions are mediated by their actions.

Effects of Glucocorticoids

Metabolic Effects. Glucocorticoids exert numerous peripheral effects that lead to changes in metabolic activities. For example, one of cortisol's main functions in the periphery is to make energy stores available for use throughout the body. This happens through multiple processes. Cortisol increases the expression of the enzymes that are responsible for a process known as gluconeogenesis, and this occurs primarily in the liver leading to the increased synthesis of glucose. Cortisol also decreases glucose uptake by cells in muscles and adipose tissues while also stimulating the release of fatty acids from adipose tissue leading to further increase in gluconeogenesis. The results are a net increase in plasma concentrations of glucose as well as amino acids.

Immune Effects. As a group of hormones, glucocorticoids are known to suppress the immune response and are used to suppress inflammation. These effects take place because glucocorticoids increase the expression of anti-inflammatory proteins, while they also suppress the expression of pro-inflammatory proteins. The increased levels of cortisol in response to stress have been seen as a mechanism to regulate changes in immune activity caused by stress. This is consistent with the hypothesis that stress-related glucocorticoids activity helps curtail the activity of endogenous cytokines and other stress-reactive immune functions. This action helps prevent the occurrence of harmful effects that may be produced by an unchecked immune response. Because of the immune suppression effects of glucocorticoids, they have been used in the treatment of various conditions that involve increased immune activity.

Central Effects. Glucocorticoids have a wide range of effects on central nervous system functions. These central effects are also implicated in the development of affective disorders. One of the primary functions of glucocorticoids is regulating their own release. For example, cortisol regulates its own secretion through its feedback effects on the pituitary, hippocampus, medial region of the frontal cortex, and central amygdala. Cortisol action includes modifying CRF expression leading to reduced release. Cortisol also decreases secretion of ACTH and pro-opiomelanocortin (POMC) from the pituitary. Because it influences gene expression of adrenergic receptors, it regulates effects of catecholamines.

There has been also evidence that cortisol affects cognitive functions, and studies that involved blocking glucocorticoids activity lead to impairment in the recall of emotionally relevant information. It has also been shown that fear learning associated with high cortisol levels leads to stronger consolidation of this memory and that memory performance materials not related to stress are reduced by glucocorticoids. Studies have also shown that formation of long-term memories occurs efficiently when glucocorticoid levels are mildly elevated. Higher levels or absence of glucocorticoids using adrenalectomy was associated with poor formation of long-term memories. Stress hormones have also been associated with reduced retrieval of stored information.

Research has demonstrated dysregulation in cortisol release usually characterized by high basal levels and low sensitivity to cortisol feedback in patients with depression. There is also evidence that antidepressants help restore the normal pattern of cortisol release. Patients with depression have greater ACTH and cortisol secretion, greater free cortisol in urine, and increased cortisol and CRF levels in cerebrospinal fluid compared with nondepressed controls. This dysregulation is also evidenced by greater incidence of escape from a test called dexamethasone suppression test, which test the feedback effects of cortisol on the brain.

Cardiovascular Effects. Glucocorticoids have various cardiovascular effects. For example,

cortisol may increase cardiac output by its effects in enhancing beta adrenergic sensitivity and increasing the synthesis of epinephrine. The latter effect is caused by cortisol's effect in stimulating phenylethanolamine-*N*-methyltransferase (PNMT), resulting in increased epinephrine synthesis, and by inhibiting the catabolic actions of catechol-*o*-methyltransferase (COMT) on catecholamines. Cortisol may act in the kidneys to increase the plasma volume by causing a fluid shift from intracellular to extracellular compartments increasing volume retention, and contributing to increased stroke volume and cardiac output. In addition, cortisol increases alpha adrenergic sensitivity contributing to increased vascular resistance and leading to increased blood pressure.

Cross-References

- ▶ ACTH
- ▶ Cortisol
- ▶ Hormones
- ▶ Metabolism
- ▶ Stress

References and Readings

al'Absi, M., & Arnett, D. K. (2000). Adrenocortical responses to psychological stress and risk for hypertension. *Biomedicine & Pharmacotherapy, 54*, 234–244.

Cahill, L., & McGaugh, J. L. (1998). Mechanisms of emotional arousal and lasting declarative memory. *Trends in Neuroscience, 21*, 294–299.

de Quervain, D., et al. (1998). Stress and glucocorticoids impair retrieval of long-term spatial memory. *Nature, 394*, 787–790.

Lupien, J. S., et al. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience, 10*, 434–445.

Glucometer

- ▶ Glucose Meters and Strips

Glucose

Michael James Coons
Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Synonyms

Glycemia; Hyperglycemia; Hypoglycemia

Definition

Glucose is a monosaccharide (i.e., simple sugar), which is an essential form of energy for cells and organs in the human body. Glucose is an important carbohydrate that is imperative for the survival of organisms.

Description

Glucose is the usable form of energy in humans. Following ingestion, carbohydrates in the stomach are broken and converted to glucose. Glucose then passes through the stomach and enters the bloodstream. Glucose metabolism triggers insulin secretion from the ß-cells in the pancreas, which are responsible for endogenous insulin production. Insulin circulating in the bloodstream binds to receptors facilitating the uptake of glucose into the red blood cells. Glucose can then be used for energy, or converted to glycogen for storage in the liver, muscles, and fat. Glucose metabolism is a homeostatic mechanism that is essential for human survival.

Blood glucose is measured on a continuum. The normal range of blood glucose is between 4.0 and 7.0 mmol/L, or 82–110 mg/dL. When concentrations fall below the lower bound, it is considered to be a state of hypoglycemia (i.e., low blood glucose). When concentrations surpass the upper bound of this range, it is considered to be

a state of hyperglycemia (i.e., high blood glucose). Among healthy individuals, actions of the pancreas maintain blood glucose homeostasis. When the availability of metabolized carbohydrates is low, resulting in a decline in plasma glucose concentration, the α-cells of the pancreas secrete glucagon. Glucagon is a hormone that facilitates the conversion of glycogen in the liver, muscles, and fat to glucose that is released into the bloodstream to restore euglycemia (i.e., normal blood glucose concentration). In the presence of excess circulating blood glucose, resulting in a state of hyperglycemia, insulin is secreted from the ß-cells of the pancreas to facilitate glucose uptake and conversion to glycogen for storage. Disruption of this metabolic process is a hallmark feature of diabetes (i.e., type 1 diabetes mellitus, type 2 diabetes mellitus, gestational diabetes mellitus).

Cross-References

- ▶ Glycemia: Control, Load-High
- ▶ Hyperglycemia

References and Readings

Fisher, S. J., & Kahn, C. R. (2002). Physiologic mechanisms in homeostatic control of glucose. In G. M. Besser & M. O. Thorner (Eds.), *Comprehensive clinical endocrinology* (pp. 239–254). London: Elsevier Science.

Reaven, G. M. (2002). Insulin resistance. In G. M. Besser & M. O. Thorner (Eds.), *Comprehensive clinical endocrinology* (pp. 291–302). London: Elsevier Science.

Glucose Meters and Strips

Janine Sanchez
Department of Pediatrics, University of Miami, Miami, FL, USA

Synonyms

Glucometer; Point of care testing; Self-monitoring of blood glucose

Definition

Glucose meter (glucometer) is a medical device used to determine the approximate level of glucose in the blood. It is used for monitoring at home and in the hospital. Patients obtain a small drop of blood using a fingertip lanceting device. Blood is placed on a disposable test strip, and in less than 20 s on average, a reading is given. Most meters are approximately the size of the palm of the hand. Some meters require the user to manually enter in a code specific to the test strip. If the code does not match the strip, then the glucose reading is inaccurate. The glucose value is displayed in mg/dl (USA) or mmol/l.

Meters have different features such as memory, calculation of average sugar, volume of blood sample required, back light, color, and size. Some meters allow manual entry of additional data, such as insulin dose, amounts of carbohydrates eaten, or exercise. Some link with insulin pumps and send the sugar reading directly to the pump. Information such as all readings for 3 months, average sugar at different times of the day, and percentage of high or low readings may be displayed. The software is available for doctor's offices and patients' home use.

This information is used to adjust the medical regimen (primarily insulin) as well as to monitor adherence. The goal is to obtain tight glucose control and prevent complications of abnormal glucose levels. Patients with diabetes are educated on how to maintain glucose levels within target limits using information from blood glucose measurement.

Cross-References

- ▶ Education, Patient
- ▶ Patient Care
- ▶ Patient-Reported Outcome

References and Readings

Garg, S., & Hirsch, I. B. (2010). Self-monitoring of blood glucose. *International Journal of Clinical Practice. Supplement, 2010*(166), 1–10.

Giampietro, M. E. (2011). Point-of-care testing in diabetes care. *Mini Reviews in Medicinal Chemistry, 11*(2), 178–184. 2011 Jan 11 (Epub ahead of print).
Mehta, S. N., & Wolfsdorf, J. I. (2010). Contemporary management of patients with Type 1 diabetes. *Endocrinology and Metabolism Clinics of North America, 39*(3), 573–593.

Glucose Test

► Oral Glucose Tolerance Test (OGTT)

Glucose: Levels, Control, Intolerance, and Metabolism

Michael James Coons
Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Synonyms

Glycemia; Hyperglycemia

Definition

Glucose is a monosaccharide (i.e., simple sugar), which is an essential form of energy for cells and organs in the human body. Glucose is an important carbohydrate that is imperative for the survival of organisms. A homeostatic process regulates the glucose available in the bloodstream that is used by the cells and organs of the body.

Description

Glucose is the usable form of energy for the central nervous system in humans. Following ingestion (within 3–4 h), carbohydrates in the stomach are broken and converted to glucose. Glucose then passes through the stomach and gastrointestinal tract into the bloodstream. Glucose metabolism triggers insulin secretion from the beta cells of the pancreas, which are responsible for endogenous insulin production. Insulin circulating in the bloodstream binds to receptors facilitating the uptake of glucose into the red blood cells. Glucose can then be used for energy or converted to glycogen for storage in the liver, muscles, and fat. Glucose metabolism is a homeostatic mechanism that is essential for human survival and is influenced by the actions of a variety of hormones, enzymes, and substrates.

Blood Glucose Homeostasis

Blood glucose is measured on a continuum. The normal range of blood glucose is between 4.0 and 7.0 mmol/L, or 70 and 120 mg/dL. When concentrations fall below the lower bound, it is considered to be a state of hypoglycemia (i.e., low blood glucose). Clinically, mild symptoms include trembling, heart palpitations, sweating, anxiety, hunger, nausea, and tingling, whereas severe symptoms include impaired concentration, fatigue, confusion, weakness, vision changes, difficulty speaking, dizziness, or loss of consciousness. When concentrations surpass the upper bound of this range, it is considered to be a state of hyperglycemia (i.e., high blood glucose). Clinically, mild symptoms include excessive thirst, excessive urination, fatigue, itchy skin, and, over time, weight loss. However, extreme states of hyperglycemia can trigger a state of ketoacidosis, during which individuals lose excessive amounts of electrolytes through urine and sweat that can trigger a myocardial infarction if untreated.

Among healthy individuals, actions of the pancreas maintain blood glucose homeostasis. When the availability of metabolized carbohydrates is low, resulting in a decline in plasma glucose concentration, the alpha cells of the pancreas secrete glucagon. Glucagon is a hormone that facilitates the conversation of glycogen that is stored in the liver, muscles, and fat, to glucose, which is then released into the bloodstream to restore euglycemia (i.e., normal blood glucose concentration). In the presence of excess

circulating blood glucose, resulting in a state of hyperglycemia, insulin is secreted from the beta cells of the pancreas to facilitate glucose uptake and conversion to glycogen for storage. Disruption of this metabolic homeostatic process is a hallmark feature of diabetes (i.e., type 1 diabetes mellitus, type 2 diabetes mellitus, gestational diabetes mellitus).

Glucose Intolerance and Diabetes Mellitus

In the majority of cases of type 1 diabetes mellitus (T1DM), an autoimmune process destroys the pancreatic beta cells resulting in insufficient insulin production. With both type 2 diabetes mellitus (T2DM) and gestational diabetes mellitus (GDM), the cellular receptors become resistant to the endogenous insulin produced by the pancreas. Prior to the onset of T2DM or GDM, individuals enter a prediabetic state that is characterized by impaired glucose tolerance (IGT), which is an intermediate step in disordered blood glucose metabolism. In such cases, the cellular receptors become resistant to the insulin produced by the pancreas, resulting in excess glucose circulating in the bloodstream. IGT is assessed using the 2-h oral glucose tolerance test (OGTT). During this procedure, individuals are orally administered 75 g of a glucose solution. Over the subsequent 2 hours, their plasma blood glucose is assessed. IGT is characterized by a 2-h OGTT between 7.8 and 11.1 mmol/L or 140 and 199 mg/dL. If untreated by adaptive health behavior change (e.g., nutritional therapy, increased physical activity) or pharmacotherapy, individuals with IGT will typically progress to a diabetic state. Although the pathophysiology and underlying mechanisms are different for the various diabetes subtypes, all forms of the disease result in chronic hyperglycemia if untreated.

Blood Glucose Management

Among individuals with diabetes, blood glucose control is influenced by four health behaviors including dietary intake, physical activity, medication adherence, and self-monitoring of blood glucose. Specifically, individuals must reduce their carbohydrate intake, frequently participate in moderate physical activity (i.e., 150 min per week, with no more than two consecutive days of inactivity), adhere to their medication regimen (of oral medications that aid in blood glucose metabolism or of exogenous insulin to overcome their beta cell deficiency), and regularly self-monitor their blood glucose before and after meals (particularly in the context of insulin therapy) to inform their future decisions to maintain normal blood glucose levels. Failure to maintain blood glucose in the normal range increases the risk for developing serious short-term and long-term complications associated with this disease process.

A1C is considered to be the "gold standard" measure of blood glucose control. It provides an index of the mean blood glucose levels over the previous 90–120 days. A1C is assessed through a blood sample that is analyzed in the laboratory. Over the 120-day life span of red blood cells, glucose molecules bind to hemoglobin in the red blood cells to form glycated hemoglobin. A concentration of glycated hemoglobin in the red blood cells reflects the average level of glucose that the red blood cells have been exposed to during its life span. This glycated hemoglobin is then expressed as a percentage. Optimal A1C is considered to be $\leq$6%. Higher A1C is indicative of worse glycemic control. Failure to maintain A1C $\leq$6% has been shown to increase the risk of developing serious vascular pathology and premature mortality. Specifically, poor blood glucose control (A1C $\geq$6%) can lead to blindness, renal failure, pain and loss of sensation in the extremities, myocardial infarctions, cerebrovascular accidents, and amputations. Following the assessment of individuals' A1C, these data are then used to inform clinical decision-making regarding pharmacotherapy and self-management practices.

Blood glucose metabolism is a complex homeostatic process. Various states of metabolic dysregulation can occur that require intensive pharmacological and behavioral intervention. Failure to achieve and maintain normal blood glucose levels can result in short-term and long-term complications and potentiate serious morbidity and premature mortality.

Cross-References

- ▶ Diabetes
- ▶ Glycemia
- ▶ Hyperglycemia
- ▶ Hypoglycemia

References and Readings

Barrett, E. J., & Nadler, J. L. (2002). Non-insulin dependent diabetes mellitus. In G. M. Besser & M. O. Thorner (Eds.), *Comprehensive clinical endocrinology* (pp. 303–318). London: Elsevier Science.

Booth, G. L. (2001). Short-term clinical consequences of diabetes in adults. In H. L. Gerstein & R. B. Haynes (Eds.), *Evidence-based diabetes care* (pp. 68–106). Hamilton, ON: BC Decker.

Capes, S., & Anand, S. (2001). What is type 2 diabetes? In H. L. Gerstein & R. B. Haynes (Eds.), *Evidence-based diabetes care* (pp. 151–163). Hamilton, ON: BC Decker.

Fisher, S. J., & Kahn, C. R. (2002). Physiologic mechanisms in homeostatic control of glucose. In G. M. Besser & M. O. Thorner (Eds.), *Comprehensive clinical endocrinology* (pp. 239–254). London, UK: Elsevier Science.

Gagel, R. F. (2002). Hypoglycemia and insulinomas. In G. M. Besser & M. O. Thorner (Eds.), *Comprehensive clinical endocrinology* (pp. 255–266). London, UK: Elsevier Science.

Lawson, M. L., & Muirhead, S. E. (2001). What is type 1 diabetes? In H. L. Gerstein & R. B. Haynes (Eds.), *Evidence-based diabetes care* (pp. 124–150). Hamilton, ON: BC Decker.

Mahon, J., & Dupre, J. (2001). Early detection and prevention of diabetes mellitus. In H. L. Gerstein & R. B. Haynes (Eds.), *Evidence-based diabetes care* (pp. 184–206). Hamilton, ON: BC Decker.

Nadler, J. L., McDuffie, M., & Kirk, S. E. (2002). Insulin-dependent diabetes mellitus. In G. M. Besser & M. O. Thorner (Eds.), *Comprehensive clinical endocrinology* (pp. 267–290). London, UK: Elsevier Science.

Reaven, G. M. (2002). Insulin resistance. In G. M. Besser & M. O. Thorner (Eds.), *Comprehensive clinical endocrinology* (pp. 291–302). London, UK: Elsevier Science.

Ryan, E. A. (2001). What is gestational diabetes? In H. L. Gerstein & R. B. Haynes (Eds.), *Evidence-based diabetes care* (pp. 164–183). Hamilton, ON: BC Decker.

Yale, J. F. (2001). Hypoglycemia. In H. L. Gerstein & R. B. Haynes (Eds.), *Evidence-based diabetes care* (pp. 380–395). Hamilton, ON: BC Decker.

Glycated Hemoglobin

- ▶ Glycosylated Hemoglobin
- ▶ HbA1c

Glycemia

- ▶ Glucose
- ▶ Glucose: Levels, Control, Intolerance, and Metabolism
- ▶ Hyperglycemia

Glycemia: Control, Load-High

Michael James Coons
Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Synonyms

Glucose; Hyperglycemia

Definition

Glycemia refers to the concentration of glucose circulating in the blood plasma. Glycemia is influenced by a metabolic homeostatic process. As carbohydrates are ingested, they are broken down and converted to glucose, which then enter the bloodstream. Glycemia is measured on a continuum. The normal range of blood glucose is between 4.0 and 7.0 mmol/L, or 82 and 110 mg/dL. Glycemia can be assessed in the laboratory using antecubital venipuncture, either in a fasting or nonfasting state. Among patients with diabetes, glycemia may also be assessed by using portable glucometer. With such devices, individuals lance their fingertip to acquire a droplet of blood from the capillaries in

the fingers. The blood droplet is placed on a test strip that is inserted into the device for analysis. These meters provide cross-sectional data on the state of glycemia at a single point in time. However, these devices do not provide data on glycemic variability. For individuals with diabetes receiving insulin therapy, recent technological advances have resulted in the development of continuous glucose monitoring systems (CGMS) that provide a virtually continuous assessment of glycemia. These devices are worn on the abdomen and wirelessly transmit to the receiver (a pager-like device worn on the belt). The abdominal sensors sample the interstitial fluid at a rate of once every 5 s and provide an average blood glucose concentration over a period of several minutes. Although data from CGMS are less accurate than plasma blood glucose, these devices provide information on blood glucose variability that would otherwise be unavailable outside of the laboratory setting. For the surveillance of diabetes self-management outcomes, individuals can provide a blood sample for A1C analysis. A1C is collected by a venipuncture in the medical clinic. It provides an index of the mean level of glycemia over the previous 90–120 days. Optimal A1C is considered to be $\leq$7%. Higher A1C is indicative of worse glycemic control and places individuals at risk of serious long-term vascular complications. A1C is considered to be the "gold standard" measure of achieved blood glucose control among patients with diabetes.

Cross-References

- ▶ Diabetes
- ▶ Glucose
- ▶ Hyperglycemia

References and Readings

American Diabetes Association. (2011). Standards of medical care in diabetes: 2011. *Diabetes Care, 34* (Suppl. 1), S11–S51.

Piette, J. D., & Glasgow, R. E. (2001). Education and home glucose monitoring. In H. C. Gerstein & R. B. Haynes (Eds.), *Evidence-based diabetes care* (pp. 207–251). Hamilton, ON: B.C. Decker.

Saudek, C. D., Derr, R. L., & Kalyani, L. L. (2006). Assessing glycemia in diabetes using self-monitoring blood glucose and hemoglobin A1C. *Journal of the American Medical Association, 295*, 1688–1697.

Glycemic Index

▶ Low Glycemic Index

Glycosylated Hemoglobin

Luigi Meneghini
Diabetes Research Institute, University of Miami, Miami, FL, USA

Synonyms

A1C; Glycated hemoglobin; Glycosylated hemoglobin; Hemoglobin A1c

Definition

HbA1c, or glycosylated hemoglobin, is a measure of how much glucose is irreversibly bound (glycated) to hemoglobin, and can be used to assess the degree of exposure to glycemia in the preceding 2–3 months (corresponding to the life span of the red blood cell where hemoglobin is contained). In a person with normal blood glucose levels, the amount of glycated hemoglobin is around 4–6%, representing an average blood glucose level between 70 and 120 mg/dl. In individuals with diabetes, HbA1c can be measured every 3 months with a goal of keeping the value as close to normal as possible, or at least under 7% in most patients. The higher the HbA1c, the greater the risk over time (usually measured in years) of developing microvascular complications, such as diabetic retinopathy, nephropathy, and neuropathy. HbA1c remains the best predictor for future diabetes-related chronic complications that is available in the clinical setting.

Cross-References

- ▶ Diabetes
- ▶ HbA1c
- ▶ Hyperglycemia

References and Readings

Joslin, E. P., & Kahn, C. R. (2005). *Joslin's diabetes mellitus* (14th ed.). Philadelphia: Lippincott Williams & Willkins.

Goals

- ▶ Meaning (Purpose)

Gonadal Female Hormones

- ▶ Estrogen

Goodness of Fit Hypothesis

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

This term refers to the effectiveness of matching (fitting) a coping strategy to a situation's level of controllability, in relation to adaptation to stress. This matching was proposed by Lazarus and Folkman (1984) and Forsythe and Compass (1987) and is termed the goodness of fit hypothesis (GOFH). According to the GOFH, emotion-focused coping (EFC) is more adaptive for uncontrollable and unsolvable situations, while problem-focused coping (PFC) is more adaptive in controllable and solvable situations. EFC includes denial, distraction, relaxation, or, in negative forms, catastrophizing. In contrast, PFC includes defining the problem, suggesting solutions, and implementing one. The GOFH is a major issue in the field of stress and coping and reveals the complexity of the person-situation fit, in relation to adequate responses to stress.

The GOFH has important implications for behavior medicine as well. For example, Levine et al. (1987) assessed levels of denial (an EFC) in cardiac patients and examined its relation with recovery indices during hospitalization (acute phase) and over 12 months after discharge (long term). Levine et al. found that while greater denial predicted better short-term prognosis in hospital, it predicted poorer prognosis in the long-term. These results can be seen as supporting the GOFH since during the acute phase of hospitalization, cardiac patients have less control over their situation, and hence, the ability to deny may have reduced stress-related excessive sympathetic responses, which may have reduced the risk of further cardiac events. In contrast, during recovery at home, when modifying one's lifestyle is under a patient's control, denial would impede such efforts and, thus, probably contribute to adverse health outcomes. Another more recent example is the study by Rapoport-Hubschman, Gidron, Reicher-Atir, Sapir, and Fisch (2009) that tested the relationship between coping and outcomes in in-vitro fertilization (IVF). This form of medical treatment "bypasses" women's hormonal system and, thus, constitutes a strong example of reduced control by the patient over the procedure and outcome. In their study, women with higher baseline levels of "letting go" (i.e., high EFC) had a significantly higher chance of being pregnant than those with lower levels of "letting go" coping, independent of confounders (age, number of IVF cycles, and cause of infertility). While not all studies have supported the GOFH, it has received quite a bit support, and it has important implications for teaching stress management. Specifically, people can learn to first appraise whether a situation is controllable or solvable or not and then choose to use EFC or PFC. Importantly, Forsythe and Compass (1987) also found that people with both types of coping adapt the best,

suggesting that people need to learn both EFC and PFC, and know when to use or perhaps combine both forms of coping strategies. The GOFH is an important concept and framework in stress, health, and illness.

Cross-References

- ▶ Coping Strategies
- ▶ Perceived Control

References and Readings

Forsythe, C. J., & Compass, B. E. (1987). Interaction of cognitive appraisals of stressful events and coping: Testing the goodness of fit hypothesis. *Cognitive Therapy and Research, 11*, 473–485.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.

Levine, J., Warrenburg, S., Kerns, R., Schwartz, G., Delaney, R., Fontana, A., et al. (1987). The role of denial in recovery from coronary heart disease. *Psychosom Medicine, 49*, 109–117.

Rapoport-Hubschman, N., Gidron, Y., Reicher-Atir, R., Sapir, O., & Fisch, B. (2009). "Letting go" coping is associated with successful IVF treatment outcome. *Fertility and Sterility, 92*, 1384–1388.

Grade of Activity

▶ Activity Level

Graded Exercise

Alexandre Morizio and Simon Bacon
Department of Exercise Science, Concordia University, Montreal Behavioral Medicine Centre, Montreal, QC, Canada

Synonyms

Maximal exercise test; Multistage submaximal exercise test

Definition

Graded exercise testing is a variety of exercise testing where tests are designed to be increasingly more difficult as they progress. A graded maximal exercise test would ideally progress until the participant reaches a level of maximal exertion, while a graded (multistage) submaximal exercise test would progress to a predetermined point.

While graded exercise tests are typically administered to determine a participant's functional aerobic capacity (VO_{2max}), they can also be used to diagnose certain diseases (primarily cardiovascular) when used in conjunction with other diagnostic tools, e.g., electrocardiograms (ECG), echocardiography, or nuclear medicine scanners. Graded exercise tests ideally involve large muscle groups, e.g., the hip and leg musculature, so as to more accurately determine from the results the need, delivery, and consumption of oxygenated blood. As such, the modalities most commonly used in graded exercise tests are treadmills and cycle ergometers. While cycle ergometers generally provide more advantages than treadmills, such as being less expensive, taking up less space, and providing more precise ECG readings due to reduced trunk movement, participants tend to attain higher VO_{2max} values when using treadmills (this is primarily due to the larger number of muscles which are engaged). Though less common, it is also possible to conduct graded exercise testing on equipment designed to assess specific muscle groups (e.g., arm ergometry) or activities (e.g., rowing ergometry).

Maximal and submaximal graded exercise tests have different advantages, and choosing which type of test to administer will depend on the parameter to be determined, the equipment available, and the level of expertise of the test administrators. For example, maximal graded exercise tests are more sensitive in the diagnosis of a number of cardiac problems, though the participant will need to exercise to the point of exhaustion. In contrast, a submaximal graded exercise test will provide a standardized and consistent level of exertion across all participants,

which can be very useful in a research setting. In addition, for all exercise testing, it is usually recommended that a physician be in attendance or available if a participant is at high risk of cardiovascular problems.

Cross-References

- ▶ Exercise
- ▶ Exercise Testing
- ▶ Isometric/Isotonic Exercise

References and Readings

American College of Sports Medicine, Thompson, W. R., Gordon, N. F., & Pescatello, L. S. (2010). *ACSM's guidelines for exercise testing and prescription* (8th ed.). PhiladelphiaPA: Wolters Kluwer Health/ Lippincott Williams & Wilkins.

Brawner, C. A. (2007). Graded exercise testing. In W. E. Kraus & S. J. Keteyian (Eds.), *Cardiac rehabilitation* (pp. 111–119). Totowa, NJ: Humana Press.

Heyward, V. H. (2006). *Advanced fitness assessment and exercise prescription* (5th ed.). Champaign, IL: Human Kinetics.

Nieman, D. C. (2007). *Exercise testing and prescription: A health-related approach* (6th ed.). New York: McGraw-Hill.

Graded Exercise Test

- ▶ Maximal Exercise Stress Test

Graded Exposure Counterconditioning

- ▶ Systematic Desensitization

Grave Yard Shift

- ▶ Night-Shift Workers and Health

Gravidity

- ▶ Gestation

Grief

- ▶ Bereavement
- ▶ Grieving

Grief Counseling

Jan R. Oyebode
School of Psychology, The University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Bereavement counseling; Bereavement therapy; Grief therapy

Definition

Grief counseling (also referred to as grief therapy or bereavement counseling) is the term used for the nonjudgmental counseling support that is provided for those who are suffering as a consequence of a loss through death of someone close to them. Grief counseling may also be used to assist with adjustment to a range of losses that involve a strong element of grief, such as amputation, loss of role, or loss of one's home. Bereavement therapy is a related term and may be distinguished from bereavement counseling as being provided for those who are having trouble adjusting, rather than being provided more routinely.

Grief counseling developed largely from the 1970s onward to provide support to people following bereavement in the increasingly fragmented societies of the technologically developed world, in which the religious rites,

rituals, and support of more traditional communities had been eroded. Services have traditionally been provided through professionals, volunteers, and self-help groups alike. The process of counseling offers an opportunity for the bereaved to express thoughts and feelings to someone trained and willing to listen, who has an understanding of the impact of grief.

Raphael, Minkov, and Dobson (2001) distinguish four levels of bereavement intervention:

1. Universal primary preventative intervention, that is, grief counseling being offered to all who have experienced bereavement with the aim of facilitating an adaptive response
2. Selective preventive intervention, that is, grief counseling being offered to those who may be vulnerable to difficulty in adapting due to the presence of risk factors related to their prior life, the nature of the death, or their circumstances following the death
3. Indicated preventive intervention, that is, grief counseling being offered to those who have unusually high levels of distress or a disturbing or unusual response, soon after bereavement
4. Treatment, that is, grief therapy offered to those with a complicated grief reaction who present to health services some time after the index event

Research evidence suggests that carefully targeted grief counseling at layers 2–4 of this framework is most effective, since universal provision may disturb a person's way of coping through reliance on their own resources and nonprofessional networks.

A number of scales have been developed which can be used to help in the assessment of whether someone might benefit from grief counseling (see Neimeyer and Hogan, 2001, for a useful review).

Where counseling is indicated, it may be provided at one of a number of levels (group, family, or individual) and using one of a number of approaches (e.g., nondirective or based on enhancing emotion-focused or problem-focused coping). Several approaches to level 4 treatment have been evaluated including behavior therapy, brief psychodynamic psychotherapy, hypnotherapy, and self-help. All appear to be helpful to a modest degree. This may indicate that nonspecific therapeutic factors are the key helpful ingredients in grief counseling or it may reflect individual differences in response to different approaches. Evidence-based factors to take into account in providing grief counseling include consideration of personal preferences; family, cultural, and religious contexts; enabling the person to reflect on both the past and the future; facilitating the person's search for meaning, and encouraging the person to consider both loss-oriented (e.g., feelings of grief) and restoration-oriented (e.g., dealing with the practicalities of day-to-day life without the deceased) stressors.

Cross-References

▶ Death Anxiety
▶ Grieving

References and Readings

Lendrum, S., & Syme, G. (2004). *Gift of tears: A practical approach to loss and bereavement counselling* (2nd ed.). Hove: Routledge.

Neimeyer, R. A., & Hogan, N. S. (2001). Quantitative or qualitative? Measurement issues in the study of grief. In M. S. Stroebe, R. O. Hansson, W. Stroebe, & H. Schut (Eds.), *Handbook of bereavement research: Causes, consequences and care* (pp. 89–118). Washington, DC: American Psychological Association.

Raphael, B., Minkov, C., & Dobson, M. (2001). Psychotherapeutic and pharmacological intervention for bereaved persons. In M. S. Stroebe, R. O. Hansson, W. Stroebe, & H. Schut (Eds.), *Handbook of bereavement research: Causes, consequences and care* (pp. 587–612). Washington, DC: American Psychological Association.

Worden, J. W. (2001). *Grief counselling and grief therapy* (3rd ed.). London: Tavistock.

Grief Therapy

▶ Grief Counseling

Grieving

Jan R. Oyebode
School of Psychology, The University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Bereavement; Grief; Loss

Definition

In a narrow sense, grieving is the term used to describe the experience and expression of the emotional state, of sadness, anguish, and pining, that commonly follows the death of one who was significant in the life of the bereaved, along with the somatic, cognitive, and behavioral facets of this response.

In a broader sense, grieving may be understood as referring to the whole period of time from first news of a death, through to a point when reasonable adjustment has been made; a period which may encompass a wide range of emotions (including numbness, disbelief, fear, anger, guilt, anguish, and sorrow) and a lengthy process of adaptation.

Description

Bereavement, by definition, is the loss of a person through death. However, grieving, the emotional response to bereavement and loss, may start prior to bereavement (anticipatory grief). Thus, facing death and issues of bereavement are closely intertwined.

Grief is often viewed as an individual emotional reaction, and its nature can be understood using ideas from the psychology of attachment and bonding, as the individual experiences irrevocable detachment and separation. However, bereavement affects all who knew a person, and therefore, families and social networks set the wider context for grieving and for support, and religious and cultural factors also influence the ways that grief is expressed and the way the dead are mourned and remembered. In some societies and in relation to some relationships, grief of a particular individual may not be sanctioned, this position being named "disenfranchised grief."

The end point of grief is hard to define with adaptation to life without the person who has died having a range of markers. For most people, life is never the same again, but many people report elements of growth and development through grief as well as distress and pain. However, about 15% of people experience prolonged and very distressing grief, and this is often referred to as "traumatic," "pathological," or "complicated."

A number of descriptive and theoretical frameworks have been used to try and capture the essence of grief. Parkes, in his seminal work, described common phases of response during the period of time following bereavement, usually described as (1) numbness, (2) searching and yearning, (3) disorganization and despair, and (4) reorganization. It should be noted that this framework is not intended to be applied rigidly to each individual case. A related view that places premium upon the active nature of grief is that of "grief work" (Worden). This conceptualizes the bereaved as needing to address four particular tasks: to accept the reality of the loss, to work through the pain of the loss, to adjust to the environment in which the deceased is no longer present, and to emotionally relocate the deceased and move on with life. In recent years, further concepts and models have been proposed. The most predominant at the present time is the dual process model (Stroebe & Schut, 1999) which lays emphasis on the concurrent existence of both loss-oriented stressors (related to the pain of separation) and restoration-oriented stressors (related to living life without the deceased) and considers the necessary oscillation in coping as the bereaved person responds to both of these sources of grief, addressing the emotional *and* the practical consequences. In addition, there is a recognition that rather than needing to let go, continuing bonds with the deceased can be helpful, as long as these are symbolic rather than very literal. It is also recognized that each person has their own grief narrative and that the process of

meaning-making is central to "coming to terms" (Neimeyer, 2000) with grief.

All the frameworks referred to in this entry can be found in the Handbooks of Bereavement Research named below.

References and Readings

Klass, D., Silverman, P. R., & Nickman, S. L. (Eds.). (1996). *Continuing bonds: New understandings of grief*. Washington, DC: Taylor & Francis.

Neimeyer, R.A. (2000). Searching for the meaning of meaning: Grief therapy and the process of reconstruction. *Death Studies, 24*, 541–558.

Parkes, C. M. (2006). *Love and loss: The roots of grief and its complications*. London/New York: Routledge.

Stroebe, M.S. & Schut, H. (1999). The dual process model of coping with bereavement : rationale and description. Death Studies, 23, 197–224.

Stroebe, M. S., Hansson, R. O., Stroebe, W., & Schut, H. (Eds.). (2001). *Handbook of bereavement research: Causes, consequences and care*. Washington, DC: American Psychological Association.

Stroebe, M., Stroebe, W., Hansson, R., & Schut, H. (Eds.). (2008). *Handbook of bereavement research: Advances in theory and intervention*. Washington, DC: APA.

Group Interview

► Focus Groups

Group Therapy/Intervention

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

Group therapy interventions refer to a format of several individuals taking part in a psychological intervention aimed at helping them change or deal with a long-lasting problem they are encountering, guided by a therapist or counselor. Group therapies have important advantages and are thus suitable for several types of problems, mainly those involving interpersonal issues. These include, for example, stress management, a common medical problem (cancer, heart disease), or addictions. The advantages of group over individual interventions include vicarious learning from others, knowing and being comforted by the fact that others share one's difficulties, and practicing in a safe environment constructive solutions for interpersonal problems. In behavior medicine, studies have used such a format to treat cancer patients (e.g., Andersen et al., 2008), cardiac patients (e.g., Gidron, Davidson, & Bata, 1999), and many other patient samples. In cardiac patients, one review found psychosocial interventions to reduce mortality and cardiac risk factors (Dusseldorp, van Elderen, Maes, Meulman, & Kraaij, 1999). Such group interventions can also take place at a workplace setting, enabling to solve work-related problems, for the benefit of individual workers, the team and the workplace as a whole.

Some studies have also begun to identify patient, therapist, and process variables predictive of positive outcomes following group interventions. In a unique and large-scale study on 40 groups of 266 Israeli children undergoing group therapy for emotional and behavioral problems, child bonding affected group functioning, which, in turn, affected behavioral outcomes. Similarly, therapist variables including encouragement and self-disclosure positively affected outcomes while challenging negatively affected outcomes (Shechtman & Leichtentritt, 2010). Thus, group interventions are an important medium for behavior modification and have been used in medical and health settings as well.

Cross-References

► Stress Management
► Therapy, Occupational

References and Readings

Andersen, B. L., Yang, H. C., Farrar, W. B., Golden-Kreutz, D. M., Emery, C. F., Thornton, L. M., Young, D. C., & Carson, W. E., 3rd. (2008). Psychologic

intervention improves survival for breast cancer patients: A randomized clinical trial. *Cancer, 113*, 3450–3458.
Dusseldorp, E., van Elderen, T., Maes, S., Meulman, J., & Kraaij, V. (1999). A meta-analysis of psychoeduational programs for coronary heart disease patients. *Health Psychology, 18*, 506–519.
Gidron, Y., Davidson, K., & Bata, I. (1999). The short-term effects of a hostility-reduction intervention on male coronary heart disease patients. *Health Psychology, 18*, 416–420.
Shechtman, Z., & Leichtentritt, J. (2010). The association of process with outcomes in child group therapy. *Psychotherapy Research, 20*, 8–21.

Grown

► Aging

Guided Imagery

Jennifer Cumming[1] and Giles M. Anderson[2]
[1]School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK
[2]School of Psychology, University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Mental imagery; Visualization

Definition

Guided imagery involves a practitioner verbally leading an individual through the processes of mentally representing situations in their mind. The representation can occur in one or more senses so that the person (or imaginer) experiences the sights, tastes, sounds, smells, and feelings associated with the situation. Details about people, places, and events can also be included to make the experience as realistic and vivid as possible.

To aid the process of generating the mental "images", guided imagery is usually performed with closed eyes, in a quiet environment, while the imaginer reclines in a comfortable position. Instructions on how and what to image can be delivered individually or in groups and are imparted either live or as an audio recording. Similar to hypnosis or meditation, guided imagery is often combined with music and relaxation techniques, such as deep breathing or progressive muscle relaxation, to help clear and focus the mind in preparation for the imagery.

What is imaged depends on personal preference or the technique used, such as Bonny's (1980) Method of Guided Imagery and Music (GIM). The content, however, is predominately determined by the imager's desired outcomes or reasons for using the guided imagery. For example, to feel more positive and reduce anxiety, instructions may direct the person to image a sense of calmness and therefore feel their mood improve. For pain sufferers, the imagery might lead them to focus on numbing and relief from pain or noxious symptoms. Alternatively, they might be guided to escape from the pain by imaging themselves in a pleasant location, such as a beach or the countryside.

Classed as a complementary alternative medicine (CAM), guided imagery is frequently recommended to cancer patients to help maximize pain relief in conjunction with pharmacological regimens; reduce aversion to certain treatments, such as chemotherapy; manage stress; aid in relaxation; and to empower patients to manage issues arising from their treatment. It has also been suggested that using the power of your mind to influence psychological and physiological states can help improve the quality of life and psychological well-being of individuals suffering from respiratory disease (e.g., asthma, chronic bronchitis, and emphysema), chronic pain (e.g., fibromyalgia, chronic headaches, and osteoarthritis), and hypertension. Guided imagery is also recommended as a coping strategy for perioperative patients and end-of-life palliative care to reduce anxiety. It has been used to help increase physical strength and aid in the recovery of motor function following musculoskeletal injuries and for individuals with stroke and Parkinson's disease. Further, guided imagery

has been recommended for changing health behaviors, including physical activity, smoking cessation, and weight management.

To evaluate its effectiveness, guided imagery has been compared to standard care practice in patient populations. For example, an intervention combining relaxation and guided imagery was used to improve immune function by asking patients undergoing surgery for breast cancer to mentally experience immune cells destroying cancer cells (Lengacher et al., 2008). The effects on the immune system were explained by relaxation and imagery reducing stress and leading to the release of neuropeptides and cytokines to improve the immune response. Despite these promising results, much of the available research evaluating the effectiveness of guided imagery for use with patients suffers from poor, inconsistent methodologies combined with small samples that make the efficacy of guided imagery difficult to compare across studies. However, a review of six randomized clinical trials by Roffe, Schmidt, and Ernst (2005) indicates that guided imagery can be psychology supportive and increase comfort of cancer sufferers but not improve their physical symptoms, such as nausea and vomiting. A more recent five-study review by King (2010) found some support for the use of guided imagery as an aid in alleviating the pain associated with cancer. When guided imagery is effective in controlling pain, it seems to provide patients with a source of distraction from the discomfort or serve to stimulate their relaxation. However, the pain characteristics of certain patients (i.e., the intensity of the pain) may make it difficult for them to image, which could explain why the technique is not always effective in managing pain (see Kwekkeboom, Hau, Wanta, & Bompus, 2008).

Another barrier is individual differences in the ability to create and control vivid images. Although everyone has the ability to image, there is considerable variability from person to person. Those who are more proficient in generating, transforming, and maintaining images will more likely benefit from imagery interventions. To identify who would be most helped, the Imaging Ability Questionnaire (IAQ) was developed by Kwekkeboom (2000) as a valid and reliable screening tool for cancer patients. The IAQ measures the ability to generate vivid images using various senses and to be involved or engaged in the imagery experience. Similar questionnaires have been developed for specific use with other clinical populations, such as the Kinesthetic and Visual Imagery Questionnaire (KVIQ; Malouin et al., 2007), to measure motor imagery ability in individuals with physical impairments. Although termed "ability", it is also important to note that individuals become more proficient at imaging following instruction and practice. Those who score low on imagery ability questionnaires therefore might first need training exercises to improve their skills of imaging before receiving a guided imagery program.

Cross-References

▶ Meditation

References and Readings

Ackerman, C. J., & Turkoski, B. (2000). Using guided imagery to reduce pain and anxiety. *Home Health Care Nurse, 18*, 524–530.

Bardia, A., Barton, D., Prokop, L., Bauer, B., & Moynihan, T. (2006). Efficacy of complementary and alternative medicine therapies in relieving cancer pain: A systematic review. *Journal of Clinical Oncology, 24*, 5457–5463.

Bonny, H. L. (1980). *GIM therapy: Past, present, and future implications* (GIM Monograph No. 3). Salina, KS: The Bonny Foundation.

Burns, D. S. (2001). The effect of the Bonny Method of guided imagery and music on the mood and life quality of cancer patients. *Journal of Music Therapy, 38*, 51–65.

Johnson, E. L., & Lutgendorf, S. K. (2001). Contributions of imagery ability to stress and relaxation. *Annals of Behavioral Medicine, 23*, 273–281.

Kelly, K. (2010). A review of the effects of guided imagery on cancer patients with pain. *Complementary Health Practice Review, 15*, 98–107.

King, K. (2010). A review of the effects of guided imagery on cancer patients with pain. *Complementary Health Practice Review*, *15*, 98–107.

Kwekkeboom, K. L. (2000). Measuring imagery ability: Psychometric testing of the imaging ability questionnaire. *Research in Nursing & Health, 23*, 301–309.
Kwekkeboom, K., Hau, H., Wanta, B., & Bumpus, M. (2008). Patients perceptions of the effectiveness of guided imagery and progressive muscle relaxation interventions used for cancer pain. *Complementary Therapeutics in Clinic Practice, 14*, 185–194.
Kwekkeboom, K., Huseby-Moore, K., & Ward, S. (1998). Imaging ability and effective use of guided imagery. *Research in Nursing and Health, 21*, 189–198.
Lengacher, C. A., Bennet, M. P., Gonzalez, L., Gilvary, D., Cox, C. E., Cantor, A., Jacobsen, P. B., Yang, C., & Djeu, J. (2008). Immune responses to guided imagery during breast cancer treatment. *Biological Research Nursing, 9*, 205–214.
Malouin, F., Richards, C. L., Jackson, P. L., Lafleur, M. F., Durand, A., & Doyen, J. (2007). The kinesthetic and visual imagery questionnaire (KVIQ) for assessing motor imagery in persons with physical disabilities: A reliability and construct validity study. *Journal of Neurologic Physical Therapy, 31*, 20–29.
Roffe, L., Schmidt, K., & Ernst, E. (2005). A systematic review of guided imagery as an adjuvant cancer therapy. *Psycho-Oncology, 14*, 607–617.
Schmidt, K., & Ernst, E. (2004). Assessing websites on complementary and alternative medicine for cancer. *Annals of Oncology, 15*, 733–742.
Trakhtenberg, E. C. (2008). The effects of guided imagery on the immune system: A critical review. *International Journal of Neuroscience, 118*, 839–855.
Watanabe, E., Fukuda, S., Hara, H., Maeda, Y., Ohira, H., & Shirakawa, T. (2006). Differences in relaxation by means of guided imagery in a healthy community sample. *Alternative Therapies in Health and Medicine, 12*, 60–66.

Guideline

▶ Clinical Practice Guidelines

Guidelines for Reporting Randomized Controlled Trials

▶ CONSORT Guidelines

GWA Study

▶ Genome-Wide Association Study (GWAS)

GxE

▶ Gene-Environment Interaction

GxG

▶ Gene-Gene Interaction

Gyrus/Gyri (pl)

▶ Brain, Cortex

H

Habilitation

► Rehabilitation

Habit Strength

Sheina Orbell
Department of Psychology, University of Essex, Colchester, Essex, UK

Synonyms

Habitual automaticity

Definition

A habit can be defined as a learned behavioral response to a situational cue. The repeated performance of a behavior in a specific context leads to the development of a behavioral habit that is triggered by features of the environment that have covaried frequently with past performance of the behavior. Such features of the environment might include performance locations, preceding actions in a sequence, the presence of particular people, or an internal thought or feeling. As a consequence of repetition in the same cue-contexts, a habit becomes capable of being triggered directly by perception of the cue. This is referred to as cue contingent automaticity. A person might experience his or her habit as something "I cannot help doing."

Habit strength is a function of the frequency with which an action has been repeated in a stable context and has acquired a high degree of habitual automaticity. Verplanken and Orbell (2003) developed and validated a metacognitive 12-item instrument to measure habit strength, the Self-Report Habit Index (SRHI). This is a generic instrument that asks respondents whether their performance of a target behavior occurs frequently; requires conscious awareness, thought, and effort; and is difficult to control.

From an empirical perspective, stronger habits are associated with heightened attention to cues associated with the performance of a habit and an increased likelihood of making an action slip when the cue is detected (Orbell & Verplanken, 2010). Removal of the cue (e.g., by changing one's environment) disrupts the performance of a previous habit (Wood, Tam, & Guerrero Witt, 2005). Strong habits also disrupt the ability to enact a counterhabitual intention. Strong habits may be useful in health contexts where, for example, good adherence is required and may be

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

promoted by interventions that promote repetition in stable contexts (Orbell & Verplanken, 2010).

Cross-References

▶ Intention Strength

References and Readings

Chatzisarantis, N. L., & Hagger, M. S. (2007). Mindfulness and the intention-behavior relationship within the theory of planned behavior. *Personality and Social Psychology Bulletin, 33*, 663–676.

Orbell, S., & Verplanken, B. (2010). The automatic component of habit in health behavior: Habit as cue-contingent automaticity. *Health Psychology, 29*, 374–383.

Verplanken, B., & Orbell, S. (2003). Reflections on past behavior: A self-report index of habit strength. *Journal of Applied Social Psychology, 33*, 1313–1330.

Wood, W., & Neal, D. T. (2007). A new look at habits and the habit-goal interface. *Psychological Review, 114*, 843–863.

Wood, W., Tam, L., & Guerrero Witt, M. (2005). Changing circumstances, disrupting habits. *Journal of Personality and Social Psychology, 88*, 918–933.

Habitual Automaticity

▶ Habit Strength

Habitual Performance

▶ Physical Fitness

HADS

▶ Hospital Anxiety Depression Scale

Hamilton Anxiety Rating Scale

Havah Schneider[1], Sabrina Esbitt[1] and Jeffrey S. Gonzalez[2,3]
[1]Ferkauf Graduate School of Psychology, Yeshiva University, Bronx, NY, USA
[2]Clinical Psychology, Health Emphasis, Ferkauf Graduate School of Psychology, Yeshiva University, Bronx, NY, USA
[3]Diabetes Research Center, Albert Einstein College of Medicine, Yeshiva University, Bronx, NY, USA

Definition

The Hamilton Anxiety Rating Scale (HAM-A) is a widely used 14-item clinician-administered rating tool in the public domain used to measure the severity of anxiety symptoms among individuals previously diagnosed with anxiety disorders (McDowell, 2006). The HAM-A was originally developed by Max Hamilton in 1959 as an assessment tool to evaluate anxiety symptoms among people diagnosed with "anxiety neurosis." Since that time, anxiety neurosis has been reconceptualized and the HAM-A is used among individuals with a variety of anxiety disorders (panic, phobia, and generalized) (McDowell, 2006). The 14 items reflect 13 categories of anxiety-related symptoms including anxious mood, tension, fear, insomnia, intellectual/cognitive symptoms, depressed mood, general somatic (muscular and memory symptoms), cardiovascular, respiratory, genitourinary, and gastrointestinal symptoms, with one item capturing the rater's assessment of behavioral symptoms. The HAM-A takes approximately 15–30 min to administer and score and contains two subscales – psychiatric anxiety (psychological distress) and somatic anxiety (physical symptoms of distress) (Hamilton, 1959).

The HAM-A is not designed to be used as a diagnostic tool and has poor discriminant validity between anxiety disorders and depression. Instead, it is a standard primary outcome measure used to asses the efficacy of clinical interventions for DSM-IV anxiety disorders – most commonly

generalized anxiety disorder – within psychopharmacologic randomized controlled trials and psychotherapeutic clinical trials (Roemer, 2001). It also used for monitoring anxiety symptoms during treatment. The severity of the item is determined on a five point scale (0 = not present, 4 = severe). A computerized version as well as a pen-and-paper format is available (Kobak, Reynolds, & Greist, 1993), and a structured interview guide has also been developed to standardize its administration (SIGH-A), as its administration is not predefined in the initial instrument (Williams, 1988). A six-item abbreviated scale capturing psychic anxiety, tension, restlessness, inability to relax, startle response, worry, and apprehension called the Clinical Anxiety Scale was also proposed by Snaith, Baugh, Clayden, Husain and Sipple (1982). The HAM-A has also been translated into several languages, including Spanish, German, and Polish (Roemer, 2001).

Cross-References

- ▶ Anxiety
- ▶ Anxiety and its Measurement
- ▶ Anxiety Disorder
- ▶ Stress

References and Readings

Hamilton, M. (1959). The assessment of anxiety states by rating. *British Journal of Medical Psychology, 32*, 50–55.

Kobak, K. A., Reynolds, W. M., & Greist, J. H. (1993). Development and validation of a computer-administered version of the Hamilton Rating Scale. *Psychological Assessment, 5*(4), 487–492.

Maier, W., Buller, R., Philipp, M., & Heuser, I. (1988). The Hamilton anxiety scale: Reliability, validity and sensitivity to change in anxiety and depressive disorders. *Journal of Affective Disorders, 14*(1), 61–68.

Marques, L., Chosak, A., Simon, N. M., Phan, D., Wilhelm, S., & Pollack, M. (2010). Rating scales for anxiety disorders. In P. Baer, P. A. Blais, P. Baer, & P. A. Blais (Eds.), *Handbook of clinical rating scales and assessment in psychiatry and mental health* (pp. 37–72). Totowa, NJ: Humana Press.

McDowell, I. (2006). *Measuring health: A guide to rating scales and questionnaires* (3rd ed.). New York: Oxford University Press.

Roemer, L. (2001). Measures for anxiety and related constructs. In M. M. Antony, S. M. Orsillo, & L. Roemer (Eds.), *Practitioner's guide to empirically based measures of anxiety* (pp. 49–83). New York: Springer.

Snaith, R. P., Baugh, S. J., Clayden, A. D., Husain, A., & Sipple, M. A. (1982). The clinical anxiety scale: An instrument derived from the Hamilton anxiety scale. *British Journal of Psychiatry, 141*, 518–523.

Williams, J. W. (1988). A structured guide for the Hamilton depression rating scale. *Archives of General Psychiatry, 45*, 742–767.

Hamilton Rating Scale for Depression (HAM-D)

Jeffrey S. Gonzalez[1,3], Erica Shreck[2] and Abigail Batchelder[2]
[1]Clinical Psychology, Health Emphasis, Ferkauf Graduate School of Psychology, Yeshiva University, Bronx, NY, USA
[2]Yeshiva University, Bronx, NY, USA
[3]Diabetes Research Center, Albert Einstein College of Medicine, Yeshiva University, Bronx, NY, USA

Definition

The Hamilton Rating Scale for Depression or Hamilton Depression Rating scale (HAM-D, HRSD, or HDRS) is a 21-item clinician-administered multiple-choice measure of depression symptom severity. The first 17 of the 21 items contribute to the total score (Hamilton, 1960) and items 18–21 give additional information not part of the scale, such as paranoia and diurnal variation (Hedlund & Vieweg, 1979). Other versions have been developed, ranging from 7 to 29 items (e.g., Hamilton, 1964; Williams, 1988). In all versions, symptoms are defined by anchor point descriptions (ranging from 3 to 5 possible responses), which increase in severity. Clinicians consider intensity and frequency of symptoms based on patient response and observations. A score of ≤ 7 is widely thought to indicate remission on the HAM-D_{17} (Frank et al., 1991).

The HAM-D was first published in 1960 and reviewed subsequently (Hamilton, 1964, 1980). Due to its comprehensive coverage of depressive

symptoms, strong psychometric properties (Hedlund & Vieweg, 1979), and the total score demonstrating high concurrent and differential validity as well as strong reliability (Carroll, Fielding, & Blashki, 1973), the HAM-D is considered by many to be the "gold standard" of assessing depressive symptomatology. However, most individual items demonstrate fair to poor agreement (Cicchetti & Prusoff, 1983). Use of the Structured Interview Guide, published in 1988, increased the reliability of the items (Williams, 1988). Self-report and computerized versions have been developed to improve the psychometric properties of individual items (Williams, 2001).

The HAM-D is primarily applied for research purposes to determine severity of depressive symptoms throughout treatment and in response to psychotherapy or antidepressants (O'Sullivan, Fava, Agustin, Baer, & Rosenbaum, 1997; Williams, 2001). More specifically, in the area of behavioral medicine, the HAM-D is used to measure the severity of depression in people with comorbid chronic illness. As assessment of depression can be particularly complicated in this population due to the co-occurrence of somatic features of depression and physical illness, the HAM-D has been criticized for its sensitivity to somatic symptoms (Maier & Philipp, 1985; Sutton, Baum, & Johnston, 2004). Consequently, researchers have evaluated the utility of the HAM-D for assessing depression in chronic illness. An early study assessing somatic comorbidity in a sample of elderly patients found that eight of the HAM-D scale items rated as positive scores for depression by psychiatrists were rated by internists as being related to somatic conditions (Linden, Borchelt, Barnow, & Geiselmann, 1995). Additionally, researchers compared depression rating scales in chronic fatigue syndrome and found that the HAM-D overestimated the number of depressed patients (Henderson & Tannock, 2005). Nevertheless, a more recent study evaluated the scale in depressed participants with multiple sclerosis and found that the majority of items (12 out of 17) captured depressive symptoms and adequately differentiated from somatic symptoms (Moran & Mohr, 2005). Results suggest that this co-occurrence must be considered when using the HAM-D in behavioral medicine settings.

References and Readings

Carroll, B. J., Fielding, J. M., & Blashki, T. G. (1973). Depression rating scales: A critical review. *Archives of General Psychiatry, 28*, 361–366.

Cicchetti, D. V., & Prusoff, B. A. (1983). Reliability of depression and associated clinical symptoms. *Archives of General Psychiatry, 40*, 987–990.

Frank, E., Prien, R., Jarrett, R., Keller, M., Kupfer, D., Lavori, P., et al. (1991). Conceptualization and rationale for consensus definitions of terms in major depressive disorder. *Archives of General Psychiatry, 48*, 851–855.

Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry, 23*, 56–62.

Hamilton, M. (1964). A rating scale for depressive disorders. *Psychological Reports, 14*, 914.

Hamilton, M. (1980). Rating depressive patients. *Journal of Clinical Psychiatry, 41*, 21–24.

Hedlund, J. L., & Vieweg, B. W. (1979). The Hamilton rating scale for depression: A comprehensive review. *Journal of Operational Psychiatry, 10*, 149–165.

Henderson, M., & Tannock, C. (2005). Use of depression rating scales in chronic fatigue syndrome. *Journal of Psychosomatic Research, 59*, 181–184.

Linden, M., Borchelt, M., Barnow, S., & Geiselmann, B. (1995). The impact of somatic morbidity on the Hamilton depression rating scale in the very old. *Acta Psychiatrica Scandinavica, 92*(2), 150–4.

Maier, W., & Philipp, M. (1985). Improving the assessment of severity of depressive states: A reduction of the Hamilton depression scale. *Pharmacopsychiatry, 18*, 114–5.

Moran, P. J., & Mohr, D. C. (2005). The validity of beck depression inventory and Hamilton rating scale for depression items in the assessment of depression among patients with multiple sclerosis. *Journal of Behavioral Medicine, 28*(1), 35–41.

O'Sullivan, R. L., Fava, M., Agustin, C., Baer, L., & Rosenbaum, J. F. (1997). Sensitivity of the six-item Hamilton depression rating scale. *Acta Psychiatrica Scandinavica, 95*, 379–384.

Sutton, S., Baum, A., & Johnston, M. (2004). *The SAGE handbook of health psychology*. London: Sage.

Williams, J. B. (1988). A structured interview guide for the Hamilton depression rating scale. *Archives of General Psychiatry, 45*, 742–747.

Williams, J. B. (2001). Standardizing the Hamilton depression rating scale: Past, present, and future. *European Archives of Psychiatry and Clinical Neuroscience, 25*, 6–12.

Handgrip Strength

Yori Gidron
Faculty of Medicine and Pharmacy,
Free University of Brussels (VUB), Jette,
Belgium

Definition

This term refers to a common measure used often in rehabilitation medicine to determine the maximum forearm muscular isometric strength. Given that muscle strength has general characteristics, the handgrip strength test may often indicate general muscular strength. The test includes a dynamometer, with a scale in kilogram, where people are asked to perform their maximal press with their hand. Different protocols exist concerning the angle of the arm and hand in relation to the body, the number of pressing trials, and the duration of pressure, normally lasting 3–5 s. This test can be used to indicate various health factors in different populations.

A review of the value of the handgrip strength test in dialysis patients found this test to correlate with general muscle mass, nutritional status (of importance in dialysis), and future complications (Leal, Mafra, Fouque, & Anjos, 2011). A review of 114 studies in the general population and 71 studies with arthritic patients found a strong age-related decline in handgrip strength, and much lower scores among arthritic patients, suggesting a relationship between inflammation and performance on this test (Beenakker et al., 2010). In some pain patients, this test is also helpful in assessment of their condition. For example, handgrip strength is lower in patients with fibromyalgia, and is inversely related to their levels of pain, fatigue, and stiffness (Aparicio et al., 2010). This test was used in several cohort studies to predict risk of death. For example, in elderly French women, a low handgrip test score significantly predicted risk of mortality, independent of confounders (Rolland et al., 2006). In patients with congestive heart failure, low handgrip strength also predicted risk of death, independent of confounders (Izawa et al., 2009). Furthermore, handgrip scores also prospectively predict decline in activity of daily living and in cognitive performance in the elderly (Taekema, Gussekloo, Maier, Westendorp, & Craen, 2010). Thus, the handgrip test is a simple, rapid, and objective test which provides information on important physical factors and has predictive validity in relation to functional, cognitive, and vital status measures.

Cross-References

▶ Functional Capacity, Disability, and Status

References and Readings

Aparicio, V. A., Carbonell-Baeza, A., Ortega, F. B., Ruiz, J. R., Heredia, J. M., & Delgado-Fernández, M. (2010). Handgrip strength in men with fibromyalgia. *Clinical and Experimental Rheumatology, 28*, S78–S81.

Beenakker, K. G., Ling, C. H., Meskers, C. G., de Craen, A. J., Stijnen, T., Westendorp, R. G., et al. (2010). Patterns of muscle strength loss with age in the general population and patients with a chronic inflammatory state. *Ageing Research Reviews, 9*, 431–436.

Izawa, K. P., Watanabe, S., Osada, N., Kasahara, Y., Yokoyama, H., Hiraki, K., et al. (2009). Handgrip strength as a predictor of prognosis in Japanese patients with congestive heart failure. *European Journal of Cardiovascular Prevention and Rehabilitation, 16*, 21–27.

Leal, V. O., Mafra, D., Fouque, D., & Anjos, L. A. (2011). Use of handgrip strength in the assessment of the muscle function of chronic kidney disease patients on dialysis: A systematic review. *Nephrology, Dialysis, Transplantation, 26*(4), 1354–1360. Epub 2010 Aug 13.

Rolland, Y., Lauwers-Cances, V., Cesari, M., Vellas, B., Pahor, M., & Grandjean, H. (2006). Physical performance measures as predictors of mortality in a cohort of community-dwelling older French women. *European Journal of Epidemiology, 21*, 113–122.

Taekema, D. G., Gussekloo, J., Maier, A. B., Westendorp, R. G., & de Craen, A. J. (2010). Handgrip strength as a predictor of functional, psychological and social health: A prospective population-based study among the oldest old. *Age and Ageing, 39*, 331–337.

Happiness

▶ Well-Being: Physical, Psychological, Social

Happiness and Health

Sarah D. Pressman[1] and Emily D. Hooker[2]
[1]Department of Psychology, University of Kansas, Lawrence, KS, USA
[2]Department of Psychology and Social Behavior, University of California, Irvine, Irvine, CA, USA

Synonyms

Physical well-being; Positive affect; Positive emotion; Subjective well-being

Definition

Positive emotions (including happiness) arise as the result of pleasurable engagement with the environment and may present themselves in a variety of forms (e.g., enthusiasm, calm, contentment). Traditionally, physical health is defined as the objective absence of disease or illness, but can also include perceptions of wellness.

Description

While the concept that happiness is tied to better health is not novel and is widely accepted by the public, the research in this area remains in its infancy. Due to the surge of interest in positive psychology over the last decade, researchers are beginning to unveil the predictive and protective effects of positive emotions on health. There are however many remaining critical research questions. This section will focus on the most robust and striking findings in the literature on positive emotions and physical health, in addition to a brief discussion on some of the important methodological concerns for the field.

What Is Positive Affect?

While there is some debate in the literature as to what adjectives and precise feelings make up positive affect (PA), it is typically considered to be the general positive emotions or feelings (e.g., happiness, enthusiasm, calm, or contentment) resulting from pleasurable interactions with the environment. These feelings may persist for long periods of time and define an individual's general disposition (often called trait PA) or they may be transient moments of emotion that last for minutes or days, typically referred to as state PA, positive mood, or emotion (emotion being the shorter lasting of the two). Research on PA and health primarily focuses on trait PA given its more likely *long-lasting* effects on physical well-being; however, on occasion, studies will assess the effects of state PA (commonly assessed via a one-time mood assessment asking questions like "How happy are you this week?"). While shorter time assessments of positive feelings are less likely to influence long-term health outcomes, they are known to have transient effects on physiology and are highly correlated with dispositional measures of PA.

Measuring Positive Affect

PA is most frequently assessed via self-report scales asking about the frequency, duration, or intensity of positive feelings. There are a host of different scales to do this with wide discrepancies between them. In the health field, the most frequently used multi-item tool is the 20-item Positive and Negative Affect Schedule (PANAS), which assesses affect by having individuals rate the degree to which each emotion word (e.g., enthusiastic or irritable) describes their typical mood with flexibility in the assessed time period covered. This scale focuses on aroused emotions and is therefore not useful for individuals interested in assessing the health impact of low-energy states (e.g., calm). There are however many other mood adjective checklists that include low-arousal emotions such as those using Circumplex Models of Emotion (Russell, 1980), which includes measures of both arousal and valence, or the extended 60-item version of the PANAS (the PANAS-X)

(Watson & Clark 1999). Studies have also utilized single-item questionnaires (e.g., "Are you happy?"), confederate report, positive items drawn from other scales (e.g., depression measures), or even autobiographical writing samples. Given the known high levels of social desirability and response bias to emotion scales, future research would benefit from greater use of unobtrusive and non-self-report methodologies to determine an individual's level of PA. One other critical measurement concern relates to the role of negative affect (NA) in the PA health association. At the trait level, PA and NA are often weakly correlated; however, they are sometimes considered to be opposite ends of the same spectrum by many researchers. It may be the case that benefits of PA on health are simply attributable to the absence of NA. The majority of studies do not test for the independence of these affect variables in relation to their health impact; however, those that do frequently report that PA is beneficial to health irrespective of NA. Also critical is to better understand what types of PA are beneficial to health. Given the divergent physiological impacts of high- versus low-arousal emotions (e.g., ecstasy versus relaxation), it is not unrealistic to anticipate differential health results. Nevertheless, this is rarely considered and frequently unmeasured due to the choice of affect items within scales.

Associations Between Positive Affect and Health

In their major review, Pressman and Cohen (2005) evaluated the results of over 150 studies on PA and health and physiological outcomes. They consistently found that greater PA was associated with increased longevity in individuals older than 55, in studies with years to decades of longitudinal follow-up. For example, in one creative study by Danner, Snowdon, and Friesen (2001), autobiographical writing samples from 180 young nuns entering the convent were coded for positive and negative emotion word usage. At a 50-year follow-up time point, researchers found that nuns who used higher levels of positive words lived almost 11 years longer than their counterparts who used the fewest positive emotion words. This finding was not attributable to negative word usage. Similar results have been demonstrated in multiple studies of healthy, community-dwelling older individuals revealing that those individuals who report greater amounts of PA at baseline live *years* longer than their less positive counterparts.

There is also consistent evidence that positive emotions are protective against a multitude of morbidity outcomes including decreased falls and injuries, reduced heart attack and stroke incidences, fewer hospitalizations for coronary complications, and improved pregnancy outcomes. An exemplary example of this is the viral challenge work of Cohen, Doyle, Turner, Alper, and Skoner (2003). In this study, PA (determined via interviews averaged over several weeks) was found to prospectively predict the decreased likelihood of developing an objective cold (and cold symptoms) after being experimentally exposed to a novel virus. These results were independent of the influence of trait NA, which was only tied to the *perception* of having a cold as opposed to actual incidence. This finding was replicated, in that positive emotion styles predicted fewer objective flu cases and fewer flu symptoms reported when the flu virus was experimentally administered.

Additional findings from the literature generally show that cross-sectionally, individuals with higher PA report fewer symptoms and generally feel better. What remains unknown is whether this is a true physiological process (e.g., altered opioid levels) or whether PA simply leads to altered attention to or perception of symptoms. Similarly, it may also be true that feeling of health lead to greater positive emotion. Most studies to date do not address these mechanistic and directional questions.

Finally, survival studies of diseased patients have provided *some* indication that PA may lead to improved health outcomes, but not in every circumstance. Research on those with early-stage life-threatening diseases (e.g., HIV, stage I–II breast cancer) indicates that PA may lengthen life. This may be due to physiological changes (outlined below) or due to greater adherence to treatments and/or positive behavioral changes,

but these mediators have not been thoroughly evaluated. To date, there is little and mixed evidence regarding the effects of PA in late stage disease (e.g., stage IV breast cancer, end-stage renal disease). There are several possible reasons for this: high PA during the end stages of life may indicate unusual or inappropriate coping and possible underreporting of important symptoms. It is also likely that the small physiological changes tied to PA may be too weak to alter the course of disease in its late stages.

How Could Positive Affect Improve Health?

Pressman and Cohen (2005) proposed two pathways by which PA might benefit health and prevent disease. The first is the main effect hypothesis, which contends that PA influences health via its positive influences on health practices, physiological functioning (e.g., immune, cardiovascular, endocrine), and social relationships (also known to have health benefits). The second theory indicates that PA may be tied to better health via its beneficial impact on the stress response. Specifically, it may ameliorate the negative impact of stress by altering perceptions of severity, reducing detrimental physiological responses, and by helping individuals build resources (e.g., physical health, social support) to aid in coping and stress recovery. It is likely that both pathways play some role, although to date no one has directly contrasted the pathways in a single study. There is, however, growing evidence for both types of connections.

Critiques and Future Directions

Future research needs to distinguish what *types* of positive factors are most important to health outcomes and when. To date, most studies focus on "happiness"; however, there is equally good evidence for multi-adjective scales in addition to related positive constructs (e.g., optimism, life satisfaction). It is also important for researchers to better understand at what intensity and frequency positive emotions must be felt to show real physiological benefits and to what extent changes are independent from negative feelings. There is also a need to better understand the mediators of the PA health association and to have studies that prospectively test both health and physiological pathways together. Finally, it is an exciting notion to consider the possibility that PA-inducing interventions might improve health in a meaningful fashion, but it is too soon to determine whether or not these types of studies have robust effects.

General Conclusions

Happiness and other positive emotions have been linked to a greater lifespan, reduced disease susceptibility, improved health perceptions, and better outcomes for those with early-stage diseases. Meanwhile, researchers continue to explore the extent to which positive emotion can be beneficial, when it is most important in disease prevention and treatment, and finally the mechanisms by which it is most helpful.

Cross-References

- ▶ Affect
- ▶ Affect Arousal
- ▶ Emotions: Positive and Negative
- ▶ Health Psychology
- ▶ Mood
- ▶ Optimism, Pessimism, and Health
- ▶ Positive Affect Negative Affect Scale (PANAS)
- ▶ Positive Affectivity
- ▶ Positive Psychology
- ▶ Well-Being

References and Readings

Cohen, S., Doyle, W. J., Turner, R. B., Alper, C. M., & Skoner, D. P. (2003). Emotional style and susceptibility to the common cold. *Psychosomatic Medicine, 65*, 652–657.

Danner, D. D., Snowdon, D. A., & Friesen, W. V. (2001). Positive emotions in early life and longevity: Findings from the nun study. *Personality Processes and Individual Differences, 80*(5), 804–813.

Diener, E., & Emmons, R. A. (1985). The independence of positive and negative affect. *Journal of Personality and Social Psychology, 47*(5), 1105–1117.

Diener, E., Larsen, R. J., Levine, S., & Emmons, R. A. (1985). Intensity and frequency: Dimensions underlying positive and negative affect. *Journal of Personality and Social Psychology, 48*(5), 1253–1265.
Diener, E., & Lucas, R. E. (2000). Subjective emotional well-being. In M. Lewis & J. M. Haviland-Jones (Eds.), *Handbook of emotions* (pp. 325–334). New York: Giliford Press.
Pressman, S. D., & Cohen, S. (2005). Does positive affect influence health? *Psychological Bulletin, 131*(6), 925–971.
Russell, J. A. (1980). A circumplex model of affect. *Journal of Personality and Social Psychology, 39*, 1161–1178.
Watson, D., & Clark, L. A. (1999). *The PANAS-X: Manual for the positive and negative affect schedule-expanded form.* Iowa City, IA: University of Iowa, Department of Psychology. Retrieved January 4, 2011 from http://www.psychology.uiowa.edu/Faculty/Watson/Watson.html

Hardiness

▶ Locus of Control
▶ Resilience
▶ Resilience: Measurement
▶ Salutogenesis
▶ Williams LifeSkills Program

Hardiness and Health

Deborah J. Wiebe
Division of Psychology, Department of Psychiatry, Southwestern Medical Center, University of Texas, Dallas, TX, USA

Synonyms

Personality hardiness

Definition

Hardiness is a personality construct composed of three traits – control, commitment, and challenge – that are theorized to make one resilient in the face of stress. Individuals high in hardiness tend to believe and act as if life experiences are controllable (control), to engage meaningfully in life activities and to appraise these activities as purposeful and worthy of investment even in the face of adversity (commitment), and to view change in life as a challenge toward growth and development rather than as a threat to security (challenge). Based on existential personality theory, the combination of these characteristics is believed to provide individuals with the courage and motivation to cope adaptively with life stress, thereby buffering its adverse effects on health.

Description

Hardiness has historical significance because it played a significant role in the re-emergence of research examining the relationship between personality and health, and it foreshadowed the current positive psychology movement that focuses on transformation, growth, and resilience in the face of adversity (e.g., optimism, benefit finding, posttraumatic growth). Hardiness was developed by Maddi and Kobasa (Kobasa, 1979; Kobasa, Maddi, & Kahn, 1982) out of a longitudinal study of executives at Illinois Bell Telephone who were facing work upheaval due to deregulation. Executives were studied before, during, and after deregulation to identify characteristics of those who remained healthy and thrived in this time of heightened life stress versus those who showed signs of strain. Individuals who displayed little strain differed from their high strain counterparts on the characteristics of control, commitment, and challenge.

Associations Between Hardiness and Health

Evidence has accumulated across the decades to suggest that hardiness is associated with lower levels of physical and psychological strain following exposure to stress. Higher hardiness has been associated with lower reports of physical symptoms and psychological distress in both cross-sectional and longitudinal analyses. Such associations have been found across samples experiencing diverse stressors including

school-related stress in undergraduates, work-related stress among business executives, bus drivers and lawyers, and military personnel undergoing stressful military procedures. The characteristics of hardiness have also been consistently associated with better performance under stress as revealed in higher GPAs, athletic performance, and leadership skills.

Research has also examined the biobehavioral mechanisms by which hardiness may attenuate adverse responses to stress. There is compelling evidence that characteristics of hardiness facilitate adaptive cognitive appraisals in the face of stress. For example, high hardy individuals make more positive appraisals when experiencing laboratory-induced threat, and appraise the same life stressors as less threatening and more controllable than do low hardy individuals. Hardiness is also associated with more adaptive coping characterized by higher problem-focused and support-seeking coping, better health behaviors, and lower avoidance coping. Consistent with hardiness theory, these more positive perceptions of stress and active versus passive coping strategies have been found to mediate associations between hardiness and health.

Controversies Regarding Hardiness and Health Associations

Despite such encouraging findings, numerous criticisms of this literature have led some researchers to question the evidence supporting an association between hardiness and health. Concerns have centered on: (a) problems with the measurement of hardiness, (b) problems with the measurement of health outcomes, and (c) inconsistent evidence that hardiness "buffers" the adverse effects of stress.

Measurement of hardiness. Progress in the field has been hampered by a number of problems with the measurement of hardiness. First, the measure of hardiness has gone through multiple iterations and no standard measure of hardiness exists. The use of multiple measures makes it difficult to evaluate findings across studies. Second, the existing measures have not consistently supported the three-factor structure theorized to underlie the hardiness construct, raising questions about whether hardiness should be examined as a single composite variable. Research that has examined the three constructs individually suggests that control and commitment are more consistently associated with lower strain than is challenge. No study has provided compelling evidence that all three components are necessary to promote adaptive responses to stress. Third, the items on the initial hardiness scales were negatively keyed, raising questions about whether the scale was measuring the absence of maladaptive traits (e.g., neuroticism) rather than the presence of adaptive traits. Construct validity studies have demonstrated that hardiness scores are strongly correlated with neuroticism, and that some associations between hardiness and lower strain are reduced or eliminated when shared variance with neuroticism is statistically controlled. The most recent version of the hardiness scale – Personal Views Survey III-Revised (PVS III-R) – appears to have partially addressed these issues. However, the psychometric properties of this scale have not been published in a peer-reviewed journal.

Measurement of physical health outcomes. Another concern with the hardiness and health literature is that health outcomes are commonly measured with self-reported somatic complaints or other subjective signs of strain, rather than with objective signs of illness. Such outcomes are imperfect measures of health; they are heavily influenced by illness cognition and illness behavior processes that occur with heightened distress. The use of such health measures combined with the overlap between measures of hardiness and neuroticism have raised concerns that hardiness-health associations reflect shared variance with neuroticism. Few published studies have examined associations between hardiness and more objective signs of physical health (e.g., psychophysiological reactivity to stress; blood pressure; immune function; mortality), and those that exist have yielded inconsistent findings.

Evidence of stress buffering. Although developed in the context of work-related stress, the question of whether hardiness "buffers" the adverse health effects of stress is not fully answered. If hardiness works by buffering stress,

its associations with health outcomes should be stronger under high versus low stress conditions, as evidenced by a statistical interaction between hardiness and stress. Many studies have not been designed to test this stress-buffering hypothesis (e.g., hardiness is often tested among samples exposed only to high levels of stress), and those that have provide inconsistent support for stress buffering. Nevertheless, the consistent finding of adaptive perceptions of stress noted above suggests that hardiness may reduce one's level of psychological stress even in the face of objectively similar stressful life events.

Conclusion

The construct of hardiness continues to be studied in a variety of settings around the world, and interventions to increase levels of hardiness have been developed. This recent wave of hardiness research has focused on psychological strain and performance-based outcomes more than on physical health outcomes, but may provide answers to some of these ongoing controversies.

Cross-References

- ▶ Benefit Finding
- ▶ Biobehavioral Mechanisms
- ▶ Construct Validity
- ▶ Coping
- ▶ Coping Styles
- ▶ Health Behaviors
- ▶ Individual Differences
- ▶ Life Events
- ▶ Mediators
- ▶ Neuroticism
- ▶ Optimism, Pessimism, and Health
- ▶ Passive Coping Strategies
- ▶ Perceived Control
- ▶ Perceptions of Stress
- ▶ Personality
- ▶ Positive Psychology
- ▶ Posttraumatic Growth
- ▶ Problem-Focused Coping
- ▶ Psychological Stress
- ▶ Psychometric Properties
- ▶ Resilience
- ▶ Self-Report
- ▶ Social Support
- ▶ Somatic Symptoms
- ▶ Stress
- ▶ Stress: Appraisal and Coping
- ▶ Stressor
- ▶ Symptoms
- ▶ Work-Related Stress

References and Readings

Funk, S. C. (1992). Hardiness: A review of theory and research. *Health Psychology, 11*, 335–345.

Kobasa, S. C. (1979). Stressful life events, personality and health: An inquiry into hardiness. *Journal of Personality and Social Psychology, 37*, 1–11.

Kobasa, S. C., Maddi, S. R., & Kahn, S. (1982). Hardiness and health: A prospective study. *Journal of Personality and Social Psychology, 42*, 168–177.

Maddi, S. R. (2006). Hardiness: The courage to grow from stress. *The Journal of Positive Psychology, 1*, 160–168.

Maddi, S. R., & Khoshaba, D. M. (2001). *HardiSurvey III-R: Test development and internet instruction manual*. Irvine, CA: Hardiness Institute.

Wiebe, D. J., & Williams, P. G. (1992). Hardiness and health: A social psychophysiological perspective on stress and adaptation. *Journal of Social and Clinical Psychology, 11*, 238–262.

Harm Minimization

- ▶ Harm Reduction

Harm Reduction

Deborah Rinehart
Denver Health and Hospital Authority, Denver, CO, USA

Synonyms

Harm minimization; Risk reduction

Definition

Harm reduction is a public health framework that refers to policies, programs, and practices that focus on reducing potentially adverse health, social, and economic consequences related to engagement in high-risk behaviors. Harm reduction has been controversial as it focuses on preventing or reducing harm and not necessarily on preventing or eliminating risky behavior. Although it has been used in many different settings, harm reduction is most often associated with issues related to substance use and became a more prominent framework in the mid-1980s as a public health response to the HIV epidemic among injection drug users.

In the field of substance abuse, harm reduction provides an alternative to abstinence. The harm reduction framework recognizes that there are many people who are unable or unwilling to stop using illicit drugs. Subsequently, it focuses on reducing the societal and individual harms that may occur as a result of drug use. Needle exchange programs are an example of a harm reduction approach to HIV among injection drug users. These programs focus on providing clean syringes so that individuals who continue to inject drugs do not get infected with HIV. Through a nonjudgmental approach, education, and offering clean equipment, the programs strive to prevent adverse health outcomes (e.g., HIV infection) among injection drug users. These programs do not encourage or promote drug use but instead offer realistic options to individuals who are unable to quit their drug use. This framework has been proven to be effective in reversing and preventing the HIV epidemic among injection drug users (Des Jarlais, 2010).

The harm reduction framework encompasses multiple levels as policy, environments, and individual behaviors can all be targeted and modified to reduce harm. The framework acknowledges that risky behaviors occur along a continuum ranging from minimal risk to excessive risk. The goal is to identify feasible and realistic options along this continuum that can be adopted to reduce risk. Instead of seeking to criminalize or moralize behavior, harm reduction seeks to meet individuals in their current situation and identify ways to reduce the harmful outcomes to society and the individual that may be a result of engaging in risky behavior. According to a recent editorial in the International Journal of Drug Policy, harm reduction started as a public health strategy informed by social justice and over time has increasingly drawn attention to structural issues and the need to reform policy so that disenfranchised populations can avoid harm (Stimson & O'Hare, 2010).

Cross-References

▶ HIV Infection

References and Readings

(2010). Special issue: Commentaries on harm reduction: Looking back, look forward. *International Journal of Drug Policy* 21(2).

Des Jarlais, D. C. (2010). Learning from HIV epidemics among injection drug users. Harm reduction: moving through the third decade. *International Journal of Drug Policy, 21*, 97–99.

Harm Reduction Journal

Inciardi, J. A., & Harrison, L. D. (Eds.). (2000). *Harm reduction: National and international perspectives*. Thousand Oaks, CA: Sage.

Marlatt, A. (Ed.). (1998). *Harm reduction: Pragmatic strategies for managing high-risk behaviors*. New York: Guilford Press.

Stimson, G., & O'Hare, P. (2010). Harm reduction: Moving through the third decade. *International Journal of Drug Policy, 21*, 91–93.

Harmful Drinking

▶ Binge Drinking

Hayman, Laura L.

Laura L. Hayman
College of Nursing & Health Sciences, University of Massachusetts Boston, Boston, MA, USA

Biographical Information

Laura L. Hayman earned her BSN, MSN, and PhD at the University of Pennsylvania. Her program of research and scholarship focuses on primary prevention of obesity and cardiovascular disease (CVD) in children, adolescents, and families. Her research, in collaboration with colleagues from several disciplines, has included clinical, school, and population-based studies of biobehavioral risk factors for CVD. Her recent work combines both individual/clinical and community-based approaches to identifying children at risk for obesity and cardiometabolic conditions, and theory-based interventions designed to increase physical activity and promote healthy lifestyle behaviors.

Hayman has served on numerous national and international interdisciplinary advisory and expert panels relevant to primary prevention of obesity and CVD in childhood and adolescence. She has also served in leadership roles in the American Heart Association, the Society of Behavioral Medicine, and the Preventive Cardiovascular Nurses Association. She holds fellowships in the American Heart Association, the American Academy of Nursing, the Society of Behavioral Medicine, and the Academy of Behavioral Medicine Research.

Major Accomplishments

Christian R. & Mary F. Lindback Award for Distinguished Teaching, University of Pennsylvania, 1983

Katharine A. Lembright Award for Achievement in Cardiovascular Nursing Research, American Heart Association, 1997

Fellow, American Heart Association, Council on Cardiovascular Disease in the Young, and Council on Nutrition, Physical Activity and Metabolism, 2003

Member, Academy of Behavioral Medicine Research, 2006

C. Tracy Orleans Distinguished Service Award, Society of Behavioral Medicine, 2007

Distinguished Achievement Award, Council on Cardiovascular Nursing, American Heart Association, 2009

Spirit of Nursing Award, College of Nursing and Health Sciences, University of Massachusetts, Boston, 2010

National Meritorious Achievement Award, American Heart Association, 2010

References and Readings

Balagopal, P. B., de Ferranti, S. D., Cook, S., Daniels, S. R., Gidding, S. S., Hayman, L. L., McCrindle, B. W., Mietus-Snyder, M. L., Steinberger, J., & On behalf of the American Heart Association Committee on Atherosclerosis, Hypertension and Obesity in Youth of the Council on Cardivascular Disease in the Young, Council on Nutrition, Physical Activity and Metabolism, and Council of Epidemiology and Prevention. (2011). Nontraditional risk factors and biomarkers for cardiovascular disease: Mechanistic, research, and clinical considerations for youth: A scientific statement from the American Heart Association. *Circulation, 123*(23), 2749–2769.

Borawski, E. A., Trapl, E. S., Adams-Tufts, K., Hayman, L. L., Goodwin, M. A., Lovegreen, L. D., & Cole, M. L. (2009). Taking be proud! Be responsible! to the suburbs: A replication study. *Perspectives on Sexual and Reproductive Health, 41*(1), 12–22.

Daniels, S. R, Pratt, C. A., Hayman, L. L. (2011). Reduction of risk for cardiovascular disease in children and adolescents. *Circulation, 124*(5):1673–1687.

Erinosho, T., Dixon, L. B., Young, C., Brotman, L. M., Hayman, L. L. (2011). Nutrition practices and children's dietary intakes at 40 childcare centers in New York City. *Journal of the American Dietetic Association, 111*(9):1391–1397.

Estabrooks, P. A., Fisher, E. B., & Hayman, L. L. (2008). What is needed to reverse the trends in childhood obesity? A call to action for society of behavioral medicine. *Annals of Behavioral Medicine, 36*(3), 209–216.

Fisher, E. B., Fitzgibbon, M. L., Glasgow, R. E., Haire-Joshu, D., Hayman, L. L., Kaplan, R. M., Nanney, M. S., & Ockene, J. K. (2011). Behavior matters. *American Journal of Preventive Medicine, 40*(5), e15–e30.

Fletcher, G., Grundy, S., & Hayman, L. L. (Eds.). (1999). *Obesity: Impact on cardiovascular disease*. New York: Futura.

Glasgow, R. E., Green, L. W., Klesges, L. M., Abrams, D. B., Fisher, E. B., Goldstein, M. G., Hayman, L. L., Ockene, J. K., & Orleans, C. T. (2006). External validity: We need to do more. *Annals of Behavioral Medicine, 31*(2), 105–108.

Hayman, L. L. (2010). Starting young: Promoting a healthy lifestyle with children. *Journal of Cardiovascular Nursing, 25*(3), 228–232.

Hayman, L. L., Helden, L., Chyun, D. A., & Braun, L. T. (2011). A life course approach to cardiovascular disease prevention. *Journal of Cardiovascular Nursing, 26*(4), S22–S34.

Hayman, L. L., Mahon, M., & Turner, R. J. (Eds.). (2002a). *Health and behavior in childhood and adolescence*. New York: Springer.

Hayman, L. L., Mahon, M. M., & Turner, R. J. (Eds.). (2002b). *Chronic illness in children: An evidence-based approach*. New York: Springer.

Hayman, L. L., Meininger, J. C., Coates, P. M., & Gallagher, P. R. (1995). Nongenetic influences of obesity on risk factors for cardiovascular disease during two phases of development. *Nursing Research, 44*(5), 277–283.

Hayman, L. L., Meininger, J. C., Daniels, S. R., McCrindle, B. W., Helden, L., Ross, J., Dennison, B. A., Steinberger, J., & Williams, C. L. (2007). Primary prevention of cardiovascular disease in nursing practice: Focus on children and youth. *Circulation, 116*(3), 344–357.

Hayman, L. L., Meininger, J. C., Stashinko, E. E., Gallagher, P. R., & Coates, P. M. (1988). Type A behavior and physiological cardiovascular risk factors in school-age twin children. *Nursing Research, 37*(5), 290–296.

Hayman, L. L., Williams, C. L., Daniels, S. R., Steinberger, J., Paridon, S., Dennison, B., & McCrindle, B. (2004). Cardiovascular health promotion in the schools: A statement for health and education professionals and child health advocates from the Committee on Atherosclerosis, Hypertension, and Obesity in Youth (AHOY) of the Council on Cardiovascular Disease in the Young, American Heart Association. *Circulation, 110*(15), 2266–2275.

Maglione, J. L., & Hayman, L. L. (2009). Psychosocial correlates of physical activity in low-income college students. *Research in Nursing and Health, 32*, 634–646.

Meininger, J. C., Hayman, L. L., Coates, P. M., & Gallagher, P. (1988). Genetics or environment? Type A behavior and other cardiovascular risk factors in twin children. *Nursing Research, 37*(6), 341–346.

Meininger, J. C., Hayman, L. L., Coates, P. M., & Gallagher, P. R. (1998). Genetic and environmental influences on cardiovascular disease risk factors in adolescents. *Nursing Research, 47*(1), 11–18.

Shi, L., Morrison, J. A., Wiecha, J., Horton, M., & Hayman, L. L. (2011). Healthy lifestyle factors associated with reduced cardiometabolic risk. *British Journal of Nutrition, 105*, 747–754.

HbA1c

Luigi Meneghini
Diabetes Research Institute, University of Miami, Miami, FL, USA

Synonyms

A1C; Glycated hemoglobin; Glycosylated hemoglobin; Hemoglobin A1c

Definition

HbA1c, or glycosylated hemoglobin, is a measure of how much glucose is irreversibly bound (glycated) to hemoglobin, and can be used to assess the degree of exposure to glycemia in the preceding 2–3 months (corresponding to the life span of the red blood cell where hemoglobin is contained). In a person with normal blood glucose levels, the amount of glycated hemoglobin is around 4–6%, representing an average blood glucose level between 70 and 120 mg/dl. In individuals with diabetes, HbA1c can be measured

every 3 months with a goal of keeping the value as close to normal as possible, or at least under 7% in most patients. The higher the HbA1c, the greater the risk over time (usually measured in years) of developing microvascular complications, such as diabetic retinopathy, nephropathy, and neuropathy. HbA1c remains the best predictor for future diabetes-related chronic complications that is available in the clinical setting.

Cross-References

- ▶ Diabetes
- ▶ Hyperglycemia

References and Readings

Joslin, E. P., & Kahn, C. R. (2005). *Joslin's diabetes mellitus* (14th ed.). Philadelphia: Lippincott Williams & Willkins.

Head Injury

▶ Traumatic Brain Injury

Headache with Aura

▶ Migraine Headache

Headaches, Types of: Cluster, Migraine, and Tension

Hiroe Kikuchi
Department of Psychosomatic Research, National Institute of Mental Health, National Center of Neurology and Psychiatry, Tokyo, Japan

Definition

Cluster headache, migraine, and tension-type headache are three major types of primary headaches. Primary headaches are headaches with no apparent underlying organic disease process.

Description

The International Classification of Headache Disorders, 2nd edition (Headache Classification Subcommittee of the International Headache Society, 2004), is a widely used classification of headaches, and it contains diagnostic criteria for headaches. Cluster headache, migraine, and tension-type headache are classified as primary headaches, and their diagnosis is based on the pain characteristics and associated symptoms. Generally, neuroimaging is not necessary for the diagnosis; however, it is considered to exclude underlying abnormalities of the brain in some cases. Assessment of psychosocial aspects is also important especially in migraine and tension-type headache because psychosocial factors can be precipitating and aggravating factors of headache and headache may affect psychosocial condition.

In these headaches, treatment consists of acute therapy and prophylactic therapy. Prophylactic therapy is important because frequent use of analgesics places patients at risk for medication overuse headache.

Cluster Headache

Cluster headache is a headache which is severe, strictly unilateral, and orbital, supraorbital, or temporal in location. Attacks usually occur in series for a period of weeks or months which is called a cluster period. Cluster periods are separated by remission periods which are usually months to years. The attack lasts 15–180 min, and its frequency ranges from once per 2 days to eight times a day. The attack accompanies ipsilateral autonomic symptoms such as ptosis, miosis, lacrimation, conjunctival injection, rhinorrhoea, nasal congestion, and forehead and facial sweating. Pain is severe enough to disturb daily activities, and most patients are restless or agitated during attack.

The prevalence of cluster headache is less than 1%, and prevalence is three to four times higher in men than in women (May, 2005).

Pathophysiological involvement of hypothalamus is suggested by time pattern of attacks. Neurovascular factors are also important.

Acute therapy for cluster headache includes inhalation of pure oxygen and triptan. Subcutaneous injection and nasal spray are preferable to oral administration. As preventive therapy for cluster headache, verapamil is established. Lithium, methysergide, and corticosteroids are also used. Non-pharmacological treatment is generally ineffective.

Migraine

Migraine is further classified into two major subtypes: migraine without aura and migraine with aura.

Migraine without aura is recurrent headache disorder whose pain is generally unilateral, pulsating (throbbing), moderate to severe in intensity, aggravated by daily physical activities, and accompanied by nausea and/or photophobia and phonophobia. The attack lasts 4–72 h, and its median frequency is 1.5 per month. Migraine with aura is characterized by a complex of reversible focal neurological signs (visual, sensory, motor, or speech signs) which gradually progresses in 5–20 min and last for less than 60 min generally before headache. Typically, headache of the same quality as migraine without aura follows aura, but sometimes, the quality of headache is different and even headache can be absent. Symptoms such as fatigue, difficulty in concentrating, neck stiffness, sensitivity to light or sound, nausea, blurred vision, yawning, pallor, or emotional lability sometimes occur several hours to 2 days prior to migraine (either with or without aura), and they are called premonitory symptoms. Migraine may be aggravated (i.e., increased in the severity or frequency in a relatively long term) by psychosocial stress, frequent intake of alcohol, and other environmental factors. An attack may be triggered by menstruation, chocolate, etc.

Prevalence has been reported to be between 5% and 25% in women and 2% and 10% in men.

Trigeminovascular theory is now a broadly accepted pathophysiological mechanism of migraine (Silberstein, 2004). Perivascular trigeminal terminals are stimulated by certain causes and vasoactive substances such as calcitonin gene-related peptide (CGRP) are released. Vessels dilate and neurogenic inflammation occurs, which leads to pain and accompanying symptom such as nausea. Central pain modulation is also thought to be involved. Cortical spreading depression is associated with aura.

Acute therapy for migraine consists of specific (triptans and ergots) and nonspecific (analgesics) (Silberstein, 2004). Triptans are $5HT_{1B/1D}$ receptor agonists and have effects of vasoconstriction and inhibition of vasoactive substances release and of neurogenic inflammation. Prophylactic therapy includes calcium channel blocker, beta-blocker, ergots, antidepressants, and anticonvulsants (Silberstein, 2004). Refraining from drinking alcohol and eating certain foods (chocolate, cheese, etc.) may also be effective for prevention of migraine attacks. Relaxation therapy, thermal and electromyography biofeedback, and cognitive behavior therapy are also used as prophylactic therapy.

Tension-Type Headache

Tension-type headache typically causes pain which is bilateral, pressing or tightening, and mild to moderate in intensity, and is not aggravated by daily physical activities. Although anorexia may accompany, neither nausea nor vomiting does. Photophobia and phonophobia can coexist.

Tension-type headache is the most common type of primary headache, and its life prevalence estimates range from 30% to 78%, and tension-type headache is slightly more in women than in men (Headache Classification Subcommittee of the International Headache Society, 2004). Its prevalence is most at 40s (Loder & Rizzoli, 2008).

Increased muscle tension was formerly thought to be a major cause of tension-type headache; however, now it is thought that peripheral factor (hypersensitivity to pain in the head and neck tissue) plays a major role in less frequent headache (i.e., infrequent and frequent episodic tension-type headache), while central factor

(alteration of pain sensitivity in the central nervous system) plays a major role in more frequent headache (chronic tension-type headache).

Acute therapy for tension-type headache is analgesics (nonsteroidal anti-inflammatory drugs (NSAIDs) and acetaminophen). Over-the-counter analgesics are commonly used. As pharmacological prophylactic therapy, amitriptyline is the most widely researched (Millea & Brodie, 2002). With less evidence, other antidepressants such as selective serotonin reuptake inhibitors (SSRIs) and tizanidine are considered as prophylactic therapy. Relaxation therapy, electromyography biofeedback, and cognitive behavior therapy are also used for prophylaxis.

Cross-References

▶ Headaches: Psychological Management

References and Readings

Headache Classification Subcommittee of the International Headache Society. (2004). The international classification of headache disorders: 2nd edition. *Cephalalgia, 24*(Suppl. 1), 9–160.

Loder, E., & Rizzoli, P. (2008). Tension-type headache. *British Medical Journal, 336*, 88–92.

May, A. (2005). Cluster headache: Pathogenesis, diagnosis, and management. *The Lancet, 366*, 843–855.

Millea, P. J., & Brodie, J. J. (2002). Tension-type headache. *American Family Physician, 66*, 797–804.

Silberstein, S. D. (2004). Migraine. *The Lancet, 363*, 381–391.

Headaches: Psychological Management

Hiroe Kikuchi
Department of Psychosomatic Research, National Institute of Mental Health, National Center of Neurology and Psychiatry, Tokyo, Japan

Definition

Psychological management of headache includes assessment of psychosocial aspects of headache, screening and treating psychiatric comorbidity, and application of psychological treatments to manage pain.

Description

Psychological management of headache has been researched mostly in tension-type headache and migraine.

When diagnosing headache, assessment of psychosocial aspects is also important because psychosocial factors can be precipitating and aggravating factors of headache and headache may affect psychosocial condition. In both migraine and tension-type headache, 50–80% of patients report that psychological stress is a precipitating or aggravating factor of headache according to some reports. Identifying individual precipitating or aggravating factors is fundamental for the prophylaxis of headache. In both migraine and tension-type headache, anxiety and depressive mood are reported to be higher than healthy controls and social activities are also affected. Overall assessments of those psychosocial conditions are necessary to understand the burden of headaches.

Comorbidity with mood disorder and anxiety disorder is also reported to be high in both migraine and tension-type headache (Holroyd, 2002). In addition, psychiatric comorbidity is reported to be a possible risk factor for chronification of headache. Therefore, screening and treating psychiatric comorbidity is also necessary for headache management.

Representative psychological treatments of headache are relaxation therapy, biofeedback therapy, and cognitive behavioral therapy (Holroyd, 2002).

In tension-type headache, relaxation therapy in the form of progressive muscle relaxation and autogenic training, electromyographic biofeedback therapy (reducing muscle activity in forehead or neck and shoulder muscles), and cognitive behavioral therapy are used. Although relaxation therapy alone is suggested to be effective, it is reported that the percentage of patients

whose headache was improved was increased when biofeedback therapy was added. Cognitive behavioral therapy is also conducted in combination with other therapies as well as alone. Cognitive behavior therapy increases the effectiveness of relaxation therapy when it is added to relaxation therapy.

In migraine, relaxation therapy in the form of progressive muscle relaxation and autogenic training, thermal biofeedback therapy (warming hand), and cognitive behavioral therapy are generally thought to be treatment options for prevention of migraine as psychological treatment. However, it is reported that cognitive behavioral therapy did not appear to enhance the effectiveness of relaxation therapy or thermal biofeedback.

In cognitive behavioral therapy, it is assumed that irrational cognition and maladaptive behavior underlie pain, psychological stress, and mood disturbances and those cognition and behavior are the targets of intervention. Usually, treatment program comprising several components is conducted and relaxation therapy is often included. Treatment aims at achieving adoptive coping behavior to pain as well as approaching psychosocial factors. Psychological treatments have a feature that they all aim at self-control in common. There is not any recommendation about which of these psychological treatment to choose for specific patients.

Psychological treatment is usually used either with or without medication. In chronic tension-type headache, the combination of cognitive behavioral therapy and tricyclic antidepressants are reported to possibly improve outcome relative to monotherapy.

The mechanism of how these psychological treatments improve headache is still unclear. Previous studies that showed the effect of psychological treatment on headache was not limited to patients with psychiatric comorbidity, and it is not likely that the improvement of comorbid psychiatric disorders fully mediates the improvement of headache.

Cross-References

▶ Headaches, Types of: Cluster, Migraine, and Tension

References and Readings

Holroyd, K. A. (2002). Assessment and psychological management of recurrent headache disorders. *Journal of Consulting and Clinical Psychology, 70*, 656–677.

Health

▶ Well-Being: Physical, Psychological, Social

Health Anxiety

Tamer F. Desouky[1], Lisa M. McAndrew[2] and Pablo A. Mora[1]
[1]Department of Psychology, The University of Texas at Arlington, Arlington, TX, USA
[2]Department of Veterans Affairs, NJ Healthcare System, East Orange, NJ, USA

Synonyms

Health phobia; Hypochondriasis

Definition

Health anxiety refers to an excessive concern or preoccupation about being ill based on the misinterpretation of somatic symptoms despite medical reassurance indicating otherwise.

Description

Health anxiety refers to an excessive concern or preoccupation about the meaning and potential

consequences of somatic symptoms. Individuals high in health anxiety are more likely to believe that their physical symptoms are signs of a serious disease than their low anxious counterparts despite medical reassurance indicating otherwise.

Several environmental, biological, and psychological (e.g., behavioral and cognitive) factors have been implicated as causes of health anxiety (Abramowitz & Braddock, 2008; Kirmayer & Looper, 2006). Evidence from research examining the environmental antecedents of health anxiety indicate that individuals, especially females, who have experienced serious illness as children, suffered the death of a loved one from a devastating medical condition, or were victims of physical, sexual, and/or emotional abuse in childhood are at higher risk of developing health anxiety when compared to individuals without a history of traumatic experiences (Stein et al., 2004). Another environmental risk factor is parental modeling of illness behaviors via observational learning (Mineka & Ben Hamida, 1998). For instance, children can learn from overprotective parents that any somatic symptom is a sign of a serious disease which can result in the regular use of emergency and/or primary care clinics to care for nonthreatening symptoms. Finally, informational transmission (e.g., media and Internet) can serve as a trigger for health anxiety and health anxious-related behaviors (Abramowitz & Braddock, 2008; Rachman, 1991). Examples of this include mass psychogenic illness, mass anxiety hysteria, "Koro" in Southeast Asia, and medical student's syndrome.

Family and twin studies suggest that heritability may play a role in the development of health anxiety and hypochondriasis; however, evidence about the specific genes involved is inconclusive (Smoller, Gardner-Schuster, & Covino, 2008). Research on the neurobiological basis of health anxiety suggests that hyperactivity in the amygdala and limbic regions and an inability of higher cortical executive areas to inhibit limbic responses may be involved in initiating and maintaining anxiety episodes (Martin, Ressler, Binder, & Nemeroff, 2010). Additionally, neuropharmacological studies have shown that either decreased inhibitory control utilizing GABA, or an increase of the excitatory neurotransmitter Glutamate, or a synergistic combination of both systems play key roles in the development of health anxiety. It must be noted, however, that these neurobiological antecedents are not unique to health anxiety but rather shared by various anxiety disorders.

The identification and understanding of psychological antecedents of health anxiety have received wide attention from researchers (Marcus, Gurley, Marchi, & Bauer, 2007). Individuals high in health anxiety have been found to: (1) have catastrophic beliefs about their somatic symptoms, (2) be more sensitive to and aware of their somatic symptoms (i.e., somatosensory amplification), and (3) be more likely to interpret their somatic symptoms as signs of a serious disease. Health-anxiety beliefs can be elicited by various triggers such as benign physical symptoms, nonthreatening disease, and hearing or reading about illnesses from different sources (e.g., friends, media). Once triggered, these beliefs are responsible for maintaining a feedback loop that results in automatic hypochondriacal thoughts which, in turn, increase attention to somatic sensations that may confirm illness (i.e., confirmatory bias). Increased somatic vigilance compounded with a confirmatory bias help perpetuate health anxiety.

Health anxiety is best understood as a continuum ranging from mild to severe. Although many people have benign health anxiety, it can become extreme and result in hypochondriasis, a somatoform disorder (Faravelli et al., 1997; Looper & Kirmayer, 2001). The prevalence of hypochondriasis in the general population (0.2%) is low, while nonclinical levels of health anxiety (6%) are more common (Looper & Kirmayer, 2001). To diagnose an individual with hypochondriasis, he or she must believe that his/her somatic symptoms result from a serious disease despite medical reassurance. These symptoms must last a minimum of 6 months and cause significant

distress or interfere with social, occupational, or other forms of functioning (American Psychiatric Association [APA], 2000). Hypochondriasis is typically treated with a combination of cognitive behavioral therapy and psychotropic medication (e.g., SSRI, Pimozide, and Clomipramine).

Health anxiety can be assessed with different tools including self-reports and structured clinical interviews. The most widely used self-reports include the Short Health Anxiety Inventory (Salkovskis, Rimes, Warwick, & Clark, 2002), the Illness Behavior Questionnaire (Pilowsky & Spence, 1994), and the Health Anxiety Questionnaire (Lucock & Morley, 1996). Structured clinical interviews include the Composite International Diagnostic Interview (World Health Organization [WHO], 1990) and Structured Clinical Interview for DSM disorders (First, Spitzer, Gibbon, & Williams, 1995). Clinical interviews should be used when the goal of assessment is the diagnosis of clinical levels of health anxiety.

Cross-References

- ▶ Anxiety
- ▶ Anxiety and Heart Disease
- ▶ Hypochondriasis
- ▶ Neuroticism
- ▶ Pain Anxiety

References and Readings

Abramowitz, J. S., & Braddock, A. E. (2008). *Psychological treatment of health anxiety & hypochondriasis: A biopsychosocial approach*. Cambridge, MA: Hogefe & Huber.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders (Rev. 4th ed.)*. Washington, DC: American Psychiatric Press.

Asmundson, G. J. G., Taylor, S., & Cox, B. J. (2001). *Health anxiety: Clinical perspectives on hypochondriasis and related conditions*. West Sussex, England: Wiley.

Barsky, A. J., & Klerman, G. L. (1983). Overview: Hypochondriasis, bodily complaints, and somatic styles. *The American Journal of Psychiatry, 140*, 273–281.

Faravelli, C., Salvatori, S., Galassi, F., Aiazzi, L., Drei, C., & Cabrasm, O. (1997). Epidemiology of somatoform disorders: a community survey in Florence. *Social Psychiatry and Psychiatric Epidemiology, 32*, 24–29.

First, M. B., Spitzer, R. L., Gibbon, M. N., & Williams, J. B. W. (1995). *Structured clinical interview for DSM-IV*. New York: New York State Psychiatric Institute, Biometrics Research Department.

Forsyth, J. P., Barrios, V., & Acheson, D. T. (2007). Exposure therapy and cognitive interventions for the anxiety disorders: Overview and newer third-generation perspectives. In D. C. S. Richard & D. L. Lauterbach (Eds.), *Handbook of exposure therapies*. San Diego: Academic Press.

Kellner, R. (1986). *Somatization and hypochondriasis*. New York: Praeger-Greenwood.

Kellner, R. (1987). *Abridged manual of the illness attitude scales*. Unpublished manual, Albuquerque: Department of Psychiatry, School of Medicine, University of New Mexico.

Kirmayer, L. J., Groleau, D., Looper, K. J., & Dao, M. D. (2004). Explaining medically unexplained symptoms. *Canadian Journal of Psychiatry, 49*(10), 663–672.

Kirmayer, L. J., & Looper, K. J. (2006). Abnormal illness behaviour: Physiological, psychological and social dimensions of coping with distress. *Current Opinion in Psychiatry, 19*(1), 54–60.

Kirmayer, L. J., & Sartorius, N. (2007). Cultural models and somatic syndromes. *Psychosomatic Medicine, 69*(9), 832–840.6.

Looper, K. J., & Kirmayer, L. J. (2001). Hypochondriacal concerns in a community population. *Psychological Medicine, 31*(4), 577–584.

Lucock, M. P., & Morley, S. (1996). The health anxiety questionnaire. *British Journal of Health Psychology, 1*, 137–150.

Marcus, D. K., Gurley, J. R., Marchi, M. M., & Bauer, C. (2007). Cognitive and perceptual variables in hypochondriasis and health anxiety: A systematic review. *Clinical Psychology Review, 27*(2), 127–139.

Martin, E. I., Ressler, K. J., Binder, E., & Nemeroff, C. B. (2010). The neurobiology of anxiety disorders: Brain imaging, genetics, and psychoneuroendocrinology. *Clinics in Laboratory Medicine, 30*(4), 865–891.

Mineka, S., & Ben Hamida, S. (1998). Observational and nonconscious learning. In W. T. O'Donohue (Ed.), *Learning and behavior therapy*. Needham Heights, MA: Allyn & Bacon.

Pilowsky, I. (1967). Dimensions of hypochondriasis. *The British Journal of Psychiatry, 113*, 89–93.

Pilowsky, I., & Spence, N. D. (1994). *Manual for the illness behavior questionnaire* (3 rd ed.). Unpublished manual, Adelaide, South Australia: Department of Psychiatry, University of Adelaide

Rachman, S. (1991). Neo-conditioning and the classical theory of fear acquisition. *Clinical Psychology Review, 11*(2), 155–173. doi:10.1016/0272-7358(91)90093-A.

Salkovskis, P. M., Rimes, K. A., Warwick, H. M., & Clark, D. M. (2002). The health anxiety

inventory: Development and validation of scales for the measurement of health anxiety and hypochondriasis. *Psychological Medicine, 32*, 843–853.
Smoller, J. W., Gardner-Schuster, E., & Covino, J. (2008). The genetic basis of panic and phobic anxiety disorders. *American Journal of Medical Genetics. Part C, Seminars in Medical Genetics, 148C*(2), 118–126. doi:10.1002/ajmg.c.3017.
Stein, M. B., Lang, A. J., Laffaye, C., Satz, L. E., Lenox, R. J., & Dresselhaus, T. R. (2004). Relationship of sexual assault history to somatic symptoms and health anxiety in women. *General Hospital Psychiatry, 26*(3), 178–183. doi:10.1016/j.genhosppsych.2003.11.003.
Warwick, H. M., & Salkovskis, P. M. (1990). Hypochondriasis. *Behaviour Research and Therapy, 28*, 105–117.
World Health Organization. (1990). *Composite international diagnostic interview (CIDI)*. Geneva, Switzerland: Author.

Health Assessment

► Physical Examination

Health Assessment Questionnaire

Bonnie Bruce
Division of Immunology and Rheumatology, Stanford University Department of Medicine, Palo Alto, CA, USA

Synonyms

Activities of daily life assessment; Disability assessment; Self-reported patient outcome measure

Definition

The Health Assessment Questionnaire (HAQ) was developed three decades ago by James F. Fries, MD, and colleagues at Stanford University (Fries, Spitz, Kraines, & Holman, 1980) as a model of patient-reported outcome (PRO) assessment for assessing physical function. Three reviews examined the HAQ's history, its reliability, validity, and applicability (Bruce and Fries, 2003; Ramey, Fries, & Singh, 1995; Ramey, Raynauld, & Fries, 1992).

The HAQ has been administered globally and validated in patients with a wide variety of rheumatic diseases, HIV/AIDS, and in studies of normal aging, in diverse disciplines and different cultures, and in dozens of languages without impacting reliability or validity with properly designed adaptations. The HAQ is usually self-administered. However, it can be administered face-to-face or over telephone by a trained interviewer. Further, the HAQ has been validated for Internet administration (Bruce, Fries, & Lingala, 2011). The HAQ is available online (The Arthritis, Rheumatism, and Aging Medical Information System, 2011).

The original HAQ was developed using classical test theory methodology, is sensitive to change, and a good predictor of future disability and costs. However, it did not benefit from use of modern psychometric approaches. Modern methods, such as Item Response Theory (IRT) (Embertson & Reise, 2000), which quantitatively assess item properties, enable development of more precise instruments (Rose, Bjorner, Becker, & Fries, 2008).

Recently, items in the HAQ, along with the SF-36's PF-10, have undergone extensive revamping using both classical and IRT methods as part of the Patient-Reported Outcomes Measurement Information System (PROMIS) (Reeve et al., 2007). PROMIS is part of the National Institutes of Health (NIH) Roadmap Initiative aimed at re-engineering the clinical research enterprise. Work in PROMIS resulted in a 20-item revised HAQ and the IRT-derived PROMIS PF-20, both of which more precisely measure physical function and are available for use on the PROMIS website (http://www.nihroadmap.nih.gov) (US Department of Health and Human Services, 2011). Investigation of the psychometric functions showed that instruments utilizing these items are more patient-centered, more validly translatable, and have better clarity in diversely educated groups. In addition, they also show responsiveness and precision that is

better than the parent instruments, the original HAQ and PF-10 (Fries, Krishnan, Rose, Lingala, & Bruce 2011).

Cross-References

- ▶ Health Economics
- ▶ SF-36

References and Readings

Bruce, B., & Fries, J. (2003). The Stanford Health Assessment Questionnaire (HAQ) a review of its history, issues, progress, and documentation. *Journal of Rheumatology, 30*(1), 167–178.

Bruce, B., Fries, J.F., & Lingala, B. (2011) Internet versus Mailed Administration of the Health Assessment Questionnaire Disability Index (HAQ). *Arthritis and Rheumatism*, (Abstract, in Press).

Embretson, S. E., & Reise, S. P. (2000). *Item response theory for psychologists*. London: Lawrence Erlbaum.

Fries, J. F., Krishnan, E., Rose, M., Lingala, B., & Bruce, B. (2011). Improved responsiveness and reduced sample size requirements of PROMIS physical function scales with item response theory. *Arthritis Research & Therapy, 13*(5), R147.

Fries, J. F., Spitz, P., Kraines, R. G., & Holman, H. R. (1980). Measurement of patient outcome in arthritis. *Arthritis and Rheumatism, 23*(2), 137–145.

Ramey, D., Fries, J., & Singh, G. (1995). The Health Assessment Questionnaire 1995 – Status and review. In B. Spilker (Ed.), *Quality of life and pharmacoeconomics in clinical trials* (2nd ed., pp. 227–237). Philadelphia: Lippincott-Raven.

Ramey, D. R., Raynauld, J. P., & Fries, J. F. (1992). The Health Assessment Questionnaire 1992: Status and review. *Arthritis Care and Research, 5*(3), 119–129.

Reeve, B. B., Hays, R. D., Bjorner, J. B., Cook, K. F., Crane, P. K., Teresi, J. A., Thissen, D., et al. (2007). Psychometric evaluation and calibration of health-related quality of life item banks: Plans for the patient-reported outcomes measurement information system (PROMIS). *Medical Care, 45*(5 Suppl 1), S22–S31.

Rose, M., Bjorner, J. B., Becker, J., & Fries, J. F. (2008). Preliminary evaluations of a physical function item bank support the methods and advantages of the patient reported outcomes measurement information system (PROMIS). *Journal of Clinical Epidemiology, 61*, 17–33.

The Arthritis, Rheumatism, and Aging Medical Information System (2011) ARAMIS: HAQ. Retrieved November 17, 2011, from http://aramis.stanford.edu/HAQ.html

US Department of Health and Human Services. National Institute of Health. Division of Program Coordination. *Patient-Reported Outcome Measurement Information System (PROMIS)®*. Retrieved November 17, 2011, from https://commonfund.nih.gov/promis/

Health Behavior Change

- ▶ Behavior Change
- ▶ Behavior Change Techniques
- ▶ Behavioral Medicine
- ▶ Health Behaviors
- ▶ Health Communication
- ▶ Health Education
- ▶ Health Promotion and Disease Prevention
- ▶ Lifestyle, Modification
- ▶ Population Health

Health Behavior Predictors

- ▶ Psychosocial Predictors

Health Behavior Variables

- ▶ Psychosocial Variables

Health Behaviors

- ▶ Aerobic Exercise
- ▶ Alcohol Consumption
- ▶ Alcohol Abuse and Dependence
- ▶ Healthy Eating
- ▶ Illness Behavior
- ▶ Lifestyle
- ▶ Lifestyle, Healthy
- ▶ Meditation
- ▶ Medication Compliance
- ▶ Relaxation: Techniques/Therapy
- ▶ Tobacco Cessation
- ▶ Tobacco Use

Health Beliefs

▶ Beliefs
▶ Illness Cognitions and Perceptions

Health Beliefs/Health Belief Model

Tana M. Luger
Department of Psychology, University of Iowa, Iowa City, IA, USA

Definition

Rosenstock's Health Belief Model (HBM) is a theoretical model concerned with health decision-making. The model attempts to explain the conditions under which a person will engage in individual health behaviors such as preventative screenings or seeking treatment for a health condition (Rosenstock, 1966).

Description

Under the HBM, a person's likelihood for health behavior is assumed to be related to four main variables. First, action is more likely if the person perceives himself to be *susceptible* to or at risk for the condition. For example, if Lucy has a history of breast cancer in her family, she may see herself as more susceptible to developing breast cancer, and thus, be more likely to get a mammogram each year. Second, the likelihood for action depends on the *perceived seriousness* of the condition. Seriousness may be judged based on the amount of emotional arousal produced by thinking about the condition as well as the anticipated physical, social, and psychological consequences of developing the condition. For example, Lucy's mother passed away from breast cancer so she deems it to be a serious condition requiring preventative action. Third, the *perceived benefits* of performing the action are considered. Lucy considers the effectiveness of a mammogram in detecting breast cancer when determining whether to get the screening. Finally, the *perceived barriers* of performing the action are weighed. Lucy knows that a mammogram can be uncomfortable and scheduling an appointment is inconvenient. However, for Lucy, the benefits outweigh the barriers. Additional modifying variables like age and sex have been introduced with the assumption that they influence the above beliefs.

The variables of HBM are intended to measure a person's psychological readiness or intentions to act (Kirscht, 1988), and on the whole, research has found the HBM to be predictive of people's individual health behaviors (Janz & Becker, 1984). Self-reported susceptibility, benefits, barriers, and severity were shown to be correlated with health behavior outcomes such as attending preventative screening, seeking medical care, and utilizing health clinics. However, the HBM has been unable to consistently predict adherence to a medical treatment regimen or terminating an unhealthy behavior such as smoking (Kirscht, 1988). Additionally, while the variables of HBM may measure a person's individual level of readiness, the optimal level of readiness for health behavior change is still unknown.

Further critiques state that HBM ignores self-efficacy (a person's belief that he has control over a particular behavior) which has been shown to play a large role in behavior change (Kirscht, 1988). Thus, while the predictive validity of HBM with regard to health behaviors seems firm, the usefulness of focusing only on HBM factors in interventions has been contested (Davidhizar, 1983; Kirscht, 1988).

Cross-References

▶ Adherence
▶ Health Behaviors
▶ Self-efficacy

References and Readings

Davidhizar, R. (1983). Critique of the health-belief model. *Journal of Advanced Nursing, 8*, 467–472.

Janz, N. K., & Becker, M. H. (1984). The health belief model: A decade later. *Health Education Quarterly, 11*, 1–47.
Kirscht, J. P. (1988). The health belief model and predictions of health actions. In D. S. Gochman (Ed.), *Health behavior: Emerging research perspectives* (pp. 27–41). New York: Springer.
Rosenstock, I. M. (1966). Why people use health services. *Milbank Memorial Fund Quarterly, 44*, 94–127.
Rosenstock, I. M. (1974). Historical origins of the health belief model. *Health Education Monographs, 2*, 328–335.

Health Care

Peter Allebeck
Department of Public Health Sciences, Karolinska Institute, Stockholm, Sweden

Definition

Health care is a general term comprising services provided to improve health in the general population as well as to cure diseases and relieve symptoms in diseased patients. Health care may denote the organization of services (e.g., private vs public health care), a facility (e.g., hospital or health care center), as well as the actual delivery of care (e.g., to provide health care or to obtain health care). The term may comprise preventive services, such as vaccination, and mother and child care as well as curative services.

Health Care Access

Peter Allebeck
Department of Public Health Sciences, Karolinska Institute, Stockholm, Sweden

Definition

Health care access is the extent to which patients and groups have access to health care. It depends on factors related to general living conditions in society, and to organization of health services as well as factors in individuals. General conditions comprise availability overall of economic resources and how they are distributed, manpower for health care, and availability of health care facilities and transport systems to reach them. Organization of health services comprises how they are organized in primary and secondary care, adequate staff at various levels, geographical distribution, and insurance system to cover costs. Factors in individuals and groups are related to their knowledge about health care services, capacity to pay for fees and transport, and knowledge of the language and other cultural codes needed to access health care.

Health Care Costs

▶ Health Economics

Health Care System

Peter Allebeck
Department of Public Health Sciences, Karolinska Institute, Stockholm, Sweden

Definition

The concept “health system” has been developed and defined by the World Health Organization (WHO), and is now widely used internationally by scientists and policy makers. The WHO defines health system as “all organizations, people, and actions whose primary intent is to promote, restore, or maintain health.” The concept thus includes not only public and private health care facilities and staff, but also health insurance organization, water control, occupational health, and safety legislation. The WHO has described a health system framework consisting of six building blocks: Service delivery; Health workforce; Information; Medical workforce, vaccines, and technologies, Financing, and Leadership/Governance.

Cross-References

▶ Health Insurance: Comparisons

References and Readings

WHO. (2007). *Everybody's business: Strengthening health systems to improve health outcomes*. Geneva: Author.

World Health Report. (2000). *Health systems: Improving performance*. Geneva: WHO.

Health Care Utilization

Olveen Carrasquillo
Division of General Medicine, Miller School of Medicine, University of Miami, Miami, FL, USA

Definition

Health Care Utilization is the quantification or description of the use of services by persons for the purpose of preventing and curing health problems, promoting maintenance of health and well-being, or obtaining information about one's health status and prognosis.

Description

Health Care Utilization refers to the use of health care services. People use health care for many reasons including preventing and curing health problems, promoting maintenance of health and well-being, or obtaining information about their health status and prognosis.

Utilization is often reported in a variety of different methods:

1. The number of services used over a period of time divided by a population denominator (e.g., in 2008, there were 320.1 ambulatory Care Visits to Physicians' Offices per 100 persons living in the USA).
2. The percentage of persons who use a certain service over individuals eligible for that service in a period of time (in the USA in 2008, 75% of all women aged 18 years and over reported having a Pap smear in the last 3 years).
3. An aggregate number without a denominator (in 2008, there were 39.9 million discharges from US hospitals).

Health care utilization can vary by many factors. Sociodemographically persons at extremes of age (very old or very young) have higher health care utilization. Women also have higher utilization than men, partially explained by need for obstetric and gynecologic care. By race and ethnicity, members of minority groups have lower utilization of certain health care services. For example, despite a higher burden of cardiovascular disease, after adjusting for factors that predict utilization, African-Americans are less likely to receive invasive cardiac procedures. Latinos are less likely to have colorectal cancer screening tests. By health status, persons with poorer health have higher utilization.

However, of all the factors that drive utilization, perceived need for health care by the patient is probably the single most important independent factor. In addition, since many people may not fully know how medical conditions are prevented, diagnosed, or treated, perceived need also includes the perceptions on what health care is needed for a particular person by their providers and others who make health care recommendations to patients. In addition to need, there are many other factors that also impact utilization. Often these are conceptualized as predisposing, enabling, and need related factors. Examples of predisposing factors include a person's propensity to seek care as well as cultural norms on health care seeking behaviors. Ability to pay or health care coverage is the most important enabling factor. However, other important enablers include accessibility and location of services, language and cultural barriers, and availability of resources to appropriately provide such services.

Data on utilization can be gathered and compiled from various sources. One is administrative or claims data collected from those delivering

health care services or serving as payers of those health care services (such as insurers). An example is data on the number of cardiac catheterizations performed among Medicare beneficiaries which can be examined from the Medicare Provider Analysis and Review (MEDPAR) files. Data can also be collected from providers using surveys. An example of this type of utilization data is the CDC's National Ambulatory Medical Care Survey in which a representative sample of office-based providers are queried to provide data on health care services delivered over a 1-week period. Another example is the Nationwide Inpatient Sample containing discharge abstracts from a 20% stratified sample of US community hospitals (part of AHRQ's Healthcare Cost and Utilization Project). A limitation of these methods is that collecting and compiling accurate such data through these approaches can be resource intensive, particularly in countries with multi-payer and delivery systems. Also, it will not capture services delivered outside the health care sector being sampled. An example is data on alternative medicine.

Another approach used in many countries to collect information on healthcare utilization is through population-level surveys using self-reported data from patients themselves. In the USA, examples are the CDC's National Health Interview Survey and AHRQ's Medical Expenditure Panel Survey. Since these are based on patient self-reports, accuracy is always a concern. Thus, careful attention needs to be paid in design, collection, and analysis of data from such surveys so that the data presented is valid and accurate.

Utilization data is used for a variety of purposes. Cross-sectional data can be used to compare services received across different settings, to relate provider characteristics to patient utilization, to compare utilization rates among subpopulations, and to assess how the health care delivery system is being used and by whom. It can provide interested parties with information to help determine if utilization is appropriate or inappropriate, high or low quality, and expensive or inexpensive, and highlight areas that may warrant in-depth examination. For example, data on a higher than expected rate on cesarean sections or less use of cancer screening tests by certain population subgroups may highlight areas in need of attention. Longitudinally, health care utilization data is also used to monitor changes in the use of health care resources and to forecast future health care expenditures, or as the basis for projecting future healthcare needs such as facilities, personnel, or supplies.

Cross-References

- ▶ Health Care Costs
- ▶ Health Care System
- ▶ Health Economics
- ▶ Medical Utilization

References and Readings

A comprehensive listing of sources of health utilization data for the United States can be found at the Partners in Information Access for the Public Health Workforce at http://phpartners.org/health_stats.html

Aday, A. L., & Awe, W. C. (1997). Health services utilization models. In D. S. Gochman (Ed.), *Handbook of health behavior research* (Vol. 1, pp. 153–177). New York: Plenum Press.

Andersen, R. (2008). National health surveys and the behavioral model of health services use. *Medical Care, 46*(7), 647–653.

Andersen, R., & Newman, J. F. (2005). Societal and individual determinants of medical care utilization in the United States. *The Milbank Quarterly, 83*(4), 1–28.

One comprehensive source of comparable statistics on health care utilization among industrialized countries is found in the OECD interactive database at www.oecd.org/health/healthdata

Health Communication

Alfred L. McAlister
Behavioral Sciences, University of Texas School of Public Health, Austin, TX, USA

Synonyms

Health education; Health promotion; Social marketing

Definition

In the context of behavioral medicine, health communication is best defined as transmission or exchange of information designed to modify behaviors related to health. This can be envisioned broadly to encompass communication in medical settings (e.g., patient counseling to increase adherence to regimens), in communities and populations (e.g., media and community outreach campaigns to increase condom use), and in the political sphere (e.g., internet messaging platforms designed to spur advocacy to influence policies that affect health).

Description

Modalities include (1) face to face, telephone, and telecommunicated interpersonal communication between individuals and groups; (2) text and graphic messaging in print and electronic form, e.g., newspaper stories, posters, leaflets, websites; (3) audiovisual messaging in mass communication, e.g., television and documentary film; and (4) new media integrating multiple modalities through interactive web-based and mobile applications.

Skills training and guidelines for health communication are widely available online (e.g., U.S. Centers for Disease Control, 2011). Key tasks for effective health communication include assessment of audiences' health literacy, composition of appropriate messages through cultural competency, selection of channels of communication to effectively reach defined audience segments, and creation of message content to influence specified intermediate cognitive or emotional factors to achieve measurable changes in behavior. Audience research is central to effective health communication, as observations, interviews, focus groups, and surveys can be used to prioritize the segmentation of audiences within populations and identify most suitable channels and forms of communication for those audiences, and the knowledge, attitudes, perceptions, feelings, competencies and other factors that will provide the most effective message content for achieving behavior change.

Health communication is an important part of health education and promotion (Glanz, Rimer, & Viswanath, 2008), which also is concerned with changing behavior through both communication and the modification of environmental circumstances to facilitate or incentivize healthy behaviors. However, changing environmental circumstances requires effective media advocacy and related political communication skills. Social marketing (Andreason, 2006), in which techniques and strategies from advertising and sale of consumer products are adopted for noncommercial purposes, has become the dominant professional model for much health communication and been associated with numerous successful behavior change campaigns in both the developed and developing world (Wakefield, Loken, & Hornik, 2010). Alternatives and complements to the marketing model include behavioral journalism (McAlister, 2000) and the use of narrative storytelling (Kreuter et al., 2007), illustrated by reality-based television programs that follow individuals through the process of smoking cessation or weight loss, and edutainment in the form of radio dramas to promote family planning.

The basic theoretical foundation for health communication was articulated by William McGuire (2001) in his classic communication matrix model which considers how channels, sources, and message content influence exposure, attention, comprehension, yielding to persuasion, skill acquisition, trial of new behaviors, and long-term behavior change. This kind of sequential analysis of communication effects on individual behavior change has been elaborated in the transtheoretical model (Prochaska & DiClemente, 2005), which highlights specific processes in particular steps such as emotional arousal in the initial contemplation of change and feelings of self-efficacy and competence in the acquisition and maintenance stages of behavior change. Health communication effects on sequential processes in behavior change on the societal level are described in Rogers' (2003) diffusion of innovation model, which distinguishes between early adopters (who acquire innovations after being exposed to them via

media communication) and later adopters (who are more influenced by peer modeling, interpersonal communication, and conformity pressures).

The integrative model of behavioral prediction (Fishbein & Cappella, 2006) classifies mediating psychological factors that are influenced when communication yields behavior change: (1) modification of belief and expectations regarding behavior outcomes and values, (2) increases in perceived "normative" social pressure and anticipations of social sanctions, and (3) rise in "self-efficacy" expectations regarding personal or collective ability and competence. The latter factor is central to Bandura's (2001) social cognitive theory, which emphasizes a dual link comprised of peer modeling via mass media and social reinforcement in learning to perform healthy behaviors. Social cognitive theory also provides a formulation for self-management training methods widely used in patient education and behavioral counseling. Motivational interviewing (Rollnick, Miller, & Butler, 2007) is another notable theory-based technique for interpersonal communication to change behavior.

Innovation in health communication, while largely based on theoretical foundations noted above, has followed emerging technologies. Computer and web-based interactivity has opened new capacities for communicators to precisely segment differentiated audiences and tailor messages to their observed preferences. Internet and mobile applications for gaming have become notable modalities for health communication (Read & Shortell, 2011). New platforms for interactive social media and mobile messaging are providing opportunities for more compelling communication with patients in behavioral medicine, communities in health promotion, and advocates for policies to strengthen public health (Korda & Itani, 2011).

Cross-References

- ▶ Cultural Competence
- ▶ Health Literacy
- ▶ Motivational Interviewing
- ▶ Social Marketing

References and Readings

Andreason, A. R. (2006). *Social marketing in the 21st century*. Thousand Oaks, CA: Sage.

Bandura, A. (2001). Social cognitive theory of mass communication. *Media Psychology, 3*(3), 265–299.

Fishbein, M., & Cappella, J. N. (2006). The role of theory in developing effective health communications. *Journal of Communication, 56*, S1–S17.

Glanz, K., Rimer, B. K., & Viswanath, K. (2008). *Health behavior and health education* (4th ed.). San Francisco: Wiley.

Korda, H., & Itani, Z. (2011). Harnessing social media for health promotion and behavior change. *Health Promotion Practice*. doi:10.1177/1524839911405850, online.

Kreuter, M. W., Green, M. C., Cappella, J. N., Slater, M. D., Wise, M. E., Storey, D., et al. (2007). Narrative communication in cancer prevention and control. *Annals of Behavioral Medicine, 33*(3), 221–235.

McAlister, A. (2000). Action-oriented mass communication. In J. Rappaport & E. Seideman (Eds.), *Handbook of community psychology* (pp. 379–396). New York: Plenum.

McGuire, W. J. (2001). Input and output variables currently promising for constructing persuasive communications. In R. Rice & C. Atkin (Eds.), *Public communication campaigns* (3rd ed., pp. 22–48). Thousand Oaks, CA: Sage.

Prochaska, J. O., & DiClemente, C. C. (2005). The transtheoretical approach. In J. C. Norcross & M. R. Goldfried (Eds.), *Handbook of psychotherapy integration* (2nd ed., pp. 147–171). New York: Oxford University Press.

Read, J. L., & Shortell, S. M. (2011). Interactive games to promote behavior change in prevention and treatment. *Journal of the American Medical Association*. doi:10.1001/jama.2011.408, online.

Rogers, E. M. (2003). *Diffusion of innovations* (5th ed.). New York: Free Press.

Rollnick, S., Miller, W. R., & Butler, C. C. (2007). *Motivational interviewing in health care: Helping patients change behavior*. New York: Guilford Press.

U.S. Centers for Disease Control (2011). Gateway to health communication and social marketing practice. www.cdc.gov/healthcommunication/

Wakefield, M. A., Loken, B., & Hornik, R. C. (2010). Use of mass media campaigns to change health behavior. *Lancet, 376*(9748), 1261–1271.

Wallack, L., Dorfman, L., Jernigan, D., & Themba, M. (1993). *Media advocacy and public health: Power for prevention*. Thousand Oaks, CA: Sage.

Health Consequences of Smoking

► Smoking and Health

Health Departments

► Health Care
► Health Care Access

Health Disparities

Kristine M. Molina
Department of Psychology, University of Miami, Miami, FL, USA

Synonyms

Health inequalities; Health inequities

Definition

A health disparity is defined as an observed difference in health outcomes (e.g., diabetes) or health status between the most advantaged group in a given category (e.g., the wealthiest) and all other groups in that category. Observed differences in the health outcomes are not only limited to differences between better- and-worse-off groups, given they reflect varying levels of social advantage and disadvantage (Braveman, 2006).

Description

The term "health disparity" is often times used interchangeably with the terms "health inequality" or "health inequity." The use of "health disparity" is most common in the United States, whereas the other terms are most often used outside of the United States (Carter-Pokras & Baquet, 2002). The underlying distinction between these terms is that the latter ones distinguish between health differences that are unfair, unjust, and unavoidable. To illustrate, differences in health among men and women that are due to sex-specific problems (e.g., ovarian cancer) would be attributed to biological variation and therefore unavoidable, whereas health differences due to social or environmental factors (e.g., socioeconomic status, unequal access to resources), for example, would be considered unjust and avoidable (Whitehead, 1992). However, the term "health inequality" requires both an ethical and moral consideration regarding what constitutes a difference as "unavoidable" and "unjust/unfair," and therefore leaves its definition open to interpretation (Braveman, 2006; Carter-Pokras & Baquet, 2002).

Health disparities are typically thought of as referring to racial/ethnic disparities in health status. This is partly due to the long legacy of racism and racial inequality in the United States, for example. In fact, it has been consistently argued that health disparities must not be stripped from the social, cultural, political, and historical contexts in which they occur. However, differences in health can be present along other social dimensions, other than those based on race and ethnicity. These may include differences in health indicators with respect to gender, socioeconomic status (e.g., education, occupation, income), disability, age, and sexual orientation, among other characteristics. Several social determinants of health disparities have been identified, including but not limited to socioeconomic status (e.g., education, income, poverty), residential segregation, differential access to resources, lack of health insurance, and differential exposure to different types of stressors. These factors can both initiate as well as sustain health disparities. Further, systematic differences in health outcomes, such as those noted for African Americans across a number of health indicators (e.g., heart disease, certain types of cancer, diabetes, HIV, infant mortality), can further compromise the health status of already disadvantaged social groups (Myers, 2009).

There is no clear consensus on how to best measure health disparities, given the differences that exist in the use of the terms "health disparities," health inequalities," and "health inequities." Likewise, different approaches to measuring health disparities exist given the research question one is trying to answer. Nonetheless, a direct way of measuring a health difference is by comparing the health of one group (the reference group) with the health of another group(s). In general, one could compare the non-minority to the majority population (e.g., non-Hispanic whites compared to Asian Americans); compare a group against the general population; or compare differences among segments of the population. Other considerations include subgroup comparisons; for example, comparing differences within the Latino category (e.g., Mexican Americans against Cuban Americans). However, for any of these approaches, lack of clarity exists in who the reference group should be, although the most widely used approach has been to identify specific social groups a priori and examine differences in health status between them (Carter-Pokras & Baquet, 2002). Additionally, the U.S. National Center for Health Statistics has advocated for using the group with the most favorable rate in a given health indicator as the reference category, since it can potentially avoid dealing with concerns regarding who is considered to be the "most" socially advantaged group (Braveman, 2006; Keppel, Pearcy, & Klein, 2004). Other approaches to measuring health disparities have included obtaining relative indicators of health (e.g., black–white ratios), as well as measuring the distribution across individuals in a population on health status (similar to the measurement of income distribution in a population) (Asada, 2005). Depending on the field and question of interest, any of these approaches may be used, though they all have advantages and disadvantages to them.

Importantly, health disparities do not equate to *health care disparities*. In contrast to a health disparity, the Institute of Medicine, in its report "Unequal treatment: Confronting Racial and Ethnic Disparities in Health Care," defines a health care disparity as a difference in access to and quality of health care treatment between population groups that are not justified by access-related factors, treatment preferences, or the underlying health condition(s) of the population groups. Important to note is that limited access to treatment and good quality health care services play a significant role in observed differences in health status, particularly in producing racial/ethnic health disparities (Institute of Medicine, 2002).

Significantly, although specifying that both a difference in health exists and that they are socially patterned are important first steps to addressing the problem, they alone are not enough to reduce or eradicate health disparities. Indeed, addressing health disparities will require multi- and interdisciplinary research approaches to better understand the causes of specific types of health disparities, as well as multilevel interventions that target social disparities known to contribute to differences in health status (Braveman, 2006; Myers, 2009).

Cross-References

- ▶ Ethnic Differences
- ▶ Gender Differences
- ▶ Minority Health
- ▶ Racial Inequality in Economic and Social Well-being
- ▶ Social Class
- ▶ Social Epidemiology

References and Readings

Asada, Y. (2005). A framework for measuring health inequity. *Journal of Epidemiology and Community Health, 59*, 700–705.

Braveman, P. (2006). Health disparities and health equity: Concepts and measurement. *Annual Review of Public Health, 27*, 167–194.

Carter-Pokras, O., & Baquet, C. (2002). What is a "health disparity"? *Public Health Reports, 117*, 426–434.

Institute of Medicine. (2002). *Unequal treatment: Confronting racial and ethnic disparities in health care*. Washington, DC: National Academy Press.

Keppel, K. G., Pearcy, J. N., & Klein, R. J. (2004). *Measuring progress in Healthy People 2010* (Healthy

People 2010 statistical notes, Vol. 25). Hyattsville, MD: National Center for Health Statistics.
Myers, H. F. (2009). Ethnicity-and socio-economic status-related stresses in context: An integrative review and conceptual model. *Journal of Behavioral Medicine, 32*, 9–19.
Whitehead, M. (1992). The concepts and principles of equity in health. *International Journal of Health Services, 22*, 429–445.

Health Economics

Stephen Birch[1] and Amiram Gafni[2]
[1]Clinical Epidemiology and Biostatistics (CHEPA), McMaster University, Hamilton, ON, Canada
[2]Department of Clinical Epidemiology and Biostatistics, Centre for Health Economics and Policy Analysis, McMaster University, Hamilton, ON, Canada

Definition

Health economics applies the principles of economics to address problems of health and health care. It identifies the factors that contribute to the health of individuals and populations and identifies the most productive ways of using whatever resources are available for improving health.

Description

Health economics is an area of economics that applies the principles of the discipline of economics to address problems of health and health care. Both health and health care are commodities with characteristics that make them different from standard goods and services that are bought and sold in private markets. This means they require particular attention from economists in order to consider the use of resources devoted to producing health care and changing health.

Health as a Commodity

Health cannot be purchased directly. Instead it is "produced" by the levels and combinations of health "determinants," that is, factors that influence health and illness. Some of these factors can be purchased directly (e.g., exercise equipment, healthy foods, health care), while others may be in the form of public goods (or bads) such as air pollution. The individual may have little control over exposure to some of these determinants.

Although *health care* is an important determinant of health, other factors might also influence an individual's health, for example, an individual's genes, his lifestyle (Does he smoke?), his places of home and work, his diet and activity levels as well as limitations placed on choices about many of these factors by income and wealth. The relationship between health determinants and health outcomes is often complex and conditional on other health determinants. For example, the improvement in health produced from a heart bypass procedure may depend on the environment in which an individual lives and works (Are there factories close by that pollute the air that he breathes? Is he exposed to unhealthy work conditions?), the lifestyle he follows (Does he smoke?), the skill levels of the doctors treating him, etc. Economics provides a means of analyzing the production of health both at the level of an individual but also in terms of the production of health in populations.

The estimation of health production functions (the relationship between health determinants and health outcomes) enables us to consider the following:

1. The returns to investment in health determinants across a range of *different levels* of investment. For example, is the relationship between the quantity of health care and the health outcome produced constant for all levels of health care or does the change in health produced from health care change with the level of investment in health care? This is similar to the dose–response relationship in clinical research.
2. Whether the returns to investment differ among a range of *different health determinants*.

For example, does investing resources in public health programs to reduce smoking produce more health outcomes than investing the same amount of resources in additional cardiac care services?

3. Whether the return to investment in a particular health determinant is conditional on the levels of other health determinants. For example, is the health outcome associated with a public health program to reduce smoking conditional on the socioeconomic circumstances of the population targeted by the program.

Health Care as a Commodity

Health care represents a range of services aimed at improving health or reducing the risks of health loss. It is often labor intensive requiring the inputs of a mix of skilled professionals (physicians, nurses, dentists, etc.) together with nonlabor inputs such as capital equipment (hospitals, beds, diagnostic and surgical equipment) into a health care production function. The production function represents the particular technology (or production process) used to combine inputs to produce health outcomes. For example, primary care physicians may work independently, or in groups or as part of multidisciplinary health care teams.

Often opportunities arise for substitution between inputs. For example, nurse practitioners are trained to be able to perform services provided by family physicians. The production of primary care services could be changed by deploying more nurse practitioners and fewer family physicians. Decisions about the choice of production function need to be informed by evidence of the difference in outcomes and costs of the different ways of producing primary care services. Substitution can also occur between human and physical capital often as a result of new technologies. Cataract replacement surgery used to involve an inpatient stay requiring considerable inputs of physician time. The introduction of new laser technology has reduced the amount of physician time required, with the procedure now taking only a few minutes delivered in an outpatient clinic.

In addition, health care often involves episodes of care that are made up of a complex series of complementary services (e.g., prevention, treatment, and rehabilitation). The health outcomes of each item of care within an episode may not be simply additive. Failure to provide one element of the package of services may undermine the outcomes of the other elements.

Both the demand and supply of health care are complex issues that cannot be analyzed in the same way as many other commodities. Because of the complex nature of the association between health care use and health outcomes, individuals are unable to determine what services they need to address their health problems. Instead they rely on the advice of their health care provider. In an unregulated market, any individual could set themselves up as an "expert" in diagnosing the cause of an individual's health problem, recommending a treatment and delivering that care. However, changing provider as a result of poor advice would not avoid the potentially profound consequences of poor health care decisions (serious injury, illness, disability, or death). Supply is, therefore, organized through a system of strict licensure that involves restrictions on entry to the market to individuals with defined qualifications as well as professional codes of practice in order to protect the public interest.

Health care is not demanded for its own intrinsic value. On the contrary, individuals would generally prefer to not consume health care since it is often unpleasant, uncomfortable, or painful. Instead, the demand for health care is derived from the demand for the health outcomes it is expected to produce. Providers, in addition to being a major input in the supply of health care, also influence (or induce) the demand for health care through their role as advisor, or agent, of the patient. Supplier-induced demand is not a problem per se because the whole purpose of a licensure system is to have "experts" advising individuals what services they need to improve their health. However, it can become a problem where the earnings of providers respond to the

level and type of health care delivered. As a result changes in levels of services used over time need not reflect (only) changes in need for those services among patients but also responses of providers to income opportunities. This means that the traditional market of supply and demand does not exist for health care and hence market mechanisms fail to achieve the socially optimum allocation of health care resources.

Health care economics is that part of health economics concerned with the supply of health care and the evaluation of health care services and patient uptake of and compliance with treatment. In the context of scarce health care resources, it considers the impact on the health and well-being of individuals and populations of using the available resources in alternative ways by comparing both the effects (outcomes) and costs of different health care interventions (Economic evaluation). Such evaluations are, in isolation, simply descriptive information on the expected rate of return on additional investment (what extra outcome can be produced by investing more resources in this particular treatment?). In addition, consideration needs to be given to the opportunity cost of the additional investment (what has to be forgone in order to provide the additional investment required) and how to ensure the services supported by the additional investment will be produced by providers and consumed by patients in the way intended. Hence, health care economics extends beyond the area of economic evaluation of health care interventions to also incorporate the study of the behavior of providers and consumers. So, for example, there may be interest in introducing a new screening service. Health care economics would involve inter alia the following:

1. Estimating the additional costs and effects of the new service compared to existing practice
2. Calculating the expected rate of return of the additional costs
3. Considering the alternative ways of supporting the additional investment within the existing resource constraint and the forgone effects associated with taking the resources required from these other uses
4. Analyzing the behavior of providers and patients when presented with the opportunity to deliver/use the new service

This final set of challenges involves studying the funding, planning, management, and delivery of health care. Health problems can be caused by problems associated with low income and wealth, and health problems can lead to reductions in income and wealth as they can restrict normal activities. As a result an individual's need for health care is greatest when his ability to pay for health care is lowest. Health economics is, therefore, concerned with addressing this "conundrum" by analyzing alternative approaches for funding provision, allocating resources, and managing performance.

References and Readings

Birch, S., Jerrett, M., & Eyles, J. (2000). Heterogeneity in the determinants of health and illness: The example of socioeconomic status and smoking. *Social Science and Medicine, 51*, 307–317.

Drummond, M., Sculpher, M., Torrance, G., O'Brien, B., & Stoddart, G. (2005). *Methods for the economic evaluation of health care programmes*. New York: Oxford University Press.

Evans, R. (2005). *Strained mercy. The economics of Canadian Health Care*. Toronto: Butterworths.

Evans, R., & Stoddart, G. (1990). Producing health, consuming health care. *Social Science and Medicine, 31*, 1347–1363.

Gafni, A., & Birch, S. (2006). Incremental cost-effectiveness ratios (ICERs): The silence of the lambda. *Social Science and Medicine, 62*, 2091–2100.

Morris, S., Devlin, N., & Parkin, D. (2007). *Economic analysis in health care*. Chichester: John Wiley.

Health Education

Linda C. Baumann and Alyssa Karel
School of Nursing, University of Wisconsin-Madison, Madison, WI, USA

Synonyms

Patient education

Definition

The World Health Organization defines health education as "any combination of learning experiences designed to help individuals and communities improve their health, by increasing their knowledge or influencing their attitudes." Because knowledge alone may not be powerful enough to motivate change, health education works to enhance knowledge, attitudes, and skills to positively influence health behaviors of individuals and communities.

Adult learning theory is an important construct to consider for effective health education. Malcolm Knowles has identified five crucial assumptions about the characteristics of adult learners. These characteristics are (1) self concept, as a person matures, they move from a dependent personality to a self-directed one; (2) experience, an accumulation of experiences are a resource for learning; (3) readiness to learn, an adult's readiness to learn is oriented to the tasks of their social roles; (4) orientation to learning, adult learning shifts from subject-centered to problem-centered; and (5) motivation, an adult learner's motivation to learn is internal.

Health education is provided in a variety of settings and can be targeted at individuals, groups, or larger populations. Although health education is generally considered primary prevention as a health promotion strategy, it can also occur at the secondary and tertiary levels. Health education at the primary prevention level is aimed at educating to promote healthy behaviors and to prevent the occurrence of illness or injury; at the secondary and tertiary levels, health education focuses on teaching strategies to detect problems early by identifying risk factors, and rehabilitation to optimize function and prevent complications of disease.

Health education is a dynamic process that requires planning and evaluation of interventions. Important steps include assessing the need for education of a target population, setting learner-centered goals and objectives, implementing the educational intervention, and evaluating and revising education to meet the targeted goals. Other considerations needed for education to be effective include one's readiness to learn, and personal, cultural, political, and environmental factors that may impact learning.

Cross-References

- ▶ Behavior Change
- ▶ Behavioral Intervention
- ▶ Community-Based Health Programs
- ▶ Education, Patient
- ▶ Empowerment
- ▶ Health Behavior Change
- ▶ Health Communication
- ▶ Health Promotion and Disease Prevention
- ▶ Intervention Theories
- ▶ Lifestyle Changes
- ▶ Risk Factors and Their Management
- ▶ Theory of Planned Behavior

References and Readings

Allender, J. A., Rector, C., & Warner, K. D. (2010). *Community health nursing: Promoting and protecting the public's health*. Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins.

Knowles, M. (1973). *The adult learner: Neglected species*. Houston, TX: Gulf Publishing Company.

Knowles, M., & Associates. (1984). *Andragogy in action: Applying modern principles of adult Learning*. San Francisco: Jossey-Bass.

Smith, M. K. (2009). Andragogy. *The encyclopedia of informal education*. Retrieved April 25, 2011, from http://www.infed.org/lifelonglearning/b-andra.htm

Taylor, C., Lillis, C., LeMine, P., & Lynn, P. (2008). *Fundamentals of nursing: The art and science of nursing care* (6th ed.). Philadelphia: Lippincott Williams & Wilkins.

The Coalition of National Health Education Organizations. (2011). *What is health education?* Retrieved April 19 2011, from http://www.cnheo.org/PDF%20files/health_ed.pdf

Wallace, R. B., Kohatsu, N., & Last, J. M. (2007). *Public health & preventive medicine* (15th ed.). New York: McGraw Hill Medical.

World Health Organization. (2011). *Health education*. Retrieved April 19, 2011, from http://www.who.int/topics/health_education/en/

Health Inequalities

► Health Disparities

Health Inequities

► Health Disparities

Health Information Record

► Electronic Health Record

Health Insurance

► Health Insurance: Comparisons

Health Insurance Portability and Accountability Act (HIPAA)

Howard Sollins
Attorneys at Law, Ober, Kaler, Grimes & Shriver, Baltimore, MD, USA

Synonyms

Patient protection

Definition

Health Insurance Portability and Accountability Act of 1996 (HIPAA)

HIPAA is a federal law that addresses a variety of health care subjects in various titles. These address health insurance coverage, enrollment and preexisting conditions, fraud and abuse, administrative simplification, electronic billing and coding for health care services, and the protection of certain individually identifiable health information that is obtained by "covered entities." These titles affect how health care claims are documented and billed and amended laws governing health insurers. Tax laws were amended to establish medical savings accounts and address the deductibility of health insurance premiums by self-employed individual, long-term care insurance, and provide other benefits. With respect to fraud and abuse, HIPAA also, for example, provide for advisory opinions, increased and expanded fraud and abuse investigation and enforcement penalties and tools for regulatory agencies and outline when inducements by health care providers to Medicare and certain other health care beneficiaries are prohibited. The use, disclosure, and retention of protected health information is addressed under both a privacy rule and a security rule. Covered without limitation entities include health care providers such as physicians, nurse practitioners, physician assistants, psychologists, health care facilities such as hospitals, nursing homes and pharmacies, health insurance companies and health plans, and entities that process nonstandard health information they receive from another entity into a standard format (a health information clearing house). There are also regulations governing the sharing and use and accounting of information by business associates of covered entities. Any discussion of HIPAA should also include reference to the statutes and regulations enacted under the Health Information Technology for Economic and Clinical Health (HITECH) Act, enacted as part of the American Recovery and Reinvestment Act of 2009. Subtitle D of the HITECH Act addresses the privacy and security concerns associated with the electronic transmission of health information, in part, through several provisions that strengthen the civil and criminal enforcement of the HIPAA rules.

Cross-References

► Patient protection

References and Readings

Access to health information of individuals. Retrieved from http://www.nlm.nih.gov/hmd/manuscripts/phi.pdf

Health Insurance: Comparisons

Catharina Hjortsberg
The Swedish Institute for Health Economics, Lund, Sweden

Synonyms

Health care system; Health insurance; Risk pooling

Definition

The term *health insurance* is generally used to describe a form of insurance that pays for medical expenses of the insured. The main aim of a health insurance is to spread the financial risk arising from ill-health. Health insurance may apply to a limited or comprehensive range of medical services and may provide for full or partial payment of the costs of specific services. Moreover, the insurance could be provided either through a governmental national insurance program, or from private for-profit or non-for-profit insurance companies. Some health care systems rely more on private health insurance than others, for example, the health care system in the United States.

Description

A *health care system* includes all activities and structures whose primary purpose is to influence health in its broadest sense. The goals for health systems are good health, responsiveness to the expectations of the population, and fair financial contribution (World Health Organization (WHO), 2000). *Health care systems* are organized differently, that is, the way health care is provided and how it is financed differs between countries. Most health care systems are characterized by both providers (primary care centers, hospitals), who supply health care services, and purchasers (insurers, health authorities) who buy health care for a certain population. Health care systems can be funded via social health insurance, general taxation to the state, county or municipality, direct or out-of-pocket payments, voluntary or private health insurance, and donations or community health insurance. Most countries' systems constitute a combination of these. One common feature is that all health care systems implicitly *pool* the risks associated with individual health care needs (WHO, 2000). A risk pool allows a large group of people to share the risk that they may become ill and need expensive care. Funds dedicated for health care are collected through pre-payment (e.g., via insurance) and are managed in such a way as to ensure that the risk of having to pay for health care is borne by all the members of a pool and not by each contributor individually.

Since improving access to health care services has been a fundamental objective of health systems in the past 30 years, most countries today have a *national health insurance*, which means that it insures a national population. By having a national health insurance governments ensure that people have access to affordable health services without risking, ending up in financial difficulties. Very often national health insurance systems are established by national legislation. The clear benefit of national insurance is that the pool of pools is very large representing the national population. The contribution from the individual is regulated by the government and paid into the pool over a lifetime. This is different from *private health insurance*, where the price is set in a competitive insurance market and health risks are matched with the price of the insurance.

National health insurance can be administered by the public sector, the private sector, or a combination of both. These insurance programs differ both in terms of how the money is collected, and how the services are provided. Even if several countries raise part of the revenue for health in the same way, they may operate differently in how they pool funds and how they purchase and provide services. This is why the

traditional way of categorizing health financing systems into tax-based or social health insurance is not longer useful (WHO, 2010). In some countries, payment is made by the government (or local governments) directly from tax revenue, for example, Canada and Sweden. In other countries, for example, the UK, an additional contribution is collected for all workers, paid by employees and employers based on their earnings. In both of these cases the collection is administered by the government. The collection of compulsory contributions can also be administered by non-profit organizations like in the case of France. This is sometimes related to as a single-payer health care system. The health care providers may be either publicly or privately owned. The Netherlands, on the other hand, has adopted a completely different funding approach, where competing health insurance funds receive the compulsory contributions. These insurance funds can be either public bodies, private for-profit companies or non-profit companies. They are all obliged to provide a minimum standard of coverage and are not allowed to discriminate between patients by charging different rates according to age, occupation, or previous health status. Other countries' national health insurance plans, for example, Germany and Belgium, are largely funded by contributions by employers and employees to sickness funds. With these programs, funds usually come from three sources (private, employer-employee contributions, and national/subnational taxes). These funds are usually not for profit; institutions run entirely for the benefit of their members.

Some national insurance plans also provide compensation for loss of work due to ill-health, or covering things such as pensions, unemployment, and occupational retraining.

Countries' national health insurance systems also differ in terms of the amount of out of pocket payments that are required. Out-of-pocket expenses are direct outlays of cash made by the patient when seeking care, which may or may not be later reimbursed. Many countries still rely greatly on out-of-pocket payments from individuals to health service providers to fund their health systems. Some countries have abolished out-of-pocket payments completely, for example, the United Kingdom, and the patient is not paying anything when seeking health care, while in other countries patients are expected to pay part of their medical expenses and to pay more for higher level of services, for example, as provided in Singapore. In other countries like France, patients pay medical bills and are later reimbursed by sickness insurance funds. These outlays made by patients, usually just represent a symbolic part of the real cost of services in developed countries, as in the majority of these the government subsidizes basic healthcare. However, in many developing countries these financial outlays made by patients lead to severe financial difficulties as a consequence. A high proportion of the world's poor have no access to health services merely because they cannot afford to pay at the time they need them (Preker et al., 2004).

Separate to national health insurance there is in many countries also the possibility to have private health insurance. Private health insurance schemes are financed through private health premiums, that is, payments that a policyholder agrees to make for coverage under a given insurance policy. A contract is issued by an insurer to the covered person. Commonly private health insurance is voluntary; however, it can be compulsory for employees as part of their working conditions. Premiums paid by the covered person are non-income-related, although the actual purchase of private health insurance can in some cases be subsidized by the government. An important distinction between private and national health insurance is that the pool of financing is not channeled nor administered through the government. Private health insurance can be a controversial form of insurance because of the conflict between the need for the insurance company to make profit versus the need of its customers to remain healthy, which many view as a basic human right.

Some countries' health systems depend greatly on private health insurance, for example, the United States (US) which has a complex health care system. In the USA, public programs (e.g., Medicare, Medicaid, and Veterans Health Administration) provide coverage for health care of those

citizens that meet their eligibility requirements. The US insurance system has made discussions, and in 2010, a new law came into force making it mandatory to have health insurance.

The health sector is extremely complex, and health care expenditure represents a major use of a nation's resources and has been growing during the last three decades. Factors such as an aging population, the increased personal use of health care, and medical advances that have opened the way for more treatment options and diagnostics have contributed to a rise in the demand for health care and increased the costs for health care. At the same time enormous pressure is being put on the health system in terms of their capacity to improve outcomes and ensure consumer satisfaction, in an equitable, efficient, and financially sustainable manner. Health financing is, therefore, an issue of growing importance.

Cross-References

▶ Health Care System

References and Readings

Preker, A., Carrin, G., Dror, D., Jakab, M., Hsiao, W., & Arhin-Tenkorang, D. (2004). Rich–poor differences in health care financing. In A. Preker & G. Carrin (Eds.), *Health financing for poor people: Resource mobilization and risk-sharing*. Washington, DC: World Bank.

World Health Organization. (2010). *The World Health Report – Health systems financing: The path to universal coverage*. Geneva: World Health Organization.

World Health Organization. (2000). *The World Health Report 2000. Health systems: Improving performance*. Geneva: World Health Organization.

Health Literacy

Lee Sanders
Center for Health Policy and Primary Care Outcomes Research, Stanford University, Stanford, CA, USA

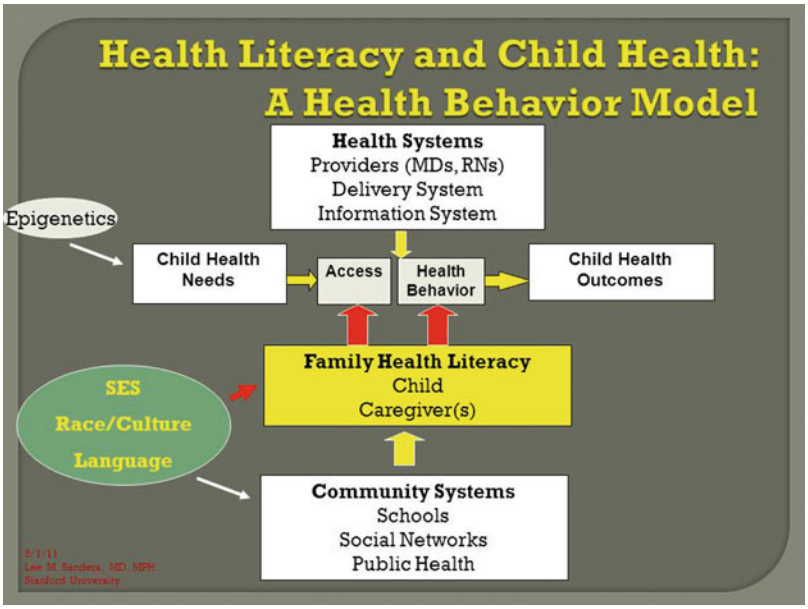
Synonyms

Literacy

Definition

Health literacy refers to an individual's ability to understand and use written and spoken health information. An applied version of the broader construct of "literacy" (i.e., the ability to read and understand all written information), health literacy encompasses the tasks necessary for a patient to navigate the modern medical system. This includes tasks such as dosing medication according to written instructions, interpreting a food label, following an immunization schedule, completing personal health information at the doctor's office, understanding a patient-centered brochure, completing health-insurance forms, or finding health information on the Internet. At least one in three US adults has limited health literacy (Kutner, Greenberg, Jin, & Paulsen, 2006; Nielsen-Bohlman, Panzer, & Kindig, 2004; Yin et al., 2009). According to the 2003 National Assessment of Adult Literacy (NAAL), 78 million US adults (36% of the population) are unable to perform "basic" health literacy tasks, such as using an immunization schedule, following recommendations from a preventive-health brochure, and interpreting a growth chart (Doak, Doak, & Root, 1996; Rich, 2004). Unfortunately, most health information in the USA is written with a complexity of form and language too difficult for most US adults to understand (Davis et al., 1994; Kutner et al., 2006; Rothman, Housam, & Weiss, 2006; Sanders, Federico, Klass, Dreyer, & Abrams, 2009; Yin et al., 2009)

Health literacy is a critical and potentially modifiable factor that influences health behaviors and may help reduce health disparities. Health literacy may contribute to the health-behavior model of health outcomes by attenuating the relationship between social factors and health behaviors. Many of the leading sources of morbidity and health disparities (e.g., preterm birth, obesity, chronic lung disease, cardiovascular disease, type 2 diabetes, mental health disorders, and cancer) are the result of literacy-sensitive health behaviors acquired across the life course (e.g., physical activity, nutrition, smoking,



Health Literacy, Fig. 1 This conceptual model proposes collective health literacy ("Family Health Literacy") and institutional health literacy (the "Health Systems") as modifiable determinants of child health outcomes. Note the contribution of other social determinants (e.g., SES, culture, language) as moderating factors and of health behaviors as mediators

risky sexual behaviors). Recent studies among adults have established an independent association between lower health literacy and decreased access to preventive-care services, increased use of urgent care services, increased risk for depression, and worse chronic-illness outcomes. Controlling for income, gender, and age, several studies have demonstrated that adults with limited literacy skills are significantly less likely than those with stronger skills to receive basic preventive care, including vaccines, weight management, and screening for breast, cervical, and prostate cancer (Bennett et al., 1998; Scott, Gazmararian, Williams, & Baker, 2002; Schillinger et al., 2002). In similarly adjusted analyses, children living with low-literacy caregivers have decreased access to primary preventive care, are more likely to be uninsured, less likely to access needed social services, less likely to be breastfed, and more likely to be exposed to second-hand tobacco smoke (Sanders et al., 2009). Adolescents who read below grade level are at an increased risk for violent and aggressive behavior, substance use, and sexually transmissible illnesses (Abrams & Dreyer, 2009)

As a result of these research findings, leading government agencies and national medical organizations – including the National Institutes of Health, the Institute of Medicine, and the Agency for Research in Healthcare Quality – have developed guidelines that call for more strategic attention to individuals' health literacy as a way of addressing major health disparities and public health challenges in the USA (Kutner et al., 2006; Nielsen-Bohlman et al., 2004; Yin et al., 2009). Experimental, clinical, community-based, and policy approaches to attenuating literacy-related health disparities have been proposed and tested. Evidence suggests that the most effective solutions apply to simplifying systems of care, particularly in the domains of medication delivery, chronic-illness management, and informed consent (Doak et al., 1996; Edwards, Elwyn, & Mulley, 2002; Rich, 2004; Sanders, Thompson, & Wilkinson, 2007; Weiss, Francis, Senf, Heist, & Hargraves, 2006). The most innovative and effective strategies apply interdisciplinary solutions that integrate cognitive behavioral theory, visual images, cultural sensitivity, and new interactive technologies.

Cross-References

- ▶ Community-Based Participatory Research
- ▶ Informed Consent

References and Readings

Abrams, M. A., & Dreyer, B. P. (2009). *Plain language pediatrics: Health Literacy strategies and communication resources for common pediatric topics*. Elk Grove Village, IL: American Academy of Pediatrics.

Bennett, C. L., Ferreira, M. R., Davis, T. C., Kaplan, J., Weinberger, M., Kuzel, T., et al. (1998). Relation between literacy, race, and stage of presentation among low-income patients with prostate cancer. *Journal of Clinical Oncology*, 16, 3101–3104.

Davis, T. C., Mayeaux, E. J., Fredrickson, D., Bocchini, J. A., Jacson, R. H., & Murphy, P. W. (1994). Reading ability of parents compared with reading level of pediatric patient education materials. *Pediatrics*, 93, 460–468.

Doak, C. C., Doak, L. G., & Root, J. H. (1996). *Teaching patients with low literacy skills* (2nd ed.). Philadelphia: Lippincott.

Edwards, A., Elwyn, G., & Mulley, A. (2002). Explaining risks: Turning numerical data into meaningful pictures. *BMJ, 324*(7341), 827–830.

Kutner, M., Greenberg, E., Jin, Y., Paulsen, C. (2006). *The health literacy of America's adults: Results from the 2003 National Assessment of Adult Literacy (NCES 2006-483)*. U.S. Department of Education. Washington, DC: National Center for Education Statistics.

Nielsen-Bohlman, L., Panzer, A., & Kindig, D. A. (2004). *Health literacy: A prescription to end confusion*. Washington, DC: National Academies.

Rich, M. (2004). Health literacy via media literacy: Video intervention/prevention assessment. *American Behavioral Scientist, 48*(2), 165–188.

Rothman, R. L., Housam, R., Weiss, H., Davis, D., Gregory, R., Gebretsadik, T., et al. (2006). Patient understanding of food labels: The role of literacy and numeracy. *American Journal of Preventive Medicine*, 31(5), 391–398.

Sanders, L. M., Federico, S., Klass, P., Dreyer, B., & Abrams, M. A. (2009). Health literacy in pediatrics: A systematic review. *Archives of Pediatrics & Adolescent Medicine, 163*(2), 131–140.

Sanders, L. M., Thompson, V. T., & Wilkinson, J. D. (2007). Caregiver health literacy and the use of child health services. *Pediatrics, 119*(1), 86–92.

Schillinger, D., Grumach, K., Piette, J., Wang, F., Osmond, D., Daher, C., Palacios, J., Sullivan, G. D., & Bindman, A. B. (2002). Association of health literacy with diabetes outcomes. *Journal of the American Medical Association*, 288, 475–482.

Scott, T. L., Gazmararian, J. A., Williams, M. V., & Baker, D. W. (2002). Health literacy and preventive health care use among medicare enrollees in a managed care organization. *Medical Care, 40*, 395–404.

Weiss, B. D., Francis, L., Senf, J. H., Heist, K., & Hargraves, R. (2006). Literacy education as treatment for depression in patients with limited literacy and depression: A randomized controlled trial. *Journal of General Internal Medicine, 21*(8), 823–828.

Yin, H. S., Johnson, M., Mendelsohn, A. L., Abrams, M. A., Sanders, L. M., & Dreyer, B. P. (2009). The health literacy of parents in the US: A nationally representative study. *Pediatrics, 124*(3), S289–S298.

Health Navigators

- ▶ Promotoras

Health Outcomes Research

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

Health outcomes research refers to the entire evaluation of all health professionals' efforts in ameliorating patients' health conditions, in relation to various dimensions of health. Furthermore, it refers to the work not only of health professionals but of health-related organizations and their policies as a whole as well. Thus, in contrast to research on the effectiveness of specific medical or health interventions on specific health indices (e.g., blood pressure, survival), health outcomes research is a broader concept, both on the side of the intervention agents as well as on the side of the outcomes. Health outcomes research thus can guide health policy makers, health economists, as well as specific types of health professionals and clinicians.

The setting or context of health outcomes research includes clinics, hospitals, patients' homes, or even entire regions. The typical measures used in health outcomes research are also broader than those commonly used in medical intervention trials. While in the latter, outcomes mainly include physiological parameters (e.g., HbA1C, tumor markers, pulmonary functioning), health outcomes research additionally focuses on patients' satisfaction with health care, daily functioning, and, more broadly speaking, on their well-being. In addition, health outcomes research uses the method of meta-analysis to summarize and infer from multiple, yet comparable, clinical studies of a given treatment. Measures of functional status include physical (e.g., carrying), role (e.g., being a parent or worker), and social functioning (e.g., taking part in social events) in general or as influenced by a disease. The Brief Pain Inventory assesses the level of interference of pain in these factors. Measures of well-being include dimensions such as mental health, health perceptions, pain, and general life satisfaction. A common measure assessing most of these dimensions is the SF-36 (Aaronson et al., 1992). This measure is also used in health economics studies to assess quality-adjusted life years, a major outcome in health outcomes research, as it encompasses survival time with its quality.

Cross-References

► Randomized Clinical Trial

References and Readings

Aaronson, N. K., Acquadro, C., Alonso, J., Apolone, G., Bucquet, D., Bullinger, M., et al. (1992). International quality of life assessment (IQOLA) project. *Quality of Life Research, 1*, 349–351.

Health Phobia

► Health Anxiety

Health Plan

► Health Policy/Health-Care Policy

Health Planning

► Health Policy/Health-Care Policy

Health Policy/Health-Care Policy

Erin N. Marcus[1] and Olveen Carrasquillo[2]
[1]Division of General Internal Medicine, University of Miami, Miller School of Medicine, Miami, FL, USA
[2]Division of General Medicine, Miller School of Medicine, University of Miami, Miami, FL, USA

Synonyms

Health plan; Health planning; Health program; Health strategy

Definition

A nation's, state's, city's, or other community's decisions regarding matters affecting health.

Description

Health policy refers to the systematic and/or organized approach to decision making regarding matters affecting health by a national or regional governmental entity (such as state or city) or other organized group (company, agency, etc.) (Patrick & Erickson, 1993). Most often, the term is used synonymously with health-care policy. However, the latter more narrowly applies to decisions affecting the formal medical system. Ideally the goal of such decision making would

be toward improving the well-being of members of the community (Bodenheimer & Grumbach, 2009; Weiner, Famadas, Waters, & Gikic, 2008). Further, such a decision-making process should be largely informed and driven by factual knowledge and evidence from the natural and social sciences. However, in reality, health policy is heavily influenced by many factors outside the scientific realm such as economic and political forces. In addition, such policies also heavily reflect a region's and society's ethics and values (Bodenheimer & Grumbach, 2009; Weiner et al., 2008). Thus, health policies vary widely around the world.

An example is the ways countries choose to finance their formal health-care sector, for which there are five major approaches: direct taxation; social health insurance, with mandatory premiums; voluntary or private health insurance; out-of-pocket payments; and charitable care (World Health Organization, 2005). In some countries such as Norway, the vast majority of health care is financed through direct government taxation. In Taiwan, social health insurance, financed by a payroll tax, covers nearly all health care. In the USA, a mixed market exists, with the government paying for slightly under half of all costs, and most of the rest covered by private insurance and/or out-of-pocket payments. In extremely poor countries, such as Mali, much of the care is provided by charitable organizations (World Health Organization, 2005).

Health-care policy also includes decisions around how health care is organized and delivered and the amount of money that should be devoted to health care. While it is generally agreed that countries spending less than $60 per person annually on health care have difficulty providing minimal essential services (World Health Organization, 2005), absolute funding levels do not necessarily correlate with health or health-care outcomes. For example, the USA spends nearly twice as much as most other developed countries on health care, yet is often ranked lower than many other countries with respect to measures of health outcomes and access to care. How health-care funding is allocated is also a major focus of health policy deliberations. In many countries, more population-based medical interventions, such as immunization programs, are prioritized as it is felt that these may result in a more efficient allocation of limited health-care resources. In poorer countries, such population level interventions may also be administered by transnational organizations such as the World Health Organization or nongovernmental organizations operating at the national, state, or local levels (World Health Organization, 2005).

Another example of differences in health-care policies across countries is evident with respect to counseling and therapies meant to promote behavior change, such as dietary counseling or counseling to promote tobacco cessation. Given extensive evidence that such programs can reduce complications related to obesity and long-term tobacco use, many countries provide coverage for such services. In contrast, until recently many insurance plans in the USA provided limited or no coverage for counseling directed at shaping health behaviors.

Health policy also includes programs and legislation which may influence health-related behaviors but are not typically considered part of the formal health-care sector. An example is outdoor smoking bans, which not only protect nonsmokers from secondhand smoke but are also associated with decreases in tobacco use among smokers. Laws allowing police to issue tickets to drivers of cars with unbelted passengers are associated with increased seat belt usage and a corresponding decrease in motor vehicle accident-related deaths. Another example is land-use policies that create pedestrian-friendly built environments that promote healthy behaviors, such as walking, and result in lower obesity rates (U.S. Centers for Disease Control). Often times, the health effects of such policies may not even be apparent at the time they are implemented. For example, the intention of a federally mandated decrease in highway speed limits in the 1970s was to improve conservation of fuel, but resulted in fewer automobile accident-related deaths. Thus, while health-care policy often dominates health policy deliberations, interventions

outside of the formal health-care delivery system may also have a major influence on health (Connolly, 2008).

Cross-References

- ▶ Centers for Disease Control and Prevention
- ▶ Community-based Health Programs
- ▶ Health Departments
- ▶ Institute of Medicine
- ▶ National Cancer Institute
- ▶ National Heart, Lung, and Blood Institute
- ▶ National Institute of Diabetes and Digestive and Kidney Diseases
- ▶ National Institute of Mental Health
- ▶ National Institute of Nursing Research
- ▶ National Institute on Aging
- ▶ National Institute on Alcohol Abuse and Alcoholism
- ▶ National Institutes of Health
- ▶ Robert Wood Johnson Foundation
- ▶ Smoking Prevention Policies and Programs
- ▶ Tobacco Control
- ▶ World Health Organization (WHO)

References and Readings

Bodenheimer, T. S., & Grumbach, K. (2009). *Understanding health policy: A clinical approach*. New York: McGraw Hill.

Centers for Disease Control. About healthy places. Available at: http://www.cdc.gov/healthyplaces/about.htm. Accessed Oct. 17, 2011.

Connolly, C. (2008). Overcoming obstacles to health: A report from the Robert Wood Johnson foundation. Retrieved October 13, 2011, from http://www.rwjf.org/files/research/obstaclestohealth.pdf

Patrick, D., & Erickson, P. (1993). *Health status and health policy: Quality of life in health care evaluation and resource allocation*. New York: Oxford University Press.

Weiner, J. P., Famadas, J. C., Waters, H. R., & Gikic, D. (2008). Managed care and private health insurance in a global context. *Journal of Health Politics, Policy and Law, 33*(6), 1107–1131.

World Health Organization. (2005). Strategy for Health Care Financing for Countries of the Western Pacific and Southeast Asian Regions 2006–2010. Geneva: World Health Organization. Available at: http://203.90.70.117/PDS_DOCS/B0131.pdf. Accessed Oct. 16, 2011.

Health Program

- ▶ Health Policy/Health-Care Policy

Health Promotion

- ▶ Health Communication

Health Promotion and Disease Prevention

- ▶ Centers for Disease Control and Prevention
- ▶ Health Communication
- ▶ Health Education
- ▶ Health Literacy
- ▶ Health Policy/Health-Care Policy
- ▶ Healthy Cities
- ▶ Healthy Eating
- ▶ HIV Prevention
- ▶ Prevention: Primary, Secondary, Tertiary
- ▶ Preventive Care
- ▶ Preventive Medicine Research Institute (Ornish)
- ▶ Worksite Health Promotion

Health Psychology

Vincent Tran
University of Texas, Southwestern Medical Center, Dallas, TX, USA

Synonyms

Behavioral medicine; Medical psychology; Psychosomatic medicine

Definition

Health psychology is a relatively new subfield of psychology that studies psychological factors related to how people stay healthy, why they become ill, and how they respond when they do become ill. The American Psychological

Association's official definition of health psychology comes from Matarazzo (1982): "Health Psychology is the aggregate of the specific educational, scientific, and professional contributions of the discipline of psychology to the promotion and maintenance of health, the prevention and treatment of illness, the identification of etiologic and diagnostic correlates of health, illness, and related dysfunction and to the analysis and improvement of the health care system and health policy formation."

Health psychology emphasizes the biopsychosocial model where physical well-being and disease reflect a complex set of interrelated processes including biological factors (e.g., genetics, hormonal fluctuations), psychological factors (e.g., mood, personality, health behaviors), and social factors (e.g., cultural norms, health policy, social support). Health psychologists may focus their professional activities on consultation, intervention, public health policy and administration, and/or research. They commonly collaborate with other health care professionals in multidisciplinary settings in order to provide optimal care for patients and to improve health care systems, policy, and public health. From its inception, health psychology has had a dual focus on research and practice, reflecting the philosophy of the broader discipline of psychology.

The field of health psychology was formally recognized in the USA in 1978 with the establishment of the Division of Health Psychology (Division 38) within the American Psychological Association. A confluence of factors contributed to the development of the field of health psychology at this time including (a) research demonstrating compelling mind-body associations (e.g., Neal Miller's work on the conditioning of physiological processes), (b) recognition that the leading causes of mortality (e.g., coronary heart disease) could be prevented, delayed, or treated through health behavior change, and (c) the possibility to curb health care costs through prevention and low-cost behavioral initiatives. The mission of Division 38 was – and still is – to advance the contributions of psychology as a discipline to understanding health and illness through basic and clinical research, to promote education and services in the psychology of health and illness, and to inform the psychological and biomedical community of these research and service activities. Parallel movements and related fields have developed over the years but remain distinct from that of health psychology. Behavioral medicine is an interdisciplinary organization devoted to integrating biomedical and psychosocial factors in health and illness, in contrast to the intradisciplinary focus of health psychology; health psychologists engage in behavioral medicine when they collaborate with colleagues outside of psychology (e.g., medicine, nursing, public health, etc.). Medical psychology, or clinical health psychology, is a term most commonly used to describe the work conducted by clinical psychologists who practice in medical settings. Another interdisciplinary field, psychosomatic medicine, which developed somewhat earlier than health psychology and behavioral medicine, focuses similarly on understanding biobehavioral links between psychology, psychiatry, internal medicine, physiology, and other disciplines.

Division 38 (Health Psychology) of the American Psychological Association, the Society of Behavioral Medicine, and the American Psychosomatic Society are organizations that promote research and practice of health psychology and related fields. Many scholarly journals are dedicated to disseminating research generated by health psychologists. The official journal of Division 38 is Health Psychology, but there are also international journals that publish peer-reviewed research in health psychology (e.g., Journal of Health Psychology; Psychology and Health; Health Psychology Review). Health psychology research is also routinely published in journals linked to the interdisciplinary organizations of behavioral medicine (Annals of Behavioral Medicine) and psychosomatic medicine (Psychosomatic Medicine). Finally, consistent with the goal of informing the biomedical community about the research and service activities of health psychologists, health psychologists increasingly publish their research in relevant medical journals.

Cross-References

- ▶ Behavioral Medicine
- ▶ Medical Psychology
- ▶ Psychosomatic

References and Readings

Belar, C. D., McIntyre, T. M., & Materazzo, J. D. (2003). Health Psychology. In D. B. Freedheim & I. B. Weiner (Eds.), *Handbook of psychology: History of psychology* (pp. 451–464). Hoboken, NJ: Wiley.

Brannon, L., & Feist, J. (2000). *Health psychology: An introduction to behavior and health* (4th ed.). Belmont, CA: Wadsworth/Thomson Learning.

Health Psychology. (2012). About Division 38. *Health Psychology (APA, Division 38)*. Retrieved from http://www.health-psych.org/LandingDivision.cfm

Health Psychology. (2012). Education & Training. *Health Psychology (APA, Division 38)*. Retrieved from http://www.health-psych.org/LandingEducation.cfm

Marks, D. F., Murray, M., Evans, B., Willig, C., Woodall, C., & Sykes, C. M. (2005). *Health psychology: Theory, research and practice* (2nd ed.). Thousand Oaks, CA: Sage.

Matarazzo, J. D. (1982). Behavioral health's challenge to academic, scientific, and professional psychology. *American Psychologist, 37*, 1–14.

Nezu, A. M., Nezu, C. M., & Geller, P. A. (Eds.). (2003). *Handbook of psychology: Health psychology* (Vol. 9). Hoboken, NJ: Wiley.

Health Risk

- ▶ Cancer Risk Perceptions

Health Risk (Behavior)

Peter Allebeck
Department of Public Health Sciences, Karolinska Institute, Stockholm, Sweden

Definition

Health risk is a comprehensive term covering all phenomena that carry a hazard to health of individuals or communities. Health risks can be divided into social, environmental, and behavioral risks. Another, partly overlapping, division is into health risks at population level, community level, or individual level. However, it is important to understand that health risks operate on various levels, so even if one talks about health risks on individual level, such as an individual's smoking, alcohol consumption, or lack of physical activity, these are often related to social and environmental conditions under which people live. Behavioral health risks, such as those related to eating habits, physical activity, stress, etc., have often been referred to lifestyle risk factors, although more specific terms are usually preferred.

Health Science

- ▶ Occupational Therapy

Health Strategy

- ▶ Health Policy/Health-Care Policy

Health Survey Questionnaire

- ▶ SF-36

Health Systems

- ▶ Health Insurance: Comparisons

Health-Related Quality of Life

Maartje de Wit and Tibor Hajos
Medical Psychology, VU University Medical Center, Amsterdam, North Holland, The Netherlands

Synonyms

Quality of life

Definition

In 1948, the World Health Organization defined health as "the state of complete physical, mental and social well-being and not merely the absence of disease or infirmity" (World Health Organization [WHO], 1948). Since then, QoL has become increasingly important in health-care practice and research. The term "health-related quality of life" (HRQoL) narrows QoL to aspects relevant to health. However, HRQoL is a comprehensive and complex concept for which no universally accepted definition is available (Fayers & Machin, 2000). Two aspects of HRQoL are central in most definitions. First, it is a multidimensional concept that can be viewed as a latent construct which describes the physical, role functioning, social, and psychological aspects of well-being and functioning (Bullinger, 1991; Calman, 1987; Spilker, 1990). Second, in contrast to QoL, HRQoL can include both objective and subjective perspectives in each domain (Testa & Simonson, 1996). The objective assessment focuses on what the individual can do, and it is important in defining the degree of health. The subjective assessment of QoL includes the meaning to the individual; essentially it involves the translation or appraisal of the more objective measurement of health status into the experience of QoL. Differences in appraisal account for the fact that individuals with the same objective health status can report very different subjective QoL.

HRQoL as an Outcome

It has become clear in the last decade that HRQoL is an important outcome variable on its own independent of medical outcomes. HRQoL outcomes can guide decisions on alternative treatments or effectiveness of interventions at a *patient group level* (Koot, 2001). In clinical research trials in children, HRQoL has long been neglected as an outcome, but this changed rapidly over the last 10 years (Clarke & Eiser, 2004). An important step towards a more structured and frequent use of patient-reported outcomes (PROs) in drug development is represented by the US Food and Drug Administration (FDA) guidance, issued in 2006. This describes how the FDA evaluates PROs, including HRQoL, to be used as effectiveness end points in clinical trials (U.S. Department of Health and Human Services, 2006). This guidance emphasizes the importance of considering HRQoL separate from medical effectiveness. From the *individual patient perspective*, HRQoL can guide the choice of best treatment, made by the patient himself/herself and the health-care professionals (Koot, 2001). Evaluating the impact of diabetes on the adolescents' HRQoL and vice versa can help both the patient and physician decide on the optimal individual treatment (de Wit et al., 2008).

HRQoL in Children

Attention to the QoL of children has evolved rapidly from the 1980s. Advances in medical care have changed the emphasis in pediatric medicine from the diagnosis and management of infectious disease to prevention and control of chronic conditions. This means that health-care professionals should have insight into the child's views and experiences. Early attempts to rate children's QoL were based on data provided by mothers as children are often regarded as unreliable respondents. However, children and parents do not necessarily share similar views about the impact of illness. As children grow older and develop their own life, the HRQoL reports of parents become of less relevance. It has been shown that parents and children agree more on objective domains of HRQoL (i.e., physical functioning) than on subjective domains, like emotional and social functioning (Eiser & Morse, 2001; Janse, Sinnema, Uiterwaal, Kimpen, & Gemke, 2008). Therefore, the child's HRQoL is included more and more in decisions about their care and treatment.

Cross-References

▶ Quality of Life
▶ Quality of Life: Measurement

References and Readings

Bullinger, M. (1991). Quality of life – definition, conceptualization and implications – a methodologists view. *Theoretic Surgery, 6*, 143–149.

Calman, K. (1987). Definitions and dimensions of quality of life. In N. Aaronson, J. Beckman, J. Bernheim, & R. Zittoun (Eds.), *The quality of life of cancer patients* (pp. 81–97). New York: Raven.

Clarke, S.-A., & Eiser, C. (2004). The measurement of health-related quality of life (QOL) in paediatric clinical trials: A systematic review. *Health and Quality of Life Outcomes, 2*(1), 66.

de Wit, M., Delemarre-van de Waal, H. A., Bokma, J. A., Haasnoot, K., Houdijk, M. C., Gemke, R. J., et al. (2008). Monitoring and discussing health-related quality of life in adolescents with type 1 diabetes improve psychosocial well-being: A randomized controlled trial. *Diabetes Care, 31*(8), 1521–1526.

Eiser, C., & Morse, R. (2001). Can parents rate their child's health-related quality of life? Results of a systematic review. *Quality of Life Research, 10*(4), 347–357.

Fayers, P., & Machin, D. (2000). *Quality of life. Assessment, analysis and interpretation*. Chichester: Wiley.

Janse, A. J., Sinnema, G., Uiterwaal, C. S., Kimpen, J. L., & Gemke, R. J. (2008). Quality of life in chronic illness: Children, parents and paediatricians have different, but stable perceptions. *Acta Paediatrica, 97*(8), 1118–1124.

Koot, H. M. (2001). The study of quality of life: Concepts and methods. In J. L. Wallander & H. M. Koot (Eds.), *Quality of life in child and adolescent illness. Concepts, methods and findings* (pp. 3–17). East Sussex: Brunner-Routledge.

Spilker, B. (1990). *Quality of life assessment in clinical trials*. New York: Raven.

Testa, M. A., & Simonson, D. C. (1996). Assessment of quality-of-life outcomes. *The New England Journal of Medicine, 334*(13), 835–840.

U.S. Department of Health and Human Services (2006). Patient-reported outcome measures: Use in medical product development to support labeling claims. *Guidance for Industry*. Retrieved July, 2008, from http://www.fda.gov/cder/guidance/5460dft.pdf

World Health Organisation. (1948). *The constitution of the World Health Organisation*. Washington, DC: WHO.

Healthy Cities

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

The term healthy cities (HC) refers to a policy and activity at the village, town, or city levels to promote health. This follows the World Health Organization's (WHO) conceptualization of health as one's physical, psychological, and social well-being and current health professionals' growing emphasis on people's self-care. Furthermore, HC reflect the emerging need to allocate resources to disease prevention and to the maintenance of health and well-being, beyond treatment of existing illnesses alone. When this is done at the level of a town or city, "peer pressure" becomes positive and can influence people toward more healthy lifestyles including balanced diets, physical activity, smoking cessation, moderate alcohol consumption, and provision of communal social support. Furthermore, recognizing that environmental factors influence health (e.g., crowding, pollution), HC also provide an excellent opportunity to change one's environment in order to foster health and well-being. Such initiatives are supported by the WHO via fostering programs and networks, inside and between countries (Goldstein, 2000). Additional core values in the HC project are equity, community participation, and community empowerment (Tsouros, 2009), particularly fostered by the European Healthy Cities Network (Heritage & Dooris, 2009).

An example of a HC project, which was tested, includes the Minnesota Heart Health Program (MHHP), where three intervention towns/cities were compared to three control towns/cities. The MHHP focused on health education with the aim to reduce cardiovascular morbidity and mortality. It succeeded to mobilize many community leaders, large segments of the adult population, and repeatedly exposed health-education information to residents via multiple channels of communication (Mittelmark et al., 1986). van Oers and Reelick (1992) developed quantitative indicators for evaluating HCs and also showed that such evaluation can feedback into local policy making, thus influencing health-related decisions at the city levels. According to initial findings from the European Healthy Cities Network, 80% of such cities used various forms of community participation, and more than two thirds of cities tried to empower their citizens (Heritage & Dooris, 2009). Empowerment is of

course central to health since it fosters self-efficacy, a major predictor of health outcomes (e.g., Ironson et al., 2005). This term reflects an important area of intervention for behavior medicine, where its theoretical models, methodological rigor, and clinical practice could contribute to societies' health at the "macro" level.

Cross-References

- ▶ Health Behavior Change
- ▶ Prevention: Primary, Secondary, Tertiary
- ▶ Self-Care

References and Readings

Goldstein, G. (2000). Healthy cities: Overview of a WHO international program. *Reviews on Environmental Health, 15*, 207–214.

Heritage, Z., & Dooris, M. (2009). Community participation and empowerment in healthy cities. *Health Promotion International, S1*, i45–i55.

Ironson, G., Weiss, S., Lydston, D., Ishii, M., Jones, D., Asthana, D., et al. (2005). The impact of improved self-efficacy on HIV viral load and distress in culturally diverse women living with AIDS: The SMART/EST Women's project. *AIDS Care, 17*, 222–236.

Mittelmark, M. B., Luepker, R. V., Jacobs, D. R., Bracht, N. F., Carlaw, R. W., Crow, R. S., et al. (1986). Community-wide prevention of cardiovascular disease: Education strategies of the Minnesota Heart Health Program. *Preventive Medicine, 15*, 1–17.

Tsouros, A. (2009). City leadership for health and sustainable development: The World Health Organization European Healthy Cities Network. *Health Promotion International, S1*, i4–i10.

van Oers, J. A., & Reelick, N. F. (1992). Quantitative indicators for a healthy city – The Rotterdam local health information system. *Journal of Epidemiology and Community Health, 46*, 293–296.

Healthy Eating

Sheah Rarback
Department of Pediatrics, University of Miami, Miami, FL, USA

Synonyms

Food pyramid

Definition

A healthy diet is one that maintains a state of well-being and reduces the risk of chronic diseases such as obesity, cancer, heart disease, and diabetes. A healthy food intake will have an adequate amount and balance of macronutrients (protein, carbohydrates, and fats), micronutrients (vitamins and minerals), and fluids. This goal can be accomplished with different dietary patterns.

Description

A healthy diet produces an appropriate body weight. Maintaining a healthy weight, with a body mass index between 18.5 and 24.9, is achieved by balancing total calorie intake with calorie requirements. The 2010 report of the Dietary Guidelines for Americans states that too many calories from foods high in solid fats and added sugars are contributing to obesity. These same foods offer few nutrients other than calories. Eating with mindfulness and an awareness of what, when, and how much is eaten is a useful technique for weight loss and maintenance. Limiting solid fats such as butter and lard and using limited amounts of mono- and polyunsaturated fats from plants and seeds support healthy weight and normal blood lipids.

The base of a heart-healthy intake is nutrient-rich plant foods. In addition to essential vitamins and minerals, plant foods provide phytonutrients. Phytonutrients are chemical compounds that occur naturally in plants. Phytonutrients have a beneficial effect on health but are not yet established as essential nutrients. Examples of phytonutrients are lutein and zeaxanthin in dark greens that reduce the risk of cataracts and sulforaphane in broccoli that reduces the risk of cancer. It is recommended that half of the food consumed at a meal be plant based.

The recommendation for dietary fiber is between 25 and 35 g a day. Dietary fiber assists with weight management, control of blood glucose levels, and healthy blood cholesterol levels. Dry beans, whole grains, fruits, and vegetables with skin are sources of fiber. Many different

foods, including breakfast cereals and yogurts, are fortified with extra fiber. The Nutrient Facts Label on all packaged foods lists fiber content.

Dietary protein provides essential amino acids to build body proteins and is also a calorie source. Most Americans are eating the required 0.8 g protein/kg body weight/day. Major sources of protein are lean meats, chicken, fish, dry beans, and soy products. Proteins from dry beans and soy products have the added benefit of fiber.

For a healthy diet, sodium intake should be less than 2,300 mg for healthy adults and less than 1,500 mg for individuals with hypertension, African Americans, and middle-aged and older adults. Seventy-five percent of sodium intake comes from processed and fast food. Increasing plant-based foods, cooking and eating at home, and using low-sodium canned products assist in reducing sodium intake.

Potassium helps to reduce the impact of sodium on blood pressure and is deficient in the average American diet. Most fruits and vegetables are good sources of potassium. Excellent sources are bananas, melon, oranges, spinach, fat-free milk, tomatoes, and vegetable juice.

Most fluid requirements are met through water and beverages, and a lesser amount through food. Adequate fluid is necessary for maintaining body temperature, lubricating joints, protecting spinal cord and other sensitive tissues, and ridding the body of waste. Greater fluid intake is necessary in hot climates, among physically active people, during illness such as a fever, diarrhea, or vomiting. Primary source of fluid should be water and calorie-free drinks.

A healthy meal is one half vegetables and fruits, ¼ whole grains, and ¼ lean meats or high-protein plant foods. The Food Guide Pyramid is a resource for further information about portion sizes, meal plans, and food tracking.

Cross-References

- ▶ Cholesterol
- ▶ Eating Behavior
- ▶ Fat, Dietary Intake
- ▶ Nutrition
- ▶ Nutrition Data System for Research (NDSR)

References and Readings

Anderson, A., Harris, T., Tylavsky, F., Perry, S., Houston, D., Hue, T., et al. (2011). Dietary patterns and survival of older adults. *Journal of the American Dietetic Association, 111*, 84–91.

Drewnowski, A., Darmon, N., & Briend, A. (2004). Replacing fats and sweets with vegetables and fruits-a question of cost. *American Journal of Public Health, 94*, 1555–1559.

Gao, S., Beresford, S., Frank, L., Schreiner, P., Burke, G., & Fitzpatrick, A. (2008). Modification to the healthy eating index and its ability to predict obesity: The multi-ethnic study of atherosclerosis. *American Journal of Clinical Nutrition, 88*, 64–69.

Rowe, S., Alexander, N., Almeida, N., Black, R., Burns, R., & Bush, R. (2011). Translating the dietary guidelines for Americans 2010 to bring about real behavior change. *Journal of the American Dietetic Association, 111*, 28–39.

USDA. (2011) Dietary Guidelines. www.dietaryguidelines.gov

Healthy Eating Guide

- ▶ MyPlate

Healthy Lifestyle

- ▶ Lifestyle, Active

Healthy-Years Equivalents (HYEs)

- ▶ Benefit Evaluation in Health Economic Studies

Hearing Disturbances

- ▶ Tinnitus and Cognitive Behavior Therapy

Hearing Impairment

► Hearing Loss

Hearing Impairment (Noise Pollution Related)

Margaret Wallhagen
Department of Physiological Nursing, University of California San Francisco School of Nursing, San Francisco, CA, USA

Synonyms

Noise-related hearing loss

Definition

Hearing impairment is the alteration in the perception and interpretation of sound secondary to changes in the auditory system as a result of exposure to noise.

Description

Hearing is a complex phenomenon that depends on the capture, transmission, and interpretation of sound waves from the environment. Ultimately, hearing occurs in the brain where incoming information is synthesized and interpreted. However, appropriate and accurate transmission of sound has to occur for correct interpretation to be possible. Exposure to a single loud noise or chronic exposure to noise above a sound pressure level of 85 dB (decibels) can damage the inner elements of the ear responsible for the translation of sound into signals that can be transmitted by the auditory nerve to the central nervous system. Although other components may be affected, of special concern is the impact that loud noise can have on the hair cells within the cochlea that transform sound into electrical signals that can be transmitted via the auditory nerve to the auditory cortex and related structures The level of noise exposure needed to cause permanent hearing loss may vary with the individual because of genetic or other environmental conditions. The hair cells that are responsive to high-frequency sounds, located near the base of the cochlea, are especially vulnerable. Thus, hearing loss as a result of noise exposure tends to present with a distinct audiogram pattern which usually includes a "notch" at about 4,000 Hz (Hertz; 4 kHz). A "notch" is a drop in hearing acuity at a given frequency, indicating that the sensory receptors responsive to that frequency have been damaged. Current data suggest that the damage from noise may be caused by oxidative stress. Individuals vary in their susceptibility to noise exposure, and other concurrent exposures, such as to certain medications or environmental toxins, may act synergistically with noise to further damage the hair cells. Strategies are being tested to try and protect the sensitive inner ear components before noise exposure and/or to minimize the traumatic response to the noise exposure. However, most current efforts are aimed at preventing exposure to excessive noise or minimizing the level of exposure. For example, the Occupational Safety and Health Administration has established specific requirements for hearing protection in settings where noise exceeds a given level as well as a permissible noise exposure.

Cross-References

► Hearing Impairment

References and Readings

Henderson, D., Bielfeld, E. C., Harris, K. C., & Hu, B. H. (2006). The role of oxidative stress in noise-induced hearing loss. *Ear & Hearing, 27*, 1–19.

Konings, A., Van Laer, V., & Van Camp, G. (2009). Genetic studies on noise-induced hearing loss: A review. *Ear & Hearing, 30*, 151–159.

National Institute on Deafness and Other Communication Disorders. (2008). *Noise-induced hearing loss*. (NIDCD Fact Sheet. Publication No. 08-4233, Updated December 2008). Bethesda, MD: Author.

Occupational Safety & Health Administration, Regulations (Standards 29 CFR), Part Number 1910, Standard Number 1910.95, Occupational noise exposure. Retrieved May 1, 2012, from http://www.osha.gov/pls/osha.gov/pls/oshaweb/owadisp.show_document?p_table=STANDARD&p_id=9735

Hearing Loss

Margaret Wallhagen
Department of Physiological Nursing, University of California San Francisco School of Nursing, San Francisco, CA, USA

Synonyms

Hearing impairment

Definition

A decrease, alteration, or distortion in the perception of sound.

Description

Hearing involves the perception and interpretation of sounds transmitted from the environment and occurs within the central nervous system. Sound waves enter the ear canal and stimulate the ear drum or tympanic membrane. The movement of the tympanic membrane changes the acoustic energy into mechanical energy that can then be transmitted by the three tiny inner ear bones or ossicles to the oval window of the inner ear or cochlea. The mechanical energy transmitted to the oval window initiates movement of the fluid within the inner ear. The sound energy now carried by the inner ear fluid stimulates sensitive hair cells and is transformed into electrochemical energy that can be transmitted by the auditory nerve to the auditory centers of the central nervous system. It is at the central level that sound is perceived and interpreted. Disturbances anywhere in the transmission process can lead to hearing loss. Thus, hearing loss can be conductive (disturbance in transmission from the external environment to the inner ear), sensorineural (involving alterations in the inner ear and/or auditory nerve), mixed conductive/sensorineural, or central. The cause of hearing loss can be multifactorial. Conductive loss can be caused by cerumen or wax in the ear canal which blocks sound transmission. The accumulation of wax is more common in older adults because of changes in its consistency with age. Conductive loss can also occur as a result of changes in the middle ear related to the ossicles or other diseases. Sensorineural losses can be caused by noise, ototoxic agents (e.g., environmental toxins or ototoxic medications), chronic conditions, or age. Chronic conditions such as diabetes mellitus are increasingly recognized as associated with increased incidence of hearing loss, possibly through their impact on the cardiovascular system. Age-related hearing loss is usually called "presbycusis" and is characterized initially by loss in the perception of high-frequency tones. Because consonants (such as s, p, t) are high frequency while vowels (a, e, i, o, u) are low frequency, individuals often feel they can "hear but not understand" or that individuals mumble because they are only hearing the vowels in any given word. This leads to distortion and misinterpretations. For example, "time" and "dime" may be confused. Hearing loss can occur at any time in the life cycle but becomes increasingly common with age. While hearing loss is usually not considered a life-threatening condition, it is associated with multiple negative outcomes including isolation, depression, and altered interpersonal relationships. There is also increasing interest in the relationship between hearing loss and cognition. Approaches to the treatment of hearing loss usually focus on enhancing amplification in the frequencies that are most affected. Newer hearing aids have a greater capacity to be individualized but remain aids. Individuals need to understand that they will need to relearn how to hear and to get use to hearing sounds that they may not have heard in some time. The underlying damage to the ear is

not repaired. In addition to hearing aids, other assistive listening devices are available to facilitate communication or participation in activities or events (510).

References and Readings

Chisolm, T. H., Johnson, C. E., Danhauer, J. L., Portz, L. J. P., Abrams, H. B., Lesner, S., et al. (2007). A systematic review of health-related quality of life and hearing aids: Final report of the American Academcy of Audiology Task Force on the health-related quality of life benefits of amplification in adults. *Journal of the American Academy of Audiology, 18*, 151–183.

Donaldson, N., Worrall, L., & Hickson, L. (2006). Older people with hearing impairment: A literature review of spouse's perspective. *Australian and New Zealand Journal of Audiology, 26*(1), 30–39.

Issacson, B. (2010). Hearing loss. *Medical Clinics of North America, 94*, 973–988.

Martin, F. N., & Clark, J. G. (2000). *Introduction to audiology*. Needham Heights: Allyn & Bacon.

Wallhagen, M. I. (2008). The relationship between hearing impairment and cognitive function: A 5 year longitudinal study. *Research in Gerontological Nursing, 1*(2), 80–86.

Wallhagen, M. I. (2010). The stigma of hearing loss. *The Gerontologist, 50*(1), 66–75.

Wallhagen, M. I., Pettengill, E., & Whiteside, M. (2006). Sensory impairment in older adults: Part 1: Hearing loss. *The American Journal of Nursing, 106*(10), 40–48.

Wallhagen, M. I., Strawbridge, W. J., Shema, S. J., & Kaplan, G. A. (2004). Impact of self-assessed hearing loss on a spouse: A longitudinal analysis of couples. *Journal of Gerontology: Social Sciences, 59B*(3), S190–S196.

Heart

Siqin Ye
Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Definition

The heart is the principle organ of the circulatory system and is responsible for pumping blood through the pulmonary and systemic circulations.

Description

In humans, the heart has four structural chambers: two upper ones, the left and right atria, serving as reservoirs for blood return from the pulmonary and systemic venous vasculature, respectively; and two lower chambers, the left and right ventricles, that eject blood forward into the aorta and the pulmonary arteries. In a typical cycle, peripheral venous blood returns to the right atrium via the superior and inferior venae cavae, and then empties into the right ventricle through the tricuspid valve, both passively and with contribution from right atrial contraction. Contraction of the right ventricle then opens the pulmonic valve and propels the deoxygenated blood through the main pulmonary artery on the way to the lungs. After passing through lung alveoli where gas exchange takes place, oxygenated blood from the pulmonary circulation eventually drains into the left atrium through the four pulmonary veins, and from there fills the left ventricle via passage across the mitral valve. Contraction of the left ventricle then opens the aortic valve, and oxygenated blood is pumped into the aorta to perfuse the other organs of the body. The blood supply to the heart itself is provided by the coronary arteries: the left main artery, arising from the left coronary cusp at the root of the aorta, and giving rise to the left anterior descending and the left circumflex arteries; and the right coronary artery, arising from the right coronary cusp. Venous return from the heart drains into the right atrium via the coronary sinus (Kusumoto, 2005).

Anatomically, the heart lies within the mediastinum and is encapsulated by the pericardial sac, which normally contains small amounts of serous fluid to minimize friction as the heart moves during each contraction. At the cellular level, the heart is composed of myocytes, which contain sarcomere units that form the basic contractile elements. The myocytes also possess gap junctions that allow propagation of electrical impulses from cell to cell. In addition, there are specialized myocytes with unique electrical properties that form the conduction system of the heart. Electrical impulses are typically

initiated in the sinoatrial node (SA node), located within the right atrium, then travel to the atrioventricular node (AV node), which lies along the inferior-posterior aspect of the interatrial septum near the opening of the coronary sinus. The AV node allows conduction from the atria to the ventricles, and its refractory properties help to prevent rapid ventricular response in case of atrial arrhythmias. From the AV node, the electrical impulses travel rapidly down the bundle of His and then the right, left anterior and left posterior bundle branches, eventually arriving at the Purkinje fibers that reach the rest of the heart. It is this carefully timed activation sequence that makes possible the ordered and efficient contraction that occur with each heartbeat (Kusumoto, 2005).

Numerous disease processes are known to affect the heart. Early in life, abnormalities of cardiac development can lead to various forms of congenital heart disease. Disorders of the conduction system can manifest as tachyarrhythmias and bradyarrhythmias, and can present at any age, either in isolation or in conjunction with other cardiac diseases. Valvular heart disease may involve stenotic or regurgitant lesions of any of the four cardiac valves or combinations thereof. The clinical syndrome of heart failure, defined as the inability of the heart to generate enough forward flow except at elevated filling pressures, can occur through diverse mechanisms including ischemia due to coronary artery disease to genetic disorders such as hypertrophic cardiomyopathy. Ischemic heart disease caused by coronary atherosclerosis is currently the most frequently diagnosed cardiac disorder, in part due to the increased life expectancy and changes in lifestyle and diet that occurred in the past century. Taken as a whole, cardiovascular diseases represent one of the most significant global public health burdens of the modern age, accounting for 30% of all deaths worldwide and up to 40% of all deaths in the industrialized world (American Heart Association Statistics Committee and Stroke Statistics Subcommittee, 2010; Gaziano, 2008).

Cross-References

▶ Heart Disease

References and Readings

American Heart Association Statistics Committee and Stroke Statistics Subcommittee. (2010). Heart disease and stroke statistics 2010 update: A report from the American Heart Association. *Circulation, 121*, e46–e215.

Gaziano, J. M. (2008). Global burden of cardiovascular disease. In P. Libby, R. O. Bonow, D. L. Mann, D. P. Zipes, & E. Braunwald (Eds.), *Braunwald's heart disease: A textbook of cardiovascular medicine* (pp. 1–22). Philadelphia: Saunders Elsevier.

Kusumoto, F. M. (2005). Cardiovascular disorders: Heart disease. In S. J. McPhee, V. R. Lingappa, & W. F. Ganong (Eds.), *Pathophysiology of disease: An introduction to clinical medicine* (5th ed., pp. 259–299). New York: McGraw-Hill Professional.

Heart and Estrogen/Progestin Replacement Study (HERS)

Jonathan Newman
Columbia University, New York, NY, USA

Definition

The heart and estrogen/progestin replacement study (HERS) was a randomized, double-blinded placebo-controlled trial of the effect of 0.625 mg of conjugated estrogens plus 2.5 mg of medroxyprogesterone acetate daily on coronary heart disease (CHD) event risk among more than 2,700 postmenopausal with women with known CHD.

Description

The primary outcome of this trial was CHD events (nonfatal myocardial infarction (MI) plus CHD-related death). Secondary cardiovascular outcomes included coronary revascularization, unstable angina, congestive heart failure,

resuscitated cardiac arrest, stroke or transient ischemic attack, and peripheral arterial disease.

Overall, during more than 4 years of follow-up, there were no significant differences between the hormone and placebo groups in the primary outcome of CHD events (nonfatal MI plus CHD-related death) or in any secondary outcomes.

However, post-hoc analyses showed a statistically significant time effect, wherein participants taking hormone therapy experienced more CHD events in the first year of treatment, and fewer in years 3–5. This led the investigators to speculate that the early risk due to estrogen/progestin treatment may be due to a prothrombotic, proischemic, or proarrhythmic effect which was gradually outweighed by the beneficial effects of hormone therapy, potentially mediated by observed changes in low- and high-density lipoprotein cholesterol.

This apparent pattern of an early increase in CHD events followed by a later decrease led to the recommendation that women with CHD should not start hormonal therapy for the purposes of preventing CHD events, but that those who were taking hormones could continue to do so. As reported in HERS II, women in HERS tended to follow this advice and many of those randomized to hormones during the trial continued with open-label treatment. This provided an opportunity to continue outcome surveillance for several years (HERS II).

The HERS II study contained both cardiovascular and non-cardiovascular components. Participants from the initial HERS study were followed for an additional 4 years. The HERS cardiovascular study demonstrated the absence of late benefit of hormone therapy, effectively answering the important question raised by the original HERS study of whether the lower CHD event rate observed during the final years of the trial suggested the presence of a long-term benefit of hormone replacement therapy. The non-cardiovascular component of the HERS study demonstrated an increase in venous thromboembolism and biliary tract surgery over the near 7 years of follow-up and demonstrated unfavorable trends in the incidence of some cancers and incidence of fractures.

The follow-up study (HERS II) has been critiqued because randomized assignment was no longer blinded in HERS II, and event ascertainment may have been influenced by the knowledge of randomization assignment. Regardless, the HERS I/II provided useful information regarding the short- and long-term use of hormone replacement therapy, and supported the larger, more definitive trials on hormone replacement and cardiovascular and non-cardiovascular events, such as the Women's Health Initiative.

References and Readings

Grady, D., Applegate, W., Bush, T. L., et al. (1998). Heart and estrogen/progestin replacement study (HERS): Design, methods and baseline characteristics. *Controlled Clinical Trials, 19*, 314–335.

Grady, D., Herrington, D., Bittner, V., et al. (2002). Cardiovascular disease outcomes during 6.8 years of hormone therapy heart and estrogen/progestin replacement study follow-up (HERS II). *Journal of the American Medical Association, 288*, 49–57.

Hulley, S., Furberg, C., Barrent-Connor, E., et al. (2002). Noncardiovascular disease outcomes during 6.8 years of hormone therapy heart and estrogen/progestin replacement study follow-up (HERS II). *Journal of the American Medical Association, 288*, 58–66.

Hulley, S., Grady, D., Bush, T., et al. (1998). Randomized trial of estrogen plus progestin for secondary prevention of coronary heart disease in postmenopausal women. *Journal of the American Medical Association, 280*, 605–613.

Heart Attack

▶ Acute Myocardial Infarction

Heart Bypass Surgery

▶ Bypass Surgery

Heart Disease

William Whang
Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Definition

Heart disease encompasses a range of conditions, including coronary artery disease, heart failure or inadequate cardiac output, and cardiac arrhythmia. See ▶ Coronary Heart Disease (CHD) and ▶ Congestive Heart Failure for further explanations.

Cross-References

- ▶ Atrial Fibrillation
- ▶ Congestive Heart Failure
- ▶ Coronary Heart Disease

Heart Disease and Cardiovascular Reactivity

Mark Hamer
Epidemiology and Public Health, Division of Population Health, University College London, London, UK

Synonyms

Cardiac risk factor

Definition

There has been a long-standing notion that exaggerated responses to mental stress are linked to the development of future heart disease (Hamer & Malan, 2010). Although not clinically meaningful in themselves, if heightened responses to stress are elicited on a regular basis, they might become clinically relevant over time. Existing work has largely focused on cardiovascular reactivity to stress as a tool to predict future risk. Blood pressure and heart rate responses to mental stress are largely augmented by the sympathetic nervous system and release of catecholamines. The issue of whether stress reactivity contributes to the progression of underlying disease or only to the incidence of clinical cardiac events has led to research involving indicators of subclinical disease. Several studies have indicated that heightened blood pressure and heart rate responses to laboratory-induced stressors predict future progression of subclinical atherosclerosis and hypertension in initially healthy participants, independently from conventional risk factors such as blood cholesterol, resting blood pressure, and smoking (Chida & Steptoe, 2010). Few studies have examined the association between cardiovascular reactivity and the incidence of clinical cardiac events, and the available evidence is equivocal. Since individuals cannot be randomized to being high or low cardiovascular reactors to stress, it is impossible to prove or disprove beyond doubt that exaggerated reactivity is a causal risk factor in cardiovascular disease. On balance, there is substantive evidence to support the reactivity hypothesis although effect sizes are small and the clinical utility is questionable. Reasons for inconsistencies in the data include variability in the type of mental stressors employed and individual differences in responses that might be influenced by factors such as personality, race and ethnicity, genetics, chronic background stress, and lifestyle habits.

From a mechanistic standpoint, stress-induced blood pressure surges that contribute to increased shear stress in the arteries could promote endothelial damage and inflammatory responses that are thought to play a role in atherogenesis. Endothelial dysfunction plays a key role in the initiation of atherosclerosis because nitric oxide production from healthy endothelial cells has an anti-atherogenic effect by inhibiting cellular adhesion, migration, and proliferation responses. Individual differences in blood pressure reactivity have also been linked with activation patterns in corticolimbic brain areas (e.g., divisions of the

cingulate cortex, insula, and amygdale) that are jointly involved in processing stressors and regulating the cardiovascular system (Gianaros & Sheu, 2009). Thus, this data might provide the vital piece in the missing jigsaw puzzle explaining the interaction between brain and body.

Cross-References

- ▶ Blood Pressure Reactivity or Responses
- ▶ Cold Pressor Test
- ▶ Physiological Reactivity
- ▶ Stroop Color-Word Test

References and Readings

Chida, Y., & Steptoe, A. (2010). Greater cardiovascular responses to laboratory mental stress are associated with poor subsequent cardiovascular risk status: A meta-analysis of prospective evidence. *Hypertension, 55*(4), 1026–1032.

Gianaros, P. J., & Sheu, L. K. (2009). A review of neuroimaging studies of stressor-evoked blood pressure reactivity: Emerging evidence for a brain-body pathway to coronary heart disease risk. *NeuroImage, 47*(3), 922–936.

Hamer, M., & Malan, L. (2010). Psychophysiological risk markers of cardiovascular disease. *Neuroscience and Biobehavioral Reviews, 35*(1), 76–83.

Heart Disease and Emotions: Anger, Anxiety, Depression

Timothy W. Smith
Department of Psychology, University of Utah, Salt Lake City, UT, USA

Definition

The negative emotions of anger, anxiety, and depression exist in several different forms – stable individual differences in negative emotionality (i.e., personality traits), brief emotional episodes, symptoms of emotional distress, and emotional disorders. Each of these emotion constructs has been examined as an influence on the development and course of coronary heart disease, a common cause of death resulting from the progression of atherosclerosis in the arteries that otherwise supply blood to the myocardium.

Description

Negative emotions have been suggested as an influence on the development and course of coronary heart disease (CHD) for centuries since the earliest descriptions of this common and costly medical condition. CHD begins as early as childhood or adolescence, with the development of fatty streaks within the walls of the coronary arteries. Over decades, these progress to more substantial coronary artery lesions, with the buildup of lipids and inflammatory processes at these sites. After decades of asymptomatic or silent progression of coronary artery disease (CAD), the symptoms of CHD appear in mid to later adulthood in the form of angina pectoris (chest pain due to myocardial ischemia), myocardial infarction, or sudden coronary death. Recent research indicates that negative emotion may play a role in CHD at each stage of this decades-long process. However, the various specific negative emotions and forms of these emotion characteristics are closely related, and it is not clear if they have independent or overlapping effects on the development and course of CHD (for reviews, see Smith, 2010; Suls & Bunde, 2005).

Virtually all major conceptual descriptions of basic emotions distinguish among anxiety and related emotions (e.g., fear), depression and related emotions (e.g., sadness), and anger. In contrast to a state of calm, anxiety is characterized by nervousness, tension, and apprehension. In the extreme, it is characterized by dread. Depression is characterized by sadness, sorrow, and unhappiness, and in the extreme by despair. Anger varies in intensity from irritation or annoyance to the extreme of rage. In terms of accompanying cognitive content, anxiety is associated with perceptions of threat and vulnerability to potential harm. In contrast, depression is associated with

a sense of loss, separation from important others, failure, and hopelessness. Anger is associated with thoughts of interpersonal transgression, frustrated goals, and unfair victimization or mistreatment.

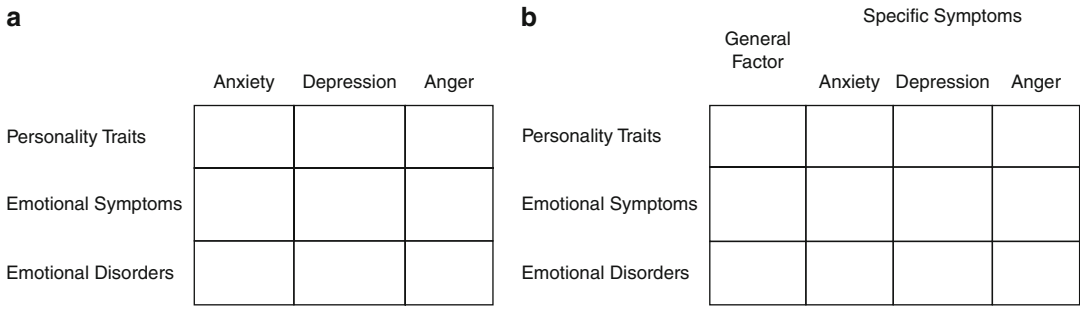
These affective phenomena take several forms, ranging from brief episodes well within the range of normal experience to much more enduring and maladaptive conditions. Episodes of negative emotion (e.g., anger during a pointed disagreement) have been examined as precipitants or "triggers" of acute coronary events (e.g., myocardial infarction, sudden coronary death), and the related findings suggest that these episodes can indeed evoke such life-threatening events, although this is most likely limited to individuals who have advanced CAD (Bhattacharyya & Steptoe, 2007). Episodes of negative emotion can be distinguished from related moods, in that emotions are more intense, briefer, and much more strongly related to physiological changes and the activation of associated behavioral response tendencies.

There are stable individual differences in the tendency to experience negative emotions and moods, such as the Five-Factor Model trait of neuroticism versus emotional stability. This trait contrasts individuals who are prone to anxiety, depression, anger, and related characteristics (e.g., self-consciousness, feelings of vulnerability or inferiority) with individuals who are generally calm. A closely related construct – negative affectivity – differs from neuroticism by including only the affective elements of this broad trait. In these trait models, individual differences in the tendency to experience anxiety, depression, and anger are seen as lower level, more specific aspects or "facets" of the broader trait.

These emotions are also seen in various forms of psychopathology. Anxiety disorders (e.g., generalized anxiety disorder, social phobia) and mood disorders (e.g., major depressive disorder) are common, and intense and destructive anger is the major feature of intermittent explosive disorder. Individuals can report elevated symptoms of anxiety, depression, or anger without reaching the severity, duration, or level of related impairment necessary to qualify for a diagnosed emotional disorder. Such elevations in symptoms are presumed to be less enduring than the negative emotionality seen in related personality characteristics, and diagnosable emotional disorders are seen as qualitatively distinct from personality traits in their severity, related features, and levels of general impairment or dysfunction. Hence, anxiety, depression, and anger are typically conceptualized and studied as distinct emotions, as are their various forms in emotional episodes, symptoms, disorders, and personality traits.

If these distinctions were in fact firm, the common approach of studying one emotional risk factor for CHD at a time would not pose a problem. Unfortunately, these distinctions are quite difficult to support empirically (Smith, 2010; Suls & Bunde, 2005). There is a high degree of correlation among anxiety, depression, and anger, and this is true in each of the various forms. Further, the distinctions between the various forms of negative emotions are problematic. Symptoms of emotional distress are closely correlated with related personality traits, and scores on such symptom inventories are more stable than should be the case if they were unrelated to personality (Suls & Bunde; Watson, 2009). Neuroticism and negative affectivity are closely associated with anxiety and mood disorders (Weinstock & Whisman, 2006), and symptoms of emotional disorders often appear to take the form of continuous severity distributions rather than the discrete structure implied by categorical models of emotional disorder (Haslam, 2007). Emotional disorders co-occur so frequently that alternative diagnostic frameworks involving "distress disorders" have been proposed (Watson, 2009). Hence, when one specific negative affect risk factor is related to the development or course of CHD, this association could easily involve another negative affect and/or another form of negative affect. This lack of specificity complicates the design and implementation of risk-reducing interventions, in that it is not clear which specific negative affect or which form of negative affect should be targeted.

Reviews of the many studies of negative emotional factors and the development and course of CHD suggest reliable associations for depression, anxiety, and anger when these characteristics are



Heart Disease and Emotions: Anger, Anxiety, Depression, Fig. 1 (**a**) Conceptual framework for overlapping negative affective risk factors (**b**) Alternative framework including general and specific aspects of negative affect (Reprinted with permission from Smith (2010) (Fig. 12.1, p. 161))

considered separately (Chida & Steptoe, 2009; Nicholson, Kuper, & Hemingway, 2006; Suls & Bunde, 2005). However, the far fewer studies attempting to parse the potentially overlapping effects of these negative emotional phenomena have produced mixed results. Studies of preclinical, asymptomatic atherosclerosis that have measured multiple negative affective characteristics have sometimes found that depression but not anxiety or anger predicts disease severity (Stewart et al., 2007), whereas others have found that anxiety and anger but not depression are related to CAD (Smith, Uchino et al., 2008). Studies of multiple indicators of negative affect as predictors of the initial occurrence of CHD have produced evidence of the unique or independent effects of anxiety (Phillips et al., 2009; Kubzansky et al., 2006; Shen et al., 2008), depression (Grossardt et al., 2009; Phillips et al., 2009), and general emotional distress (Boyle et al. 2006; Kubzansky et al., 2006). In studies of patients with CHD, the evidence is again mixed. Some studies suggest that anxiety and depression are independent predictors of recurrent cardiac events (Frasure-Smth & Lespérance 2008), whereas others suggest that anxiety but not depression has an independent effect (Tully et al., 2008).

Additional research is clearly needed to sort out the independent versus overlapping effects of the various negative emotions and their various forms, across the multiple phases of the development and course of CHD. The design of future studies must attend to the fact that anxiety, depression, and anger are correlated, and the overlap across various forms of these emotions is considerable. Figure 1 presents two conceptual models that can guide these efforts. In the first (panel A), anxiety, depression, and anger are each considered and assessed separately, as are the various forms of these emotional constructs. In the second, the general factor (i.e., general emotional distress) is considered and assessed directly, as are the unique aspects of anxiety, depression, and anger. For example, depression differs from anxiety and anger in its associated low levels of positive affect and physiological activation (Watson, 2009), and anger involves approach motivation (e.g., overcoming obstacles in frustrated goals, redress social transgressions or mistreatment), whereas anxiety is mostly associated with avoidance motives (i.e., minimize harm). Additional studies that measure these multiple aspects and forms of negative emotion could help define targets for risk-reducing interventions more precisely.

Interventions for CHD patients focused on a specific negative affect (e.g., depression) generally have not been found to be effective in reducing recurrent coronary events (ENRICHD Investigators, 2003). In contrast, more general interventions focusing on stress and negative emotion broadly considered have been found to be effective in improving the prognosis of CHD patients (Linden, Phillips, & Leclerc, 2007). If supported in future research, it is possible that this pattern of intervention results indicates that it is the general or overlapping aspects of negative emotion – rather than the specific aspects – that

are important risk factors and important intervention opportunities. However, much additional research is needed.

Cross-References

- ▶ Anger Management
- ▶ Anger, Measurement
- ▶ Anxiety and Heart Disease
- ▶ Anxiety Disorder
- ▶ Depression: Measurement
- ▶ Depression: Symptoms
- ▶ Depression: Treatment
- ▶ Type A Behavior
- ▶ Type D Personality

References and Readings

Bhattacharyya, M. R., & Steptoe, A. (2007). Emotional triggers of acute coronary syndromes: Strength of evidence, biological processes, and clinical implications. *Progress in Cardiovascular Diseases, 49*, 353–365.

Boyle, S. H., Michalek, J. E., & Suarez, E. C. (2006). Covariation of psychological attributes and incident coronary heart disease in U.S. Air Force veterans of the Vietnam War. *Psychosomatic Medicine, 68*, 844–850.

Carver, C. S., & Harmon-Jones, E. (2009). Anger is an approach-related affect: Evidence and implications. *Psychological Bulletin, 135*, 183–204.

Chida, Y., & Steptoe, A. (2009). The association of anger and hostility with future coronary heart disease: A meta-analytic review of prospective evidence. *Journal of the American College of Cardiology, 53*, 774–778.

ENRICHD Investigators. (2003). Effects of treating depression and low perceived social support on clinical events after myocardial infarction. *Journal of the American Medical Association, 289*, 3106–3116.

Frasure-Smth, N., & Lespérance, F. (2008). Depression and anxiety as predictors of 2-year cardiac events in patients with stable coronary artery disease. *Archives of General Psychiatry, 65*, 62–71.

Grossardt, B. R., Bower, J. H., Geda, Y. E., Colligan, R. C., & Rocca, W. A. (2009). Pessimistic, anxious, and depressive personality traits predict all-cause mortality: The Mayo Clinic Cohort Study of personality and aging. *Psychosomatic Medicine, 71*, 491–500.

Haslam, N. (2007). The latent structure of mental disorders: A taxometric update on the categorical vs. dimensional debate. *Current Psychiatry Reviews, 3*, 172–177.

Kubzansky, L. D., Cole, S. R., Kawachi, I., Volonas, P., & Sparrow, D. (2006). Shared and unique contributions of anger, anxiety, and depression to coronary heart disease: A prospective study in the normative aging study. *Annals of Behavioral Medicine, 31*, 21–29.

Linden, W., Phillips, M. J., & Leclerc, J. (2007). Psychological treatment of cardiac patients: A meta-analysis. *European Heart Journal, 28, 24*, 2964–2966.

Nicholson, A., Kuper, H., & Hemingway, H. (2006). Depression as an aetiologic and prognostic factor in coronary heart disease: A meta-analysis of 6362 events among 146,538 participants in 54 observational studies. *European Heart Journal, 27*, 2763–2774.

Phillips, A. C., Batty, G. D., Gale, C. R., Deary, I. J., Osborn, D., Macintrye, K., et al. (2009). Generalized anxiety disorder, major depressive disorder and their comorbidity as predictors of all-cause and cardiovascular mortality: The Vietnam Experience Study. *Psychosomatic Medicine, 71*, 395–403.

Shen, B.-J., Avivi, Y. E., Todaro, J. F., Spiro, A., Laurenceau, J., Ward, K. D., & Niaura, R. (2008). Anxiety characteristics independently and prospectively predict myocardial infarction in men. *Journal of the American College of Cardiology, 51*, 113–119.

Smith, T. W. (2010). Conceptualization, measurement, and analysis of negative affective risk factors. In A. Steptoe (Ed.), *Handbook of behavioral medicine: Methods and applications* (pp. 155–168). New York: Springer.

Smith, T. W., Uchino, B. N., Berg, C. A., Florsheim, P., Pearce, G., Hawkins, M., Henry, N., Beveridge, R., Skinner, M., Hopkins, P. N., & Yoon, H. C. (2008). Self-reports and spouse ratings of negative affectivity, dominance and affiliation in coronary artery disease: Where should we look and who should we ask when studying personality and health? *Health Psychology, 27*, 676–684.

Stewart, J. C., Janicki, D. L., Muldoon, M. F., Sutton-Tyrell, K., & Kamarch, T. W. (2007). Negative emotions and 3-year progression of subclinical atherosclerosis. *Archives of General Psychiatry, 64*, 225–233.

Suls, J., & Bunde, J. (2005). Anger, anxiety, and depression as risk factors for cardiovascular disease: The problems and implications of overlapping affective dispositions. *Psychological Bulletin, 131*, 260–300.

Tully, P. J., Baker, R. A., & Knight, J. L. (2008). Anxiety and depression as risk factors for mortality after coronary artery bypass surgery. *Journal of Psychosomatic Research, 64*, 285–290.

Watson, D. (2009). Differentiating the mood and anxiety disorders: A quadripartite model. *Annual Review of Clinical Psychology, 5*, 221–247.

Weinstock, L. M., & Whisman, M. A. (2006). Neuroticism as a common feature of the depressive and anxiety disorders: A test of the revised integrative hierarchical model in a national sample. *Journal of Abnormal Psychology, 115*, 68–74.

Widiger, T. A., & Smith, G. T. (2008). Personality and psychopathology. In O. P. John, R. W. Robbins, & L. A. Pervin (Eds.), *Handbook of personality: Theory and research* (3rd ed., pp. 743–769). New York: Guilford Press.

Heart Disease and Smoking

Scott DeBerard
Department of Psychology, Utah State University, Logan, UT, USA

Synonyms

Cardiovascular disease (CVD); Coronary heart disease (CHD); Nicotine

Definition

Heart disease is a relatively broad term which refers to many diseases associated with the heart. Coronary heart disease (CHD) refers to a narrowing or even complete occlusion of the blood vessels which supply the heart with blood and oxygen. Cardiovascular disease (CVD) is often used as a synonym for CHD. Smoking is defined as inhalation of smoke from burning tobacco via a cigarette, cigar, pipe, or other smoke delivery device (e.g., hookah).

Description

Approximately 20% of adults smoke in the United States, and this percentage has remained fairly consistent across the last 10 years (Centers for Disease Control and Prevention, 2010). It is currently estimated that over 140,000 individuals in the US will die annually due to smoking-attributable CHD (Centers for Disease Control and Prevention, 2008). There is currently compelling evidence that smoking is causally related to cardiovascular diseases. The most extensive current compilation of evidence supporting a causal link between smoking and CHD is found in the 2010 Surgeon General's Report: *How Tobacco Smoke Causes Disease: The Biology and Behavioral Basis for Smoking-Attributable Disease* (U.S. Department of Health and Human Services, 1994). What follows is a brief overview of the evidence for a causal link between smoking and CHD including a discussion of the biological processes impacted by smoking that are implicated in CHD.

There are numerous studies which demonstrate a clear dose–response relationship between number of cigarettes smoked per day and risk for developing CHD. The recent Surgeon General's Report on Smoking (Centers for Disease Control and Prevention, 2010) indicated that risk for CHD is increased across all levels of smoking and that smoking even a few cigarettes per day (less than five) resulted in increased risk of developing CHD. This report also indicated that duration of smoking is associated with increased risk of CHD-related mortality.

Exposure to secondhand smoke also increases the risk of CHD. One study determined that the relative risk of CHD for smokers is 1.78 as compared with 1.31 in nonsmokers exposed to secondhand smoke. This study also revealed that exposure to secondhand smoking resulted in similar negative circulatory changes to those which occur in smokers. There is also evidence of decreased hospital admissions due to myocardial infarction rates in locations which have banned public smoking (Barnoya & Glantz, 2005)

There are multiple physiological changes which occur in response to smoking which are thought to be associated with CHD. Cigarette smoking results in decreased blood flow to the heart and increased blood pressure and heart rate (U.S. Department of Health and Human Services, 1994). Smoking damages and impairs regeneration of the inner lining of blood vessels (endothelium). Damaged blood vessels increase the risk of developing arterial plaques (atherosclerosis) which consequentially increases the risk of developing blood clots which can lead to myocardial infarct (U.S. Department of Health and Human Services). Cigarette smoking causes inflammation in the circulatory system and also impairs vasodilation of blood vessels, both of which increase the risk for negative cardiac events (U.S. Department of Health and Human Services). Smoking can also cause platelets in the blood to become sticky, and this is thought to increase the likelihood of clot formation (U.S. Department of Health and Human Services). Importantly, similar

physiological changes have been demonstrated in persons exposed to secondhand smoke (Barnoya & Glantz 2005).

Quitting smoking will reduce the risks of developing CHD (U.S. Department of Health and Human Services). The risk for CHD and CHD-related mortality drops in half after approximately 1 year of smoking cessation. After 10 years of smoking cessation, the risk for developing CHD-related issues is essentially equivalent to that of a nonsmoker (U.S. Department of Health and Human Services). Smoking cessation at any age is also clearly associated with increased longevity (Taylor et al. 2002).

Cross-References

- ▶ American Heart Association
- ▶ Atherosclerosis
- ▶ Cardiac Rehabilitation
- ▶ Cigarette Smoking Behavior
- ▶ Coronary Heart Disease
- ▶ Ex-Smokers
- ▶ Heart Disease
- ▶ Heart Failure
- ▶ Inflammation
- ▶ Secondhand Smoke
- ▶ Smoking and Health
- ▶ Smoking Behavior
- ▶ Smoking Cessation
- ▶ Tobacco Cessation
- ▶ Tobacco Use

References and Readings

Barnoya, J., & Glantz, S. A. (2005). Cardiovascular effects of secondhand smoke: Nearly as large as smoking. *Circulation, 111*, 2684–2698.

Centers for Disease Control and Prevention. (2010). Vital signs: Current cigarette smoking among adults aged ≥18 years-United States, 2009. *Morbidity and Mortality Weekly Report, 59*(35), 1135–1140 (Accessed 2011 Sept 7).

Centers for Disease Control and Prevention. (2008). Annual smoking-attributable mortality, years of potential life lost, and productivity losses-United States, 2000–2004. *Morbidity and Mortality Weekly Report, 57*(45), 1226–1228 (Accessed 2011 Sept 7).

Taylor, D. H., Hasselblad, V., Henley, J., Thun, M. J., & Sloan, F. A. (2002). Benefits of smoking cessation for longevity. *American Journal of Public Health, 92*(6), 990–996.

U.S. Department of Health and Human Services. (1994). *How Tobacco Smoke Causes Disease: The biology and behavioral basis for smoking -attributable disease: A report of the surgeon general*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health.

Heart Disease and Stress

Peter A. Shapiro
Department of Psychiatry, Columbia University, New York, NY, USA

Definition

Stress is a hard concept to define precisely, but like obscenity (to paraphrase Supreme Court Justice Potter Stewart (*Jacobellis vs. Ohio. 378 U.S. 184, 197, 1964*)), you (sometimes, but perhaps not always) know it when you feel it. For any given system, stress implies a load that challenges the homeostasis and integrity of the system. Stress may be intrinsic to the system, for example, the weight of a suspension bridge's roadway platform, or extrinsic to the system, such as the effect of hurricanes, earthquakes, or traffic jams loading the bridge. Both intrinsic and extrinsic stressors pose the risk of bridge collapse. Whether a stressor is intrinsic or extrinsic, the system must have a capacity to withstand it in order to maintain structural and/or functional integrity. Thus for a suspension bridge, to continue the example, this capacity is built into the tensile strength of its suspension cables and the mass and strength under compression of the towers and buttresses supporting the cables and transmitting force to the surrounding ground. In behavioral cardiology, concern about stress and heart disease has focused on external psychological stressors, and related behaviors and affective and psychophysiological states, associated with

the onset and progression of heart disease, especially coronary artery disease and sudden cardiac death.

Description

For lack of a precise, gold-standard, quantifiable measure of stress as it relates to heart disease, stress is usually defined by subjective measures of perceived stress, by characterization and counts of common life experiences generally regarded as stressful, unwanted, or at least as disturbing homeostasis (e.g., daily hassles, hostile interactions, job strain, job loss, loss of a loved one, divorce, interpersonal conflict, moving to a new city, marriage, promotion to new work responsibilities), by experimental manipulations or ecological measurement of short-term challenges, and by exposures to epidemiologically significant events such as natural disasters, missile attacks during wartime, and terrorism. It is evident that stress is often associated with negatively valenced affective states such as anxiety, depression, and anger, but can also be associated with more positively experienced acute excitement and life experiences.

Evidence for the association of stress with heart disease is overwhelming. Numerous studies demonstrate that earthquakes are associated with a subsequent increase over background rates in myocardial infarction and sudden death. Iraqi missile attacks on Israel during the Gulf War in the 1990s, the destruction of the World Trade Center in New York in 2001, and sports events such as World Cup Soccer matches have been consistently observed to be associated with increased rates of acute coronary syndromes and lethal ventricular arrhythmias, even in individuals not physically endangered by proximity to the events. Acute episodes of anger appear to be an especially potent trigger of acute coronary events in vulnerable patients. In INTERHEART, a very large case–control study of first myocardial infarction involving over 24,000 subjects in 52 countries, chronic exposure to stress, measured by self-reports of problems with family relationships, work, or financial strain over a 1-year period, was associated with increased rate of MI, with population-attributable risk estimated at 12–33%. A dose–response relationship was observed between the number of stressful life events reported and risk of myocardial infarction, with an odds ratio for MI risk of 1.5 for individuals reporting two or more stressful life events compared to those reporting no stressful life events. Thus, epidemiological evidence supports the role of emotional stress both over the long term, in processes contributing to the development of coronary atherosclerosis, and in the short term, as a trigger of acute coronary events in vulnerable patients. Perceived stress is heightened in people with low socioeconomic status, which may also be associated with numerous other behavioral and physiological cardiac risk factors. Conversely, stress effects on cardiovascular risk are moderated by the presence of good social support. Studies comparing heart disease risk in men and women have found that for women, the relationship of stress at work to coronary heart disease risk is less clear-cut than in men, but stress at home and in interpersonal relationships is associated with increased risk of coronary disease-related events.

Stressful experiences result in derangements of autonomic nervous system regulation of the cardiovascular system, with reduced vagal tone and relatively increased sympathetic tone. Heart rate and blood pressure increase in response to acute mental stress; the extent of this increase depends in part on the nature of the stressor and also in part on individual characteristics such as trait anxiety and hostility. Interruption of sympathetic inflow to the left side of the heart by left stellate ganglionectomy reduces stress-induced ischemia and ventricular arrhythmias. Stressful experience is associated with hemoconcentration and increased blood viscosity, which may increase the risk of thrombosis, especially as clotting factors and platelet activation also increase during acute stress. Stressful experiences alter hypothalamic-pituitary axis function, with lasting consequences, such that childhood trauma is associated with elevated circulating levels of corticotrophin-releasing factor in adulthood, and stress in adulthood leads to hypercortisolemia.

Endothelial dysfunction and paradoxical vasoconstriction during acute mental stress are observed in atherosclerotic coronary artery segments. During mental stress, increased heart rate and blood pressure along with coronary vasoconstriction may result in myocardial ischemia, which may be manifest as ST segment changes in the electrocardiogram, reduced left ventricular ejection fraction and regional wall motion abnormalities; stress-induced impairment of left ventricular function is associated with an increase in the risk of recurrent coronary events and survival. Yet, although mental stress–induced ischemia occurs primarily in individuals with exercise-induced ischemia, it is notable that mental stress–induced myocardial ischemia is frequently asymptomatic, and occurs at a level of increased heart rate and blood pressure less than that required to achieve ischemia during exercise. Acute mental stress also induces an inflammatory response, with elevated levels of inflammatory cytokines IL-6 and TNF-alpha, which can destabilize atherosclerotic plaque and promote plaque rupture or superimposed thrombosis. The extent to which each of these mechanisms mediates the effect of stress on heart disease risk is unknown.

Sudden emotional stress can also lead to acute cardiac events in patients who do not have coronary artery disease. Takotsubo cardiomyopathy is a rare syndrome characterized by transient left ventricular dysfunction with apical ballooning and acute heart failure in response to emotional shocks, such as surprise parties or receiving unexpected bad news. The syndrome has been attributed to rapidly increasing sympathetic tone and catecholamine levels in response to the psychological stressor.

Studies of the effects of interventions to reduce stressful experience or improve resilience in managing stress on heart disease outcomes have had decidedly mixed results. Behavioral or cognitive behavioral stress management treatment groups to reduce angry responding and time urgency reduce the incidence of recurrent MI. Inclusion of "stress management" in cardiac rehabilitation programs for post-MI patients is associated with reduced recurrent cardiac events and reduced mortality, but individualized life stress monitoring and problem-solving interventions for post-MI patients resulted in no benefit in men and increased recurrent cardiac events in women in the Montreal Heart Attack Readjustment Trial (M-HART). Cognitive behavior therapy aimed at bolstering social support also had a negative effect on recurrent cardiac events in women in the ENRICHD trial. Overall, however, exercise and stress management programs appear to reduce recurrent cardiac events, improve subjective well-being, and may reduce hemodynamic and other physiological responses to stress.

Cross-References

- ▶ Coronary Artery Disease
- ▶ Social Support
- ▶ Stress

References and Readings

Albus, C. (2010). Psychological and social factors in coronary heart disease. *Annals of Medicine, 42*, 487–494.

Dimsdale, J. E. (2008). Psychological stress and cardiovascular disease. *Journal of the American College of Cardiology, 51*, 1237–1246.

Kent, L. K., & Shapiro, P. A. (2009). Depression and related psychological factors in heart disease. *Harvard Review of Psychiatry, 17*, 377–388.

Leeka, J., Schwartz, B. G., & Kloner, R. A. (2010). Sporting events affect spectators' cardiovascular mortality: It is not just a game. *American Journal of Medicine, 123*, 972–977.

Low, C. A., Thurston, R. C., & Matthews, K. A. (2010). Psychosocial factors in the development of heart disease in women: Current research and future directions. *Psychosomatic Medicine, 72*, 842–854.

Rozanski, A., Blumenthal, J. A., Davidson, K. W., Saab, P. G., & Kubzansky, L. (2005). The epidemiology, pathophysiology, and management of psychosocial risk factors in cardiac practice. The emerging field of behavioral cardiology. *Journal of the American College of Cardiology, 45*, 637–651.

Shapiro, P. A. (2011). Heart disease. In J. L. Levenson (Ed.), *Textbook of psychosomatic medicine* (2nd ed., pp. 407–440). Washington, DC: APPI.

Steptoe, A., & Brydon, L. (2009). Emotional triggering of cardiac events. *Neuroscience and Biobehavioral Reviews, 33*, 63–70.

Heart Disease and Type A Behavior

Yoichi Chida
Research Department of Epidemiology & Public Health, University College London, London, UK

Synonyms

Coronary heart disease (CHD); Type A behavior pattern (TABP)

Definition

A comprehensive meta-analysis of prospective studies between 1966 and 1998 failed to show an association between Type A behavior pattern (TABP) and coronary heart disease (CHD) (Myrtek, 2001), and since then there has been no evidence showing such an association. However, the recent meta-analytic investigation on prospective studies showed that anger and hostility, one of the key dimensions of TABP, are significantly associated with not only increased CHD events in initially healthy populations but also poor prognosis in the patients with existing CHD (Chida & Steptoe, 2009).

Description

Type A behavior pattern (TABP) was first noted by two American cardiologists, Meyer Fredman and Ray Rosenman, in 1959 (Friedman & Rosenman, 1959). They demonstrated that a group of 83 men, mainly executives, had seven times the risk of coronary heart disease (CHD) when compared with that in the other two groups of 83 men from unions and accounting firms and 46 unemployed blind men. The behavior pattern of group A is characterized by intense ambition, competitive "drive," constant preoccupation with "deadlines," and a sense of time urgency. Over time, together with input from psychologists, they refined the notion of TABP with an increasing focus on the elements of aggressiveness and easily aroused annoyance or hostility (Rosenman, 1978). Persons not showing these characteristics are labeled Type B behavior pattern. Data published in 1974 and 1976 from the Western Collaborative Group Study containing approximately 3,000 middle-aged men, exhibited a relative risk of 1.8 for nonfatal myocardial infarction (MI) or angina over 4 years in those were TABP, and a relative risk of 1.9 for fatal CHD, nonfatal MI, or angina over 8.5 years in those who were TABP (Jenkins, Rosenman, & Zyzanski, 1974; Rosenman, Brand, Sholtz, & Friedman, 1976). Nevertheless, as time passed, these original findings were not supported by an increasing number of subsequent studies (Brotman, Golden, & Wittstein, 2007; Everson-Rose & Lewis, 2005). More comprehensively, a meta-analysis of prospective studies between 1966 and 1998 failed to show an association between TABP and CHD (Myrtek, 2001), and since then there has been no evidence showing such an association.

Some researchers, therefore, changed their focus to investigate whether anger, hostility, and related constructs, one of the key dimensions of TABP, would be more closely linked to the development of CHD. Hostility is typically described as a negative attitude or cognitive trait directed toward others, anger as an emotional state that consists of feelings that vary in intensity from mild irritation or annoyance to intense fury or rage, and aggressiveness as a verbal or physical behavioral pattern manifest in yelling, intimidation, or physical assaults. These constructs, these terms often are used interchangeably and their interrelationship remains poorly delineated (Martin, Watson, & Wan, 2000; Schulman & Stromberg, 2007). Over the past 25 years, the body of research investigating associations between anger and hostility and CHD development and progression has grown. The recent meta-analytic investigation on prospective studies showed that anger and hostility are significantly associated with not only increased CHD events in initially healthy populations but also poor prognosis in the patients with existing CHD (Chida & Steptoe, 2009). The harmful effects of anger and hostility were slightly greater in the CHD patients than the healthy population

studies, making it possible that frequent anger episodes related to trait anger and hostility trait might accelerate recurrence of CHD.

If anger and hostility do influence CHD risk, effects might be primarily mediated via behavioral pathways, with anger and hostility promoting high-risk behaviors such as poor diet, less physical activity, smoking, poor sleep, or lower treatment adherence (Scherwitz et al., 1992; Shin et al., 2005; Siegler, Peterson, Barefoot, & Williams, 1992). Indeed, the apparently harmful effects of anger and hostility on CHD were no longer significant in either the healthy or disease populations after fully controlling for behavioral covariates such as smoking, physical activity or body mass index, and socioeconomic status (Chida & Steptoe, 2009). However, other unmeasured factors cannot rule out that could potentially have confounded the associations, and direct physiological pathways might also contribute. Anger and hostility may alter susceptibility to CHD via autonomic nervous dysregulation (Chida & Hamer, 2008; Thomas, Nelesen, & Dimsdale, 2004; Vella & Friedman, 2007), increases in inflammatory and coagulation factors such as interleukin-6, C-reactive protein, and fibrinogen (Markovitz, 1998; Stewart, Janicki-Deverts, Muldoon, & Kamarch, 2008), and higher cortisol levels (Steptoe, Cropley, Griffith, & Kirschbaum, 2000). Furthermore, a number of studies have demonstrated that anger and cynical hostility predict the progression of subclinical atherosclerosis (Matthews et al., 1998; Raikkonen, Matthews, Sutton-Tyrrell, & Kuller, 2004), suggesting that the associations between anger/hostility and CHD may be due to the impact of anger and hostility on the development of coronary atherosclerosis, although acute trigger effects may also contribute (Dimsdale, 2008; Mittleman et al., 1995; Möller et al., 1999).

Taken together, given a recent meta-analysis on randomized controlled trials endorsing the efficacy of psychological interventions in cardiac patients (Linden, Phillips, & Leclerc, 2007), clinical trials focusing on anger and hostility in CHD interventions are necessary for the more effective prevention and treatment of CHD.

Cross-References

- ▶ Aerobic Exercise
- ▶ Anger, Measurement
- ▶ Coronary Event
- ▶ Coronary Heart Disease
- ▶ Hostility, Measurement of

References and Readings

Brotman, D. J., Golden, S. H., & Wittstein, I. S. (2007). The cardiovascular toll of stress. *Lancet, 370*, 1089–1100.

Chida, Y., & Hamer, M. (2008). Chronic psychosocial factors and acute physiological responses to laboratory-induced stress in healthy populations: a quantitative review of 30 years of investigations. *Psychological Bulletin, 134*, 829–885.

Chida, Y., & Steptoe, A. (2009). The association of anger and hostility with future coronary heart disease: A meta-analytic review of prospective evidence. *Journal of the American College of Cardiology, 53*, 936–946.

Dimsdale, J. E. (2008). Psychological stress and cardiovascular disease. *Journal of the American College of Cardiology, 51*, 1237–1246.

Everson-Rose, S. A., & Lewis, T. T. (2005). Psychosocial factors and cardiovascular disease. *Annual Review of Public Health, 26*, 469–500.

Friedman, M., & Rosenman, R. H. (1959). Association of specific overt behavior pattern with blood and cardiovascular findings: Blood cholesterol level, blood clotting time, incidence of arcus senilis, and clinical coronary artery disease. *Journal of the American Medical Association, 169*, 1286–1296.

Jenkins, C. D., Rosenman, R. H., & Zyanski, S. J. (1974). Prediction of clinical coronary heart disease by a test of the coronary-prone behaviour pattern. *The New England Journal of Medicine, 290*, 1271–1275.

Linden, W., Phillips, M. J., & Leclerc, J. (2007). Psychological treatment of cardiac patients: A meta-analysis. *European Heart Journal, 28*, 2972–2984.

Markovitz, J. H. (1998). Hostility is associated with increased platelet activation in coronary heart disease. *Psychosomatic Medicine, 60*, 586–591.

Martin, R., Watson, D., & Wan, C. K. (2000). A three-factor model of trait anger: Dimensions of affect, behavior, and cognition. *Journal of Personality, 68*, 869–897.

Matthews, K. A., Owens, J. F., Kuller, L. H., Sutton-Tyrrell, K., & Jansen-McWilliams, L. (1998). Are hostility and anxiety associated with carotid atherosclerosis in healthy postmenopausal women? *Psychosomatic Medicine, 60*, 633–638.

Mittleman, M. A., Maclure, M., Sherwood, J. B., Mulry, R. P., Tofler, G. H., Jacobs, S. C., et al. (1995).

Triggering of acute myocardial-infarction onset by episodes of anger. *Circulation, 282*, 1720–1725.
Möller, J., Hallqvist, J., Diderichsen, F., Theorell, T., Reuterwall, C., & Ahlbom, A. (1999). Do episodes of anger trigger myocardial infarction? A case-crossover analysis in the Stockholm Heart Epidemiology Program (SHEEP). *Psychosomatic Medicine, 61*, 842–849.
Myrtek, M. (2001). Meta-analyses of prospective studies on coronary heart disease, type A personality, and hostility. *International Journal of Cardiology, 79*, 245–251.
Raikkonen, K., Matthews, K. A., Sutton-Tyrrell, K., & Kuller, L. H. (2004). Trait anger and the metabolic syndrome predict progression of carotid atherosclerosis in healthy middle-aged women. *Psychosomatic Medicine, 66*, 903–908.
Rosenman, R. H. (1978). The interview method of assessment of the coronary-prone behaviors in the Western Collaborative Group Study. In T. M. Dembroski, S. M. Weiss, J. L. Shields, et al. (Eds.), *Coronary prone behavior* (pp. 55–69). New York: Springer.
Rosenman, R. H., Brand, R. J., Sholtz, R. I., & Friedman, M. (1976). Multivariate prediction of coronary heart disease during 8.5 year follow-up in the Western Collaborative Group Study. *The American Journal of Cardiology, 37*, 903–909.
Scherwitz, L. W., Perkins, L. L., Chesney, M. A., Hughes, G. H., Sidney, S., & Manolio, T. A. (1992). Hostility and health behaviors in young adults -the CARDIA study. *American Journal of Epidemiology, 136*, 136–145.
Schulman, J. K., & Stromberg, S. (2007). On the value of doing nothing. *Cardiology in Review, 15*, 123–132.
Shin, C., Kim, J. Y., Yi, H., Lee, H. J., Lee, J. B., & Shin, K. (2005). Relationship between trait anger and sleep disturbances in middle-aged men and women. *Journal of Psychosomatic Research, 58*, 183–189.
Siegler, I. C., Peterson, B. L., Barefoot, J. C., & Williams, R. B. (1992). Hostility during late adolescence predicts coronary risk factors at mid-life. *American Journal of Epidemiology, 136*, 146–154.
Steptoe, A., Cropley, M., Griffith, J., & Kirschbaum, C. (2000). Job strain and anger expression predict early morning elevation in salivary cortisol. *Psychosomatic Medicine, 62*, 286–292.
Stewart, J. C., Janicki-Deverts, D., Muldoon, M. F., & Kamarch, T. W. (2008). Depressive symptoms moderate the influence of hostility on serum interleukin-6 and C-reactive protein. *Psychosomatic Medicine, 70*, 197–204.
Thomas, K. S., Nelesen, R. A., & Dimsdale, J. E. (2004). Relationships between hostility, anger expression, and blood pressure dipping in an ethnically diverse sample. *Psychosomatic Medicine, 66*, 298–304.
Vella, E. J., & Friedman, B. H. (2007). Autonomic characteristics of defensive hostility: reactivity and recovery to active and passive stressors. *International Journal of Psychophysiology, 66*, 95–101.

Heart Doctor

► Cardiologist

Heart Failure

Valerie Sabol
School of Nursing, Duke University, Durham, NC, USA

Synonyms

Congestive heart failure

Definition

Heart failure is a general term used to describe a clinical syndrome characterized by shortness of breath, exertional dyspnea, paroxysmal nocturnal dyspnea, orthopnea, peripheral and/or pulmonary edema, and exercise intolerance. Since individual experiences of these clinical indicators may vary, it is important to understand how heart failure is classified. The underlying etiology and associated pathophysiology are keys to appropriate diagnosis and management.

Several categories are used to describe, organize, and classify heart failure. Acute versus chronic heart failure are terms used to describe both the onset and intensity of symptoms. Acute heart failure refers to the sudden appearance of symptoms (e.g., usually over hours or days), which have progressed to a point at which immediate or emergency medical intervention is necessary. Chronic heart failure refers to the development of symptoms over a period (e.g., months to years). Chronic symptoms represent the baseline condition or symptoms that an individual lives with on a daily basis. If the cause of the acute onset of heart failure is not reversible (e.g., left ventricular damage from a myocardial

infarction), then the heart failure may become chronic. Other classification categories include left versus right-sided heart failure.

Right-sided heart failure refers to failure of the right ventricle to pump adequately, and is most commonly caused by left-sided heart failure. It can also be caused by pulmonary disease and primary pulmonary hypertension. Left-sided heart failure refers to failure of the left ventricle to fill or empty properly, which leads to increased pressures inside the ventricle and congestion in the pulmonary vascular system. Left-sided heart failure may be further classified into systolic and diastolic dysfunction.

Systolic dysfunction is usually estimated by ejection fraction, or the percentage of the left ventricular end-diastolic volume (LVEDV) that is ejected from the ventricle in one cycle. For example, if the LVEDV is 100 mL and the stroke volume is 70 mL, the ejection fraction is 70%. Normal ejection fraction is 50–70%. Systolic dysfunction is defined as an ejection fraction of less than 40% and is caused by a decrease in heart contractility. Diastolic dysfunction, which is more common among older adults, is less well defined and more difficult to measure (i.e., left ventricular function is preserved). Although heart contractility is normal or even increased (e.g., normal or even high ejection fractions), diastolic dysfunction is caused by impaired relaxation and/or filling (e.g., fast heart rate, stiff or poorly compliant ventricle, rhythm that is poorly organized).

Diagnosis and management of heart failure begins with a clinical history and physical examination, and confirmatory evidence of cardiac dysfunction on an echocardiogram. Once a diagnosis has been made, it is important to characterize the degree of impairment using the standardized New York Heart Association (NYHA) Functional Classification. The American College of Cardiology (ACC) and the American Heart Association Guidelines (AHA) outline four stages of heart failure that are useful for organizing the prevention, diagnosis, management, and prognosis for patients with heart failure; only stages C and D are applicable to the NYHA functional classification system.

Cross-References

- ▶ Dyspnea
- ▶ Heart
- ▶ Systolic Blood Pressure (SBP)

References and Readings

American College of Cardiology/American Heart Association. (2005). Guideline update for the diagnosis and management of chronic heart failure in the adult. *Journal of the American College of Cardiology, 46*, 1116–1143.

New York Heart Association. (1964). *Diseases of the heart and blood vessels: Nomenclature and criteria for diagnosis* (6th ed.). Boston: Little, Brown and Company.

Heart Patients

- ▶ Williams LifeSkills Program

Heart Rate

Annie T. Ginty
School of Sport and Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

Synonyms

Pulse rate

Definition

Heart rate is the number of beats of the heart per unit of time. Typically it is measured in beats per minute (bpm). Heart rate is based on the time interval between one R wave and the next. The R wave is the onset of ventricular depolarization. For further details, see Andreassi (2006).

Cross-References

▶ Psychophysiology: Theory and Methods

References and Readings

Andreassi, J. L. (2006). *Psychophysiology: Human behavior and physiological response*. Hillsdale, NJ: Lawrence Erlbaum Associates.

Heart Rate Variability

Julian F. Thayer
Department of Psychology, The Ohio State University, Columbus, OH, USA

Synonyms

Autonomic; Parasympathetic; Sympathetic

Definition

Like many organs in the body, the heart is dually innervated. Although a wide range of physiologic factors determine cardiac functions such as heart rate (HR), the autonomic nervous system (ANS) is the most prominent. Resting cardiac autonomic balance favors energy conservation by way of parasympathetic dominance over sympathetic influences. In addition, the HR time series is characterized by beat-to-beat variability over a wide range, which also implicates vagal dominance as the sympathetic influence on the heart is too slow to produce beat-to-beat changes. There is an increasing interest in the study of this heart rate variability (HRV) among researchers from diverse fields. Low HRV is associated with increased risk of all-cause mortality and has been proposed as a marker for disease. In the following, I will briefly describe the nature and assessment of HRV.

Heart Rate Variability

The basic data for the calculation of all the measures of HRV is the sequence of time intervals between heart beats. This interbeat interval time series is used to calculate the variability in the timing of the heart beat. Relative sympathetic increases cause the time between heart beats (the interbeat interval) to become shorter and relative parasympathetic increases cause the interbeat interval to become longer. The parasympathetic influences are pervasive over the frequency range of the heart rate power spectrum whereas the sympathetic influences "roll-off" at about 0.15 Hz. Therefore high-frequency HRV represents primarily parasympathetic influences with lower frequencies (below about 0.15 Hz) having a mixture of sympathetic and parasympathetic autonomic influences. The differential effects of the ANS on the sinoatrial node, and thus the timing of the heart beats, are due to the differential effects of the neurotransmitters for the sympathetic (noradrenaline) and parasympathetic (acetylcholine) nervous systems. The sympathetic effects are slow, on the timescale of seconds, whereas the parasympathetic effects are fast, on the timescale of milliseconds. Therefore the parasympathetic influences are the only ones capable of producing rapid changes in the beat-to-beat timing of the heart.

Measures of HRV

A variety of measures have been used to operationalize HRV. Long-term measures like the standard deviation of all interbeat intervals in 24 h, short-term measures like the standard deviation of 5 min intervals and beat-to-beat measures like the root mean square of successive RR differences (RMSSD) have all been used. Power spectral analysis of interbeat interval time series is frequently used to quantify HRV. The power spectrum of short-term time series contains two major components, a high-frequency

(0.15–0.40 Hz) and low-frequency (0.01–0.15 Hz) component reflecting cardiac vagal influences and a mixture of vagal and sympathetic influences, respectively. RMSSD and the high-frequency component of the power spectrum are closely related and reflect vagal cardiac influence. More recently, measures derived from nonlinear dynamics have been used to describe aspects of HRV. One such measure is approximate entropy (ApEn). It quantifies the complexity or irregularity of time series data.

Cross-References

▶ Autonomic Balance

References and Readings

Ahern, G. L., Sollers, J. J., Lane, R. D., Labiner, D. M., Herring, A. M., Weinand, M. E., et al. (2001). Heart rate and heart rate variability changes in the intracarotid sodium amobarbital (ISA) test. *Epilepsia, 42*, 912–921.

Benarroch, E. E. (1997). The central autonomic network. In P. A. Low (Ed.), *Clinical autonomic disorders* (2nd ed., pp. 17–23). Philadelphia: Lippincott-Raven.

Jose, A. D., & Collison, D. (1970). The normal range and determinants of the intrinsic heart rate in man. *Cardiovascular Research, 4*, 160–167.

Lane, R. D., McRae, K., Reiman, E. M., Chen, K., Ahern, G. L., & Thayer, J. F. (2009). Neural correlates of heart rate variability during emotion. *NeuroImage, 44*, 213–222.

Levy, M. N. (1997). Neural control of cardiac function. *Baillière's Clinical Neurology, 6*, 227–244.

Pagani, M., Lombardi, F., Guzzetti, S., Rimoldi, O., Furlan, R., Pizzinelli, P., et al. (1986). Power spectral analysis of heart rate and arterial pressure variabilities as a marker of sympathovagal interaction in man and conscious dog. *Circulation Research, 59*, 178–193.

Pincus, S. M. (2001). Assessing serial irregularity and its implications for health. *Annals of the New York Academy of Science, 954*, 245–267.

Roach, D., Wilson, W., Ricthie, D., & Sheldon, R. (2004). Dissection of long-range heart rate variability. *Journal of the American College of Cardiology, 43*, 2271–2277.

Saul, J. P. (1990). Beat-to beat variations of heart rate reflect modulation of cardiac autonomic outflow. *News in Physiological Science, 5*, 32–37.

Stein, P. K., & Kleiger, R. E. (1999). Insights from the study of heart rate variability. *Annual Review of Medicine, 50*, 249–261.

Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. (1996). Heart rate variability: Standards of measurement, physiological interpretation, and clinical use. *Circulation, 93*, 1043–1065.

Thayer, J. F., & Brosschot, J. F. (2005). Psychosomatics and psychopathology: Looking up and down from the brain. *Psychoneuroendocrinology, 30*, 1050–1058.

Thayer, J. F., Hansen, A. L., & Johnsen, B. H. (2010). The non-invasive assessment of autonomic influences on the heart using impedance cardiography and heart rate variability. In A. Steptoe (Ed.), *Handbook of behavioral medicine: Methods and applications*. New York: Springer.

Thayer, J. F., & Lane, R. D. (2000). A model of neurovisceral integration in emotion regulation and dysregulation. *Journal of Affective Disorders, 61*, 201–216.

Thayer, J. F., & Lane, R. D. (2007). The role of vagal function in the risk for cardiovascular disease and mortality. *Biological Psychology, 74*, 224–242.

Heart Rate Variability (HRV)

▶ Respiratory Sinus Arrhythmia

Heavy Episodic Drinking

▶ Binge Drinking

Height

▶ Body Mass Index

Helplessness

▶ External Locus of Control
▶ Passive Coping Strategies

Hematopoietic Stem Cell Transplantation

Yoshinobu Kanda
Division of Hematology, Saitama Medical Center, Jichi Medical University, Omiya-ku, Saitama, Japan

Description

In general, hematological malignancies show good response to antineoplastic agents. However, less than half of patients with hematological malignancies can be cured with chemotherapy alone. Since some antineoplastic agents show linear dose–response relationship, increasing the dose of chemotherapy may increase the antineoplastic effects, but it is precluded by the adverse effects of antineoplastic agents, so-called dose-limiting toxicities. The dose-limiting toxicity of many antineoplastic agents is myelosuppression. Therefore, hematopoietic stem cell transplantation has been investigated to allow high-dose chemotherapy over the maximum tolerated dose by supporting the hematopoietic system with the infusion of hematopoietic stem cells from a donor (allogeneic transplantation) or the patients themselves (autologous transplantation). In addition to the effect of the high-dose chemotherapy, an immunological antineoplastic effect of donor cells, so-called graft-versus-leukemia/lymphoma effect, can be harnessed after allogeneic transplantation. Currently, the application of hematopoietic stem cell transplantation is extended to solid tumors and nonmalignant hematological disorders such as aplastic anemia.

Previously, bone marrow was the sole source of hematopoietic stem cells. However, in the 1990s, the peripheral blood stem cells, which were mobilized by the use of granulocyte-colony stimulating factor alone or in combination with chemotherapy in autologous transplantation, became widely used as a source of hematopoietic stem cells, since the hematopoietic recovery is faster after peripheral blood stem cell transplantation than that after bone marrow transplantation. In addition, cord blood cells are currently the third source of hematopoietic stem cells. A major drawback of cord blood stem cell transplantation is the longer duration between transplantation and hematopoietic recovery. However, cord blood units are readily accessible, since they are already frozen in cord blood banks and there is no need for time-consuming door coordination.

The procedure of hematopoietic stem cell transplantation starts with a conditioning regimen using high-dose chemotherapy with or without total body irradiation. The major object of the conditioning regimen is to eradicate malignant cells from the recipient. However, in allogeneic transplantation, conditioning regimens are also required to suppress host immune system to prevent rejection of donor hematopoietic cells. The latter is the sole object of the conditioning regimen in allogeneic transplantation for aplastic anemia. After the conditioning regimen, hematopoietic stem cells are infused to the recipients intravenously. In general, hematopoietic recovery is observed within 2–4 weeks. However, supportive managements to prevent or treat transplant-related complications are required at least several months (or much longer in allogeneic transplantation) after transplantation.

The major complications after allogeneic hematopoietic stem cell transplantation include toxicities directory due to the conditioning regimen, infection, and graft-versus-host disease (GVHD). Increasing the dose of chemotherapy or total body irradiation over the maximum tolerated dose may induce toxicities that are not observed after standard-dose chemotherapy. For example, a fatal cardiac toxicity may develop after high-dose cyclophosphamide as a conditioning regimen. To extend the application of allogeneic transplantation to the elderly or clinically infirm patients who cannot tolerate these conditioning regimens, reduced-intensity or non-myeloablative stem cell transplantation has been investigated. Clinical studies of reduced-intensity or non-myeloablative transplantation have shown that the incidence of transplant-related morality was decreased, but the increase in relapse rate was the problem.

Hematopoietic stem cell transplantation recipients experience various infections. In the early phase after transplantation (day 0 to day 30), neutropenia is the primary immunologic defect. In addition, mucosal damage caused by the conditioning regimen adds the risk of infection. Flora of the skin, gastrointestinal tract, and mouth are the primary causes of infection during this phase. The second phase (day 30 to day 100), infectious complications due to *Cytomegalovirus*, *Aspergillus*, and *Pneumocystis jiroveci* are common due to the impaired cellular immunity. The development of acute or chronic GVHD is associated with severely impaired cellular immunity. In the later phase (after day 100), impaired cellular and humoral immunity is the major risk factor for infectious complications, especially in allogeneic transplant recipients with chronic GVHD. The causative organisms of infectious complications in this phase include Cytomegalovirus, Varicella-zoster virus, Epstein-Barr virus, *Aspergillus*, and *Pneumocystis jiroveci*.

GVHD is an immunological reaction of the donor immunological cells targeting host antigens. Clinical manifestations of acute GVHD include erythroderma, diarrhea, and jaundice. On the other hand, chronic GVHD resembles autoimmune disorders such as scleroderma, Sjögren's syndrome, primary biliary cirrhosis, bronchiolitis obliterans, and immune cytopenias. GVHD can be observed even after human leukocyte antigen (HLA) matched transplantation, but both its incidence and severity are increased by the presence of HLA-mismatch between the donor and the recipient. A combination of calcineurin inhibitors (cyclosporine or tacrolimus) and methotrexate is the mainstay of the pharmacological prevention of GVHD, whereas steroid is the first choice of the treatment of acute GVHD. The use of these immunosuppressive agents increases the risk of infectious complications.

In general, the incidence of transplant-related mortality is less than 5% after autologous transplantation, but it exceeds 10–20% after allogeneic transplantation, even from an HLA-matched donor. In addition, transplant-related complications, especially the development of chronic GVHD, affect the quality of life of survivors after allogeneic transplantation. Therefore, the indication of hematopoietic stem cell transplantation should be considered by balancing the risk and benefit obtained after transplantation. Many clinical trials have been performed to evaluate the indication of hematopoietic stem cell transplantation in a variety of hematological disorders. However, information is not yet sufficient to identify patients who will clearly benefit from undergoing hematopoietic stem cell transplantation.

Cross-References

▶ Stem Cells

Hemodynamic

Jonathan Newman
Columbia University, New York, NY, USA

Definition

Hemodynamics is a general term referring to the movement or flow of blood. More specifically, this term refers to the measurement of and general principles governing the flow of blood in the human body. Movement or flow of blood throughout the body is dependent upon many factors. Two of the most important of these factors are the pressure differences and resistances between different elements of the circulatory system, such as the cardiac chambers, large arteries, arterioles, veins, and venules. Along with the heart rate (HR), pressure and resistance are major determinants of the amount of circulatory blood flow. Measurement of certain hemodynamic parameters, such blood pressure (BP) and heart rate (HR), can be determined noninvasively. Resistance is a calculated parameter determined by flow and pressure. For example, systemic vascular resistance is equal to the cardiac output divided by mean arterial

pressure. As will be described, cardiac output is determined by heart rate and stroke volume. These measures are important in understanding the pathophysiology of cardiovascular disease diagnosis and treatment. However, more detailed measures of cardiac structure, function, and intracardiac pressures are frequently necessary, and advanced measurement modalities are required.

Description

In the field of cardiology and cardiovascular medicine, hemodynamics are most commonly measured by two different modalities in the clinical setting: two-dimensional/Doppler echocardiography and cardiac catheterization. Each modality has limitations, and these two techniques are frequently used in concert. Importantly, absolute pressures can only be directly measured by cardiac catheterization. In contrast, absolute pressure cannot be measured by echocardiography; only pressure differences can be measured.

Echocardiography measures: Cardiac ultrasound, or echocardiography, uses ultrasonic beams through tissue. M-mode and two-dimensional (2D) echocardiography are used for structural imaging, while Doppler echocardiography uses ultrasound to record the movement of blood, enabling the assessment of hemodynamics and cardiac physiology. Each tissue has different properties that influence the amount of ultrasound beam that is reflected back to the ultrasound machine, generating a very small electrical current that can then be integrated into a composite image of the structure. Doppler ultrasound measures the difference between the scatter of ultrasound by different tissues and the frequency of the transmitted ultrasound beam. Doppler echocardiography is especially useful for the hemodynamic measurement of blood flow across stenotic valves of the heart and for measuring the pressure differences between different chambers. Assessment of blood flow is approximated by measurement of cardiac stroke volume. Stroke volume, in turn, is measured by taking the product of the velocity-time-integral (VTI, measured by Doppler echocardiography) and the cross-sectional area (CSA, measured with 2D echocardiography) between certain chambers. Cardiac output is then determined by the product of stroke volume and heart rate. The change in pressure between two cardiac chambers, a useful principle in cardiac hemodynamics, is frequently assessed using a modified form of the Bernoulli equation, which is itself a derivation of basic principles of conservation of energy in closed systems.

Catheterization measures: During catheterization, a special type of catheter is introduced into either the internal jugular vein in the neck or a femoral vein in the leg. The catheter is advanced into different chambers of the right (venous) side of the heart, the side responsible for collecting and pumping deoxygenated blood from the systemic circulation into the lungs. Typically, these catheters have an inflatable balloon built into the catheter. Inflation of the balloon in a pulmonary artery allows for the measurement of the pulmonary capillary "wedge" pressure (PCWP), which is, in most circumstances, a clinically useful approximation of intravascular fluid balance. To measure pressures in the left (arterial) side of the heart, a similar type of catheter and wire system is introduced into the right femoral artery and advanced retrograde up to and across the aortic valve into the left ventricle. Routine hemodynamic pressure measurements obtained from the right side include right atrial pressure, right ventricular pressure, pulmonary artery pressure, and PCWP. Left-sided pressure measurements include measurement of aortic pressure and left ventricular pressure. Cardiac output can be measured directly with cardiac catheterization using the thermodilution or Fick method. In the thermodilution method, cold saline is injected into the proximal port of the pulmonary artery catheter and mixes with body temperature blood in the right ventricle. As blood flows past the distal port, a small sensor records the change in temperature over time. The area under this curve is proportional to the flow in the pulmonary artery which, assuming there is no intracardiac shunt, is proportional to cardiac output. Stroke volume is determined by dividing this measured cardiac output by the heart rate.

The Fick method of cardiac output uses measures of oxygen consumption measured by either exhaled breath analysis or determined by an age, sex, height, and weight specific nomogram and is dependent on hemoglobin and levels of arterial oxygen saturation.

References and Readings

Kern, M. J., Feldman, T., & Bitar, S. (2003). Hemodynamic data. In M. J. Kern (Ed.), *The cardiac catheterization handbook* (4th ed., pp. 126–216). St. Louis: Mosby-Year Book.

Otto, C. M., & Pearlman, A. S. (Eds.). (1995). *Textbook of clinical echocardiography*. Philadelphia: WB Saunders.

Hemodynamic Response/Reactivity

► Blood Pressure Reactivity or Responses

Hemodynamic Stress Responses

► Blood Pressure Reactivity or Responses

Hemoglobin A1c

► HbA1c
► Glycosylated Hemoglobin

Hemoglobin, Glycosylated

► Glycosylated Hemoglobin

Hemostasis

► Coagulation of Blood

Hepatitis Types A, B, C

Carrie Brintz
Department of Psychology, University of Miami, Coral Gables, FL, USA

Definition

Hepatitis involves inflammation of the liver and is most commonly caused by a number of viruses. Hepatitis can be an acute or a chronic condition. Hepatitis A is an acute viral infection of the liver due to the hepatitis A virus (HAV), and is the least serious of the hepatitis viruses. Hepatitis B and C are viral infections of the liver due to the hepatitis B virus (HBV) and the hepatitis C virus (HCV). Both hepatitis B and hepatitis C can become chronic infections and result in chronic liver disease.

Description

HAV is the main cause of acute viral hepatitis and is endemic worldwide. The virus is spread by the fecal-oral route, and very rarely by blood transfusion, and is often contracted from a contaminated water or food source. Major epidemics of hepatitis A are uncommon in the United States of America (USA), and most common in regions of the world with poor sanitary hygiene and overcrowding. The severity of the illness tends to increase with age. More than 80% of childhood cases of hepatitis A are asymptomatic, which encourages the spread of the virus. Adults with hepatitis A are more likely to have clinical symptoms such as jaundice (a yellowing of the skin and whites of the eyes). Symptoms tend to appear 2–6 weeks after exposure to HAV, and mild symptoms may last several months, particularly in adults. The virus does not remain in the body when the infection is gone. Vaccination for HAV is available, but there are no specific treatments for unvaccinated individuals who are exposed to the virus or develop the infection.

HBV is also found worldwide. It is spread parenterally and by sexual contact. Hepatitis B infection is most prevalent in sub-Saharan Africa, China, and Southeast Asia. Less than 1% of people in modern Europe, the USA, and Canada carry the virus, and in these areas, horizontal transmission occurs predominantly in younger individuals (ages 15–39) by sexual contact or intravenous drug use. Vertical transmission of the virus from mother to child and horizontal transmission from household members to unimmunized children occurs more frequently in high prevalence countries and helps maintain the virus in these areas. Most cases of hepatitis B are acute, but a small percentage of individuals will not clear the virus from their bodies and develop chronic infection. Hepatitis B infections are typically asymptomatic, especially in infancy through young adulthood, but early symptoms of acute hepatitis may occur such as jaundice, appetite loss, low-grade fever, and nausea and vomiting. Symptoms may not appear for up to 6 months after infection with HBV. Chronic infection with HBV can result in cirrhosis and hepatocellular carcinoma (HCC), also known as primary liver cancer. Up to 40% of individuals with chronic infection die from liver-related causes. A vaccination for HBV is available, and unvaccinated individuals exposed to the virus may be treated with Hepatitis B Immune Globulin. Those who develop chronic liver disease may need liver transplantation.

Like HAV and HBV, HCV is a worldwide endemic. It is primarily spread parenterally, while vertical and sexual transmissions of HCV are much less common than in HBV. Intravenous drug use is the most common cause of the virus. More than 80% of acute HCV cases lead to chronic infection, with long-term infection often leading to cirrhosis and HCC. As with hepatitis A and B, hepatitis C infection is usually asymptomatic, with the possible appearance of symptoms such as jaundice, dark urine, abdominal pain, and others. There is currently no vaccination for HCV. However, there are treatments such as antiviral medications that have a high likelihood of removing the virus from the blood or preventing the development of cirrhosis or liver cancer. Chronic hepatitis C with comorbid alcohol liver disease is the most common reason for liver transplantation in the USA.

Relevant to the field of Behavioral Medicine, the behavioral nature of numerous risk factors for exposure to viral hepatitis has led to research examining relevant psychosocial and behavioral predictors of viral hepatitis and its symptoms, as well as behavioral interventions for the prevention of viral hepatitis. Behavioral interventions researched include those targeting risk factors for viral hepatitis such as intravenous drug use and unsafe sex practices that could result in exposure to viral hepatitis.

Cross-References

- ▶ Infectious Diseases
- ▶ Inflammation
- ▶ Needle Exchange Programs
- ▶ Sexual Risk Behavior

References and Readings

A.D.A.M. Medical Encyclopedia [Internet]. Hepatitis A. Atlanta, GA: A.D.A.M., Inc.; c1997–2011; [last reviewed 2010 Nov 23; cited 2011 Dec 6]. Retrieved from http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0001323/

A.D.A.M. Medical Encyclopedia [Internet]. Hepatitis B. Atlanta, GA: A.D.A.M., Inc.; c1997–2011; [last reviewed 2010 Nov 23; cited 2011 Dec 6]. Retrieved from http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0001324/

A.D.A.M. Medical Encyclopedia [Internet]. Hepatitis C. Atlanta, GA: A.D.A.M., Inc.; c1997–2011; [last reviewed 2010 Nov 23; cited 2011 Dec 6]. Retrieved from http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0001329/

Fagan, E. A., & Harrison, T. J. (2000). *Viral hepatitis: A handbook for clinicians and scientists*. New York: Springer.

Ryder, S., & Beckingham, I. (2001). Acute hepatitis. *British Medical Journal, 322*(7279), 151–153.

Hepatocyte Stimulating Factor

- ▶ Interleukins, -1 (IL-1), -6 (IL-6), -18 (IL-18)

Herbal Medicines

Timothy Whittaker
The International Register of Herbalists & Homeopaths, Cinderford, Glos., UK

Definition

Herbal medicine is the use of plants to treat human illnesses and debility. Parts used can be roots, bark, flowers, seed, leaves, or sometimes total aerial parts.

Description

Until early twentieth century, materials required for herbal medicine were collected in the countryside, dried by hanging in a warm room, and cut into small pieces to facilitate infusion in hot water, the resulting liquor being consumed as a dose. Mid-twentieth century saw the introduction of fluid extracts where 1 ml of finished liquid preparation represented 1 g of the starting material (dried herb). The industrial process to make these incurred considerable use of heat, very deleterious to fragile plant chemistry via hydrolysis, and/or oxidation. The last quarter of the century saw a substantial swing to the use of tinctures, aqueous-alcoholic preparations, made by steeping the dried herb in the liquor for a few weeks, cold, and pressing the residue to separate the liquid. Tinctures are typically 1:5, i.e., 1 g herb represented by 5 ml tincture. These products are very versatile for practitioner use, as the dose can be varied precisely from one prescription to the next, as the patient requires.

The herbal practitioner diagnoses and then writes his/her prescription. This will comprise a mixture of tinctures (and sometimes water) to make a 5-ml or 10-ml spoonful dose, using from 3 to about 25 per prescription. Experience teaches that there is a high degree of synergy accompanying the use of many together, with the advantage of an absolute minimum of adverse reactions as far less is used of each.

Some modern teachers have promoted the use of single plant extracts, standardized by assay of a single chemical constituent, dried to a powder, and formulated into tablets. This approach fails to appreciate the wide diversity of constituents in a plant (often several hundred), *all* of which work together therapeutically.

Significant behavior modifiers include hops (*Humulus lupulus*), scullcap (*Scutellaria lateriflora*), valerian (*Valeriana officinalis*), kava (*Piper methysticum*), and vervain (*Verbena officinalis*), as sedatives and hypnotics; St. John's wort (*Hypericum perforatum*) and Rhodiola rosea as antidepressants; and the ginsengs, Chinese or Korean (*Panax ginseng*) and Siberian (*Eleutherococcus senticosus*). The latter group acts as stamina providers, called adaptogens, because of their ability to improve the body's response to stress. Most of the named plants have added support from orthodox clinical trials. There are other less-studied herbs also in this area of therapeutics.

References and Readings

Mills, S. Y. (1993). *The essential book of herbal medicine*. London: Penguin Arkana.

Heritability

Jennifer Wessel
Public Health, School of Medicine, Indiana University, Indianapolis, IN, USA

Definition

Heritability, or h^2, is the fraction of the total variation in a phenotype that can be explained by genetic factors. Heritability is not about cause.

Description

Interpretation

Heritability has values between 0 and 1. A heritability of 0 would be a trait where none of

the phenotypic variance is due to genetic factors, whereas a heritability of 1 would be a trait where all of the variance can be explained by genetic factors.

Heritability is based on measuring the correlations of phenotypes within families. Families share more than their genes; they also share environments. Therefore, the correlations may not simply reflect their shared genetic profiles but also environmental (behavioral) exposures. Heritability can also be affected by other factors such as the population (ethnicity, age, gender, location, etc.) from which it was measured (measurement error) and their environmental exposures.

Calculations

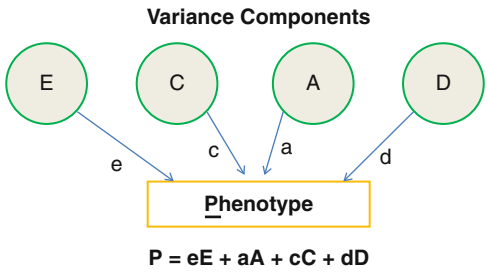
Twin studies are commonly used to measure heritability, as well as family studies. In the classic twin model, heritability is calculated by:

$$h^2 = 2(r_{mz} - r_{dz}),$$

which is the difference between intraclass correlations of a quantitative trait between monozygotic twins and dizygotic twins.

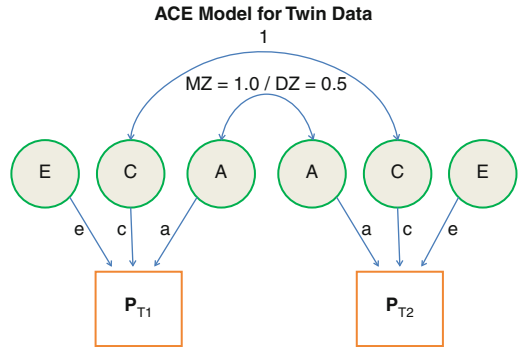
Intraclass correlations are subject to low power and large standard errors, necessitating the development of newer methods which take advantage of variances and covariances.

Path analysis or structural equation modeling estimates the genetic and environmental parameters that give the best fit of the variances and covariances observed in twins or families.



where E is the unique environment of each twin, C is the shared environment within the twin pairs, A is the additive genetic effects, and D is the dominant genetic effects.

Path analysis, or the ACE model, allows a diagrammatic representation between the linear models of the relationships between variables.



where MZ is monozygotic twins that share 100% of their genomes, and DZ is dizygotic twins that share 50% of their genomes, on average. It is assumed that both of the twins in a pair share 100% of their shared environment.

The ACE model can parse the variance of a trait into additive genetic, shared environmental, and non-shared environmental influences on the basis of the trait covariance observed among both monozygotic and dizygotic twin pairs. This model can accommodate hypothesis testing, both continuous and categorical variables, multiple variables, and more complex pedigrees or questions.

Cross-References

- ▶ Dizygotic Twins
- ▶ Monozygotic Twins

References and Readings

Elston, R. C., Olson, J. M., & Palmer, L. (2002). *Biostatistical genetics and genetic epidemiology* (1st ed.). Chichester: Wiley.

Neale, M. C., & Cardon, L. R. (1992). *Methodology for genetic studies of twins and families*. Dordrecht: Kluwer.

Nussbaum, R. L., Mc Innes, R. R., & Willard, H. F. (2001). *Genetics in medicine* (6th ed.). Philadelphia: W.B. Saunders.

Spector, T. D., Snieder, H., & MacGregor, A. J. (2000). *Advances in twin and sib-pair analysis* (1st ed.). London: Greenwich Medical Media.

Herpes Simplex Virus (HSV) Infection

▶ Genital Herpes

Heterogeneity

▶ Diversity

Heterozygous

Laura Rodriguez-Murillo[1] and Rany M. Salem[2]
[1]Department of Psychiatry, Columbia University Medical Center, New York, NY, USA
[2]Broad Institute, Cambridge, MA, USA

Definition

Humans have two alleles on homologous chromosomes for each gene or trait, one from their mother and one from their father. If the allele inherited from the mother is different from the allele inherited from the father, then the individual is heterozygous for that trait or gene. Alleles are variants of a gene and can also lead to a differential manifestation of the trait or disease they codify. Recessive alleles are not observed in the phenotype of heterozygous individuals.

The degree of heterozygosity (proportion of genes that are heterozygous by individual and by population) is used as a measure of genetic diversity in populations. The higher the mean heterozygosity is across the genome and across individuals in one population, the higher the genetic diversity.

Cross-References

▶ Allele
▶ Dominant Inheritance
▶ Gene
▶ Genotype
▶ Homozygous
▶ Recessive Inheritance

References and Readings

Lewis, R. (2005). *Human genetics. Concepts and applications* (7th ed.). Boston: McGraw-Hill Science/Engineering/Math.
Strachan, T., & Read, A. P. (2003). *Human molecular genetics* (3rd ed.). London/New York: Garland Science/Taylor & Francis Group.

Hidden Variable

▶ Latent Variable

Hierarchical Linear Modeling (HLM)

Yutaka Matsuyama
Department of Biostatistics, School of Public Health, The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Synonyms

Covariance components model; Linear mixed-effects model; Multi-level analysis; Random-coefficient model

Definition

Hierarchical linear modeling (HLM) is a particular regression model that is designed to take into account the hierarchical or nested structure of the data. HLM is also known as multi-level modeling, linear mixed-effects model, or covariance components model (Leyland & Goldstein, 2001).

Description

HLM has historically been used in educational research where hierarchies occur naturally: students nested within classrooms, classrooms nested within schools, and schools nested within districts (Sullivan, Dukes, & Losina, 1999). Recent advances in statistical computing capabilities have made this model more available to researchers across a variety of disciplines. For example, in organizational psychology research, data from individuals must often be nested within teams or other functional units. For repeated measures or longitudinal data, time can be considered as another level which occurs within subjects (Fitzmaurice, Laird, & Ware, 2004). For assessing differences in mortality rates across hospitals relative to a specific condition or procedure, data are collected on random samples of patients nested within each hospital. In this application, it might be appropriate to adjust for covariates at both the patient level (such as patient age, patient gender, and the severity of the disease) and at the hospital level (such as hospital size and hospital teaching status) (Austin, Yu, & Alter, 2003). These hierarchical data structures are often seen in many medical research applications.

HLM is a more advanced form of traditional linear regression models which were developed making certain assumptions about the nature of the dependency structure among the observed responses. For example, in a simple linear regression model, $y_i = b_0 + b_1 x_i + e_i$, where y_i is the response for individual i; the standard assumption is that the y_i given the covariate x_i is independently identically distributed, that is, the error term e_i is independent among individuals. In many real-life situations, however, one has data structures, whether observed or by design, for which this assumption does not hold.

Suppose, for example, that the response variable is the birth weight of a baby and the predictor is maternal age and data are collected from a large number of maternity units located in different physical and social environments. One would expect that the maternity units would have different mean birth weights so that knowledge of the maternity unit already conveys some information about the baby. A more suitable model for these data is

$$y_{ij} = b_0 + b_1 x_{ij} + u_j + e_{ij},$$

where another subscript j was added to identify the maternity unit and a unit-specific effect u_j was included to account for mean differences among units. If one assumes that the maternity units are randomly sampled from a population of units, then the unit-specific effect is a random variable and the above model becomes a simple example of a two-level model. Its complete specification, assuming normality for random variables, can be written as follows:

$$y_{ij} = b_0 + b_1 x_{ij} + u_j + e_{ij},$$

$$u_j \sim \mathrm{N}\left(0, {s_u}^2\right),\ e_{ij} \sim \mathrm{N}\left(0, {s_e}^2\right),$$

$$\mathrm{cov}\left(u_j, e_{ij}\right) = 0,$$

where ${s_u}^2$ represents the degree of heterogeneity among maternity units (between-unit variance) and ${s_e}^2$ is the pure random error variance (within-unit variance). In this model, the correlation among birth weights in the same unit can be written as ${s_u}^2/({s_u}^2 + {s_e}^2)$, which is known as an intra-cluster correlation. This lack of independence, arising from two sources of variation at different levels of the data hierarchy (births and maternity units), contradicts the traditional linear model assumption, and an HLM is a suitable regression model for this situation.

The above most simple HLM can be elaborated in a number of directions, including the addition of further covariates (both births and units levels) or levels of nesting. An important direction is where the coefficient of covariates is also allowed to have a random distribution, for example, the age relationship can vary across units. This is particularly important for assessing the covariate-by-unit interaction. The regression coefficients b are usually referred to as fixed effects parameter, and the unit-specific effects u_j are referred to as random effects parameter, and the above model is often referred to as a mixed-effects model or random coefficient model.

The parameters of fixed effects and variance components (s_u^2 and s_e^2) in HLM are obtained via a restricted maximum likelihood (REML) estimation method (Cnaan, Laird, & Slasor, 1997; Leyland & Goldstein, 2001). The random effects are estimated using shrinkage estimators, which is referred to as an empirical Bayes estimator or a best linear unbiased predictor (Fitzmaurice et al., 2004). This estimator is essentially an optimally weighted linear combination of the estimated overall mean and unit-level mean. The degree of shrinkage toward the overall means depends on the magnitude of the relative magnitude of s_u^2 and s_e^2. When s_e^2 is relatively large and the within-unit variability is greater than the between-unit variability, more weight is assigned to overall mean. On the other hand, when the between-unit variability is large relative to the within-unit variability, more weight is given to the unit-specific observed response.

There are two major specialized software packages for conducting HLM analysis, one is MLwiN and the other is HLM. MLwiN (version 2.21) can be found via the links provided on the Centre for Multilevel Modelling (CMM), which is a research center based at the University of Bristol, with Internet address http://www.cmm.bristol.ac.uk/. HLM (version 6.08) is available from Scientific Software International, Inc., where HLM has its homepage with Internet address http://www.ssicentral.com/hlm/index.html. HLM analysis can also be conducted using general purpose packages such as SAS or SPSS. SAS (version 9.2) has full-fledged possibilities for HLM analysis, using PROC MIXED or PROC GLIMMIX.

Cross-References

► Multilevel Modeling

References and Readings

Austin, P. C., Yu, J. V., & Alter, D. A. (2003). Comparing hierarchical modeling with traditional logistic regression analysis among patients hospitalized with acute myocardial infarction: Should we be analyzing cardiovascular outcomes data differently? *American Heart Journal, 145*, 27–35. doi:10.1067/mhj.2003.23.

Cnaan, A., Laird, N. M., & Slasor, P. (1997). Tutorial in biostatistics: Using the general linear mixed model to analyse unbalanced repeated measures and longitudinal data. *Statistics in Medicine, 16*, 2349–2380. doi:10.1002/(SICI)1097-0258(19971030)16:20<2349::AID-SIM667<3.0.CO;2-E.

Fitzmaurice, G. M., Laird, N. M., & Ware, J. H. (2004). *Applied longitudinal analysis*. New Jersey: John Wiley & Sons.

Leyland, A. H., & Goldstein, H. (Eds.). (2001). *Multilevel modelling of health statistics*. Chichester, England: John Wiley & Sons.

Sullivan, L. M., Dukes, K. A., & Losina, E. (1999). Tutorial in biostatistics: An introduction to hierarchical linear modeling. *Statistics in Medicine, 18*, 855–888. doi:10.1002/(SICI)1097-0258(19990415)18:7<855::AID-SIM117<3.0.CO;2-7.

Hierarchy of Evidence

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Evidence hierarchy

Definition

The concept of the "hierarchy of evidence" refers to a tabular representation (sometimes presented as a pyramid) of the relative strengths of various investigational methodologies in providing the evidence that is used in evidence-based medicine and evidence-based behavioral medicine.

Byar (cited by Piantadosi, 2005) listed different types of medical studies in terms of the (increasing) strength of the evidence provided by them: case report, case series, database analysis, observational study, controlled clinical trial, and replicated clinical trials (independent verification of treatment efficacy estimates), referring to this progression as a "hierarchy of strength of evidence."

While the concept is simple, there can be disagreement on the order of some methodologies,

particularly toward the top of the pyramid. Randomized controlled clinical trials have typically been placed at the top, and many researchers still agree with this positioning. Others regard systematic reviews and meta-analyses as providing stronger evidence since they combine information garnered from more than one study (although many authors note the considerable care that is necessary when publishing a systematic review or meta-analysis). One such order, therefore, might be:

- Systematic reviews and meta-analyses
- Randomized controlled trials
- Cohort studies
- Case-control studies
- Cross-sectional surveys
- Case reports
- Expert opinions

More detailed accounts of several of these methodologies (listed in the "Cross-References" section) can be found in the encyclopedia.

Cross-References

▶ Case-Control Studies
▶ Meta-Analysis
▶ Randomized Clinical Trial

References and Readings

Piantadosi, S. (2005). *Clinical trials: A methodologic perspective* (2nd ed.). Hoboken, NJ: Wiley.

Webb, P., Bain, C., & Pirozzo, S. (2005). *Essential epidemiology: An introduction for students and health professionals*. New York: Cambridge University Press.

High Blood Pressure

▶ Blood Pressure, Elevated
▶ Hypertension

High Blood Pressure Medications

▶ Antihypertensive Medications

High Cholesterol

▶ Dyslipidemia

High-Risk Drinking

▶ Binge Drinking

HIPAA

▶ Confidentiality

Hispanic Community Health Study/Study of Latinos

Neil Schneiderman
Department of Psychology, Behavioral Medicine Research Center, University of Miami, Coral Gables, FL, USA

Synonyms

Epidemiological studies; Ethnicity

Description

Behavioral, psychosocial, and sociocultural factors pose adverse cardiovascular disease (CVD) risks similar in magnitude to those caused by traditional risk factors such as smoking, high cholesterol, or hypertension. Little research has examined the risk and protective factors that contribute to CVD outcome of Hispanics/Latinos in the United States or that may lead to differential effects across subpopulations of Hispanics/Latinos. Consequently, the Hispanic Community Health Study/Study of Latinos (SOL), sponsored by the National Institutes of Health, was initiated to provide the largest, comprehensive, multicenter, community-based, longitudinal cohort study

of Hispanic/Latino health ever conducted in the continental United States. Objectives of the study include: (a) characterizing the health status and disease burden of the largest minority population in the United States; (b) describing the positive and negative consequences of their immigration and acculturation to the mainstream United States in relation to lifestyle, environmental factors, and access to health care; and (c) identifying likely causal factors of disease in this diverse population.

By June 2011, 16,000 men and women, 18–74 years of age, who self-identified as being Hispanic or Latino completed a 6.5 h baseline clinical exam. The participants were recruited from a stratified random sample of households in defined communities in the Bronx, Chicago, Miami, and San Diego. These communities were chosen so that the overall sample would include adequate representation from Central and South American, Cuban, Mexican, Puerto Rican, and Dominican ancestral backgrounds.

The study assessed risk factors for and prevalence of heart, lung, blood, and sleep disorders, kidney and liver dysfunction, diabetes, cognitive impairment, dental problems, and hearing disorders. Among the physical exam procedures employed were electrocardiogram, blood pressure, comparison of ankle and arm blood pressures to detect evidence of peripheral artery disease, evaluation of pulmonary function, physical activity assessed by activity monitors worn for a week by participants, and disordered breathing overnight to evaluate sleep interruption due to sleep apnea. Questionnaires assessed health histories of participants and their families, information about acculturation, social variables, education, occupation, smoking, nutrition, alcohol consumption, sleep, physical activity, and prescription and nonprescription drug use.

During annual follow-up phone calls, deaths, hospitalizations, and emergency department visits that occurred since the baseline examination are identified and followed up by examining hospital charts and death certificates. These records include documentation of acute myocardial infarction, heart failure, resuscitated cardiac arrest, cardiac revascularization, stroke, transient ischemia attacks, and asthma.

In general, SOL is designed to inform health care providers, the public health community, and the Hispanic/Latino population about the prevalence of impaired health in that population, the likely causes of such impairments as well as the measures needed to improve Hispanic/Latino health in the United States. In addition to the SOL parent study, the NIH has also sponsored ancillary studies that allow expansion of the major objectives of SOL. Thus, for example, an ancillary sociocultural study based on 5,280 of the original SOL participants is examining the influence of such factors as socioeconomic status (e.g., effects of cumulative deprivation during the lifespan), sociocultural factors (e.g., ethnic identity, fatalism), psychosocial risk (e.g., stress, negative emotions), and protective factors (e.g., social resources, family cohesion). In summary, SOL is providing the most comprehensive study of Hispanic/Latino health that has ever been conducted in the continental United States.

Cross-References

- Cardiovascular Disease
- Cardiovascular Disease Prevention
- Cultural Factors
- Epidemiology
- Ethnicity
- Health Disparities
- Hispanic/Latino Health

References and Readings

LaVange, L. M., Kalsbeek, W., Sorlie, P.D., Avilés-Santa, L. M., Kaplan, R. C., Barnhart, J., et al. (2010). Sample design and cohort selection in the Hispanic community health study/study of Latinos. *Annals of Epidemiology, 20*, 642–649.

Sorlie, P. D., Avilés-Santa, L., Wassertheil-Smoller, S., Kaplan, R. C., Daviglus, M. L., Giachello, A. L., et al. (2010). Design and implementation of the Hispanic community health study/study of Latinos. *Annals of Epidemiology, 20*, 629–641.

Hispanic Health

▶ Hispanic/Latino Health

Hispanic/Latino Health

John Ruiz, Mariana Garza and Lauren Smith
Department of Psychology, University of North Texas, Denton, TX, USA

Synonyms

Hispanic health

Definition

The terms *Hispanic* and *Latino/a* are used to collectively refer to people from Mexico, Central and South America, Spain, and Spanish-speaking or influenced countries. According to the 2010 Census, Latinos comprised 16.3% of the total US population, making them the largest racial/ethnic minority group in the USA (Pew Hispanic Center, 2011). Like other minorities, Latinos have a significant health risk factor profile marked by educational, economic, and disease challenges. Despite these disparities, Latino's appear to live longer than non-Hispanic Whites, an epidemiological phenomenon commonly referred to as the *Hispanic or Latino Mortality Paradox*.

Description

Background

Defining Latinos in the USA

Latinos in the USA are a heterogeneous group representing more than 23 nationalities and speaking at least 12 languages and dialects (Pew Hispanic Center, 2010). Approximately two-thirds (66%) of US Latinos are of Mexican descent followed by Puerto Ricans (8.9%), Cubans (3.5%), Salvadorans (3.3%), and Dominicans (2.8%). Latinos are largely defined and differentiated from other groups by cultural factors. In particular, Latino culture is *collectivistic*, meaning they place an emphasis on the needs of the group over those of the individual. Several specific values including the valuing family (*familismo)* valuing and warm interpersonal relationships (*personalismo)* and valuing interpersonal harmony (*simpatico*) facilitate strong social cohesion (Zea, Quezada, & Belgrave, 1994).

Demographics

Latinos account for approximately 16.3% of the total US population, accounting for 56% of the nation's population growth over the last decade (Pew Hispanic Center, 2011). By the year 2050, the Latino population is projected to triple in size to approximately 29% of the US population. Of the 50.5 million Latinos currently residing in the USA, an estimated 38% are foreign-born (Pew Hispanic Center, 2010). Importantly, foreign-born should not be equated with illegal immigration as unauthorized immigrants account for only 4% of the US population and, thus, a small percentage of the Latino community. Rather, native births are by far the largest source of growth in the US Latino population. Nearly one in five children in the United States is of Latino descent with approximately 89% of those children born in the USA, making them citizens by birth (Fry & Passel, 2009). It is estimated that by the year 2025, 30% of all children born in the USA will be of Latino descent.

The demographic profile of US Latinos differs significantly from other racial/ethnic groups. For example, Latinos are by far the youngest group with a median age of 27 years compared to 41 years for non-Hispanic Whites, 32 years for non-Hispanic Blacks, 36 years for non-Hispanic Asians, and a median age of 36 years for the total US population (Pew Hispanic Center, 2010). Latinos are less likely to have ever been married compared to non-Hispanic Whites (66.3% vs. 76.7%) but more likely to live in a "household" or family unit (81% vs. 69%). Latino households tend to be larger than non-Hispanic White households, and US-born Latinos are more likely to

live with family members than non-Hispanics of any race (Pew Hispanic Center, 2010).

Socioeconomic Profile

Socioeconomic status (SES) is among the most robust psychosocial modifiers of physical health and mortality. Similar to other racial/ethnic minorities, Latinos experience significant SES disparities relative to non-Hispanic Whites and the US population in general. For example, nearly one in five (19.5%) Latinos is classified as living in poverty, a rate comparable to the percentage of non-Hispanic Blacks (21.9%) and significantly higher than non-Hispanic Whites (8.2%; Pew Hispanic Center, 2010). Latinos have the lowest median income for full-time, year-round work relative to other racial/ethnic groups, which may be due in part to their overrepresentation in manual labor jobs and underrepresentation in management and technology (U.S. Bureau of Labor Statistics, 2009).

Education is an important determinant of future earning potential and is an integral part of the conceptualization of SES. Latinos are at a significant educational disadvantage relative to other racial/ethnic groups including other minorities. For example, nearly 40% of Latinos have less than a high school diploma, a rate that is twice that of any other racial/ethnic group and markedly higher than the national average of 15.1%. In addition, the college graduation rate for Latinos is just 12.9% compared to 17.7% of non-Hispanic Blacks, 30.7% of non-Hispanic Whites, and the national average of 27.7%. Together with the economic disparities, Latinos are at or near the bottom of most of the major SES indicators – a profile associated with significant health risk.

Latino Health Status

Perceived Health

Latinos' self-reported health ratings are typically lower than those from non-Hispanic Whites (Perez-Stable, Napoles-Springer, & Miramontes, 1997). For example, in a large survey of California residents, Latinos were less likely than non-Hispanic Whites to rate their health as "excellent" or "very good" (50.2% vs. 30.2%) and more likely to rate their health as "fair" (9.9% vs. 27.5%). This finding is consistent with the challenges of lower SES, although some data suggests that SES does not explain the entire effect.

Diabetes Diabetes is amongst the most common chronic diseases in the USA with an incidence rate of approximately 8.3% and rising (Center for Disease Control [CDC], 2011). Racial and ethnic minorities suffer a disproportionate burden of the diabetes epidemic (CDC, 2011). In general, Latinos are nearly 1.7 times as likely to have diabetes as compared to non-Hispanic Whites (11.8% vs. 7.1%) and are at 50% greater risk of dying from diabetes-related complications. There is considerable heterogeneity in diabetes risk among Latino subgroups with persons of Puerto Rican and Mexican descent twice as likely to have diabetes compared to non-Hispanic Whites (13.8% and 13.3%, respectively) whereas individuals of Cuban descent experience prevalence rates more similar to non-Hispanic Whites (7.6%). End-stage renal disease (ESRD) is a major complication arising from diabetes. Latinos are nearly 1.7 times more likely to initiate ESRD treatment compared to non-Hispanic Whites.

Cardiovascular Disease Cardiovascular disease (CVD) is the leading cause of death in the United States, accounting for approximately 34.3% of all deaths annually (American Heart Association, 2010). Race-related disparities in CVD incidence and mortality are well documented. The CDC and the Office of Minority Health report that African Americans are 1.5 times more likely to have hypertension (high blood pressure) and 1.3 times more likely to die from heart disease compared to Whites. Although far less Latino comparative health data is available, there are important differences to consider. Latinos are more likely to be overweight and have uncontrolled diabetes, two robust risk factors for heart disease. However, the age-adjusted mortality rate from heart disease is significantly lower for Latinos compared to non-Hispanic Whites (165.0 vs. 239.8 per 100,000). Emerging

data suggests that although the incidence rate of heart disease is similar between Latinos and non-Hispanic Whites, the rate at which the disease progresses to critical endpoints, such as heart attacks, is significantly slower for less acculturated Latinos.

Cancer Overall cancer incidence is approximately 30% lower for Latinos relative to non-Hispanic Whites (U.S. Cancer Statistics Working Group, 2011). This advantage is evident among both men and women although there is heterogeneity by cancer type. For example, Latina women are at 33% lower risk for breast cancer and Latino men are 23% less likely to have prostate cancer relative to their non-Hispanic White counterparts. However, Latinos are twice as likely to develop stomach and liver cancer relative to non-Hispanic Whites. In addition, Latina women have an 80% greater risk of being diagnosed with cervical cancer than non-Hispanic women. Unfortunately, the relative survival rate of Latinos following diagnosis is unclear due to a lack of available data. What is known is that relative mortality rates mirror incidence rates with Latinos more likely to die from stomach and liver cancers compared to non-Hispanic Whites.

Infectious Conditions Latinos suffer disproportionate rates of a range of infectious conditions. For example, Latinos account for approximately 75% of newly diagnosed tuberculosis cases and are at twice the risk of hepatitis A, twice the risk for gonorrhea and syphilis, and three times the risk for chlamydia compared to non-Hispanic Whites (CDC, 2009a). With respect to HIV/AIDS, Latinos accounted for approximately 17% of all HIV/AIDS cases despite accounting for less than 15% of the US population in 2008 (CDC, 2009b). Comparatively, Latinos are 3 times as likely to be diagnosed with HIV/AIDS relative to non-Hispanic Whites. Although disparities exist for both sexes, Latina women are 5 times more likely to be diagnosed with HIV compared to non-Hispanic White women. The AIDS-related mortality rate for Latino men is approximately 2.5 times, and for Latina women, 3.6 times the rate of non-Hispanic White men and women, respectively.

Despite their lower SES profile, infant/childhood immunization rates are comparable between Latinos and non-Hispanic Whites (CDC, 2009c). However, there are critical differences in the vulnerable 65 and older age category. At a time when risk increases, Latinos are 20% less likely to get the annual influenza (flu) vaccination and 40% less likely to be immunized for pneumonia (CDC, 2010). Such differences may increase vulnerability disparities in later life.

The Hispanic Mortality Paradox Substantial evidence suggests Latinos live longer than non-Hispanic Whites, an epidemiological paradox given the relative differences in SES and mortality risk factors. This phenomenon was first identified by Markides (1983) who noticed that among community samples in the Southwestern United States, Latino mortality rates were more similar to non-Hispanic Whites than to non-Hispanic Blacks despite substantial SES differences. Subsequent national cohort data consistently demonstrates a Latino mortality advantage relative to other racial/ethnic groups, including non-Hispanic Whites. For example, the 2006 CDC report on mortality by race/ethnicity indicates that the age-adjusted death rate for Latinos is approximately 27.4% lower than for non-Hispanic Whites and 43.7% lower than for non-Hispanic Blacks (Heron et al., 2009). Hence, the Hispanic mortality paradox refers to the epidemiological finding that Latinos appear to live longer than non-Hispanic Whites despite the lower SES of the former relative to the latter.

Multiple explanations have been postulated to explain these paradoxical findings. The most common criticism concerns data reliability. Critics argue that the national cohort data is highly suspect as it is based on representative statistics. For example, the Latino mortality rate is estimated by entering the annualized Latino mortality (determined by number of death certificates indicating Hispanic ethnicity in a given year) in the numerator and the corresponding Latino population size (determined via census estimates) into the denominator. The major

concern is that the numerator relies on accurate recording of Hispanic ethnicity on death certificates and that underreporting biases a more favorable ratio. However, recent data suggests that ethnic misclassification is rare and comparable for Latinos and non-Hispanics (Arias, Eschbach, Schauman, Backlund, & Sorlie, 2010). In addition, two general explanatory hypotheses, the *salmon bias hypothesis* (i.e., Latinos possibly returning to their ancestral country of origin prior to death) and the *healthy immigrant hypothesis* (i.e., biased Latino health in the USA due to better health among those who successfully migrate) have been largely debunked by available data.

An alternative to representative estimates is to examine longitudinal studies where individuals are tracked over time. A recent systematic review and meta-analysis of the longitudinal literature further supports the validity of the paradox (Ruiz, Steffen, & Smith, 2011). Across 58 identified studies involving over 4.6 million participants, Latinos were found to have a 17.5% lower risk of mortality compared to other racial groups. The advantage was most evident in initially healthy samples and in the context of cardiovascular disease. In conjunction with the consistent national cohort data, it may be time to turn the corner on questioning the validity of the paradox and direct efforts to identifying the mechanisms leading to these surprising outcomes.

It is important to note that a mortality advantage is not synonymous with better health. As the risk factor data illustrates, Latinos experience high rates of diabetes, infectious diseases, and several types of cancer. They are less likely to have access to regular medical care, and when they do seek care, it is often in more emergent conditions. Hence, Latinos may live longer but suffer a lower health-related quality of life.

Future Directions

The Hispanic mortality paradox illustrates the heterogeneity in minority health. Despite a risk profile similar to African American/Blacks, Hispanics have significantly different disease incidence, burden, and mortality outcomes. Such differences necessitate research specifically on the health and disease course of Latinos. It is possible that traditional risk factors simply are not the best predictors for disease and mortality among Latinos. It is also possible that Latinos may have resilience advantages at important time points in the disease course such as resistance to initial incidence, slower disease progression, or the ability or resources to more effectively recover from acute incidences. Additional research is also needed to understand the moderating and mediating factors specific to Latinos. Such studies may encompass the role of individual level factors (e.g., personality, experiences of stress), social (e.g., social network size and function) and cultural factors (e.g., cultural values, collectivism), as well as genetic/biological differences that may influence health outcomes. Finally, models are needed to understand the interplay between diverse factors and their impact on Latino health.

Cross-References

▶ Health Disparities

References and Readings

American Heart Association. (2010). Heart disease and stroke statistics 2010 update: A report from the American Heart Association. *Circulation, 121*, e46–e215.

Arias, E., Eschbach, K., Schauman, W., Backlund, E., & Sorlie, P. (2010). The Hispanic mortality advantage and ethnic misclassification on US death certificates. *American Journal of Public Health, 100*, S171–S177.

Centers for Disease Control and Prevention. (2009a). *Sexually transmitted diseases in the United States, 2008: National surveillance data for Chlamydia, gonorrhea, and syphilis*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention.

Centers for Disease Control and Prevention (2009b). *HIV/AIDS surveillance Report, 2007*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention.

Centers for Disease Control and Prevention (2009c). *National Immunization Survey (NIS) – Children 19–35 months old*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention.

Centers for Disease Control and Prevention. (2010). *Healthy People 2010 Database. Table 14-29d*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention. http://wonder.cdc.gov/data2010/

Centers for Disease Control and Prevention. (2011). *National diabetes fact sheet: National estimates and general information on diabetes and prediabetes in the United States, 2011*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention.

Fry, R., & Passel, J. S. (2009). *Latino children: A majority are U.S.-born offspring of immigrants*. Washington, DC: Pew Hispanic Center.

Heron, M., Hoyert, D. L., Murphy, S. L., Xu, J., Kochanek, K. D., & Tejada-Vera, B. (2009). Deaths: Final data for 2006. *National vital statistics reports, 57*. Hyattsville, MD: National Center for Health Statistics.

Markides, K. S. (1983). Mortality among minority populations: A review of recent patterns and trends. *Public Health Reports, 98*, 252–260.

Perez-Stable, E. J., Napoles-Springer, A., & Miramontes, J. M. (1997). The effects of ethnicity and language on medical outcomes of patients with hypertension. *Medical Care, 35*, 1212–1219.

Pew Hispanic Center. (2010). *Statistical Portrait of Hispanics in the United States, 2008*. http://pewhispanic.org/factsheets/factsheet.php?FactsheetID=58

Pew Hispanic Center (2011). Census 2010: 50 Million Latinos: Hispanic Account for more than half of nations' growth in past decade. March 24, 2011 (http://www.pewhispanic.org/files/reports/140.pdf). Washington, D.C.: Pew Hispanic Center.

Ruiz, J. M., Steffen, P.,& Smith, T. B (2011). The Hispanic mortality paradox: Resolving discrepancies through a quantitative analysis of the prospective literature. Paper presented to the American Psychosomatic Society Meeting, Spring 2011, San Antonio, TX.

Smith, D. P., & Bradshaw, B. S. (2006). Rethinking the Hispanic paradox: Death rates and life expectancy for U.S. non-Hispanic white and Hispanic populations. *American Journal of Public Health, 96*, 1686–1692.

U.S. Bureau of Labor Statistics. (2009). *Labor force characteristics by race and ethnicity, 2008*. U.S. Department of Labor, Report 1020. http://www.bls.gov/cps/cpsrace2008.pdf

U.S. Cancer Statistics Working Group (2011). *United States Cancer Statistics: 1999–2007 Incidence and Mortality Web-based Report*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention and National Cancer Institute. Available at: www.cdc.gov/uscs.

Zea, M. C., Quezada, T., & Belgrave, F. Z (1994). Cultural values and adjustment to disability among Latinos. *Journal of Social Behavior and Personality*, 9, 195–200

Histamine

Scott DeBerard
Department of Psychology, Utah State University, Logan, UT, USA

Synonyms

Substance H

Definition

Histamine is a biogenic amine which is involved in a number of biological processes including immune system, gastrointestinal, and nervous system functioning. Histamine is typically released in response to a pathogen or allergen and serves to increase capillary permeability allowing fluid and other immune system cells to leave the capillaries and enter tissues. The fluid will contain white blood cells which can attack and minimize the effect of the pathogen. In response to an allergen, histamine results in typical allergy symptoms of runny nose, congestion, sneezing, and watery eyes. Antihistamines are medications which block receptor sites for histamine and can reduce such allergy symptoms. Histamines are also thought to be implicated in memory, learning, and sleep regulation.

Cross-References

▶ Immune Function
▶ Immunity

References and Readings

Jones, B. L., & Kearns, G. L. (2011). Histamine: New thoughts about a familiar mediator. *Clinical Pharmacology and Therapeutics, 89*(2), 189–197.

Mohammed, S., Khardori, N., Khan, R. A., & Tripathi, T. (Eds.). (2010). *Biomedical aspects of histamine: Current perspectives*. New York: Springer.

Nuutinen, S., & Panula, P. (2011). Histamine in neurotransmission and brain diseases. *Advances in Experimental Medicine and Biology, 709*, 95–107.
Smuda, C., & Bryce, P. J. (2011). New developments in the use of histamine and histamine receptors. *Current Allergy and Asthma Reports, 11*(2), 94–100.

HIV Infection

Nicole Overstreet
Social Psychology, University of Connecticut, Storrs, CT, USA

Synonyms

AIDS: Acquired immunodeficiency syndrome; Sexually transmitted disease/infection (STD/STI)

Definition

HIV infection can be characterized as a condition caused by the human immunodeficiency virus (HIV) (National Center for Biotechnical Information [NCBI], 2010a). HIV is a retrovirus that weakens the body's immune system by infecting and replicating in CD4 + T-cells (Morris & Cilliers, 2008). CD4 + T-cells stimulate lymphocytes that assist the body in attacking antigens and fighting off disease. When HIV infection occurs, the virus gains entry to host T-cells by attaching to CD4 receptors via glycoprotein (gp120). gp120 allows HIV to bind to CD4 receptors and kill off helper T-cells. During this process, HIV's genetic material integrates its viral DNA into the cell's host DNA and allows the virus to replicate in the immune system (Morris & Cilliers, 2008).

Description

HIV Infection: Prevalence

Since its co-discovery by Drs. Luc Montagnier and Robert Gallo in 1983, millions of people have been infected with HIV. Current prevalence estimates reveal that there are approximately 33.3 million people living with HIV worldwide (UNAIDS, 2010). HIV prevalence varies greatly by region: HIV disproportionately affects sub-Saharan Africa, a region that accounts for 68% of the global total of persons living with HIV (22.5 million); HIV prevalence has nearly tripled in Eastern Europe and Central Asia (1.4 million) and has risen in North America (1.5 million), Western and Central Europe (820,000), Oceania (57,000), and in the Middle East and North Africa (460,000); HIV prevalence remains relatively stable in South and Central America (1.4 million), the Caribbean (240,000), and Asia (4.9 million); however, there is considerable variation in HIV prevalence within these regions (UNAIDS, 2010).

HIV Infection: Incidence

Advances in HIV prevention have markedly reduced the number of new HIV infections. However, there are noticeable differences in HIV incidence trends worldwide, and incidence patterns vary within countries. Many new cases of HIV infection are concentrated among people who inject drugs, commercial sex workers, and men who have sex with men (UNAIDS, 2010). HIV incidence is particularly high in developing countries (Merson, 2006). However, countries with higher incomes are not immune from the HIV epidemic. In the United States, the epidemic disproportionately affects racial and ethnic minorities and men who have sex with men (MSM), especially in urban areas of the Northeast, West Coast cities, and small towns in the South (El-Sadr, Mayer, & Hodder, 2010). Higher HIV incidence in Western Europe can also be attributed to increases in risky sexual behavior among MSM. Concentrated epidemics, particularly among people using injection drugs and sex workers, contribute to the growing epidemic in Eastern Europe and Central Asia (UNAIDS, 2010). HIV incidence by region is as follows (UNAIDS, 2010):

- Sub-Saharan Africa: 1.8 million
- South and Southeast Asia: 270,000
- Eastern Europe and Central Asia: 130,000
- Central and South America: 92,000

- East Asia: 82,000
- Middle East and North Africa: 75,000
- North America: 70,000
- Western and Central Europe: 31,000
- Caribbean: 17,000
- Oceania: 4,500

Stages of HIV Infection

Within 2–4 weeks after HIV infection, individuals may experience acute retroviral syndrome (ARS) or acute HIV infection. Acute HIV infection can appear flu-like and may be accompanied by a number of symptoms such as sore throat, headache, fever, and swollen lymph glands (National Center for Biotechnical Information [NCBI], 2010b). It may take several months for individuals to exhibit ARS symptoms; however, many people never develop ARS or exhibit symptoms associated with the syndrome. During this phase, the virus is actively replicating and is highly transmissible (NCBI, 2010b). After primary HIV infection, the illness may be asymptomatic in which there are no major symptoms of HIV. Individuals vary in their detectable HIV viral load but can still transmit the virus to others. The asymptomatic stage can last up to 10 years. Over time, the body's immune system weakens from fighting the virus and transitions into symptomatic HIV infection. In this period, individuals exhibit symptoms typical of chronic HIV infection (e.g., diarrhea, fatigue, fever, weight loss). Acquired immunodeficiency syndrome (AIDS) is the final stage of HIV and is defined by <200 CD4 + T-lymphocyte cell counts per microliter of blood (CDC, 1993). The body's immune system is severely damaged and is susceptible to opportunistic infections (e.g., Kaposi's sarcoma). Individuals vary in their progression through these phases. Adherence to antiretroviral medications plays a vital role in suppressing HIV viral replication and the progression of AIDS. There are two types of HIV infection – HIV-1 and HIV-2: both types of HIV weaken the body's immune system; however, HIV-2 has a longer asymptomatic phase with slower and milder immunodeficiency and is predominately found in West Africa (CDC, 2010).

Two classification systems are currently used to assess the progression of HIV infection: the Centers for Disease Control (CDC) classification system and the World Health Organization (WHO) Clinical Staging and Disease Classification System.

The CDC classification system (CDC, 1993) categorizes the progression of HIV infection into three CD4 + T-lymphocyte categories and three clinical categories. These categories are accompanied by particular illnesses or conditions. The corresponding CD4 + T-lymphocyte categories are as follows:

1. Category 1: ≥500 cells/mL
2. Category 2: 200–499 cells/mL
3. Category 3: <200 cells/mL

These categories correspond to CD4 + T-lymphocyte counts per microliter of blood in which an acute HIV infection (≥500 cells/mL) progresses to acquired immunodeficiency syndrome (AIDS) (<200 cells/mL). The clinical categories of HIV infection and corresponding illnesses are as follows:

1. Category A (asymptomatic/acute HIV): Asymptomatic or acute retroviral syndrome/acute HIV infection (acute HIV infection occurs 2–4 weeks after HIV infection, and its symptoms include fatigue, fever, headache, sore throat, and decreased appetite).
2. Category B (symptomatic conditions): Conditions include, but are not limited to, cervical dysplasia, shingles, and pelvic inflammatory disease.
3. Category C (AIDS-indicator conditions): Presence of opportunistic infections such as Kaposi's sarcoma and *Mycobacterium tuberculosis*.

In settings with limited capability to test for CD4 viral loads, the WHO Clinical Staging and Disease Classification System (WHO, 2007) may be used to determine the progression of HIV infection and the appropriate antiretroviral therapy. This classification system is categorized into four stages ranging from primary HIV infection to AIDS-indicator illnesses. The stages of HIV infection and some accompanying illnesses are as follows:

1. Clinical stage 1 (asymptomatic): Asymptomatic, persistent generalized lymphadenopathy

2. Clinical stage 2 (mild symptoms): Unexplained weight loss, fungal nail infections
3. Clinical stage 3 (advanced symptoms): Unexplained severe weight loss, oral hairy leukoplakia
4. Clinical stage 4 (severe symptoms): HIV wasting syndrome, Kaposi's sarcoma

HIV Transmission

HIV is primarily spread through the following modes of transmission (CDC, 2010): unprotected sexual contact with a person who has HIV infection (unprotected oral sex is lower risk for transmitting HIV than vaginal or anal sex; unprotected anal sex is riskier than vaginal sex; unprotected receptive anal sex is riskier than unprotected insertive anal sex; having multiple sex partners also increases risk of HIV infection, especially in the presence of other sexually transmitted diseases during sex), sharing needles or syringes used to prepare illicit drugs for infection, and being born to an infected mother (HIV can be passed from mother to child through pregnancy, birth, or breast-feeding). Less common modes of HIV infection are through blood transfusions. These modes of HIV transmission occur by the transfer of semen, blood, vaginal fluid, or breast milk.

HIV Diagnosis

HIV is typically diagnosed by a set of blood tests (HIV ELISA) that detect the presence of HIV antibodies but can also be diagnosed by tests that identify HIV's genetic material (CDC, 2010). The presence of HIV antibodies marks the body's response to HIV infection. A "window period" between HIV infection and seroconversion (presence of detectable HIV antibodies) can range from 2 weeks to 3 months, in which an individual's immune system may not produce enough antibodies for the antibody test to detect. HIV is highly transmissible during this period even if the virus is not yet detectable. Most people will develop detectable antibodies within 2–8 weeks of HIV infection, and HIV test should be considered positive after a second confirmatory HIV test (CDC, 2010; Puren, 2008).

HIV Treatment

There is no cure for HIV infection. However, antiretroviral therapy can suppress HIV viral replication and the progression of AIDS. These medications help people living with HIV to lead longer and healthier lives. Inconsistent adherence allows HIV to acquire resistance to antiretroviral therapy. There are considerable barriers in HIV prevention and treatment, but global efforts continue to address these challenges.

Cross-References

▶ HIV Prevention
▶ Sexual Risk Behavior

References and Readings

Centers for Disease Control and Prevention. (1993). 1993 Revised classification system for HIV infection and expanded surveillance case definition for AIDS among adolescents and adults. *Morbidity and Mortality Weekly Report, 269*, 729–730. Available online at www.cdc.gov/mmwr/preview/mmwrhtml/00018871.htm.

Centers for Disease Control and Prevention. (2010). *Basic information about HIV and AIDS*. Retrieved from http://www.cdc.gov/hiv/topics/basic/index.htm

El-Sadr, W. M., Mayer, K. H., & Hodder, S. L. (2010). AIDS in America-Forgotten but not gone. *New England Journal of Medicine, 362*, 967–969.

Merson, M. H. (2006). The HIV-AIDS pandemic at 25-The global response. *New England Journal of Medicine, 354*, 2414–2417.

Morris, L., & Cilliers, T. (2008). Viral structure, replication, tropism, pathogenesis and natural history. In S. S. Abdool Karim & Q. Abdool Karim (Eds.), *HIV/AIDS in South Africa* (pp. 79–88). New York: Cambridge University Press.

National Center for Biotechnical Information. (2010a). *HIV infection*. Retrieved from http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0001627#adam_000602.disease.signs-and-tests

National Center for Biotechnical Information. (2010b). *Acute HIV infection*. Retrieved from http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0001629/

Puren, A. J. (2008). HIV diagnostics. In S. S. Abdool Karim & Q. Abdool Karim (Eds.), *HIV/AIDS in South Africa* (pp. 89–108). New York: Cambridge University Press.

UNAIDS. (2010). *Global report: UNAIDS report on the global AIDS epidemic 2010*. Retrieved from http://www.unaids.org/globalreport/Global_report.htm

World Health Organization. (2007). *WHO Case definitions of HIV for surveillance and revised clinical staging and immunological classification of HIV-related disease in adults and children*. Retrieved from http://www.who.int/hiv/pub/guidelines/HIVstaging150307.pdf

HIV Prevention

Jason W. Mitchell
Center for AIDS Intervention Research, Medical College of Wisconsin, Milwaukee, WI, USA

Synonyms

AIDS prevention

Definition

HIV prevention is the science of designing, implementing, and evaluating the effectiveness of interventions, programs, and services that aim to prevent an individual from acquiring HIV.

Description

The acquired immune deficiency syndrome (AIDS) was first recognized in 1981 and is caused by the human immunodeficiency virus (HIV) (CDC, 2010). Since the 1980s, science has significantly increased our knowledge about HIV and AIDS. We now know how HIV is transmitted from one person to another, the physiological processes of how the virus replicates once acquired, which medications help stop the virus from replicating, why individuals are more susceptible to opportunistic infections, and how best to prevent HIV transmission between two individuals. As a result, the field of HIV prevention science has emerged to better understand how individuals can help protect themselves from acquiring HIV, and among those who are living with HIV, how best to improve their adherence to treatment and reduce the transmission of HIV to others.

Efforts to reduce new HIV cases are based on how the virus is transmitted. There are three primary modes of transmission, including sexual transmission, transmission through blood, and mother-to-child transmission (Avert, 2011). In detail, an individual may acquire HIV from infected donated blood and organ products, vertically from mother-to-child during labor and/or from feeding the infant breast milk, sexually from having unprotected anal or vaginal intercourse, and from sharing contaminated works when injecting drugs or being stuck with a contaminated needle. Accordingly, HIV prevention interventions, programs, and services are designed to target specific populations based on their primary risk factor(s) for acquiring HIV. For example, self-identified heterosexual individuals who may be more likely to have unprotected intercourse, otherwise known as "high-risk heterosexuals," would be targeted for a particular HIV prevention program that was specifically designed for them. Another HIV prevention program may target HIV-negative men who have sex with men (MSM) to help them sustain from having unprotected anal intercourse (UAI) with multiple sex partners of HIV-positive and/or unknown serostatus. As such, HIV prevention interventions, programs, and services are typically designed and implemented to reach certain populations that engage in behaviors that put them at risk for acquiring HIV.

HIV prevention also has evolved to include individuals living with HIV. For instance, people living with HIV may want help with adherence to their medications, finding appropriate doctors for their treatments, access to care, assistance with disclosing their HIV serostatus, or learning how to help protect others from acquiring HIV from them. In sum, HIV prevention interventions,

programs, and services include two broad types: (1) primary prevention which aims to prevent individuals from acquiring HIV and (2) secondary prevention which helps individuals living with HIV to maintain their health and to reduce the possibility of transmitting HIV onto others (Avert, 2011).

Moreover, the implementation of these interventions, programs, and services is further divided based on the context of the implementation and how many individuals will be targeted. Specifically, interventions are classified as individual-level, group-level, or community-level interventions. Each of these levels describes the population context of the intervention for the primary or secondary HIV prevention services that would be provided to the participants. For example, an individual-level, primary HIV prevention intervention would focus on reducing HIV risk for each individual who participates in that particular intervention.

Many interventions, programs, and services of HIV prevention include an aspect of psycho-behavioral modification and/or a biomedical approach. Several theories of behavior change and models have been developed or modified for the prevention of HIV. For example, many studies and interventions aimed at preventing HIV have used constructs from the health belief model (Rosenstock, Strecher, & Becker, 1994), social cognitive theory (Bandura, 1997), theory of reasoned action (Fishbein & Ajzen, 1975), theory of planned behavior (Ajzen, 1991), transtheoretical model (Prochaska, DiClemente, & Norcross, 1992), AIDS risk reduction model (Catania, Kegeles, & Coates, 1990), and the information-motivation-behavioral skills model (Fisher & Fisher, 1992). Constructs from these theories and models are used to help participants and their networks change their beliefs, attitudes, perceived ability, and norms to lower their risk for acquiring HIV. Scientists and other researchers also use these constructs to better understand individuals' thoughts, actions, and feelings about HIV and/or risk behaviors.

In contrast, the biomedical approach uses a "test and treat" approach that may or may not include a psycho-behavioral component to it. The biomedical approach emphasizes the importance of being tested for HIV. The frequency of being tested for HIV will depend on the individual needs, her or his risk, and access to receiving the HIV test. Because an individual's viral load is highest after initially being infected, the premise of the approach is to link HIV-positive individuals with care as soon as possible and, preferably, before the possibility of transmitting the virus to others. Regardless of which HIV prevention approach is used, only correct and consistent condom use for anal and vaginal intercourse, being in a long-term mutually monogamous relationship with an uninfected partner, or abstaining from anal and vaginal intercourse will significantly prevent an individual from contracting HIV (CDC, 2010).

In addition to testing, consistent condom use, and modifying behaviors to reduce HIV risk, new advances with treatments have led to the development of other promising HIV prevention methods. Current studies are assessing the efficacy and cost-effectiveness of administering a pill or inserting a microbicide (i.e., gel or foam) inside an anus or vagina as additional modes of preventing HIV transmission (AVAC, 2011). These new biomedical advances are still being studied in a variety of populations around the world.

The success of the psycho-behavioral and biomedical approaches to preventing primary and secondary HIV infections is not without debate. Evaluation of HIV prevention interventions is crucial for determining what works best in HIV prevention. For example, the US Centers for Disease Control and Prevention (2007) provides a list of interventions that have been shown to prevent HIV among a variety of at-risk populations. These interventions are called evidence-based interventions (EBIs). Further, new studies that examine the different contexts of HIV are constantly needed in order to address how the epidemic changes with time across the world. The Further Readings section provides additional information about HIV prevention.

References and Readings

Ajzen, I. (1991). The theory of planned behavior. *Organizational Behavior and Human Decision Processes, 50*, 179–211.
AVAC. (2011). *Global advocacy for HIV prevention*. Retrieved February 8, 2011 from http://www.avac.org/
Avert. (2011). *Introduction to HIV prevention*. Retrieved February 8, 2011 from http://www.avert.org/prevent-hiv.htm
Bandura, A. (1997). *Self-efficacy: The exercise of control*. New York: Freeman.
Catania, J. A., Kegeles, S. M., & Coates, T. J. (1990). Towards an understanding of risk behavior: A AIDS risk reduction model (ARRM). *Health Education Quarterly, 17*, 53–72.
Centers for Disease Control and Prevention. (2007). *Prevention research synthesis*. Retrieved February 8, 2011 from http://www.cdc.gov/hiv/topics/research/prs/prs_rep_debi.htm
Centers for Disease Control and Prevention. (2010). Retrieved February 8, 2011 from http://www.cdc.gov/hiv/topics/basic/index.htm
Fishbein, M., & Ajzen, I. (1975). *Belief, attitude, intention, and behavior: An introduction to theory and research*. Reading, MA: Addison-Wesley.
Fisher, W. A., & Fisher, J. D. (1992). Changing AIDS-risk behavior. *Psychological Bulletin, 111*, 455–474.
Prochaska, J., DiClemente, R. J., & Norcross, J. (1992). In search of how people change. *American Psychologist, 47*, 1102–1114.
Rosenstock, I. M., Strecher, V. J., & Becker, M. H. (1994). The health belief model and HIV risk behavior change. In R. J. DiClemente & J. L. Peterson (Eds.), *Preventing AIDS: Theories and methods of behavioral interventions*. New York: Plenum Press.

HIV Status

► Serostatus: Seronegative and Seropositive

HIV Wasting

► Cachexia (Wasting Syndrome)

HMG-CoA Reductase Inhibitors

► Statins

Holistic Medicine

► Integrative Medicine

Home Health Care

Olveen Carrasquillo[1] and Osvaldo Rodriguez[2]
[1]Division of General Medicine, Miller School of Medicine, University of Miami, Miami, FL, USA
[2]Miami VA Healthcare System, Miami, FL, USA

Definition

Home health care is the rendering of predominantly medically related services to patients in a home setting rather than in a medical facility. The goals of home health care include helping patients recover from an injury or illness and restoring, maintaining, or increasing their ability to tend to their everyday needs at home.

Description

There are a wide range of services that fall under the definition of home health care. These include more skilled care often focused on a particular condition such as wound care for pressure ulcers or surgical wounds, physical and occupational therapy, speech-language therapy, patient and caregiver education such as for daily insulin injections, other intravenous injections, and monitoring serious or difficult to control chronic illness such as diabetes and congestive heart failure. However, it may also include a broader range of services such as social services or assistance from a home health aide. Examples of home health aide services include help with basic daily activities like getting in and out of bed, dressing, bathing, eating, and using the bathroom, as well as help with light housekeeping, laundry, shopping, and cooking for the patient.

In addition to such formalized services, the term can also include informal health-care

services provided by spouses, family, friends, and neighbors often similar in scope to those done by paid home health aides. In some European countries, such informal care is partially funded, while in most other countries, it is typically unreimbursed. One estimate from the USA is that one of every five adults in the United States provides some sort of such informal home care to someone else.

Historically, most health care was traditionally provided in a patient's home. For centuries, midwives, medicine men, and traveling doctors all performed their services in a patient's home. Sometimes, sick people went to a doctor's office, which was usually an extension of the provider's home or a small clinic, but more often than not, the health-care specialists visited the sick in their home. It was only at the start of the twentieth century with the advancement in medical knowledge, treatments, and technology, not feasible to be provided in the home setting, that led to movement of health service delivery for very sick patients to a hospital setting. Of note, in many countries, particularly those with fewer resources where hospital growth and development has been more limited, many very sick patients are still not able to receive hospital-based care and remain in their home, often cared, to the extent possible, by family and friends.

In the latter half the twentieth century, patient preferences and rising hospital care costs have led to initiatives in many advanced countries to shift some care back away from hospitals to a patients home. Thus, governments and other payers began reimbursing for services aimed at fostering home health-care practices. At present, in the USA, where nearly 12 million persons receive home health care at an estimated cost of over $70 billion, the Center for Medicare and Medicaid services is the largest payer for home health care. CMS is also responsible for regulating most home health companies and ensuring compliance. Many states also have additional regulations, licensing and certification requirements, and compliance measures. However, determination of eligibility for coverage and length of services is often a challenge. For example, under CMS, to qualify for reimbursement for home health services, an individual has to be "confined to his home." Operationalizing this definition is often difficult as policy makers need to balance ensuring patients needing services are not denied home care while at the same time limiting potential fraud and abuse. Which services are covered (e.g., skilled or unskilled), for how many hours per day, and for how long are decisions that have tremendous implications and need to be balanced between need and available resources.

Given the extensive evidence that such care is often less costly and sometimes of higher quality than that delivered in an inpatient facility, there will continue to be large growth in the home health-care sector. Particularly given the aging of the population in many countries, governments and policy makers will need to continue to foster, develop, and strengthen innovative programs aimed at facilitating home-based care.

Cross-References

- ▶ Aging
- ▶ Disability
- ▶ Family, Caregiver
- ▶ Health Care
- ▶ Patient Care

References and Readings

Bercovitz, A., Moss, A., Sengupta, M., Park-Lee, E. Y., Jones, A., Harris-Kojetin, L. D., et al. (2011). *An overview of home health aides: United States, 2007* (National Health Statistics Reports No. 34). Hyattsville, MD: National Center for Health Statistics.

Ellenbecker, C. H., Samia, L., Cushman, M. J., & Alster, K. (2008). Patient safety and quality in home health care. In R. G. Hughes (Ed.), *Patient safety and quality: An evidence-based handbook for nurses*. Rockville, MD: Agency for Healthcare Research and Quality (US).

Medicare and Home Health Care, Centers for Medicare & Medicaid Services. CMS Product No. 10969 Revised May 2010.

Tarricone, R., & Tsouros, A. D. (2008). *Home care in Europe*. Copenhagen: World Health Organization. ISBN 978 92 890 4281 9.

The National Association for Home Care & Hospice. (2010). Basic statistics about home care: Updated 2010. Retrieved from www.nahc.org

Homeostasis

Jens Gaab
Clinical Psychology and Psychotherapy, Department of Psychology, University of Basel, Basel, Switzerland

Synonyms

Equilibrium; Milieu interieur

Definition

Tendency and/or ability of a system to strive for and maintain stability of vital internal functions/parameters under unstable, external, or internal circumstances by means of negative or positive feedback.

Description

The necessity of a constant internal stability in face of external instability as a requirement of a free and independent life has first been stated by Claude Bernard, a French physiologist (1813–1878). Walter Bradford Cannon (1871–1945), physiologist at Harvard, then first coined the term "homeostasis," which he based on four propositions: (1) Constancy needs mechanisms to maintain constancy, (2) any change will be automatically resisted, (3) controlling mechanisms are interacting and cooperative, and (4) homeostasis is organized. Thus, homeostasis is the result of a complex interaction of multiple physiological – as well as psychological and behavioral – processes in order to secure and maintain set levels. Formally, the components of a homeostatic system encompass receptors that assess and relay information afferent to a central control which then send efferent signals to subordinated effectors, such as muscles, glands, organs, or systems. The responses are then monitored and controlled by positive and/or negative feedback. Examples for homeostasis can be seen in the regulation of body temperature by redistribution of blood flow (cold feet and hands), activation of muscular (shivers) and behavioral responses (put on a sweater), or in the regulation of blood glucose concentration (release of insulin and glucagon) as well as in more complex interactions between perceived challenges in social situations and emotional, behavioral, as well as neuroendocrine responses and their impact on the situational circumstances. The consequences of the regulatory responses are manifold and encompass permissive, stimulatory, suppressive, as well as preparative actions.

The concept of homeostasis have been extended by the concept of allostasis (McEwen, 1998), which states that stability is not only achievable through regulation, but also through change. Thus, external or internal circumstances are not only met by counterregulatory activation of the respective response but also lead to adaptive changes in the activity and reactivity of the respective system. For example, enduring stress not only leads to enhanced output of adrenocortical steroids, but also to changes in the negative feedback regulation of the affected neuroendocrine system, depending on the duration and the extent of the stressor (for review: Sapolsky, Romero, & Munck, 2000).

Cross-References

- ▶ Allostasis, Allostatic Load
- ▶ Neuroendocrine Activation
- ▶ Stress

References and Readings

McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *The New England Journal of Medicine, 338*(3), 171–179.

Sapolsky, R. M., Romero, L. M., & Munck, A. U. (2000). How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. *Endocrine Reviews, 21*(1), 55–89.

Homocysteine

Siqin Ye
Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Synonyms

$HSCH_2CH_2CH(NH_2)CO_2H$

Definition

Homocysteine is an amino acid that acts as the intermediary during the synthesis of cysteine from dietary methionine in the human body.

Description

Homocysteine is an amino acid that acts as the intermediary during the synthesis of cysteine from dietary methionine in the human body. Homocysteine is not obtained from the diet but rather formed from the demethylation of methionine. It can then condense with the amino acid serine to form cystathionine, in a step catalyzed by the enzyme cystathionine β-synthase that requires vitamin B_6 as a cofactor. Cystathionine is then cleaved by cystathionine γ-lyase to produce cysteine and alpha-ketobutyrate. Alternatively, homocysteine can be converted back into methionine by donation of a methyl group from methyltetrahydrofolate, which is derived from folic acid. This reaction is catalyzed by the enzyme methionine synthase and requires vitamin B_{12} as an essential cofactor (Konkle, Simon, & Schafer, 2008).

In patients with the medical condition homocystinuria, deficiencies in key enzymes of homocysteine metabolism such as those described above lead to dramatically elevated levels of plasma homocysteine, which then spill over and are detected in urine. In addition to abnormalities of neurological and skeletal development, these patients also suffer from premature atherosclerosis and thrombophilia and have high incidences of venous thromboembolism, cerebrovascular disease, and peripheral vascular disease, beginning as early as the second and third decade of life. Because of this, it has been suggested that elevated homocysteine may play a causative role in promotion of atherosclerotic disease. Both in vitro and in vivo studies have tended to support this hypothesis, demonstrating that homocysteine can increase oxidative stress, cause endothelial dysfunction, promote inflammation, and induce smooth muscle proliferation (Ridker & Libby, 2008; Undas, Brozek, & Szczeklik, 2005). Furthermore, multiple large, prospective epidemiological studies have also concluded that even mildly elevated levels of homocysteine may carry increased risks for both initial and recurrent cardiovascular events (Homocysteine Studies Collaboration, 2002; Wald, Law, & Morris, 2002).

These observations have led to interest in homocysteine reduction as a strategy for cardiovascular disease prevention. Since B-complex vitamins (folic acid, vitamin B_6, and vitamin B_{12}) play a vital role in homocysteine metabolism and are often insufficiently obtained from dietary sources, several large, randomized clinical trials have been carried out to test the efficacy of B-complex vitamin supplementation in patients with cardiovascular disease. Despite uniform reduction of homocysteine levels in the intervention groups, however, none of the trials were able to demonstrate any decrease in incidence of cardiovascular events. Most recently, a meta-analysis was performed by the Cochrane Collaboration in 2009 that included eight randomized clinical trials with a total of 24,210 participants, confirming that homocysteine lowering with B-complex vitamin supplementation does not significantly reduce the risk of fatal or nonfatal myocardial infarction, stroke, or death from any cause (Martí-Carvajal, Solà, Lathyris, & Salanti, 2009). Given the inconsistency between these results and previous experimental and epidemiological data, there remains considerable debate on whether the homocysteine hypothesis is itself incorrect or whether there are other negative effects of B-complex vitamin supplementation

that may have countered the potential benefit of homocysteine reduction (Loscalzo, 2006).

References and Readings

Homocysteine Studies Collaboration. (2002). Homocysteine and risk of ischemia heart disease and stroke: A meta-analysis. *Journal of the American Medical Association, 288*, 2015–2022.

Konkle, B. A., Simon, D., & Schafer, A. I. (2008). Hemostasis, thrombosis, fibrinolysis, and cardiovascular disease. In P. Libby, R. O. Bonow, D. L. Mann, D. P. Zipes, & E. Braunwald (Eds.), *Braunwald's heart disease: A textbook of cardiovascular medicine* (pp. 2049–2078). Philadelphia: Saunders Elsevier.

Loscalzo, J. (2006). Homocysteine trials-clear outcomes for complex reasons. *The New England Journal of Medicine, 354*, 1629–1632.

Martí-Carvajal, A. J., Solà, I., Lathyris, D., & Salanti, G. (2009). Homocysteine lowering interventions for preventing cardiovascular events. *Cochrane Database of Systematic Reviews* (4), CD006612. doi:10.1002/14651858.CD006612.pub2

Ridker, P. M., & Libby, P. (2008). Risk factors for atherothrombotic disease. In P. Libby, R. O. Bonow, D. L. Mann, D. P. Zipes, & E. Braunwald (Eds.), *Braunwald's heart disease: A textbook of cardiovascular medicine* (pp. 1003–10026). Philadelphia: Saunders Elsevier.

Undas, A., Brozek, J., & Szczeklik, A. (2005). Homocysteine and thrombosis: From basic science to clinical evidence. *Thrombosis and Haemostasis, 94*(5), 907–915.

Wald, D. S., Law, M., & Morris, J. K. (2002). Homocysteine and cardiovascular disease: Evidence on causality from a meta-analysis. *British Medical Journal, 325*, 1202–1202.

Homozygous

Laura Rodriguez-Murillo[1] and Rany M. Salem[2]
[1]Department of Psychiatry, Columbia University Medical Center, New York, NY, USA
[2]Broad Institute, Cambridge, MA, USA

Definition

An allele is one of the possible variants of a gene or position in the DNA sequence. All humans have two alleles on homologous chromosomes for each gene or trait. If these two alleles are identical, the person is considered to be homozygous for that trait or gene.

Cross-References

▶ Allele
▶ Dominant Inheritance
▶ Gene
▶ Heterozygous
▶ Recessive Inheritance

References and Readings

Lewis, R. (2005). *Human genetics. Concepts and applications* (7th ed.). Boston: McGraw-Hill Science/Engineering/Math.

Strachan, T., & Read, A. P. (2003). *Human molecular genetics* (3rd ed.). London/New York: Garland Science/Taylor & Francis Group.

Hooking Up

▶ Sexual Hookup

Hopelessness

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

This is an important reaction to situations involving lack of, or reduced, control. The literature at times confuses helplessness with hopelessness, with the former included in the latter. Hopelessness refers to the belief that one lacks control over outcomes (response-outcome noncontingency or helplessness) and to the expectation of a negative future (negative outcome expectancies or pessimism), as conceptualized by Everson et al. (1996). Hopelessness includes thus two cognitions, rather than feelings. In the general

population, hopelessness is quite a stable phenomenon and is predicted by economic difficulties and unemployment. However, positive changes in one's living condition may protect people from becoming hopeless (Haatainen et al., 2003).

Multiple ways exist to assess hopelessness including a 2-item scale by Everson et al. (1996), the Beck Hopelessness Scale, and other measures.

Hopelessness is a major precursor or depression, particularly in people with a negative attribution style (attributing a negative event to an internal, stable, and global cause; Sweeney, Anderson, & Bailey, 1986). Hopelessness has also been found to be a significant predictor of suicide. Given the difficulty to prevent suicidal behavior, conducting preventative interventions among high-hopeless people may have important preventative value for public health.

Hopelessness is a risk factor of death from coronary heart disease and cancer and significantly predicts progression of carotid atherosclerosis. In Everson et al.'s (1996, 1997), Everson, Kaplan, Goldberg, Salonen, and Salonen (1997) studies, controlling for known cardiac risk factors had little effect on the prognostic role of hopelessness, leading them to conclude that other, not tested factors, could explain these relationships. One model proposes that hopelessness predicts poor prognosis in cancer by its link to brain and systemic interleukin-1 (IL-1) since blocking brain IL-1 prevents helplessness, since brain IL-1 promotes peripheral metastases, and since peripheral IL-1 at tumor sites promotes tumor cell proliferation, angiogenesis, and metastasis (Argaman et al., 2005). Thus, hopelessness could serve as an important predictor and therapeutic target in behavior medicine intervention trials since it is a risk factor of both mental and physical illnesses.

Cross-References

- ▶ Depression: Symptoms
- ▶ Perceived Control

References and Readings

Argaman, M., Gidron, Y., & Ariad, S. (2005). Interleukin-1 may link helplessness hopelessness with cancer progression: A proposed model. *International Journal of Behavioral Medicine, 12*, 161–170.

Everson, S. A., Goldberg, D. E., Kaplan, G. A., Cohen, R. D., Pukkala, E., Tuomilehto, J., et al. (1996). Hopelessness and risk of mortality and incidence of myocardial infarction and cancer. *Psychosomatic Medicine, 58*, 113–121.

Everson, S. A., Kaplan, G. A., Goldberg, D. E., Salonen, R., & Salonen, J. T. (1997). Hopelessness and 4-year progression of carotid atherosclerosis. The Kuopio ischemic heart disease risk factor study. *Arteriosclerosis, Thrombosis, and Vascular Biology, 17*, 1490–1495.

Haatainen, K. M., Tanskanen, A., Kylmä, J., Honkalampi, K., Koivumaa-Honkanen, H., Hintikka, J., et al. (2003). Stable hopelessness and its predictors in a general population: A 2-year follow-up study. *Suicide & Life-Threatening Behavior, 33*, 373–380.

Sweeney, P. D., Anderson, K., & Bailey, S. (1986). Attributional style in depression: A meta-analytic review. *Journal of Personality and Social Psychology, 50*, 974–991.

Hormone System

▶ Endocrinology

Hormone Theory of Aging

▶ Neuroendocrine Theory of Aging

Hormone Therapy

▶ Hormone Treatment

Hormone Treatment

Oliver T. Wolf
Department of Cognitive Psychology
Ruhr-Universität Bochum, Bochum, Germany

Synonyms

Hormone therapy; Menopausal hormone therapy

Definition

Serum concentrations of the female gonadal sex steroid hormones estradiol and progesterone decrease sharply after ▶ menopause due to the stop of ovarian hormone production. In order to counteract this decline, several forms of hormone (replacement) treatment (or therapy; HT or HRT) have been developed. It has been suggested that this hormone regime can treat menopausal symptoms and could potentially prevent several age-associated disorders. Women with an intact uterus can be treated with a combined regimen of estrogens and progestins. The progestin is added in order to prevent endometrial hyperplasia. Women with a hysterectomy can be treated with estrogen only preparations. Oral route of administration is still the most commonly used route as of today, but other approaches (e.g., transdermal applications) are available.

Sex steroid treatment after menopause has beneficial effects on hot flushes and urogenital atrophy. Moreover it has been shown to prevent osteoporosis, fractures, and ▶ diabetes. Adverse effects include venothrombotic episodes, stroke, and ▶ breast cancer. Negative effects are more pronounced when the combined treatment is administered, when patients are older, and when treatment is continued for longer periods of time (more than 5 years). For reviews see Maki et al. (2010) and Santen et al. (2010).

Due to the widespread action of sex steroids in the brain, beneficial effects of hormone therapy on mood and cognition have been postulated. These could be of relevance for the prevention and/or treatment of menopausal depression and ▶ dementia. Beneficial effects on cognition or mood might occur if treatment is started during or directly after menopause ("window of opportunity hypothesis"). However large randomized controlled clinical trials in this age group are still lacking. In contrast to the postulated beneficial effects, adverse effects were observed in the women's health initiative (WHI) study. In older women, the combined treatment with estrogens and progestins increased the risk for stroke and dementia. More clinical trials with cognitive and affective measures are needed before recommendations can be provided based on solid empirical evidence. For reviews see Maki et al. (2010) and Santen et al. (2010).

As of today, hormone therapy is recommended by the food and drug administration (FDA) for the treatment of menopausal symptoms only. The FDA recommends that hormone therapy be used at the lowest doses for the shortest duration needed to achieve treatment goals.

Cross-References

- ▶ Breast Cancer
- ▶ Depression
- ▶ Diabetes
- ▶ Menopause

References and Readings

Maki, P. M., Freeman, E. W., Greendale, G. A., Henderson, V. W., Newhouse, P. A., Schmidt, P. J., et al. (2010). Summary of the National Institute on Aging-sponsored conference on depressive symptoms and cognitive complaints in the menopausal transition. *Menopause, 17*, 815–822.

Santen, R. J., Allred, D. C., Ardoin, S. P., Archer, D. F., Boyd, N., Braunstein, G. D., et al. (2010). Postmenopausal hormone therapy: An Endocrine Society scientific statement. *The Journal of Clinical Endocrinology and Metabolism, 95*, s1–s66.

Hormones

- ▶ Leptin
- ▶ Neuropeptide Y (NPY)

Hospice

Andrea Croom
Department of Psychology, University of Texas Southwestern Medical Center, Dallas, TX, USA

Synonyms

Hospice care; Hospice programs; Hospice services

Definition

Hospice is a specialized form of palliative care; however, whereas palliative care can be provided at multiple stages of illness, hospice care is reserved for patients who are estimated to have less than 6 months to live. The philosophy of hospice care is to provide comfort and control pain symptoms for patients in the last phases of incurable disease while holistically providing for the psychological, social, and spiritual needs of the patient and their loved ones. Hospice care also involves some components of home health care, which focuses on patient rehabilitation and medical management. Home health care focuses on the patient and their physical needs while hospice includes a focus on the family and also addresses emotional and spiritual needs.

Description

History

The word "hospice" stems from the Latin word "hospitium" meaning hospitality, lodging, or guesthouse. The concept of a hospice can be traced back to medieval times when it referred to a place of shelter and rest for weary or ill travelers on a long journey. There is evidence of facilities like modern hospices dating back to the 1800s. For example, Jeanne Garnier founded several hospices in France as early as 1842 and the Irish Sisters of Charity opened hospices in Ireland and England in the 1800s to provide care for dying or incurable patients. The modern hospice movement began in the 1960s when Dr. Cicely Saunders established St. Christopher's Hospice near London to provide pain management and compassionate care for the dying. The hospice movement was propelled by Dr. Elisabeth Kubler-Ross' book, *On Death and Dying*, which was published in 1969 and provided a personal glimpse into the lives of terminally ill patients. During the 1970s the Hospice movement gained attention in the United States when Kubler-Ross testified at the first national hearings on the subject of death with dignity (1972); the first hospice organization was established in the United States in New Haven, Connecticut (1974); and the first hospice legislation was introduced by Senators Frank Church and Frank E. Moss (1974). It was not until 1983 that the Medicare hospice benefit was officially introduced and in 1986 was made permanent by Congress. In the past two decades, hospice organizations have continued to evolve from volunteer-based, grassroots organizations into health care companies with paid staff and quality practices. Today there are more than 4,000 hospice programs in the United States and over one million people in the United States receive hospice services each year. Hospice organizations are also widespread internationally.

Goals and Services

People choose hospice when the disease is not responding to treatment (incurable) or when the treatment has detrimental effects on quality of life. While many hospice patients are diagnosed with cancer (approximately 40%), hospice services are also available to patients with pulmonary disease, heart disease, neurological disorders, Alzheimer's disease, AIDS, and other life-limiting illnesses. The majority of patients who receive hospice services are over the age of 65 (approximately 80%). In the United States, hospice services have historically been more widely utilized by Caucasian patients (greater than 80% of hospice patients in 2005); however, hospice utilization among ethnic and racial minorities has been increasing in recent years. On average, patients spend 69 days on the hospice service before dying (median length = 21 days). In the United States, the majority of hospice services are funded through the Medicare hospice benefit (84% of hospice stays), although private insurers and other medical programs (e.g., Veteran's Administration) offer hospice benefits.

To qualify for the Medicare hospice benefit, a physician and the hospice medical director must certify that the patient has less than 6 months to live if the disease were to run its normal course. Then the patient or a durable medical power of attorney signs a statement declaring that they would like to be admitted to hospice. By signing the statement, the patient agrees to no longer

receive curative treatment related to their terminal illness. Medicare will still pay for covered benefits for any health needs that are not related to the patient's terminal illness. A terminally ill patient may receive hospice care for as long as necessary when a physician certifies that he or she has a life expectancy of 6 months or less; however, recertification of life expectancy is required at regular intervals (approximately 60 days). At any time, patients or their family members have the right to revoke the hospice benefit and resume Medicare coverage of the benefits that were waived when hospice care was elected. The patient can reelect to return to receiving hospice coverage at a later date. The hospice agency can discharge a patient if their health improves and they no longer have a 6-month prognosis.

Hospice services can be provided in multiple settings. More than 90% of the hospice services provided in the United States are based in patients' homes. Home hospice typically requires that a family member or loved one be established as the primary caregiver and be home with the patient at all times. Members of the hospice service will have regularly scheduled visits with the patient and caregiver as well as being available 24 hours a day, 7 days a week by phone to handle emergencies or questions. Hospice services can also be provided in hospitals and nursing homes or other long-term care facilities. These services can be provided on specialized hospice units, by trained nursing staff that can care for hospice patients, or through arrangements made with independent community-based hospice services that provide care inside of the hospital or nursing home facilities. These services can be a good option for patients who want hospice care but do not have primary caregivers to take care of them at home. Finally, many communities have free-standing, independently owned hospices that feature inpatient care facilities as well as home care hospice services.

Hospice affirms the concept of palliative care as an intensive program that enhances comfort and promotes the quality of life for individuals and their families. When cure is no longer possible, hospice recognizes that a peaceful and comfortable death is an essential goal of health care. Hospice philosophy recognizes that death is a natural part of the life cycle and that people have a human right to die in comfort and with dignity. The goal of hospice is not to prolong life or hasten death, but to allow people to achieve high levels of quality of life at the end of their life. Hospice care is also holistic and aims to treat the whole person rather than just focusing on the disease. The expected outcome is relief from distressing symptoms, lower levels of pain, and/or enhanced quality of life. Hospice philosophy is a family-centered approach and aims to assist the family and loved ones with the dying process as much as the patient. Patients and family members are encouraged to be in control of decision making and treatment planning. Finally, hospice philosophy promotes the idea that palliative care should be available to all individuals and their families without regard to age, gender, nationality, race, creed, sexual orientation, disability, diagnosis, availability of a primary caregiver, or ability to pay.

To provide holistic care, hospice services are provided by an interdisciplinary team of doctors, nurses, social workers, counselors, home health aides, clergy, physical and occupational therapists, and trained volunteers among others. Hospice organizations typically become the primary care coordinators during end-of-life care. Services are provided during the last stages of illness, throughout the dying process, and to family members during the first year after the patient's death. Hospice services provide medical treatment to relieve pain and to control other physical symptoms. Patients on hospice can continue to receive palliative radiation and chemotherapy that are designed to reduce pain. Hospice services provide needed medications, medical supplies, and equipment, as well as special services such as speech therapy, physical therapy, or nutritional consultation when indicated. Hospice services also provide assistance with basic needs of daily living by providing limited home health aide and supervision/training for family members. Family coaching and family conferences are common to help patients and their family members understand the illness, how to care for the patient, and improve communication about end-of-life issues.

Hospice staff may assist the patient with unfinished legal or financial business and in making funeral arrangements. Patients who need additional care are able to receive inpatient care as well as brief periods of respite care (up to 5 days), so that caregivers can attend special events or get rest from caregiving responsibilities. Counselors and clergy provide emotional support and address the spiritual needs of patients and their family members. Family members are able to receive bereavement care up to 13 months after the patient's death, which might include counseling, supportive phone calls, referrals to community resources, support groups, and/or memorial services.

Cross-References

- ▶ End-of-Life Care
- ▶ Home Health Care
- ▶ Palliative Care

References and Readings

American Academy of Hospice and Palliative Medicine (AAHPM). Published in 2012 in Glenview, IL. Retrieved from www.aahpm.org.

Connor, S. R. (2009). *Hospice and palliative care: The essential guide* (2nd ed.). New York: Routledge.

Fine, P. G. (2008). *The hospice companion*. New York: Oxford University Press.

Forman, W. B., Kitzes, J. A., Anderson, R. P., & Sheehan, D. K. (2003). *Hospice and palliative care: Concepts and practice* (2nd ed.). Sudbury, MA: Jones and Bartlett.

International Association for Hospice and Palliative Care (IAHPC). Published in 2012 in Houston, TX. Retrieved from www.hospicecare.com

Kubler-Ross, E. (1969). *On death and dying*. New York: Scribner.

National Hospice and Palliative Care Organization (NHPCO). Published in 2012 in Alexandria, VA. Retrieved from www.nhpco.org.

Saunders, D. C., & Kastenbaum, R. (Eds.). (1997). *Hospice care on the international scene*. New York: Springer.

Hospice Care

- ▶ Hospice

Hospice Programs

- ▶ Hospice

Hospice Services

- ▶ Hospice

Hospices

- ▶ Palliative Care

Hospital Anxiety

Hollie B. Pellosmaa and Tamer F. Desouky
Department of Psychology, The University of Texas at Arlington, Arlington, TX, USA

Synonyms

Fear of hospitals; Hospital stress; Nosocomephobia; Operative anxiety

Definition

Hospital anxiety refers to the anxious response and unusual preoccupation about noxious consequences (real or not) that can result from visiting hospitals or from undergoing medical procedures in a hospital.

Description

Hospital anxiety and related concepts (e.g., "fear of hospitals" and "hospital stress") have been widely examined in studies investigating barriers to both seeking medical care (Tod, Read, Lacey, & Abbott, 2001) and blood and organ donation

(e.g., Boulware et al., 2002), and preparation for medical procedures (Johnston, 1980). These fears, if irrational, can lead individuals to avoid appropriate medical care when needed or to disregard medical instructions. Individuals with excessive fear of hospitals (i.e., nosocomephobia) often avoid hospitals which results in skipping or missing appointments scheduled at hospitals and avoid visiting friends/relatives who are hospitalized. These fears are often founded on the belief that visiting a hospital may result in serious illness and even death. Original work focused on nonspecific anxious responses, but recent work has expanded the study of "hospital anxiety" by focusing on specific triggers such as blood, needles, injuries, and medical personnel.

Early studies examined the affective responses that both adult and pediatric patients experienced in hospital settings (e.g., hospital stress). The aim of this work was to understand the effects of anxiety and stress experienced before and/or after undergoing a medical procedure on medical outcomes (e.g., slow recovery post-surgery, increased pain and physical symptoms). Pre- and postoperative anxiety was found to disturb sleep patterns (Kain & Caldwell-Andrews, 2003), reduce adherence to medical recommendations (Mathews & Ridgeway, 1981), and increase pain sensitivity (Chapman & Cox, 1977). Among children, self-report data also showed an association between preoperative anxiety and postoperative negative behaviors (e.g., eating/sleeping problems) (Melamed, Dearborn, & Hermecz, 1983).

Although it is well established that anxiety levels are indirectly and directly associated with physiological and psychological factors that influence health outcomes, the mechanisms are poorly understood (Kiecolt-Glaser, Page, Marucha, MacCallum, & Glaser, 1998). For example, psychoneuroimmunological data suggest that anxiety may delay wound healing by indirectly influencing immune function. Specifically, it has been proposed that individuals who report high levels of anxiety when undergoing medical procedures tend to experience increased pain and may require more anesthesia during surgery and more analgesics during recovery than low anxiety individuals (Maranets & Kain, 1999). This increased use of anesthesia and analgesics, in turn, can result in endocrinological and immunological changes that delay wound healing. Similarly, postoperative anxiety can delay recovery from surgical procedures by influencing adherence to medical recommendations. Existing data suggest that specific anxiety and fear (e.g., fear of pain, cardiophobia, fear of injury) and not general anxiety are the key determinants of maladaptive behaviors (Berlin et al., 1997).

Several individual and situational factors have been implicated as antecedents of hospital anxiety. Although the specific genetic contribution to hospital anxiety is currently unknown, twin studies suggest that genetic predisposition accounts for an important proportion of the variance in anxiety disorders (Hettema, Neale, & Kendler, 2001). Personal experience and learning may lead to specific anxiety/fear. For instance, needle phobia is often the result of an aversive experience with needles involving doctors and/or dentists. These fears can also be the result of watching another individual experience a negative reaction to the medical procedures (Willemsen, Chowdhury, & Briscall, 2002). Other individual factors associated with increases in hospital anxiety include sex (being female), moderate to intense levels of depressive symptoms, and a history of chronic medical conditions (Caumo et al., 2001). Adults undergoing surgical procedures also indicated that they experienced anxiety surrounding uncertainty of the outcomes of the procedure (Caumo et al.). Among situational factors, poor or lack of communication with hospital personnel and health care providers, as well as loss of autonomy and control, are considered some of the most stressful aspects of hospitalization that could lead individuals to experience anxiety (Volcier, 1974).

Assessments of hospital anxiety and related concepts can be conducted in multiple ways. Researchers often explore three main aspects of anxiety, patients' cognitions, emotional state, and situation-specific anxieties. The most commonly used self-report measures of anxiety for adults include Spielberger's State-Trait Anxiety

Inventory and the Hospital Anxiety and Depression Scale (HADS). Spielberger's State-Trait Anxiety Inventory for Children and the Children's Manifest Anxiety Scale have been widely used to assess anxiety among children. Observational tools used often include the Clinical Anxiety Scale, Yale Preoperative Anxiety Scale, and the Palmar Sweat Index. Other instruments that assess specific aspects of hospital anxiety (e.g., the Injection Phobia Scale Anxiety, Blood Injection Symptom Scale, Medical Fears Survey, and the Disgust Scale) can provide important information about the specific triggers that lead to patients' anxious responses.

Interventions to reduce hospital anxiety have a long history. Melamed and collaborators used behavioral modeling to reduce pre- and postoperative anxiety and negative behaviors among children (Melamed & Siegel, 1975). Initial work with adults focused on the reduction of pain and anxiety caused by stressful and painful medical procedures (e.g., endoscopy). Work with adults conducted by Johnson and Leventhal (1974) demonstrated that accurate sensory and procedural information successfully reduced anxiety and pain caused by medical procedures. This type of intervention has been shown to be effective across various types of medical procedures (Suls & Wan, 1989). Principles from these earlier works are at the core of more recent and innovative psychological interventions to reduce pre- and postoperative anxiety. Exposure techniques have been associated with reduction in fear of blood-injection illness in adults (Olatunji, Smits, Connolly, Willems, & Lohr, 2007); whereas behavioral therapies (e.g., modeling) can reduce anxiety levels in children with needle phobias (Willemsen et al., et al., 2002).

Cross-References

- ▶ Anxiety
- ▶ Fear and Fear Avoidance
- ▶ HADS
- ▶ Health Anxiety
- ▶ Trait Anxiety

References and Readings

Berlin, I., Bisserbe, J. C., Eiber, R., Balssa, N., Sachon, C., Bosquet, F., et al. (1997). Phobic symptoms, particularly the fear of blood and injury, are associated with poor glycemic control in type I diabetic adults. *Diabetes Care, 20*(2), 176–178. doi:10.2337/diacare.20.2.176.

Boulware, L. E., Ratner, L. E., Ness, P. M., Cooper, L. A., Campbell-Lee, S., LaVeist, T. A., et al. (2002). The contribution of sociodemographic, medical, and attitudinal factors to blood donation among the general public. *Transfusion, 42*(6), 669–678. doi:10.1046/j.1537-2995.2002.00120.x.

Caumo, W., Schmidt, A. P., Schneider, C. N., Bergmann, J., Iwamoto, C. W., Bandeira, D., et al. (2001). Risk factors for preoperative anxiety in adults. *Acta Anaesthesiologica Scandinavica, 45*(3), 298–307. doi:10.1034/j.1399-6576.2001.045003298.x.

Chapman, C. R., & Cox, G. B. (1977). Anxiety, pain, and depression surrounding elective surgery: A multivariate comparison of abdominal surgery patients with kidney donors and recipients. *Journal of Psychosomatic Research, 21*(1), 7–15. doi:10.1016/0022-3999(77)90020-4.

Hettema, J. M., Neale, M. C., & Kendler, K. S. (2001). A review and meta-analysis of the genetic epidemiology of anxiety disorders. *The American Journal of Psychiatry, 158*(10), 1568–1578. doi:10.1176/appi.ajp. 158.10.1568.

Johnson, J. E., & Leventhal, H. (1974). Effects of accurate expectations and behavioral instructions on reactions during a noxious medical examination. *Journal of Personality and Social Psychology, 29*(5), 710–718.

Johnston, M. (1980). Anxiety in surgical patients. *Psychological Medicine: A Journal of Research in Psychiatry and the Allied Sciences, 10*(1), 145–152.

Kain, Z. N., & Caldwell-Andrews, A. A. (2003). Sleeping characteristics of adults undergoing outpatient elective surgery: A cohort study. *Journal of Clinical Anesthesia, 15*(7), 505–509. doi:10.1016/j.jclinane.2003.02.002.

Kendall, P. C. (2006). *Child and adolescent therapy: Cognitive-behavioral procedures*. New York: Guilford Press.

Kiecolt-Glaser, J., Page, G., Marucha, P., MacCallum, R., & Glaser, R. (1998). Psychological influences on surgical recovery: Perspectives from psychoneuroimmunology. *American Psychologist, 53*(11), 1209–1218.

Kulik, J. A., Mahler, H. I. M., & Moore, P. J. (1996). Social comparison and affiliation under threat: Effects on recovery from major surgery. *Journal of Personality and Social Psychology, 71*(5), 967–979.

Lehrer, P. M., Woolfolk, R. L., Sime, W. E., & Barlow, D. H. (2008). *Principles and practice of stress management*. New York: Guilford Press.

Mahler, H. I. M., & Kulik, J. A. (2002). Effects of a videotape information intervention for spouses on spouse distress and patient recovery from surgery. *Health Psychology, 21*(5), 427–437.

Maranets, I., & Kain, Z. N. (1999). Preoperative anxiety and intraoperative anesthetic requirements. *Anesthesia and Analgesia, 89*(6), 1346.
Mathews, A., & Ridgeway, V. (1981). Personality and surgical recovery: A review. *British Journal of Clinical Psychology, 20*(4), 243–260.
Melamed, B. G., Dearborn, M., & Hermecz, D. A. (1983). Necessary considerations for surgery preparation: Age and previous experience. *Psychosomatic Medicine, 45*(6), 517–525.
Melamed, B. G., & Siegel, L. J. (1975). Reduction of anxiety in children facing hospitalization and surgery by use of filmed modeling. *Journal of Consulting and Clinical Psychology, 43*(4), 511–521.
Mineka, S., & Zinbarg, R. (2006). A contemporary learning theory perspective on the etiology of anxiety disorders: It's not what you thought it was. *American Psychologist, 61*(1), 10–26.
Olatunji, B., Smits, J., Connolly, K., Willems, J., & Lohr, J. (2007). Examination of the decline in fear and disgust during exposure to threat-relevant stimilu in blood-injection-injury phobia. *Journal of Anxiety Disorders, 21*(3), 445–455.
Öst, L., Hellstrom, K., & Kaver, A. (1992). One versus five sessions of exposure in the treatment of injection phobia. *Behavioral Therapy, 23*, 263–282.
Suls, J., & Wan, C. (1989). Effects of sensory and procedural information on coping with stressful medical procedures and pain: A meta-analysis. *Journal of Consulting and Clinical Psychology, 57*(3), 372–379.
Tod, A. M., Read, C., Lacey, A., & Abbott, J. (2001). Barriers to uptake of services for coronary heart disease: Qualitative study. *British Medical Journal, 323*(7306), 214.
Volcier, B. (1974). Patients' perceptions of stressful events associated with hospitalization. *Nursing Research, 23*(3), 235–238.
Willemsen, H., Chowdhury, U., & Briscall, L. (2002). Needle phobia in children: A discussion of aetiology and treatment options. *Clinical Child Psychology and Psychiatry, 7*(4), 609–619.
Wolitzky-Taylor, K. B., Horowitz, J. D., Powers, M. B., & Telch, M. J. (2008). Psychological approaches in the treatment of specific phobias: A meta-analysis. *Clinical Psychology Review, 28*(6), 1021–1037. doi:10.1016/j.cpr.2008.02.007.

Hospital Anxiety Depression Scale

Steven C. Palmer
Abramson Cancer Center, University of Pennsylvania, Philadelphia, PA, USA

Synonyms

HADS

Definition

The Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) is a 14-item measure designed to be used as a brief screen for depression (seven items) and anxiety (seven items) disorders among nonpsychiatric, medically ill, outpatient populations. As a screening instrument, the HADS does not provide definitive diagnosis, but is the first step in a multistage process of selecting individuals at elevated risk for disorder to be evaluated through clinical interview. For example, the HADS does not assess specific symptoms of major depressive disorder (e.g., suicidality, guilt) thought by its authors to be less prevalent in nonpsychiatric populations, functional impairment, or frequency of symptoms, and the time frame assesses the previous 7 days, rather than 2 weeks of mood disturbance. Moreover, five of the seven items in the Depression subscale assess anhedonia-related experiences, and there is no question specifically addressing low mood. The HADS attempts to improve specificity of measurement by reducing physical symptoms that confound physical and psychological disorder (e.g., fatigue, insomnia) and focusing more on behavioral and affective symptoms. Anxiety and Depression subscales can range in score from 0 to 21, with scores of 11 or higher indicating probable caseness for clinical disorders and scores of 8–10 being suggestive of caseness. However, numerous cut-points have been suggested in the literature, as have total rather than subscale scores, though this has not been recommended by the scale authors. The HADS has been used as a measure of symptom severity among individuals with an anxiety or depressive disorder to allow tracking of therapeutic response over time. Both Anxiety and Depression subscales have demonstrated adequate internal consistency (Cronbach's alpha $\geq$0.67 and $\leq$0.93) across numerous translated versions, and adequate sensitivity and specificity for disorder using a score of $\leq$8 (both approximately 0.80; Bjelland, Dahl, Haug, & Neckelmann, 2002), though positive predictive values have been shown to be substantially lower (e.g., 0.17 for depressive disorder) in

medical patients (Silverstone, 1994, cited in Parker & Hyett, 2010).

Cross-References

- ▶ Anxiety and Its Measurement
- ▶ Depression: Measurement
- ▶ Screening

References and Readings

Bjelland, I., Dahl, A. A., Haug, T. T., & Neckelmann, D. (2002). The validity of the hospital anxiety and depression scale. An updated literature review. *Journal of Psychosomatic Research, 62*(2), 69–77.

Parker, G., & Hyett, M. (2010). Screening for depression in medical settings: Are specific scales useful? In A. J. Mitchell & J. C. Coyne (Eds.), *Screening for depression in clinical practice: An evidence-based guide* (pp. 191–202). Oxford: Oxford University Press.

Zigmond, A. S., & Snaith, R. P. (1983). The hospital anxiety and depression scale. *Acta Psychiatrica Scandinavica, 67*(6), 361–370.

Hospital Stress

- ▶ Hospital Anxiety

Hostile Affect

- ▶ Trait Anger

Hostility

Kimberly M. Henderson, Susan A. Everson-Rose and Cari J. Clark
Department of Medicine, University of Minnesota, Minneapolis, MN, USA

Synonyms

Affective hostility; Aggression; Anger; Cynical distrust; Cynical hostility; Mistrust

Definition

Hostility is a multidimensional personality trait with distinct cognitive, affective, and behavioral features. The cognitive component of hostility is evident in the habitual patterns of cynical mistrust and negative, suspicious attitudes and beliefs that hostile individuals have toward their interpersonal network and the community at large. The affective (emotional) component reflects the internal and external expression of anger and contempt, which may vary in degrees from moderate to high. The behavioral component may manifest through mannerisms and actions that perpetuate interpersonal conflicts, such as aggression and irritability. Although distinctions can be made among the cognitive, affective, and behavioral components of hostility, they often are interrelated and co-occur.

Description

The role of hostility and anger in cardiovascular disease (CVD) and more general health and well-being has a long history. For centuries, anecdotal reports and historical observations suggested that strong negative emotions, and particularly feelings of anger, aggressive tendencies, and hostile attitudes, were related to poor health (Everson-Rose & Lewis, 2005; Gallo & Matthews, 2003). These beliefs were supported in early psychoanalytic and psychodynamic reports that identified such personality characteristics in patients with hypertension and heart disease (Everson-Rose & Lewis, 2005). In the mid-1900s, this literature and related observations prompted work on identifying and assessing "coronary-prone behavior"; indeed, hostility, anger, and aggression were identified as critical tenets of coronary-prone behavior, which later came to be redefined as Type A behavior pattern (Friedman & Rosenman, 1971). Type A individuals are described as highly ambitious, competitive, time-urgent, impatient, quick-tempered, and tightly wound. The seminal work by Drs. Rosenman and Friedman in the Western Collaborate Group Study provided clear evidence

identifying Type A behavior as a risk factor for coronary heart disease (CHD) (Rosenman et al., 1975). Indeed, this study and initial empirical confirmation of the study findings prompted the medical community to accept Type A as the first psychosocial factor clearly identified as a risk factor for CHD (Cooper, Detre, & Weiss, 1981). However, the reproducibility of these findings was limited and negative findings on Type A and CHD risk began to appear in the literature. Consequently, researchers began to consider whether one of the components of Type A was driving the adverse effects previously identified for the broader Type A construct. Strong empirical evidence suggests that hostility is the "toxic" component of Type A behavior pattern (Williams et al., 1980), and an independent predictor of CVD (Miller, Smith, Turner, Guijarro, & Hallet, 1996).

Measurement

The development of valid and robust tools for measuring hostile characteristics and traits enabled systematic investigations of the impact of hostility on health. The Cook and Medley Hostility scale (CMHS) (Cook & Medley, 1954) was derived from the MMPI nearly 60 years ago and remains one of the most commonly used measures of hostility. It originally was devised to identify teachers who had poor rapport with their students, but subsequently was adopted as a more general indicator of hostility following its reported association with angiographic evidence of CHD (Williams et al., 1980), and CVD events and mortality (for reviews, see Everson-Rose & Lewis, 2005, and Hemingway & Marmot, 1999). The CMHS consists of 50 true-false items, where 1 point is assigned for each "hostile" response and higher scores indicate greater hostility. Several subsets of items on the CMHS have been used to measure specific components of hostility, the most common among them being cynicism. Other commonly used measures include the Buss-Durkee Hostility Inventory, Multidimensional Anger Inventory, and the Potential for Hostility measure derived from the Type A Behavior Pattern structured interview. Each measure is designed to broadly assess hostility and expressions of anger; however, these scales are not completely overlapping in terms of the dimensions of hostility that they capture (e.g., cynicism, hostile affect, anger expression, anger suppression). The potential for these measures to tap into different components of hostility should be taken into consideration when relating them to health outcomes.

Hostility and Health

The literature on the relationship between hostility and health is expansive, and spans across several disciplines. Most of these observations focus on the relationship between hostility (and its components) and the risk for poor cardiovascular outcomes. The literature relating hostility to non-cardiovascular diseases remains scarce – limited evidence has tied hostile traits to cancer (Tindle, et al., 2009), cognitive function (Barnes et al., 2009), and general health (Adams, 1994). In contrast, numerous epidemiological studies have investigated hostility in relation to various indicators of CVD. Literature reviews summarizing this work (Everson-Rose & Lewis, 2005; Gallo & Matthews, 2003; Miller et al., 1996) highlight significant inconsistencies between study findings. Some of the studies reviewed found that higher levels of hostility are associated with CHD incidence, presence and progression of atherosclerosis, hypertension, peripheral arterial disease, and all-cause and CVD mortality. However, the literature is not unequivocal – other studies have reported marginal and/or null findings between hostility and risk for CHD or CVD (Hemingway & Marmot, 1999). Miller et al. (1996) conducted a meta-analysis of 45 studies relating hostility to CHD, and suggested that variations in the methodology used by these studies may account for the mixed findings in the literature, such as the type of hostility measure, the study design, and the sociodemographics of the study population. Nevertheless, Miller et al. concluded that hostility is indeed an independent predictor of CHD and all-cause mortality. More recent studies, particularly

population-based studies, provide further support for hostility as a significant risk factor for CVD (Everson-Rose & Lewis, 2005); however, among patients with documented CHD, there is limited evidence that hostility or anger predict recurrent coronary events or mortality (Hemingway & Marmot, 1999).

Sociodemographic Characteristics

Hostility is related to a number of sociodemographic characteristics, with some support for differences between groups. Consistent evidence has shown an inverse relationship between socioeconomic status (SES) and hostility. This effect is linear, and suggests that those with the lowest education, income, and occupational status are more likely to express hostile characteristics. Furthermore, there is evidence that hostility may mediate the relationship between SES and CVD (Gallo & Matthews, 2003). In addition to social and economic differences in the expression of hostility, some studies show that hostile traits may vary by race and sex. For instance, growing evidence suggests that blacks and men report higher levels of hostility than their white and female counterparts (Gallo & Matthews); other studies indicate that hostility is higher in younger adults and the elderly and lower in mid-aged adults (Barefoot, Beckham, Haney, Siegler, & Lipkus, 1993). The same study also found that education may moderate the relationship between race and hostility, with more educated blacks reporting lower levels of hostility compared to less educated blacks, and more educated whites reporting higher levels of hostility than less educated whites. Nevertheless, the current literature lacks clear evidence that differences in the expression of hostility by race or sex translate into disparities in the risk for CVD. Several studies have tested this hypothesis and found no association (Everson-Rose et al., 2006) and/or lacked the sampling power necessary to test for these interactions (Iribarren et al., 2000). Future research should include large, balanced, and diverse samples in order to further investigate potential sociodemographic differences in the risk of CVD related to hostility.

Hostility and Pathways to CVD

Hostility may confer independent risk for CVD but also may operate through indirect pathways via its relationship with a number of other psychosocial and biobehavioral risk factors. Hostility is positively correlated with negative emotional states, including depression, anger, and anxiety, and inversely associated with positive psychosocial factors and personality traits, including social support and optimism (Gallo & Matthews, 2003; Tindle et al., 2009). Additionally, a number of studies have identified behavioral risk factors related to hostility. For example, a recent meta-analysis of 27 studies found that the CMHS was significantly associated with several traditional CVD-related risk factors (Bunde & Suls, 2006), including obesity (i.e., body mass index, waist-hip ratio), alcohol consumption, and smoking. Other studies link hostility to physical inactivity and dietary/caloric intake (Scherwitz et al., 1992). Moreover, several studies have identified biological and physiological pathways that may link hostility to disease. For instance, hostility is related to metabolic dysregulation, including impaired glucose metabolism, insulin resistance, and metabolic syndrome (Bunde & Suls, 2006; Goldbacher & Matthews, 2007). Hostile attitudes, emotions, and behaviors are associated with physiological response patterns that reflect activation of several biological systems in the body. Indeed, the physiological responses induced by hostility are characteristic of the "fight or flight" response, including prolonged activation of the hypothalamic-pituitary adrenal (HPA) axis and autonomic nervous system (ANS). For example, research shows that hostile individuals tend to have higher systolic and diastolic blood pressure, higher heart rates, as well as increased levels of stress hormones (i.e., cortisol), catecholamines (i.e., epinephrine, norepinephrine), and pro-inflammatory cytokines (i.e., IL-6 and IL-1) (Everson-Rose & Lewis, 2005). Furthermore, hostility is related to increased platelet activation and aggregation, which together with the other physiological risk factors contributes to increased atherosclerosis, and thrombus and plaque

formation (Markovitz, Matthews, Kiss, & Smitherman, 1996). On the whole, these observations support the fact that hostility clusters with other psychosocial and biobehavioral factors and may increase risk for CVD and other poor health outcomes through both direct and indirect pathways.

Summary

This entry provides a general insight into the history and research relating hostility to CVD and other health outcomes. Hostility is defined by cynical and suspicious attitudes, anger, bitterness, and varying levels of aggression and opposition. The role of hostility in health emerged from the shadow of the classic Type A behavior pattern and has gained momentum as one of the most pathogenic components of Type A. Instruments created to assess hostility have been vital to measuring the impact of hostility on health, and the CMHS stands out as a valid and reliable assessment tool. The literature on the role of hostility in cardiovascular health is somewhat mixed: evidence is more limited for a role of hostility in recurrent events or mortality among persons with existing heart disease, whereas evidence from methodologically sound, population-based studies generally provides support for the hypothesis that increased hostility is associated with excess risk of CVD. Research targeted at addressing these inconsistencies has investigated a number of potential pathways and mechanisms underlying the relationship between hostility and CVD. As a result, it is becoming more apparent that the impact of hostility on health may vary based on an individual's sociodemographic profile, and that hostility may operate through other psychosocial, behavioral, and physiological factors to influence CVD outcomes.

Cross-References

- ▶ Anger, Measurement
- ▶ Hostility, Cynical
- ▶ Hostility, Measurement of
- ▶ Interpersonal Circumplex
- ▶ Negative Thoughts
- ▶ Trait Anger

References and Readings

Adams, S. H. (1994). Role of hostility in women's health during midlife: A longitudinal study. *Health Psychology, 13*, 488–495.

Barefoot, J. C., Beckham, J. C., Haney, T. L., Siegler, I. C., & Lipkus, I. M. (1993). Age difference in hostility among middle-aged and older adults. *Psychology and Aging, 8*, 3–9.

Barnes, L. L., Mendes de Leon, C. F., Bienas, J. L., Wilsons, R. S., Everson-Rose, S. A., & Evans, D. A. (2009). Hostility and change in cognitive function over time in older blacks and whites. *Psychosomatic Medicine, 17*, 652–658.

Bunde, J., & Suls, J. (2006). A qualitative analysis of the relationship between the Cook-Medley Hostility scale and traditional coronary artery disease risk factors. *Health Psychology, 25*, 493–500.

Cook, W. W., & Medley, D. M. (1954). Proposed hostility and pharisaic-virtue scales for the MMPI. *Journal of Applied Psychology, 38*, 414–418.

Cooper, T., Detre, T., & Weiss, S. M. (1981). Coronary-prone behavior and coronary heart disease: A critical review. The review panel on coronary-prone behavior and coronary heart disease. *Circulation, 63*, 1199–1215.

Everson-Rose, S. A., & Lewis, T. T. (2005). Psychosocial factors and cardiovascular diseases. *Annual Review of Public Health, 26*, 469–500.

Everson-Rose, S. A., Lewis, T. T., Karavolos, K., Matthews, K. A., Sutton-Tyrrell, K., & Powell, L. H. (2006). Cynical hostility and carotid atherosclerosis in African American and white women: The Study of Women's Health Across the Nation (SWAN) Heart Study. *American Heart Journal, 152*, 982.e7–982.e13.

Friedman, M., & Rosenman, R. H. (1971). Type A behavior pattern: Its association with coronary heart disease. *Annals of Clinical Research, 3*, 300–312.

Gallo, L. C., & Matthews, K. A. (2003). Understanding the association between socioeconomic status and physical health: Do negative emotions play a role? *Psychological Bulletin, 129*, 10–51.

Goldbacher, E. M., & Matthews, K. A. (2007). Are psychosocial characteristics related to risk of the metabolic syndrome? A review of the literature. *Annals of Behavioral Medicine, 34*, 240–252.

Hemingway, H., & Marmot, M. (1999). Psychosocial factors in the aetiology and prognosis of coronary heart disease: Systematic review of prospective cohort studies. *British Medical Journal, 318*, 1460–1467.

Iribarren, C., Sidney, S., Bild, D. E., Lui, K., Markovitz, J. H., Roseman, J. M., & Matthews, K. (2000). Association of hostility with coronary artery calcification in young adults: The CARDIA Study. *Journal of the American Medical Association, 283*, 2546–2551.

Markovitz, J. H., Matthews, K. A., Kiss, J., & Smitherman, T. C. (1996). Effects of hostility on platelet reactivity to psychological stress in coronary heart disease patients and in healthy controls. *Psychosomatic Medicine, 60*, 633–638.
Miller, T. Q., Smith, T. W., Turner, C. W., Guijarro, M. L., & Hallet, A. J. (1996). A meta-analytic review of research on hostility and physical health. *Psychology Bulletin, 119*, 322–348.
Rosenman, R. H., Brand, R. J., Jenkins, D., Friedman, M., Straus, R., & Wurm, M. (1975). Coronary heart disease in the Western Collaborative Group Study: Final follow-up experience of 8 1/2 years. *Journal of the American Medical Association, 233*, 872–877.
Scherwitz, L. L., Perkins, L. L., Chesney, M. A., Hughes, G. H., Sidney, S., & Manolio, T. A. (1992). Hostility and health behaviors in young adults: The CARDIA Study. Coronary Artery Risk Development in Young Adults Study. *American Journal of Epidemiology, 136*, 136–145.
Tindle, H. A., Chang, Y., Kuller, L. H., Manson, J. E., Robinson, J. G., & Rosal, M. C. (2009). Optimism, cynical hostility, and incident coronary heart disease and mortality in the women's health initiative. *Circulation, 120*, 656–662.
Williams, R. B., Jr., Haney, T. L., Lee, K. L., Kong, Y. H., Blumenthal, J. A., & Whalen, R. E. (1980). Type A behavior, hostility, and coronary atherosclerosis. *Psychosomatic Medicine, 42*, 539–549.

Hostility, Cynical

Susan A. Everson-Rose, Cari J. Clark and Kimberly M. Henderson
Department of Medicine, University of Minnesota, Minneapolis, MN, USA

Synonyms

Cynicism; Cynical distrust; Cynical hostility

Definition

Hostility is a relatively stable personality trait that is typically characterized as a multidimensional construct with significant affective (e.g., anger), cognitive (e.g., attitudes), and behavioral (e.g., aggression) components. Hostile individuals have a suspicious, mistrustful attitude and often disparaging view of others and generally have a cynical worldview of their environment and social interactions. Thus, this type of personality disposition is often referred to "cynical hostility."

An expansive literature on personality and disease processes and health risks has developed over the past 50–60 years. Hostility has featured prominently in this literature, particularly with regard to cardiovascular disease (CVD) risk (Miller, Smith, Turner, Guijarro, & Hallet, 1996; Everson-Rose & Lewis, 2005). Though some negative studies have been reported, on balance, the available evidence from methodologically strong, population-based studies suggests that hostility is a significant risk factor for myocardial infarction and cardiovascular mortality in healthy populations (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989; Barefoot, Dodge, Dahlstrom, Siegler, Anderson, & Williams, 1991; Everson et al., 1997). Less support has been shown for the role of hostility in recurrent CVD or mortality in patients with diagnosed heart disease (Hemingway & Marmot, 1999). Recent studies in older adult populations have looked at hostility in relation to other health outcomes. Accruing evidence shows that hostility levels are associated with worse metabolic function and glucose regulation (Niaura, Todaro, Stroud, Spiro, Ward, & Weiss, 2002; Shen, Countryman, Spiro, & Niaura, 2008), higher levels of inflammation (Graham et al., 2006), poorer lung function (Kubzansky, Sparrow, Jackson, Cohen, Weiss, & Wright, 2006), and lower levels of cognitive function (Barnes et al., 2009).

Cross-References

▶ Anger, Measurement
▶ Hostility, Measurement of

References and Readings

Barefoot, J. C., Dodge, K. A., Dahlstrom, W. G., Siegler, I. C., Anderson, N. B., & Williams, R. B., Jr. (1991). Hostility patterns and health implications: Correlates of Cook-Medley Hostility Scale scores in a national survey contact and ability to predict survival. *Health Psychology, 10*, 18–24.

Barefoot, J. C., Dodge, K. A., Peterson, B. L., Dahlstrom, W. G., & Williams, R. B., Jr. (1989). The Cook-Medley hostility scale: Item contact and ability to predict survival. *Psychosomatic Medicine, 51*, 46–57.

Barnes, L. L., Mendes de Leon, C. F., Bienias, J. L., Wilson, R. S., Everson-Rose, S. A., & Evans, D. A. (2009). Hostility and change in cognitive functions over time in older blacks and whites. *Psychosomatic Medicine, 71*, 652–658.

Everson, S. A., Kauhanen, J., Kaplan, G. A., Goldberg, D. E., Julkunen, J., Tuomilehto, J., et al. (1997). Hostility and increased risk of mortality and acute myocardial infarction: The mediating role of behavioral risk factors. *American Journal of Epidemiology, 146*, 142–152.

Everson-Rose, S. A., & Lewis, T. T. (2005). Psychosocial factors and cardiovascular diseases. *Annual Review of Public Health, 26*, 469–500.

Graham, J. E., Robles, T. F., Kiecolt-Glaser, J. K., Malarkey, W. B., Bissell, M. G., & Glaser, R. (2006). Hostility and pain are related to inflammation in older adults. *Brain, Behavior, and Immunity, 20*, 389–400.

Hemingway, H., & Marmot, M. (1999). Evidence based cardiology: Psychosocial factors in the aetiology and prognosis of coronary heart disease. Systematic review of prospective cohort studies. *British Medical Journal, 318*, 1460–1467.

Kubzansky, L. D., Sparrow, D., Jackson, B., Cohen, S., Weiss, S. T., & Wright, R. J. (2006). Angry breathing: A prospective study of hostility and lung function in the Normative Aging Study. *Thorax, 61*, 863–868.

Miller, T. Q., Smith, T. W., Turner, C. W., Guijarro, M. L., & Hallet, A. J. (1996). A metaanalytic review of research on hostility and physical health. *Psychological Bulletin, 119*, 322–348.

Niaura, R., Todaro, J. F., Stroud, L., Spiro, A., 3rd, Ward, K. D., & Weiss, S. (2002). Hostility, the metabolic syndrome, and incident coronary heart disease. *Health Psychology, 21*, 588–593.

Shen, B. J., Countryman, A. J., Spiro, A., 3rd, & Niaura, R. (2008). The prospective contribution of hostility characteristics to high fasting glucose levels: The moderating role of marital status. *Diabetes Care, 31*, 1293–1298.

Hostility, Measurement of

Susan A. Everson-Rose, Cari J. Clark and Kimberly M. Henderson
Department of Medicine, University of Minnesota, Minneapolis, MN, USA

Synonyms

Cynical distrust; Cynical hostility; Cynicism

Definition

Hostility is defined by a suspicious, mistrustful attitude and cynical disposition toward others. Considered an enduring personality characteristic, hostility is characterized by cognitive, behavioral, and affective or emotional dimensions.

Description

Measurement of hostility can be based on structured interviews, with interviewer ratings of behavioral dimensions and verbal expressions of hostility based on participants' actions and responses within the interview setting. The classic example of this method of assessing hostility is the structured interview for Type A behavior, which was developed by Drs. Meyer Friedman and Ray Rosenman, the two cardiologists who first coined the term "Type A" to describe what they perceived to be coronary-prone behavior (e.g., hostility, aggressiveness, time-urgency, and a need to be hard-driving) among their heart patients (Friedman & Rosenman, 1971). The Type A interview is administered by carefully trained interviewers using a structured format in which scenarios are presented that attempt to elicit behavioral responses consistent with Type A. For example, at varying points during the interview, the interviewer will interrupt the respondent, challenge his or her responses, abruptly change topics, and also appear to be distracted. Interviews are audiotaped for later scoring. A scoring system is used that characterizes respondents on "Potential for Hostility" (PH), among other Type A characteristics, and codes on three dimensions of hostility, including intensity of response, hostile content, and style of interaction (Dembroski & Costa, 1987). As empirical support for the Type A construct in relation to risk for coronary heart disease faltered, investigators shifted focus to what were considered the most toxic components of Type A, and in particular, emphasis was placed on hostility, anger, and aggressiveness. Despite the seeming advantages of having objective ratings of hostile behaviors made by trained observers, such as PH

ratings obtained within the context of the Type A interview, such ratings are uncommon in the current literature on personality and health. Indeed, structured or semi-structured interview formats are rarely used anymore, especially in large community- or population-based studies of health because of the considerable time and expense involved in conducting such interviews. In contrast, a vast majority of such studies typically rely on self-administered or interviewer-administered questionnaires or surveys (i.e., "self-report") to assess hostility and other personality characteristics. Among the most commonly used measures of hostility is the 50-item Cook-Medley Hostility Scale (CMHS; Cook & Medley, 1954), which was derived from the original Minnesota Multiphasic Personality Inventory (Hathaway & McKinley, 1940). Items include such statements as "It is safer to trust nobody," "Most people make friends because friends are likely to be useful to them," "I think most people lie to get ahead," and "Most people are honest chiefly through fear of being caught." Several shortened versions of the CMHS are commonly used as well; most of these versions focus on a subset of 8–13 items that specifically assess interpersonal attitudes characterized by cynicism, mistrust of others' intentions, and anger responses (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989; Greenglass & Julkunen, 1989; Everson et al., 1997). The original CMHS employed a "true/false" response format with 1 point assigned for each "true" response and a hostility score was then calculated by summing across items. Alternate response formats have been used with some of the shortened versions of the CMHS, including a 4-point Likert scale in which respondents are asked to indicate the extent to which they agree or disagree with each statement from completely agree (0) to completely disagree (3). With this format, item responses are reverse-coded and summed to create a hostility score.

Other validated self-report instruments used to assess hostility include the Buss-Durkee Hostility Inventory (BDHI; Buss & Durkee, 1957) and the Multidimensional Anger Inventory (Siegel, 1986). The BDHI includes 75 true/false items; though the items were written to assess seven aspects of hostility, as defined by the creators of the scale, studies show that responses generally reflect two broad categories related to overt verbal and physical expressions of anger and hostility and to angry or hostile thoughts and emotions. The BDHI commonly has been used by psychologists but has rarely been used in epidemiological studies assessing personality or psychosocial factors and health outcomes, largely due to its length and the availability of several shortened versions of the CMHS. Similarly, the Multidimensional Anger Inventory consists of 25 self-report items that tap into four dimensions, only one of which that is specific to hostility and the remainder which are more specific to anger. The dimensions assessed include hostile outlook, mode of anger expression (anger-in and anger-out), anger arousal, and range of anger-eliciting situations. This scale was developed in a convenience sample of college students and though also validated in a community sample of male factory workers, it has not been used widely in population- or community-based studies.

Cross-References

- ▶ Anger, Measurement
- ▶ Hostility, Cynical

References and Readings

Barefoot, J. C., Dodge, K. A., Peterson, B. L., Dahlstrom, W. G., & Williams, R. B., Jr. (1989). The Cook-Medley hostility scale: Item content and ability to predict survival. *Psychosomatic Medicine, 51*, 46–57.

Buss, A. H., & Durkee, A. (1957). An inventory for assessing different kinds of hostility. *Journal of Consulting and Clinical Psychology, 21*, 343–349.

Cook, W. W., & Medley, D. M. (1954). Proposed hostility and pharisaic virtue scales for the MMPI. *Journal of Applied Psychology, 38*, 414–418.

Dembroski, T. M., & Costa, P. T., Jr. (1987). Coronary prone behavior: Components of the type A pattern and hostility. *Journal of Personality, 55*, 211–235.

Everson, S. A., Kauhanen, J., Kaplan, G. A., Goldberg, D. E., Julkunen, J., Tuomilehto, J., & Salonen, J. T. (1997). Hostility and increased risk of mortality and acute myocardial infarction: The mediating role of behavioral risk factors. *American Journal of Epidemiology, 146*, 142–152.

Friedman, M., & Rosenman, R. H. (1971). Type A behaviour pattern: Its association with coronary heart disease. *Annals of Clinical Research, 3*, 300–312.
Greenglass, E. R., & Julkunen, J. (1989). Construct validity and sex differences in Cook-Medley hostility. *Personality and Individual Differences, 10*, 209–218.
Hathaway, S. R., & McKinley, J. C. (1940). A multiphasic personality schedule (Minnesota): I. Construction of the schedule. *Journal of Psychology, 10*, 249–254.
Siegel, J. M. (1986). The multidimensional anger inventory. *Journal of Personality and Social Psychology, 51*, 191–200.

Hostility, Psychophysiological Responses

Kimberly M. Henderson, Susan A. Everson-Rose and Cari J. Clark
Department of Medicine, University of Minnesota, Minneapolis, MN, USA

Synonyms

Aggression; Anger; Antagonism; Cynical distrust; Cynical hostility; Cynicism

Definition

Hostility has distinct cognitive components, affective or emotional components, and behavioral features. This multidimensional construct most commonly is defined by a mistrustful and suspicious attitude, cynical perceptions of others and their motives, and a negative interactional style characterized by anger, resentment, contempt, antagonism, and suspiciousness. Behavioral expressions of hostility typically include verbally and/or physically aggressive actions. The physiological consequences of hostility and its components contribute to over-activation of neurochemical and biological pathways, consistent with the stress response system, that may contribute to atherogenesis, alterations in glucose metabolism and other bodily systems, and which are harmful to cardiovascular health.

Description

A wealth of research has investigated physiologic responses related to hostility (Everson-Rose & Lewis, 2005). The emotional, behavioral, and cognitive manifestations of hostility are interrelated, although they may operate via different biologic pathways to influence health. This may well be why the literature on hostility's impact on physiological functioning reveals effects on multiple biologic systems, including documented effects on heart rate (HR), heart rate variability (HRV), systolic and diastolic blood pressure (SBP; DBP). One of the well-tested hypotheses that was formulated nearly 30 years ago to understand the linkages of psychosocial risk factors, such as Type A behavior, with coronary heart disease, focused on individual differences in physiologic responding. What came to commonly be called the cardiovascular reactivity hypothesis (Treiber et al., 2003; Smith & Ruiz, 2002) postulated that exaggerated autonomic nervous system and/or neuroendocrine activation occurred under conditions of interpersonal stress in persons with particular psychological traits or under certain emotional states. Hostility is one such psychological trait that has been a focus of the cardiovascular reactivity literature. Although not unequivocal (Suls & Wan, 1993), the vast majority of studies testing this hypothesis with regard to hostility show that, indeed, hostile persons tend to have higher HR (Jamner, Shapiro, Goldstein, & Hug, 1991), reduced HRV (Sloan et al., 1994), higher SBP and DBP (Smith & Allred, 1989; Suarez & Blumenthal, 1991), increased levels of epinephrine and norepinephrine (Zhang et al., 2006), and alterations in cortisol secretion or patterning (Pope & Smith, 1991) particularly under conditions of challenge and/or in ambiguous conditions, which are thought to be interpreted as adversarial by hostile persons (Everson, McKey, & Lovallo, 1995). Furthermore, prolonged and frequent physiological arousal experienced by hostile individuals increase the risk for endothelial injury and damage, a precursor of inflammation, platelet formation, thrombogenesis, and atherogenesis. Nonetheless, the literature on hostility and

risk for cardiovascular disease morbidity and mortality has not clearly shown that this exaggerated cardiovascular reactivity evident among hostile individuals is in fact the primary pathway by which hostility increases cardiovascular risk.

Other biologic pathways are important and also have been associated with hostility. For example, several studies have linked hostility with altered glucose metabolism, central adiposity, and metabolic dysregulation, which additionally may serve as mediators between hostility and CVD and related risk factors. In a recent review of the literature, Goldbacher and Matthews (2007) concluded that negative psychosocial factors, including depression, hostility, and anger, were important contributors to the risk of metabolic syndrome. Specifically in regards to studies investigating the role of hostility on metabolic syndrome, Goldbacher and Matthews found that hostility and its components (i.e., cynicism, aggression) related to increased risk of metabolic syndrome over time, and found longitudinal and cross-sectional evidence supporting the positive association between hostility and visceral adipose tissue, waist-hip ratio, and insulin. For example, one of the studies reported from Niaura and colleagues (2000) found that men high in hostility, as assessed by the Cook-Medley Hostility Scale, have increased insulin resistance and higher fasting insulin levels in the presence of high stress levels, and impaired glucose metabolism than men who are low in hostility. Moreover, there is evidence that ties the combined effects of high hostility and metabolic syndrome to increased risk of myocardial infarction (Todaro et al., 2005). Even so, the review of the literature revealed studies that find no relationship between hostility and metabolic syndrome (Goldbacher & Matthews, 2007), and although steps have been taken to address and conceptualize some of these inconsistencies (Zhang et al., 2006), this work is limited.

Studies also have examined neuroendocrine and genetic biomarkers related to hostility, especially those involved in serotonergic function. Williams (1994) proposed that reduced serotonin levels in the central nervous system may constitute an important brain mechanism by which hostility and related biobehavioral risk characteristics influence risk for cardiovascular diseases. In his hypothesis, he suggested that serotonin dysfunction was the underlying cause of harmful traits (e.g., hostility, anger), behaviors (e.g., excessive alcohol use, smoking), and physiological responses (e.g., heightened sympathetic arousal) which seem to cluster in certain individuals. Justification for his theory is supported by a growing number of studies that identify genetic markers and environmental stimuli that regulate serotonin levels and shape the expression of hostile traits and behaviors. Indeed, more recent studies have provided scientific evidence that individuals with alleles that code for lower CNS serotonin function are more likely to express hostile-like traits (Manuck, Flory, Ferrel, Mann, & Muldoon, 2000; Lesch et al., 1996), including aggression and neuroticism, and other negative psychosocial characteristics like depression (Siegler et al., 2008). In addition, there is some evidence that these genes may vary by race and/or sex; however, this work is limited and requires further investigation. More work is needed to understand the development of hostility and potential biomarkers that may predispose individuals to hostile traits and characteristics.

Hostility is a multidimensional personality trait that functions through several biological pathways to influence health and well-being. The expression of hostile attitudes, emotions, and behaviors are stress inducing, and particularly cardio-reactive. Indeed, the body uses autonomic and neuroendocrine pathways to interpret hostile experiences, which may manifest as heightened cardiovascular stimulation or metabolic dysregulation. In addition, the tendency for hostility to cluster with other negative psychosocial factors and harmful behaviors, led to the growing theory that low CNS serotonin function may predispose individuals to these disease-inducing traits and behaviors. In order to fully conceptualize the mechanisms that shape the relationship between hostility and disease, further research will be required that supports physiological mediators.

Cross-References

- ▶ Anger, Measurement
- ▶ Hostility, Cynical
- ▶ Hostility, Measurement of

References and Readings

Everson, S. A., Mckey, B. S. & Lovallo, W. R. (1995). Effects of trait hostility on cardiovascular response to harasment in young men. *International Journal of Behavioral Medicine*, *2*, 172–191.

Everson-Rose, S. A., & Lewis, T. T. (2005). Psychosocial factors and cardiovascular diseases. *Annual Review of Public Health, 26*, 469–500.

Goldbacher, E. M., & Matthews, K. A. (2007). Are psychosocial characteristics related to risk of the metabolic syndrome? A review of the literature. *Annals of Behavioral Medicine, 34*, 240–252.

Jamner, L. D., Shapiro, D., Goldstein, I. B., & Hug, R. (1991). Ambulatory blood pressure and heart rate in paramedics: Effects of cynical hostility and defensiveness. *Psychosomatic Medicine, 53*, 393–406.

Lesch, K. P., Bengel, D., Heils, A., Sbol, S. Z., Greenberg, B. P., Petri, S., et al. (1996). Association of anxiety-related traits with polymorphism in the serotonin transporter gene regulatory region. *Science, 274*, 1527–1531.

Manuck, S. B., Flory, J. D., Ferrel, R. E., Mann, J., & Muldoon, M. F. (2000). A regulatory polymorphism of the monoamine oxidase-A gene may be associated with variability in aggression, impulsivity, and central nervous system serotonergic responsivity. *Journal of Psychiatric Research, 95*, 9–23.

Niaura, R., Banks, S. M., Ward, K. D., Stoney, C. M., Spiro, A., Aldwin, C. M., et al. (2000). Hostility and the metabolic syndrome in older males: The Normative Aging Study. *Psychosomatic Medicine, 62*, 7–16.

Pope, M. L., & Smith, T. W. (1991). Cortisol excretion in high and low cynically hostile men. *Psychosomatic Medicine, 53*, 386–392.

Siegler, I. C., Helms, M. J., Kuhn, C. M., Surwit, R. S., James, S. A., Williams, R. B., Costa, P. T., & Brummett, B. H. (2008). Personality association of high brain serotonin in men. *American Psychosomatic Meeting Abstract, 2008*(70), A79.

Sloan, R. P., Shapiro, P. A., Bigger, J. T., Bagiella, E., Steinman, R. C., & Gorman, J. M. (1994). Cardiac autonomic control and hostility in healthy subjects. *The American Journal of Cardiology, 74*, 298–300.

Smith, T. W., & Allred, K. D. (1989). Blood-pressure responses during social interaction in high- and low-cynically hostile males. *Journal of Behavioral Medicine, 12*, 135–143.

Smith, T. W., & Ruiz, J. M. (2002) Psychosocial influences on the development and course of coronary heart disease: current status and implications for research and practice. *Journal of Consulting and Clinical Psychology*, *70*, 548–568.

Suarez, E. C., & Blumenthal, J. A. (1991). Ambulatory blood pressure responses during life in high and low hostile patients with a recent myocardial infarction. *Journal of Cardiopulmonary Rehabilitation, 11*, 169–175.

Suarez, E. C., Kuhn, C. M., Schanberg, S. M., Williams, R. B., & Zimmermann, E. A. (1998). Neuroendocrine, cardiovascular, and emotional responses of hostile men: The role of interpersonal challenge. *Psychosomatic Medicine, 60*, 78–88.

Suls, J., & Wan, C. K. (1993). The relationship between trait hostility and cardiovascular reactivity: A quantitative review and analysis. *Psychophysiology, 30*, 615–626.

Todaro, J. F., Con, A., Niaura, R., Spiro, A. I. I. I., Ward, K. D., et al. (2005). Combined effect of the metabolif syndrome and hostility on the incidence of myocardial infarction (The Normative Aging Study). *The American Journal of Cardiology, 15*, 221–226.

Treiber, F. A., Kamarck, T., Schneiderman, N., Sheffield, D., Kapuku, G., & Taylor, T. (2003). Cardiovascular reactivity and development of preclinical and clinical disease states. *Psychosomatic Medicine, 65*, 46–62.

Williams, R. B. (1994). Neurobiology, cellular and molecular biology, and psychosomatic medicine. *Psychosomatic Medicine, 56*, 308–315.

Zhang, J., Niaura, R., Dyer, J. R., Shen, B., Todaro, J. F., McCaffery, J. M., et al. (2006). Hostility and urine norepinephrine interact to predict insulin resistance: The VA Normative Aging Study. *Psychosomatic Medicine, 68*, 718–726.

Household Income

- ▶ Family, Income

HPA Axis Negative Feedback Testing

- ▶ Dexamethasone Suppression Test

HPA Axis Stimulation Tests

- ▶ Pharmacological Stress Tests

HPV

▶ Human Papillomavirus (HPV)

HR_{max}

▶ Maximal Exercise Heart Rate

$HSCH_2CH_2CH(NH_2)CO_2H$

▶ Homocysteine

HSV-1

▶ Genital Herpes

HSV-2

▶ Genital Herpes

Human Factors/Ergonomics

Jonathan Z. Bakdash[1,2,3] and Frank A. Drews[1,2]
[1]Department of Psychology, University of Utah, Salt Lake City, UT, USA
[2]Center for Human Factors in Patient Safety, VA Salt Lake City Health Care System, Salt Lake City, UT, USA
[3]U.S. Army Research Laboratory, Aberdeen Proving Ground, MD, USA

Synonyms

Applied cognitive psychology; Applied experimental psychology; Engineering psychology; Ergonomics

Definition

Human factors/ergonomics can be broadly defined as optimizing the relationships between systems and the humans using them. More specifically, it is defined as follows:

"...the understanding of interactions among humans and other elements of a system, and the profession that applies theory, principles, data, and other methods to design in order to optimize human well-being and overall system performance" (Human Factors and Ergonomics Society: About HFES, n d).

In health care, the goal of human factors is to improve patient outcomes by supporting provider performance and by increasing patient safety through the reduction of iatrogenic error. In the context of behavioral medicine, the specific goal of human factors is to promote effective clinical interventions for disease prevention and treatment and to facilitate positive changes in patient behavior. The application of human factors to health care is discussed in more detail below.

Description

In the USA, an estimated 98,000 patients die each year due to preventable medical errors (Institute Of Medicine, 1999). Generally, health care has lagged far behind other industries, notably aviation, for improvements in safety (Leape, 1994). However, some specialties in medicine (e.g., anesthesiology) have made tremendous progress in patient safety over the last decades. For example, by the late 1990s, the risk of death from anesthesia was 1/60th the mortality rate in the 1980s, which translates into a 60-fold decrease in the number of annual deaths in the USA (Kohn, Corrigan, Donaldson, & Institute of Medicine U.S. Committee on Quality of Health Care in America, 2000). This phenomenal accomplishment in anesthesiology patient safety resulted from a combination of changes, most based on the application of human factors principles. Among the changes that increased anesthesia safety were steps toward optimization and standardization of system components (equipment

and training), establishment and implementation of practice guidelines which were widely accepted by providers, hospitals, and professional organizations, and significant advances in technology (Gaba, 2000). In contrast to anesthesiology, other areas in health care have not made as much progress in patient safety. For example, approximately half to two-thirds of all annual patient deaths in the USA are caused by a preventable systemic infection that is associated with a central line (a catheter that is used to draw blood and deliver fluids and medication) placed in a patient (Institute of Medicine, 1999).

A human factors approach that pursues the goal of improving patient safety focuses not only on individual components of the system in isolation, but also takes into account the interactions of these elements with each other. Only such an integrative perspective will result in sustainable change of clinical practice and improvements in patient outcomes and patient safety.

Socio-Natural Systems Perspective of Health Care

Comprehensively addressing patient safety requires considering human performance in a range of different systems while acknowledging the differences between such systems (Durso & Drews, 2010). The safety improvements in anesthesiology were successful because human factors principles were applied to multiple elements (optimization and standardization of equipment and tasks with practice guidelines adopted by providers and organizations), widespread implementation of simulator-based training with feedback, and patient monitoring technology was improved (Gaba, 2000). This combination of interventions improved the transparency of patient outcomes through feedback with better equipment and structured training and education.

Clearly, all of the multiple system components (patients, providers, physical environment, organizational environment, tasks, and equipment/tools; adapted from Carayon, Alvarado, & Hundt, 2007) that are involved in health care need to be included in a comprehensive systemic intervention. In addition, to maximize improvements in performance and safety, it is important to acknowledge the differences and similarities between different systems (e.g., technical systems in aviation vs. socio-natural systems in health care).

For example, health care can be conceptualized by applying a socio-natural systems perspective: patients are, at the core, biological or natural systems, interacting with providers in an organizational environment that constitutes a social system, where often equipment/tools are used to accomplish a goal with the equipment/tools constituting the technical aspect of the system (Durso & Drews, 2010). Interventions to improve safety in aviation and health care are frequently compared, and there are important similarities between the two systems that allow adoption. However, the comparison faces limitations when focusing on a key distinction between the relevant system components in each domain at the microlevel: in health care, tasks are performed on a patient, a natural system. Consequently, feedback on a task, such as treating a patient, is often delayed or not accessible because causality is not directly observable. At the microlevel, tasks in aviation are performed on an aircraft, an engineered system that is designed to provide continuous feedback, making it largely transparent and highly predictable (Durso & Drews, 2010).

In health care, transparency (observation and understanding of a system) of the system component (patients) is partial, at best. One challenge is that the lack of transparency limits the opportunities for learning and reduces the ability to predict the consequences of actions. Another difference between health care and aviation can be found in the approach toward training and distinction in microlevel variability between patients and aircraft. Due to the lack of standardization in health care (Timmermans & Berg, 2003), providers are unlikely to receive detailed training and practice with particular equipment and frequently need to perform the same procedure using different brands of equipment with distinct designs. Whereas in aviation, pilots train and become certified on a specific aircraft, thus they do not encounter differences between aircraft. Furthermore, tasks in aviation are highly structured and therefore predictable, with

checklists (protocols) for routine activities and emergency situations (Wickens, Gordon, & Liu,, 1998). In health care, protocols for every possible situation and patient simply do not exist. Thus, providers perform tasks based on their training, local practices, and the specific needs of patients, because the inter-individual variability of patients is significant. This situation can be contrasted with aviation, where it is one of the most important goals of quality control in aircraft manufacturing to minimize the variability between aircraft.

Other differences between health care and aviation extend to the physical and organizational environment. The work environment in health care has the potential to negatively impact cognition because of frequent error-producing conditions: interruptions, multitasking, suboptimal equipment, and inadequate staffing (Carayon et al., 2007; Nolan, 2000). The work environment in health care sharply contrasts with aviation, where during critical phases of a flight (e.g., takeoff, landing), nonessential conversations in the cockpit are prohibited (sterile cockpit), minimizing interruptions and distractions. Finally, health care is only beginning to establish a safety culture such as that successfully established in aviation. A consequence of this early stage of safety culture adoption is that errors and "near misses" generally go unreported (Leape, 1994), limiting the opportunities for learning because reoccurring patterns are not identified.

To improve performance in a complex system, components such as equipment and training need to be optimized and standardized by applying human factors principles (Nolan, 2000, Wickens et al., 1998). Specifically, this involves reducing the cognitive demands of tasks that are performed. For example, high reliance on perception, memory, attention, and decision-making when performing a task is likely to increase the probability of suboptimal actions and do not promote optimal performance (Wickens et al.). Consequently, reducing the cognitive demand of a task can provide defenses against human error (Wickens et al.). To illustrate the application of human factors in health care, two examples will be discussed in more detail: the development of a drug display (a visual monitor) to improve medication delivery in anesthesiology and tools to improve medication adherence.

Drug Display for Medication Delivery

Delivering correct drug concentrations and doses over the course of administering anesthesia is critical to patient safety. This task has significant cognitive demands because the anesthesiologist must remember the times and amounts of past drug administrations, then use this recalled information to calculate changes in concentration over time, and finally, determine the difference between the calculated current concentrations and the appropriate levels to determine present dosages. The consequences of high cognitive demands are ample opportunities for errors.

Consequently, anesthesia drug delivery performance can be improved with a drug display consistent with human factors principles. Drews, Syroid, Agutter, Strayer, and Westenskow (2006) demonstrated the positive impact of such technology optimized by application of human factors by comparing the display to the standard approach of delivering anesthesia without such information available to the anesthesiologist. The success of this approach can be attributed to the fact that the drug display creates defenses against error by providing "knowledge in the world" which reduces demands on cognition (Norman, 2002). More specifically, the human factors principles that were implemented in the drug display are as follows (Norman, 2002; Wickens et al., 1998):

1. Affordances (perceived functions of objects: intuitive representations of medication doses over time)
2. Chunking (meaningful grouping of related items: different drugs separated, current dose levels for all drugs together)
3. Reduced effort (mental/physical task demands to perform actions: information is provided on the monitor rather than requiring cognition)
4. Structure/guidance (design, layout, and sequence: e.g., time in Western cultures goes from left to right, chunking: information in meaningful units that can be maintained in memory)

5. Visual feedback (visibility: graphical representation depicting patterns of change over time for past, current, and predicted future drug concentration levels)

The drug display is an example of the application of human factors principles to equipment design (or tool design) to increase clinical task performance and reduce human error. However, equipment and tasks are only two of many additional components that are part of health care. Macroergonomics emphasizes such a broad system-based perspective when planning interventions for improvement in performance.

Medication Adherence Tools

A socio-natural systems perspective can be applied to a problem that is highly relevant for behavioral medicine: medication adherence. Lack of medication compliance has negative effects on patient health and produces significant economic costs (Osterberg & Blaschke, 2005). There are a number of causes that can lead to nonadherence, for example, the patient does not understand how to weigh medication benefits compared to the side effects, high access costs (high purchase costs of the medication, difficulty accessing a pharmacy, and other cognitive barriers such as challenges in the complexity of coordinating the timing and dosing of a number of medications) (Osterberg & Blaschke, 2005).

One human factors approach to improve medication adherence is providing cognitive aids or tools to schedule times to take medications (Waicekauskas et al., 2010). Participants scheduled the times to take several medications using a paper or electronic daily calendar, working in pairs (one participant was the "patient" and the other participant was the "provider"). Together the dyads solved examples of scheduling simple and complex sample medication problems. Complexity was increased by adding scheduling constraints, for example, fewer possible times to take a medication because of routine activities on the calendar, requiring the medication to be taken on an empty stomach. Both, the paper and electronic versions of the tool provided support to creating more accurate medication schedules than no aid.

The medication scheduling tool may serve as an example for how human factors approaches can help develop effective cognitive tools that reduce memory and other cognitive demands in coordinating the administration of medications and may also lead to increases in compliance with medication scheduling. However, such a cognitive aid is a technical system component that will likely only raise adherence if the causes for noncompliance were either forgetting to take medication or difficulty in figuring out when and how to take medication. The limitation of this approach is clearly associated with the fact that no improvement in medication compliance can be expected for patients that do not understand how to weigh the benefits of medication versus the medication side effects. For example, blood pressure medication has significant long-term health benefits, but the benefits are not directly visible to most patients, and there are frequent negative side effects (e.g., Osterberg & Blaschke, 2005). The tradeoff between the potentially invisible or less salient therapeutic benefits of medication and noticeable side effects from the perspective of the patient illustrates the challenges associated with a natural system that lacks transparency.

Nevertheless, the efficacy of a medication scheduling tool may be further enhanced by addressing other factors that contribute to noncompliance. For example, a clinical decision support system (a system designed to improve quality of care by providing cognitive support) could be implemented in electronic medical records or paper and applied to the problem of medication compliance (e.g., Drews et al., 2010). Implementing such a support system could prompt providers to: (a) remind patients about benefits versus side effects, (b) discuss medication cost and possible alternatives, and (c) provide pharmacy locations, etc. Although applying human factors to a single system can improve patient care, the example of medication compliance demonstrates both the challenges that are associated with applying an integrative human factors approach to health care and the greater benefits to patients that come with the application of this comprehensive approach.

Cross-References

- ▶ Adherence
- ▶ Cognitions
- ▶ Risk Perception

References and Readings

Carayon, P., Alvarado, C. J., & Hundt, A. S. (2007). Work design and patient safety. *Theoretical Issues in Ergonomics Science, 8*(5), 395–428.

Drews, F. A., Syroid, N., Agutter, J., Strayer, D. L., & Westenskow, D. R. (2006). Drug delivery as control task: Improving performance in a common anesthetic task. *Human Factors, 48*(1), 85–94.

Drews, F., Weir, C., Nebeker, J., Hollnagel, E., Mallin, B., & Barrus, R. (June 2010). *A theoretical perspective to develop a medication management system*. Poster session presented at AHRQ Annual Health IT Grantee and Contractor Meeting, Washington, DC

Durso, F. T., & Drews, F. A. (2010). Health care, aviation, and ecosystems: A socio-natural systems perspective. *Current Directions in Psychological Science, 19*(2), 71–75.

Gaba, D. M. (2000). Anaesthesiology as a model for patient safety in health care. *British Medical Journal, 320*(7237), 785–788.

Human Factors and Ergonomics Society: About HFES. (n.d.). Retrieved February 18, 2011 from http://www.hfes.org/web/AboutHFES/about.html

Institute of Medicine. (1999). *To err is human: Building a safer health system*. Washington, DC: National Academies Press.

Kohn, L. T., Corrigan, J., Donaldson, M. S., & Institute of Medicine U.S. Committee on Quality of Health Care in America. (2000). *To err is human: Building a safer health system* (Vol. 6). Washington, DC: National Academies Press.

Leape, L. L. (1994). Error in medicine. *Journal of the American Medical Association, 272*(23), 1851–1857.

Nolan, T. W. (2000). System changes to improve patient safety. *British Medical Journal, 320*(7237), 771–773.

Norman, D. A. (2002). *The design of everyday things*. New York: Basic Books.

Osterberg, L., & Blaschke, T. (2005). Adherence to medication. *The New England Journal of Medicine, 353*(5), 487–497.

Timmermans, S., & Berg, M. (2003). *The gold standard: The challenge of evidence-based medicine and standardization in healthcare*. Philadelphia: Temple University Press.

Waicekauskas, K. T., Kannampallil, T. G., Kopren, K., Tan, P., Fu, W., & Morrow, D. G. (2010, September). Collaborative tools in a simulated patient-provider medication scheduling task. In *Proceeding s of the Human Factors and Ergonomics Society Annual Meeting*, 1936–1940.

Wickens, C. D., Gordon, S. E., & Liu, Y. (1998). *An introduction to human factors engineering*. New York: Wesley.

Human Genome Project

Rany M. Salem[1] and Laura Rodriguez-Murillo[2]
[1]Broad Institute, Cambridge, MA, USA
[2]Department of Psychiatry, Columbia University Medical Center, New York, NY, USA

Definition

The Human Genome Project (HGP) was an international scientific research project to map and sequence the entire human genome. The project goals were to identify all 20,000–25,000 genes and determine the sequences of the three billion base pairs that make up the haploid human genome. The project was started in 1990 and coordinated by the US Department of Energy and National Institutes of Health, and in partnership with the Wellcome Trust (UK). In 1998, Celera Corporation launched a parallel private effort to sequence the human genome. Most of the government-sponsored sequencing was performed in universities and research centers from the USA, the UK, Japan, France, Germany, and China. The public and private efforts succeeded, with a draft sequence published in 2001 (International Human Genome Sequencing Consortium, 2001; Venter et al., 2001), and completion of the HGP in April 2003 (Collins, Green, Guttmacher, & Guyer, 2003; Collins, Morgan, & Patrinos, 2003; Frazier, Johnson, Thomassen, Oliver, & Patrinos, 2003). The HGP also included parallel efforts to sequence select model organisms, such as the bacterium *Escherichia coli*, the fruit fly *Drosophila melanogaster*, and the mouse (*Mus musculus*). Sequencing model organisms served to both help to develop the technology and interpret human gene function. Data from the project are freely available to researchers.

Cross-References

- ▶ DNA
- ▶ Gene

References and Readings

Collins, F. S., Green, E. D., Guttmacher, A. E., & Guyer, M. S. (2003). A vision for the future of genomics research. *Nature, 422*(6934), 835–847.

Collins, F. S., Morgan, M., & Patrinos, A. (2003). The human genome project: Lessons from large-scale biology. *Science, 300*(5617), 286–290.

Frazier, M. E., Johnson, G. M., Thomassen, D. G., Oliver, C. E., & Patrinos, A. (2003). Realizing the potential of the genome revolution: The genomes to life program. *Science, 300*(5617), 290–293.

International Human Genome Sequencing Consortium. (2001). Initial sequencing and analysis of the human genome. *Nature, 409*(6822), 860–921. doi:10.1038/35057062.

Lewis, R. (2005). *Human genetics. Concepts and applications* (7th ed.). Boston: McGraw-Hill Science/Engineering/Math.

Strachan, T., & Read, A. P. (2003). *Human molecular genetics* (3rd ed.). London/New York: Garland Science/Taylor & Francis Group.

Venter, J. C., Adams, M. D., Myers, E. W., Li, P. W., Mural, R. J., Sutton, G. G., et al. (2001). The sequence of the human genome. *Science, 291*(5507), 1304–1351.

Human Herpesvirus-4 (HHV-4)

- ▶ Epstein-Barr Virus

Human Immunodeficiency Virus (HIV)

- ▶ AIDS: Acquired Immunodeficiency Syndrome

Human Papillomavirus (HPV)

Luis I. García
Center for AIDS Intervention Research, Medical College of Wisconsin, Milwaukee, WI, USA

Synonyms

HPV

Definition

The human papillomaviruses (HPVs) are members of the Papillomaviridae. These viruses (Papillomaviridae) are specie specific, so they can only infect humans. They are non-enveloped viruses that carry their genetic information on circular, double-stranded DNA. The virus measures approximately 55 nm in diameter. Nearly 200 types of HPV have been identified. While many of them do not produce any symptoms in humans, some strains of the virus can cause warts and in a minority of cases can lead to cancer (Bonnez & Reichman, 2009; Giuliano et al., 2011).

Description

HPV tends to infect skin or mucosal tissue, and while many HPV subtypes do not cause any symptoms, the subtypes that are known to produce symptoms can cause a multitude of skin diseases (e.g., plantar warts, common warts, epidermodysplasia verruciformis, genital warts, cervical and anal carcinoma). Each virus subtype tends to be associated with a specific pathology, for example, plantar warts tend to be caused by HPV-1 or HPV-2, while cervical carcinoma is frequently caused by HPV-16 and HPV-18. However, other HPV subtypes may cause the same diseases, although less commonly (Bonnez & Reichman, 2009).

HPV is easily recognized when it causes symptoms (i.e., clinically apparent infections); however, subclinical and asymptomatic infections are more common. In addition, individuals with past HPV infections represent an even larger group of affected persons. These three types of infections (i.e., clinical, asymptomatic, and past infections) and their relationship to each other make difficult their epidemiological study (Bonnez & Reichman, 2009). HPV is also classified by the location of the infection. For example, a significant distinction is made between infections in genitals and mucosal tissue, and non-genital infections (cutaneous infections).

In the United States, 20 million people are currently estimated to be infected and contagious

(Bonnez & Reichman, 2009), while an additional 6.2 million people between the ages of 15 and 44 become infected with genital HPV annually (Richman, Whitley, & Hayden, 2009). HPV transmission requires skin to skin contact; therefore, most genital infections are transmitted through sexual activity (Giuliano et al., 2011; Richman et al., 2009). Anal HPV infections happen most frequently through anal intercourse, although it is possible to spread the infection from one area to another by contact with hands or objects that have been exposed to infected areas (Giuliano et al., 2011). Nonsexual infections are rare. Most HPV infections (70%) resolve within a year, up to 90% within 2 years. However, individuals who do not clear their HPV infection are at risk for cervical or anal cancer, though the progression to cancer can take 10–20 years (Giuliano et al., 2011).

The three most observed cutaneous manifestations of HPV are *common warts* (71% of all cutaneous infections), *plantar warts* (34%), and *juvenile or flat warts* (4%). Common warts and flat warts are more common in children, while plantar warts are more common in adolescents. The prevalence of common warts in school-age children is estimated to be 4–20% (Bonnez & Reichman, 2009). Since contact with infected skin cells is necessary for transmission, two other vehicles of transmission besides sexual contact are possible, though rare: mother-to-child transmission and exposure to contaminated objects (Giuliano et al., 2011). Infection during birth is rare, but may lead to juvenile-onset recurrent respiratory papillomatosis (JORRP) – an infection that results in warty growths in the upper respiratory tract and can lead to obstruction of the airway in severe cases. JORRP has an incidence of 2 per 100,000 children in the United States (Giuliano et al., 2011). The other uncommon route of transmission is through contact with an object that has residual infected skin cells or with infected hands with a common wart or carrying infected cells (e.g., after scratching) (Giuliano et al., 2011). This means of transmission is more likely if there is trauma to the receiving skin, which may explain the higher frequency of cutaneous warts seen among meat handlers (Bonnez & Reichman, 2009).

Currently, there is no treatment to specifically eradicate an HPV infection; infections have to be cleared by the immune system of the infected person. Treatment of HPV focuses on removing the lesions by chemical or physical means, or by local stimulation of cytokines, to reduce or eliminate the symptoms of the infection (Bonnez & Reichman, 2009). There are two potential methods of preventing HPV transmission. Avoiding skin exposure to infected areas can reduce the likelihood of transmission. However, since HPV can be asymptomatic or subclinical (i.e., an infected individual may not have any visible skin lesions), avoiding contact with infected skin can be difficult or impossible. Condoms provide only limited protection from HPV since they protect only skin that is covered by the condom (Giuliano et al., 2011). A second method of prevention is vaccination against HPV infection, which has been proven to be quite effective (Bonnez & Reichman, 2009; The FUTURE II Study Group, 2007; Trotter & Franco, 2006). The available HPV vaccinations target only up to four HPV subtypes. However, since these HPV subtypes cause the majority of cervical cancers and genital warts, the vaccines can prevent the vast majority of clinically important and cosmetically unpleasant HPV infections (Bonnez & Reichman, 2009).

Cross-References

- ▶ Infectious Diseases
- ▶ Immune Function

References and Readings

Bonnez, W., & Reichman, R. C. (2009). Papillomaviruses. In G. L. Mandell, J. E. Bennett, & R. Dolin (Eds.), *Mandell, Douglas, and Bennett's principles & practice of infectious diseases*. Philadelphia, PA: Churchill Livingstone.

Giuliano, A. R., et al. (2011). Efficacy of quadrivalent HPV vaccine against HPV infection and disease in males. *New England Journal of Medicine, 364*, 401–411.

Richman, D. D., Whitley, R. J., & Hayden, F. G. (2009). *Clinical virology* (3rd ed.). Washington, DC: ASM Press.

The FUTURE II Study Group. (2007). Quadrivalent vaccine against human papillomavirus to prevent high-grade cervical lesions. *New England Journal of Medicine, 256*, 1915–1927.

Trottier, H., & Franco, E. L. (2006). The epidemiology of genital human papillomavirus infection. *Vaccine*, *24* (Suppl. 1), S4–S15.

Human Subject Protections

▶ Protection of Human Subjects

Human Subjects Committee

Lynnee Roane
School of Nursing, University of Maryland, Baltimore, MD, USA

Synonyms

Institutional review board (IRB); Research ethics committee

Definition

Ethical conduct of research requires an objective and prospective review and approval by a committee established for the purpose of protecting human subjects involved in research. In the United States, these human subjects committees are usually called Institutional Review Boards (IRBs). They may also be known as Ethics Review Committees.

The main responsibility of the IRB is to conduct a comprehensive scientific and ethical review of the planned research activities. The purpose of this review is to ensure the proposed research meets the federal requirements for ethical research. It determines study feasibility, scientific merit, ethical soundness, strength of study design, balance of risk and benefits, and adequacy of informed consent. The review ensures that the design is scientifically sound and does not expose subjects to unnecessary risk. A sound scientific design is essential, as it is not ethically justifiable to expose subjects to any risk, discomfort, or inconvenience if the research is poorly designed and not likely to obtain meaningful information (Council for International Organizations of Medical Sciences, 1993). The IRB is also responsible for identifying, minimizing, and eliminating conflicts of interests.

Key components of the system include the federal government's regulations, IRBs, the sponsor, and the principal investigators (PIs) conducting the research. Additionally, all investigators and members of the research team and community are charged with protecting human subjects. The federal regulations, issued by the Department of Health and Human Services (DHHS) and the Food and Drug Administration (FDA), are the cornerstone in this system of protection. The DHHS regulates all research it funds and conducts, and the FDA regulates research involving drugs, biologics, and clinical devices.

The criteria for IRB approval are that risks to research participants are minimized; risks are reasonable in relation to anticipated benefits and the knowledge that may result; selection of participants is equitable; informed consent will be sought from each participant or their legally authorized representative and appropriately documented, as required; adequate plans for monitoring data to ensure participant safety are in place; adequate provisions are made to protect the privacy of the participant and the confidentiality of the data; and additional safeguards are provided to protect the welfare and safety of vulnerable participants such as children, prisoners, pregnant women, mentally disabled persons, or economically or educationally disadvantaged individuals (U. S. Department of Health and Human Services, 2009a).

IRBs are responsible for conducting comprehensive prospective and ongoing review of all research activities at least annually. The level of scrutiny and frequency of continuing review depends upon the level of risk posed by the research activities. To best protect human

subjects, an objective review is required by a diverse group of individuals with no direct involvement in the planned research. The Federal Regulations stipulate that the IRB is composed of at least five members. The members must include a scientist, nonscientist, and at least one person who is not affiliated, and is not a family member of an affiliated person, with the institution conducting the research (U. S. Department of Health and Human Services, 2009b). These are the minimal requirements established by the federal guidelines; however, the size and composition may vary depending on the institutional requirements and resources. On occasion, it may be necessary for the IRB to invite an individual to provide expertise in a specific area of research.

The responsible and ethical conduct of research requires that the PI and all team members are appropriately trained and well qualified to conduct research with humans. Responsible for the conduct of the study, PIs must consider the participants' safety and welfare at every phase of the study including initiation, implementation, closeout, and dissemination. Fundamental in providing these protections is ensuring subject privacy and confidentiality (Steneck, 2007). Conducting research in a manner consistent with the established ethical principles is of paramount importance to protect human participants in research. Providing adequate protections requires a broad range of knowledge and a systems approach. Such a system is essential to accomplish comprehensive reviews of planned research activities and to ensure sound ethical interactions between the investigator and participant, especially in the informed consent process. Also important in the system of protection is oversight, including safety monitoring and a robust quality improvement and compliance program (Federman, Hanna, & Rodriguez, 2002).

The Belmont Report and the Protection of Human Subjects

Human subjects who participate in research benefit society by contributing to the development of new drugs, medical procedures, and a greater understanding of how we think and act (Steneck, 2007). Individuals who assume the inherent risks of participating in research deserve to be treated with dignity and respect and to have their rights as humans protected. The federal regulations just discussed are founded upon ethical principles written in the Belmont report. The ethical principles of respect for persons, beneficence, and justice are tightly integrated in the regulations. The principle of respect for persons states that the protection of autonomy and personal dignity requires informed consent of each person before they are involved in research. Special protections, to prevent coercion and undue influence, are required for individuals who are unable to make autonomous decisions, such as children, prisoners, and those with compromised decision-making capacity. Securing the well-being of research participants by minimizing risk and maximizing benefits is essential to ensuring beneficence and protecting subjects from harm. Justice requires equitable subject selection to reduce or prevent overburdening certain populations and providing access to research opportunities for others (The Belmont Report, 1979). While the federal government provides the overarching governance for research in humans, oversight and implementation of the regulations are conducted by the IRB.

Cross-References

- ▶ Ethics Committee
- ▶ Human Subjects Committee
- ▶ Informed Consent
- ▶ Institutional Review Board (IRB)

References and Readings

Council for International Organizations of Medical Sciences. (1993). *International ethical guidelines for biomedical research involving human subjects*. Geneva, Switzerland: CIOMS.

Department of Health Education and Welfare, The National Commission for the Protection of Human Subjects of Biomedical and Behavioral Research. (1979). The Belmont report ethical principles and

guidelines for the protection of human subjects of research. Retrieved June 30, 2011, from http://ohsr.od.nih.gov.proxy-hs.researchport.umd.edu/guidelines/belmont.html.

Federman, D., Hanna, K., & Rodriguez, L. (2002). A systematic approach to human research participant protection programs. In Federman, D., Hanna, K., Rodriguez, L. (Eds.), Responsible research: A systems approach to protecting research participants (pp. 45–69) The National Academies Press. Retrieved June 28, 2011, from http://www.nap.edu.proxy-hs.researchport.umd.edu/openbook.php?record_id=10508page=45.

Steneck, N. H. (2007). The protection of human subjects. In ORI introduction to the responsible conduct of research (pp. 37–49) Department of Health and Human Services.

U.S. Department of Health and Human Services. Code of Federal Regulations 45 CFR 46. 111. Protection of Human Subjects. (2009a). *Criteria for IRB approval of research*. Retrieved from http://www.hhs.gov/ohrp/humansubjects/guidance/45cfr46.html#46.111

U.S. Department of Health and Human Services. Code of Federal Regulations 45 CFR 46.107. Protection of Human Subjects. (2009b). *IRB membership*. Retrieved from http://www.hhs.gov/ohrp/humansubjects/guidance/45cfr46.html#46.107

Human Subjects Protections

► Protection of Human Subjects

Hybridoma Growth Factor

► Interleukins, -1 (IL-1), -6 (IL-6), -18 (IL-18)

Hybridoma Plasmacytoma Growth Factor

► Interleukins, -1 (IL-1), -6 (IL-6), -18 (IL-18)

Hypercholesterolemia

► Dyslipidemia

Hyperglycemia

Michael James Coons
Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Synonyms

Control; Glycemia; Load-high

Definition

Hyperglycemia is defined as a state of elevated blood plasma glucose ($\geq$7.0 mmol/L, or 120 mg/dL, before meals) and is a hallmark feature of all diabetes subtypes. Hyperglycemia results from disruptions in blood glucose metabolism, insufficient insulin production (in the case of type 1 diabetes), or the cellular resistance to the endogenous insulin produced by the pancreas (in the cases of type 2 diabetes and gestational diabetes). Hyperglycemia can be assessed at home using a portable glucometer, or in the laboratory setting by drawing a blood sample for analysis. Symptoms include excessive thirst, excessive urination, fatigue, itchy skin, and, over time, weight loss. In mild forms, individuals are often unaware of blood glucose elevations. However, in more flagrant episodes, individuals may self-detect these aforementioned symptoms. Hyperglycemia may be managed by a number of self-management behaviors including changes in dietary intake and physical activity. Among individuals with diabetes who are receiving insulin therapy, they may also administer an injection of short-acting insulin. These behaviors, in isolation or combination, will help to restore normal blood glucose levels. However, if unmanaged, extreme hyperglycemia can precipitate a state of diabetic ketoacidosis (DKA), which results in both the production of ketones and metabolic acidosis. DKA leads to excessive urination and loss of both fluid and electrolytes that can result in myocardial infarction and death. Repeated episodes of DKA may be evidence

of poor metabolic control, which potentiates morbidity and premature mortality. Precipitants for DKA include infection, abdominal crises (e.g., gastrointestinal bleeding, pancreatitis), physical trauma, and insulin omission. If unmanaged, repeated episodes of hyperglycemia lead to the development of serious vascular pathology resulting in blindness, renal failure, pain and loss of sensation in the extremities, myocardial infarctions, cerebrovascular accidents, and amputations.

Cross-References

- ▶ Diabetes
- ▶ Glucose
- ▶ Glucose: Levels, Control, Intolerance, and Metabolism
- ▶ Glycemia
- ▶ Glycemia: Control, Load-High

References and Readings

Barrett, E. J., & Nadler, J. L. (2002). Non-insulin dependent diabetes mellitus. In G. M. Besser & M. O. Thorner (Eds.), *Comprehensive clinical endocrinology* (pp. 303–318). London: Elsevier Science.

Booth, G. L. (2001). Short-term clinical consequences of diabetes in adults. In H. G. Gerstein & R. B. Haynes (Eds.), *Evidence-based diabetes care* (pp. 68–106). Hamilton, ON: BC Decker.

Nadler, J. L., McDuffie, M., & Kirk, S. E. (2002). Insulin-dependent diabetes mellitus. In G. M. Besser & M. O. Thorner (Eds.), *Comprehensive clinical endocrinology* (pp. 267–290). London: Elsevier Science.

Hyperinsulinemia

Alyssa Parker
UTSW Health Systems, South western Medical Center, Dallas, TX, USA

Synonyms

Insulin Resistance; Metabolic syndrome X; Prediabetes

Definition

Hyperinsulinemia is an indication of an underlying problem controlling blood glucose levels and is characterized by an excess of insulin circulating in the blood (Shanik et al. 2008). Insulin is produced by the pancreas and helps regulate blood glucose. Hyperinsulinemia may be caused by insulin resistance, a condition in which an individual's body becomes resistant to the effects of insulin and the pancreas compensates by producing additional insulin. Increases in insulin result in a decrease in circulating blood glucose (Buse, Polonsky, & Burant, 2008). Symptoms of hyperinsulinemia are often absent unless hypoglycemia (abnormally low glucose levels) occurs. When present, symptoms may include temporary muscle weakness, brain fog, fatigue, visual problems, headaches, shaking, and/or thirst. Treatment of hyperinsulinemia is directed at the underlying problem and typically focuses on diet and exercise (Diabetes.co.uk Team, 2011). Dieticians recommend a nutritional regimen that is low in sugar and carbohydrates, while high in protein. Additionally, regular monitoring of weight, blood glucose, and insulin is advised. In some cases, typically where obesity is present, treatment with metformin (i.e., an oral anti-diabetic drug) may be used to reduce insulin levels (Glueck, 2007). Though it is often mistaken for diabetes or hypoglycemia, hyperinsulinemia is a separate condition. Left unmonitored and untreated, however, it can develop into type 2 diabetes mellitus. A significant risk factor of hyperinsulinemia is the increased likelihood of developing a cluster of closely related abnormalities known as the metabolic syndrome (Reaven, 2005). Within this syndrome, it is believed that the more insulin resistant a person, the more likely he or she will develop some degree of glucose intolerance, high triacylglycerol and low HDL concentrations, essential hypertension, and procoagulant and proinflammatory states (Reaven, 2006), all of which are known to increase the chance of cardiovascular disease.

Cross-References

- Blood Glucose
- Diabetes
- Glucose: Levels, Control, Intolerance, and Metabolism
- Hypoglycemia
- Insulin
- Insulin Resistance (IR) Syndrome
- Metabolic Syndrome
- Type 2 Diabetes Mellitus

References and Readings

Buse, J. B., Polonsky, K. S., & Burant, C. F. (2008). Type 2 diabetes mellitus. In H. M. Kronenberg, S. Melmed, K. S. Polonsky, & P. R. Larsen (Eds.), *Williams textbook of endocrinology* (11th ed., pp. 1329–1389). Philadelphia: Elsevier Science.

Diabetes.co.uk Team. (2011). *Hyperinsulinemia.* Retrieved from http://www.diabetes.co.uk/hyperinsulinemia.html

Glueck, C. H. (2007). *Insulin resistance and hyperinsulinemia*. Retrieved from http://www.jewishhospitalcincinnati.com/cholesterol/Research/insulin_resistance.html

Reaven, G. M. (2005). The insulin resitance syndrome: Definition and dietary approaches to treatment. *Annual Review of Nutrition, 25*, 391–406.

Reaven, G. M. (2006). The metabolic syndrome: Is this diagnosis necessary? *American Journal of Clinical Nutrition, 83*(6), 1237–1247.

Shanik, M. H., Xu, Y., Skrha, J., Dankner, R., Zick, Y., & Roth, J. (2008). Insulin resistance and hyperinsulinemia: Is hyperinsulinemia the cart or the horse? *Diabetes Care, 31*(2), S262–S268.

Hyperlipidemia

Kelly Flannery
School of Nursing, University of Maryland Baltimore, Baltimore, MD, USA

Synonyms

Lipid disorder

Definition

Hyperlipidemia simply means high blood lipid levels. Lipids refer to cholesterol, cholesterol compounds, triglycerides, and phospholipids. Lipids are carried in the blood by lipoproteins (e.g., low-density lipoproteins (LDL) and high-density lipoproteins (HDL)) (American Heart Association, n.d.).

Description

Hyperlipidemia increases the risk for atherosclerosis (i.e., hardening of the arteries, which is a buildup of plaque in the arteries). Atherosclerosis leads to narrow and stiff arteries that provide a reduced blood flow (Society for Vascular Surgery, 2010) and ultimately an increased risk for high blood pressure, heart disease, stroke, and poor lower leg circulation (Society for Vascular Surgery; U.S. National Library of Medicine & National Institutes of Health, 2011a, 2011b).

Risk Factors

Hyperlipidemia has several risk factors and many are modifiable. Some risk factors include (Mayo Clinic, 2010b; Society for Vascular Surgery, 2010; U.S. National Library of Medicine & National Institutes of Health, 2011b):

- Having certain genetic disorders; for example, familial hypercholesterolemia or familial dysbetalipoproteinemia
- Having a (nuclear) family history of early heart disease (before age 55)
- Age (men >45, women >55)
- Having certain diseases such as diabetes, high blood pressure, kidney disease, hypothyroidism, Cushing syndrome, polycystic ovary syndrome
- Having a body mass index over 25 (also defined as being overweight or obese)
- Drinking alcohol excessively
- Eating diets high in unhealthy fats (i.e., saturated and trans fat)
- Eating diets high in dietary cholesterol
- Lack of exercise

Hyperlipidemia, Table 1 Cholesterol screening summary

	Why the test is important	Desired or optimal value[a]	Some possible modifiable reasons why value is not at desired or optimal value
Total cholesterol	Tells the total amount of cholesterol in blood	<200 mg/dL	Being overweight
			Eating a diet high in saturated and/or trans fats
LDL (bad) cholesterol	When LDL cholesterol is too high, plaque can form in the arteries, which can increase risk for cardiovascular disease	<100 mg/dL	Being overweight
			Eating a diet high in saturated and/or trans fats
HDL (good) cholesterol	High levels can be protective whereas low levels (<40 mg/dL for men and <50 mg/dL for women) are a significant risk factor for heart disease. HDLs also help remove LDL cholesterol	≥60 mg/dL	Smoking cigarettes
			Being inactive
			Being overweight
			Eating a diet high in trans fats
Triglycerides	Unused calories are converted to triglycerides (fat that is circulating in the blood before it is stored)	<150 mg/dL	Eating more calories than working off
			Eating too many sweets
			Drinking alcohol excessively
			Being overweight
			Having diabetes with elevated blood sugar

[a]These are general guidelines and results should be discussed with primary care provider; the table was compiled with information from American Heart Association 2010, 2011; Centers for Disease Control and Prevention, 2010; Mayo Clinic, 2011; National Heart, Lung and Blood Institute, n.d.; U.S. National Library of Medicine & National Cancer Institute, 2011

- Using certain medications such as estrogen, birth control pills, corticosteroids, beta blockers, some types of diuretics, and some types of antidepressants
- Smoking cigarettes

Screening

Hyperlipidemia causes no symptoms; therefore, screening is important (Mayo Clinic, 2010c). To screen for hyperlipidemia a blood sample is obtained and a lipid panel (e.g., total cholesterol, LDL, HDH, triglycerides) is done (Mayo Clinic, 2011). Table 1 summarizes why the test is done, the desired or optimal value, and some possible modifiable reasons why the test is not at the desired or optimal value (American Heart Association 2010, 2011; Centers for Disease Control and Prevention, 2010; Mayo Clinic, 2011; National Heart, Lung and Blood Institute, n.d.; U.S. National Library of Medicine & National Cancer Institute, 2011).

Screening Recommendations

Recommendations for when hyperlipidemia screening should begin differ across organizations but generally testing is recommended to begin for men between ages 20–35 and 20–45 for women and then checked every 5 years. Additionally, screening is recommended when someone develops certain diseases (e.g., diabetes). Guidelines for cholesterol screening for children and/or adolescents vary due to limited evidence. However, some experts say those under age 18 should be screened if risk factors are present (e.g., family history of high cholesterol) while some recommend screening all children (U.S. National Library of Medicine & National Institutes of Health, 2011a).

Treatment

There are two common treatment methods for addressing an abnormal lipid panel. Sometimes only a lifestyle change is needed. A lifestyle

change is often the first step in treating hyperlipidemia. However, if a lifestyle change is not effective (especially if LDLs are still elevated), a combination of lifestyle modification and cholesterol medication may be recommended (Society for Vascular Surgery, 2010).

Possible lifestyle recommendations (Centers for Disease Control and Prevention, 2011; Mayo Clinic, 2010a; Society for Vascular Surgery, 2010; U.S. National Library of Medicine & National Cancer Institute, 2011):

- Eating the recommended amount of fruits and vegetables
- Limiting saturated (e.g., butter, cheese, whole milk) and trans (e.g., processed foods, fried foods, commercially baked foods) fats and replacing them with monounsaturated (e.g., canola oil, olive oil, avocados) and polyunsaturated (e.g., canola oil, walnuts, salmon) fats
- Limiting dietary cholesterol intake
- Eating whole grains
- Engaging in regular exercise
- Losing weight (if overweight)
- Quit smoking cigarettes
- Limiting alcohol consumption

There are five major classes of cholesterol lowering medication, which include: (Agency for Healthcare Research and Quality, 2009)

- Vitamins and supplements
- Statins
- Bile ace binders
- Cholesterol absorption inhibitor
- Fibrates

Cross-References

- ▶ Cholesterol
- ▶ Dyslipidemia
- ▶ Lipid Abnormalities
- ▶ Lipid Metabolism
- ▶ Plasma Lipid

References and Readings

Agency for Healthcare Research and Quality. (2009). *Treating high cholesterol: A guide for adults*. Retrieved April 15, 2011, from http://www.effectivehealthcare.ahrq.gov/index.cfm/search-for-guides-reviews-and-reports/?pageaction=displayproduct&productID=351

American Heart Association. (2010). *Levels of cholesterol*. Retrieved April 15, 2011, from http://www.heart.org/HEARTORG/GettingHealthy/FatsAndOils/Fats101/Levels-of-Cholesterol_UCM_305051_Article.jsp

American Heart Association. (2011). *What your cholesterol levels mean*. Retrieved April 15, 2011, from http://www.heart.org/HEARTORG/Conditions/Cholesterol/AboutCholesterol/What-Your-Cholesterol-Levels-Mean_UCM_305562_Article.jsp

American Heart Association. (n.d.). *Hyperlipidemia*. Retrieved April 15, 2011, from http://www.americanheart.org/presenter.jhtml?identifier=4600

Centers for Disease Control and Prevention. (2010). *Nutrition for everyone: Trans fat*. Retrieved April 15, 2011, from http://www.cdc.gov/nutrition/everyone/basics/fat/transfat.html

Centers for Disease Control and Prevention. (2011). *Nutrition for everyone: Polyunsaturated fats and monounsaturated fats*. Retrieved April 15, 2011, from http://www.cdc.gov/nutrition/everyone/basics/fat/unsaturatedfat.html

Mayo Clinic. (2010a). *High cholesterol: Lifestyle and home remedies*. Retrieved April 15, 2011, from http://www.mayoclinic.com/health/high-blood-cholesterol/DS00178/DSECTION=lifestyle-and-home-remedies

Mayo Clinic. (2010b). *High cholesterol: Risk factors*. Retrieved April 15, 2011, from http://www.mayoclinic.com/health/high-blood-cholesterol/DS00178/DSECTION=risk-factors

Mayo Clinic. (2010c). *High cholesterol: Symptoms*. Retrieved April 15, 2011, from http://www.mayoclinic.com/health/high-blood-cholesterol/DS00178/DSECTION=symptoms

Mayo Clinic. (2011). *Cholesterol test: Why it's done*. Retrieved April 15, 2011, from http://www.mayoclinic.com/health/cholesterol-test/MY00500/DSECTION=why-its-done

National Heart, Lung and Blood Institute. (n.d.). *What causes high blood cholesterol?* Retrieved April 15, 2011, from http://www.nhlbi.nih.gov/health/dci/Diseases/Hbc/HBC_Causes.html

Society for Vascular Surgery. (2010). *Hyperlipidemia*. Retrieved April 15, 2011, from https://www.vascularweb.org/vascularhealth/Pages/Hyperlipidemia.aspx

U.S. National Library of Medicine, & National Cancer Institute. (2011). *Dietary fats explained*. Retrieved April 15, 2011, from http://www.nlm.nih.gov/medlineplus/ency/patientinstructions/000104.htm

U.S. National Library of Medicine, & National Institutes of Health. (2011a). *Coronary risk profile*. Retrieved April 15, 2011, from http://www.nlm.nih.gov/medlineplus/ency/article/003491.htm

U.S. National Library of Medicine, & National Institutes of Health. (2011b). *High blood cholesterol and triglycerides*. Retrieved April 15, 2011, from http://www.nlm.nih.gov/medlineplus/ency/article/000403.htm

Hypertension

Douglas Carroll
School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

High blood pressure

Definition

Hypertension has been variously defined over the years. However, systolic (i.e., peak) bloodpressure ≥140 mmHg and diastolic blood pressure ≥90 mmHg are now considered indicative of hypertension and of meriting treatment. Blood pressure ca 120/80 mmHg is deemed to be normal (normotensive). Although hypertension may be a secondary consequence of problems elsewhere, e.g., renal failure, the vast majority of cases of hypertension have no proximal and discrete cause. This is called essential hypertension and is a major focus of research in behavioral medicine. This is easy to understand given the prevalence and health consequences of hypertension. It is estimated to affect 15% and 20 % of the adult population in Western countries, with worldwide prevalence being around 10%. Concern with hypertension also reflects its association with coronary heart disease and stroke; the results of many studies testify that as blood pressure rises, so life expectancy decreases. For example, at the outset of the famous Framingham study, some 5,000 of the citizens of that small community in the USA had their blood pressures recorded; around 20% of them were hypertensive. Those with hypertension were three times more likely to go on to have a heart attack and eight times more likely to have a stroke. Given the clinical importance of hypertension, it is perhaps hardly surprising that there are several dedicated scientific journals and many texts devoted to hypertension. My favorite, however, remains Beevers and MacGregor (1995).

Cross-References

- ▶ Blood Pressure, Measurement of
- ▶ Blood Pressure
- ▶ Hypertrophy

References and Readings

Beevers, D. G., & MacGregor, G. A. (1995). *Hypertension in practice* (2nd ed.). London: Martin Dunitz.

Hypertriglyceridemia

- ▶ Dyslipidemia

Hypertrophy

Leah Rosenberg
Department of Medicine, School of Medicine, Duke University, Durham, NC, USA

Synonyms

Cardiovascular disease; Hypertension

Definition

Hypertrophy is a structural response of an anatomic chamber, particularly the right and left cardiac ventricles, to chronically increased volumes or pressures. Left ventricular hypertrophy has been shown to be independently predictive of coronary heart disease onset and is associated with common conditions such as hypertension (high blood pressure) and cardiac valvular dysfunction (Maron, Ridker, Grundy, & Pearson, 2010). While hypertrophy is initially an adaptive mechanism for the heart, the chronic enlargement leads to unsustainable pressures and deformation that impedes contraction and relaxation. Serial

measurements of hypertrophy can be helpful in primary and secondary prevention for cardiovascular events.

References and Readings

Maron, D. J., Ridker, P. M., Grundy, S. M., & Pearson, T. A. (2010) Chapter 51. Preventive strategies for coronary heart disease (Chapter). Fuster, V., O'Rourke, R. A., Walsh, R. A., Poole-Wilson, P., (Eds.) King, S. B., Roberts, R., Nash, I. S., & Prystowsky, E. N., Assoc. (Eds.), *Hurst's the heart* (12, ed).

Hypochondriasis

Molly S. Clark[1], Katherine T. Fortenberry[2] and Kate L. Jansen[1]
[1]Department of Family Medicine, University of Mississippi Medical Center, Jackson, MS, USA
[2]Department of Family and Preventative Medicine, The University of Utah, Salt Lake City, UT, USA

Synonyms

Health anxiety

Definition

Hypochondriasis is defined by the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR) (American Psychiatric, 2000), as preoccupation with fear of having or the belief that one has a serious health condition. Criteria for the diagnosis include that the belief: is often based on the misreading of physical symptoms, is not of intensity better described as delusional, leads to clinically significant impairment in functioning, is of 6 months duration or longer, and is not better accounted for by a medical or psychiatric disorder. A specifier of "with poor insight" may be added to the diagnosis if the individual does not have recognition that the fears of illness are unwarranted and unfounded.

Description

Epidemiology

The prevalence of hypochondriasis is estimated to be between 1% and 5% in the general population. However, the prevalence rates increase to 2–7% within primary care outpatient populations, which is not unexpected given that this population is likely to seek treatment (American Psychiatric, 2000). Hypochondriasis may manifest at any age, but occurrences are commonly noted in early adulthood, with no gender differences noted. The course of hypochondriasis is typically described as chronic in greater than 50% of cases, and likelihood of chronicity is increased by when the individual experiences a number of bodily sensations, believes that one has a serious illness, or has other comorbid psychiatric diagnoses (Taylor & Asmundson, 2006). Cross-cultural differences have been noted among patients diagnosed with hypochondriasis; (Taylor & Asmundson; Asmundson, Taylor, & Sevgur, 2001) for example, gastrointestinal sensations are predominant in the United Kingdom, pulmonary complaints are common in Germany, and concerns surrounding immunology and chemical sensitivities frequently occur in the USA and Canada (Taylor & Asmundson, 2006).

Evaluation

Essential features of an evaluation of hypochondriasis include a thorough medical examination to rule out any potential physiological explanation for the reported symptoms (Taylor & Asmundson, 2006). Following this evaluation, a detailed psychiatric interview should be conducted and include any recent psychosocial stressors and previous history of health-related difficulties of the patient, family member, or significant others. Interviewers should consider in the evaluation that hypochondriasis typically manifests in the forms of disease phobia, bodily preoccupation, and disease conviction. For example, a patient may present with the belief that they have cancer (disease conviction) or may have a preoccupation with gastrointestinal complaints (bodily preoccupation) and insists on laboratory tests and radiological imaging. Although a person

with hypochondriasis may exhibit symptoms in all three of these categories, symptoms that fall within a predominant category can impact patient presentation and treatment (Stewart & Watt, 2001). Adjunctive standardized assessments may be utilized but with cautious interpretation. For example, the Minnesota Multiphasic Personality Inventory-2 (MMPI-2), which has a hypochondriasis scale, does not necessarily measure the fear associated with health complaints but rather measures the degree to which one experiences physical symptoms (Taylor & Asmundson, 2006). Other measures, such as the Structured Clinical Interview for the DSM-IV (SCID), the Structured Diagnostic Interview for Hypochondriasis (SDIH), and other self-report hypochondriasis measures (e.g., Whiteley Index, Health Anxiety Questionnaire, Illness Attitudes Scale, Somatosensory Amplification Scale and Illness Behavior Scale), may be more appropriate tools in gathering diagnostic information within this patient population (Speckens, 2001; Stewart & Watt, 2001).

Treatment/Best Practices

Although there is no evidence to date on combined treatments, there is consistent evidence suggesting that cognitive behavioral therapy (CBT) and medication-based treatments independently for hypochondriasis are effective (Taylor & Asmundson, 2006). Specifically, CBT interventions that focused on exposure and response prevention, psychoeducation, and cognitive therapy were found to improve symptoms in patients with hypochondriasis, and treatment gains continued to be present in a 12-month follow-up (Asmundson, Taylor, & Sevgur, 2001; Fallon & Feinstein, 2001; Taylor & Asmundson, 2006). As an example of CBT with exposure and response prevention for hypochondriasis, a patient with a bodily preoccupation of skin rashes may be exposed to anxiety associated with thoughts of having a skin rash, and resist the typical subsequent responses to that anxiety that would usually follow (e.g., calling a doctor, asking numerous questions about the rash, researching information on the internet/medical books, etc.) until the anxiety is relieved. Finally, while there are self-help and internet resources available, there is a paucity of evidence on the effectiveness of these resources utilized independently and/or as an adjunct to other therapies described (Taylor & Asmundson, 2006).

With regard to pharmacotherapy, selective serotonin reuptake inhibitors (SSRIs) and imipramine, a tricyclic antidepressant, have been shown in both head-to-head trials and randomized control trials to be useful in patients with hypochondriasis. Of the SSRIs studied (paroxetine, fluoxetine, fluvoxamine, and nefazadone), fluoxetine appears to have greater potential for desired treatment effects. However, there are no recent studies on long-term use of these medications with persons diagnosed with hypochondriasis (Asmundson et al., 2001; Enns, Kjernisted, & Lander, 2001; Taylor & Asmundson, 2006).

In addition to these treatment choices, environmental factors may impact treatment effectiveness. For example, the relationship between the patient and treating provider may impact treatment and exacerbation of symptoms (Lipsitt, 2001). If the provider is perceived by the patient as dismissing of symptoms or as not addressing the patient's concerns, the patient may choose to move from provider to provider until such symptoms are addressed. It is also suggested that overreassurance can exacerbate symptoms because the patient may perceive that their symptoms are being dismissed, leading to patient dropout (Starcevic, 2001). Optimally, providers who communicate clearly about guidelines on when to seek treatment and who have regularly scheduled follow-ups may reduce unnecessary treatment visits. Furthermore, patient preference should be weighted heavily into the decision to utilize pharmacotherapy, psychotherapy, or combined treatments (Taylor & Asmundson, 2006).

Cross-References

▶ Health Anxiety
▶ Somatoform Disorders

References and Readings

American Psychiatric Association, (2000). *Diagnostic and statistical manual of mental disorders*, 4th ed. text Rev. (DSM-IV-TR) (pp. 504–507). Washington, DC: Americain Psychiatric Association

Asmundson, G. J. G., Taylor, S., & Sevgur, S. (2001). In B. J. Cox, G. J. G. Asmundson, S. Taylor, & B. J. Cox (Eds.), *Health anxiety: Clinical and research perspectives on hypochondriasis and related conditions* (pp. 3–21). Chichester, England: Wiley.

Enns, M. W., Kjernisted, K., & Lander, M. (2001). In G. J. G. Asmundson, S. Taylor, & B. J. Cox (Eds.), *Health anxiety: Clinical and research perspectives on hypochondriasis and related conditions* (pp. 193–219). Chichester, England: Wiley.

Fallon, B. A., & Feinstein, S. (2001). *Somatoform and factitious disorders* (pp. 27–60). Washington, DC: American Psychiatric.

Lipsitt, D. R. (2001). *Hypochondriasis* (pp. 265–290). New York: Oxford University Press.

Speckens, A. E. M. (2001). *Hypochondriasis* (pp. 61–88). New York: Oxford University Press.

Starcevic, V. (2001). *Hypochondriasis* (pp. 291–313). New York: Oxford University Press.

Stewart, S. H., & Watt, M. C. (2001). In G. J. G. Asmundson, S. Taylor, & B. J. Cox (Eds.), *Health anxiety: Clinical and research perspectives on hypochondriasis and related conditions* (pp. 95–131). Chichester, England: Wiley.

Taylor, S., & Asmundson, G. J. G. (2006). Hypochondriasis. In J. E. Fisher & W. T. O'Donohue (Eds.), *Practitioner's guide to evidenced-based psychotherapy* (pp. 313–323). New York: Springer.

Hypoglycemia

Alyssa Parker
UTSW Health Systems, South western Medical Center, Dallas, TX, USA

Synonyms

Insulin shock; Low blood glucose

Definition

Hypoglycemia is characterized by an abnormally low level of blood glucose concentration, designated as less than or equal to 70 mg/dL. Blood glucose serves as the body's main energy source (Pub Med Health, 2011). With hypoglycemia, there is an inadequate supply of glucose to the brain, resulting in impairment of function that requires immediate attention to prevent organ or brain damage, especially when blood glucose is extremely low (Cryer, 1997). Hypoglycemia occurs when an individual's glucose is metabolized too quickly, when glucose is released into the bloodstream too slowly, and when too much insulin is released into the bloodstream (Cryer, Davis, & Shamoon, 2003). Hypoglycemia is relatively common among people with type 1 diabetes, and may occur when too much insulin or diabetes medication is taken, when insufficient amounts of food are ingested, or with sudden increases in exercise. Hypoglycemia is characterized as mild (below 70 mg/dL), moderate (below 55 mg/dL), or severe (below 35–40 mg/dL); and common symptoms include: trembling, nausea, sweating, dizziness, irritability, confusion, seizures, fainting, and coma. Immediate treatment involves quick steps to return blood glucose levels to a normal range. Mild-to-moderate hypoglycemia is treated by ingesting carbohydrates, while glucagon injections, a hormone that blocks insulin and raises blood glucose, are used to treat severe cases (Wysocki, Greco, & Buckloh, 2003). People with type 1 diabetes suffer an average of two episodes of symptomatic hypoglycemia per week, and approximately one episode of severe, at least temporarily disabling, hypoglycemia per year (Cryer, 2010). Careful monitoring of blood glucose levels can help predict or prevent moderate to severe hypoglycemia in at-risk individuals.

Cross-References

- Blood Glucose
- Diabetes
- Glucose
- Glucose: Levels, Control, Intolerance, and Metabolism
- Glycemia: Control, Load-High
- Insulin
- Type 1 Diabetes Mellitus

References and Readings

Cryer, P. E. (1997). *Hypoglycemia: Pathophysiology, diagnosis, and treatment*. New York: Oxford University Press.
Cryer, P. E. (2010). Hypoglycemia in type 1 diabetes mellitus. *Endocrinology and Metabolism Clinics of North America, 39*, 641–654.
Cryer, P. E., Davis, S. N., & Shamoon, H. (2003). Hypoglycemia in diabetes. *Diabetes Care, 26*(6), 1902–1912.
Hypoglycemia (General). Retreived from http://diabetes.niddk.nih.gov/dm/pubs/hypoglycemia/
Pub Med Health (2011). *A.D.A.M. Medical Encyclopedia: Hypoglycemia*. Retreived from http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0001423/
Wysocki, T., Greco, P., & Buckloh, L. M. (2003). Childhood diabetes in psychological context. In M. C. Roberts (Ed.), *Handbook of pediatric psychology* (pp. 304–320). New York: Guilford Press.

Hypothalamic Nuclei

► Hypothalamus

Hypothalamic-Pituitary-Adrenal Axis

Jennifer Heaney
School of Sport and Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

Synonyms

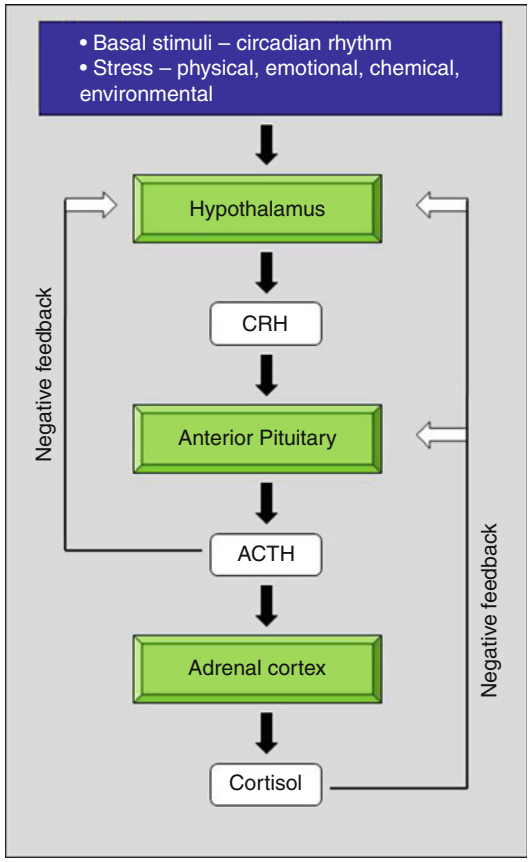
Pituitary-adrenal axis

Definition

The hypothalamic-pituitary-adrenal axis (HPA axis) is an interactive neuroendocrine unit comprising of the hypothalamus, the pituitary gland, and the adrenal glands. The hypothalamus is located in the brain and the pituitary at the base of it, whereas the adrenals are on top of the kidneys.

The HPA axis plays key roles in basal homeostasis and in the body's response to stress. The major pathway of the axis results in the production and secretion of cortisol. The hypothalamus responds to basal neural input which follows a circadian rhythm and input as a result of stress by increasing the secretion of corticotrophin-releasing hormone (CRH) from the hypothalamus. This increase in CRH acts upon the anterior pituitary gland to secrete adrenocorticotropic hormone (ACTH), which in turn circulates to the adrenal cortex to stimulate the release of cortisol into the bloodstream. The HPA axis is an example of a negative feedback loop; cortisol can reduce its own secretion via feedback to the anterior pituitary to reduce ACTH and the hypothalamus to limit the secretion of CRH. ACTH also provides negative feedback limiting its secretion via CRH.

The importance of the HPA axis to homeostasis is illustrated through the wide range of functions that cortisol is involved in, including metabolism, vascular activity, and immune and inflammatory responses. Therefore, even in the absence of stress, the production of cortisol via the HPA axis performs vital roles within the human body. In the presence of stress, be it physical, emotional, chemical, or environmental, the HPA axis governs the body's response. An increase in the production of cortisol enhances vascular activity, reduces immune responses, limits inflammation, stimulates gluconeogenesis, and inhibits nonessential functions. These effects serve to protect the body from potential damaging effects of stress, e.g., excessive immune and inflammatory response, but also act to increase resources to cope during the period of stress through changes in metabolism. While the role of cortisol in the stress response is initially protective, long-term stimulation of the HPA axis and over exposure to cortisol can be damaging, leading to immunosuppression and excessive catabolism of body tissue. Dysregulation of the HPA axis has been associated with various illnesses and disorders, both physiological and psychological. More detail on the HPA axis can be found from the following sources: Widmaier et al. (2004) and Greenspan and Forsham (1983) (Fig. 1).



The hypothalamic-pituitary-adrenal axis
CRH: Corticotrophin releasing hormone
ACTH: Adrenocorticotrophic hormone

Hypothalamic-Pituitary-Adrenal Axis, Fig. 1 The hypothalamic-pituitary-adrenal axis. *CRH* Corticotrophin releasing hormone, *ACTH* Adrenocorticotrophic hormone

Cross-References

- ▶ ACTH
- ▶ Cortisol
- ▶ Pituitary-Adrenal Axis

References and Readings

Greenspan, F. S., & Forsham, P. H. (1983). *Basic and clinical endocrinology*. Los Altos, CA: Lange Medical.

O'Riordan, F. L. H., Malan, P. G., & Gould, R. P. (1988). *Essentials of endocrinology* (2nd ed.). Oxford: Blackwell Scientific.

Widmaier, E. P., Raff, H., & Strang, K. T. (2004). *Vander, Sherman, & Luciano's human physiology: The mechanism of body function*. New York: McGraw-Hill.

Hypothalamus

Elliott A. Beaton
Department of Psychiatry and Behavioral Sciences and the M.I.N.D. Institute, University of California-Davis, Sacramento, CA, USA

Synonyms

Anterior hypothalamic area; Arcuate nucleus; Dorsal hypothalamic area; Dorsomedial nucleus; Hypothalamic nuclei; Lateral mammillary nucleus; Lateral nucleus; Lateral preoptic area; Medial mammillary nucleus; Medial preoptic area; Paraventricular nucleus; Posterior hypothalamic area; Suprachiasmatic nucleus; Supraoptic nucleus; Ventromedial nucleus

Definition

The hypothalamus is a subcortical collection of nuclei that monitor, modulate, and regulate physiology and behavior including feeding, thirst, reproduction, temperature regulation, sleep, and emotional behavior such as fear and aggression.

Description

This entry describes the hypothalamus in humans. All vertebrates have a hypothalamus, and while there is a high degree of conservation across species and especially in mammals, readers should refer elsewhere for phenotypic and functional details regarding other animals.

The hypothalamus (Latin: hypo: "below" the thalamus) is located below the thalamus on the base of the brain on both sides of the ventral portion of the third ventricle. Three main divisions of the hypothalamus are the anterior, medial

(or tuberal), and posterior regions, each made up of several nuclei. These clusters of neurons coordinate autonomic, endocrine, somatic, and behavioral information to maintain homeostasis and motivate behavior in both the short and long term. Via bidirectional connections throughout many areas of the brain, the hypothalamus regulates a host of physiological processes and related reflexes including feeding, drinking, reproduction, temperature regulation, sleep, and emotional behavior such as fear and aggression.

The hypothalamus receives sensory information from the entire body about conditions in the external world including smells and light cycles and internal information from the visceral somatosensory system. Other sensory cells within hypothalamic nuclei monitor and respond to changes in internal states and biological set points such as temperature, salinity, and glucose, in addition to other factors outside the blood-brain barrier via connections with circumventricular organs such as the area postrema. A change in homeostasis (e.g., increased body temperature) triggers a variety of responses aimed at body temperature reduction, all of which are regulated by the hypothalamus. These include moving blood to the surface of the skin, increased sweating, and increased water conservation at the kidneys.

The hypothalamus controls the endocrine system via the anterior and posterior pituitary gland. The hypothalamus is neural tissue, but it also has specialized neural cells that secrete neurohormones from nerve cell terminals into blood vessels in the pituitary gland. Although the output of these neurosecretory cells is endocrinological, they are morphologically similar to typical neurons and serve to bridge the neural and endocrine systems. Oxytocin and vasopressin are peptides produced in neuroendocrine cells in the paraventricular and supraoptic nuclei of the hypothalamus and released into general circulation directly via projections into the arterial blood within the posterior pituitary gland (also called the neurohypophysis). Other hypothalamic nuclei indirectly control the endocrine system through the production of neuroendocrine factors that inhibit or increase release of various hormones from the anterior pituitary gland (also called the adenohypophysis). For example, corticotropin-releasing factor and growth hormone-releasing factor regulate the release of adrenocorticotropin and growth hormone from the anterior pituitary respectively.

Finally, bidirectional communication between higher cortical regions and the hypothalamus occurs via the limbic system. In the temperature regulation example above, information from the hypothalamus to other limbic areas and the cortex can regulate complex behavior and motivation such as acting on the environment to generate cooling or mobility to a cooler environment. Cognition and emotion related to the situational context can also modulate hypothalamic responses.

References and Readings

Greenstein, B., & Wood, D. (2006). *The endocrine system at a glance* (2nd ed.). Malden, MA: Blackwell.

Joseph, J. T., & Cardozo, D. L. (2004). *Functional neuroanatomy: An interactive text and manual*. Hoboken, NJ: Wiley.

Kandel, E. R., Schwartz, J. H., & Jessell, T. M. (Eds.). (2000). *Principles of neuroscience* (4th ed.). New York, NY: McGraw-Hill.

Panksepp, J. (1998). *Affective neuroscience: The foundations of human and animal emotions*. New York, NY: Oxford University Press.

Hypothesis Testing

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Inferential statistical testing

Definition

Hypothesis testing is the core component of inferential statistics, a branch of statistics that is

employed to provide compelling evidence that two or more groups of numbers (data) differ from each other in a meaningful manner. It is therefore a strategy that facilitates provision of compelling evidence of the efficacy of a new behavioral medicine intervention or treatment.

Description

One of the most efficient ways to acquire new knowledge about any topic (including new behavioral medicine interventions) is to devise questions that guide our investigations. While relatively loosely formed questions can be helpful in early stages of the knowledge acquisition process, refining our knowledge is facilitated by asking more specific questions, and, in turn, acquiring more specific information and knowledge.

The scientific method is one particular method of acquiring new knowledge. In scientific research, our questions need to be asked in a particular manner. These questions are called research questions, and they lead to the development of two research hypotheses in each case. Turner (2007) provided an operational definition of a useful research question:

- It needs to be specific (precise).
- It needs to be testable.

Consider a new behavioral medicine intervention for pain management. To test its efficacy against a standard (existing) treatment, it is planned to conduct a randomized, controlled clinical trial. The research question of interest is:

- Is the new treatment statistically significantly more effective than the standard treatment?

Actually, the "pure" statistical version of this question asks whether there is a statistically significant difference in the efficacy demonstrated by the two treatments. This question allows for the fact that the new treatment could theoretically be statistically significantly ***less*** effective than the standard treatment as well as being significantly more effective. That is, use of a two-sided approach (hypothesis) is, in reality, appropriate here. However, we will proceed here with interest focused on just one of the two directions of differential performance of the new treatment.

The research hypotheses are commonly called the null hypothesis and the alternative hypothesis. In this author's opinion, the term "research hypothesis" is more informative than the term "alternative hypothesis," and so it is used here. The null hypothesis is the crux of hypothesis testing. For our research question, the null hypothesis would be:

- The new treatment is not statistically significantly more effective than the standard treatment.

The accompanying research hypothesis is:

- The new treatment is statistically significantly more effective than the standard treatment.

It is important to note that, whatever the outcome of the trial, the following statements are true:

- It is never the case that neither hypothesis is correct.
- It is never the case that both hypotheses are correct.
- It is always the case that one of them is correct, and that the other is not correct.

A helpful way of remembering which hypothesis is which, i.e., which form the null hypothesis takes and which form the research hypothesis takes, is to conceptualize that the research hypothesis states what you are "hoping" to find, and the null hypothesis states what you are not hoping to find (researchers who have developed a new treatment have an understandable tendency to want it to be better than other treatments). It must be emphasized here, however, that, while helpful, this conceptualization skates on very thin scientific ice. In strict scientific terms, hope has no place in experimental research. The goal is to discover the truth, whatever it may be, and one should not start out hoping to find one particular outcome. In the real world, this ideologically pure stance is not common for many reasons (financial and personal recognition reasons being not the least of them).

Statistical analysis of the data acquired in the trial conducted to compare the two treatments will enable us to decide between the two mutually exclusive hypotheses, i.e., the null hypothesis and the research hypothesis. A measure of pain reduction will be obtained for each person receiving

the new treatment (the test treatment group), and will also be obtained for each person receiving the standard treatment (the control treatment group). The mean (average) pain reduction will be calculated for each treatment group. The focus of attention can be captured by a treatment effect associated with the new treatment, which is calculated as follows: Treatment effect = Mean pain reduction in the test treatment group minus mean pain reduction in the control treatment group.

The concept of statistical significance becomes important here because it is possible for the mean pain reduction in the test treatment group to be numerically greater than mean pain reduction in the control treatment group, but for the difference not to attain statistical significance. To provide compelling evidence that the new treatment is "better" than the standard one, there must be a statistically significant treatment effect. The statistical methodology involved in determining the presence or absence of a statistically significant treatment effect is formulaic: The treatment effect is either of statistically significant magnitude or it is not. If it is, the appropriate test will ultimately produce a result in the form "$p < 0.05$" (see the ▶ Probability entry). If so, given that a randomized study design has been used, this evidence would allow the statement that the difference in magnitude between the mean response in the test treatment group and that in the control group (the treatment effect) is unlikely to have been due to chance, i.e., it is sufficiently great to be declared statistically significant. In this case, the null hypothesis is rejected in favor of the research hypothesis. If the result is "$p \geq 0.05$" statistical significance is not achieved. This result allows the statement to be made that the degree of difference in pain reduction between the two treatment groups could well have arisen by chance alone. In this case, we fail to reject the null hypothesis.

Statistical methodology necessitates a choice being made here: It is a forced choice paradigm. It is always the case that the process of hypothesis testing will result in one, and only one, of these two mutually exclusive actions.

The connection between the terms "hypothesis testing" and "inferential statistics" is a result of the following statement. The ultimate purpose of the results from a single clinical trial (or a group of related trials) is not to tell us precisely what happened in that trial, but to allow us to gain insight into, i.e., to infer in an educated manner, what is likely to happen in a much larger group of patients should the treatment make it into widespread clinical practice.

Cross-References

▶ Probability

References and Readings

Turner, J. R. (2007). New drug development: design, methodology, and analysis. Hoboken, NJ: Wiley.

Hypothetical Construct

▶ Latent Variable

Hypothetical Variable

▶ Latent Variable

Iatrogenic Conditions

Marie Boltz
College of Nursing, New York University, New York, NY, USA

Synonyms

Nosocomial medical errors

Definition

Iatrogenesis refers to any unintended adverse patient outcome due to a health-care intervention not related to the natural course of an illness or injury (Agency for Healthcare Research and Quality).

Description

Iatrogenic conditions may be both preventable (e.g., medical error, negligence, consumer decisions) and unpreventable (e.g., the side effects of chemotherapy). The study of iatrogenic conditions has largely been in the acute care hospital. Two Institute of Medicine reports describe the staggering prevalence and ramification of medical errors and elevated patient safety as a major concern in health care and among policymakers. The first report, *To Err is Human: Building a Safer Health System* estimated that preventable medical errors resulted in 44,000 and 98,000 deaths per year at a cost of up to $29 billion in unnecessary health-care expenses, disability, and lost income (Kohn, Corrigan, & Donaldson, 1999). The second report, *Crossing the Quality Chasm* emphasized the need to reengineer health-care delivery, and defined six aims of health care. The report called for care to be safe, effective, patient-centered, timely, efficient, and equitable (Institute of Medicine, 2001).

The rate of preventable adverse events continues to increase. In a study published in 2011, the National Center for Policy Analysis estimates that there are as many as 187,000 preventable iatrogenic deaths in hospitals and as many as 6.1 million injuries, both in and out of hospitals (Goodman, Villarreal, & Jones, 2011). Iatrogenic complications, in general, are related to medications (adverse drug events), therapeutic or diagnostic interventions, nosocomial infections, and environmental factors.

Adverse Drug Events

Inappropriate drug prescribing, polypharmacy, administration errors, and suboptimal adherence by the patient are common preventable causes of adverse drug events (ADEs). Medication reconciliation upon admission, transfer, and discharge is a key strategy to maintain medication safety. The use of electronic medical records that provide information (e.g., past diagnoses and lab studies), educational prompts, and warnings

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

(e.g., allergy alerts and contraindications) are used to reduce errors in prescribing. The Institute for Safe Medication Practices provides guidelines for the use of well-designed standard order sets, safe automated dispensing, and medication labeling both in acute and community settings (The Institute for Safe Medication Practices Guidelines).

Guidelines for rational drug prescribing, particularly critical in the older adult, are another strategy to prevent ADEs. The Beers criteria address two key areas: (1) medications or medication classes that should generally be avoided in persons 65 and older and (2) medications that should be avoided in older persons with specific medical conditions. The Centers for Medicare & Medicaid Services (CMS) have incorporated the Beers criteria into regulatory guidelines in long-term care. The Joint Commission (TJC) describes prescribing that is incompatible with the criteria as a potential sentinel event in hospitals. While the Beers criteria for inappropriate medications are an accepted guideline for assessing *potential* inappropriate medications, they need to be used in conjunction with clinical assessment of the individual patient's clinical presentation and needs (Swagerty & Brickley, 2005).

Patient understanding of medications needs to be addressed as a potential barrier to medication adherence. The National Quality Forum (NQF) identified the "teach back" method as an essential safe practice to improve health care (National Quality Forum, 2005). Asking the patient to recount what he or she has been told can help gauge the level of understanding and facilitate individualized educational approaches. This method is recommended for all types of provider settings.

Therapeutic or Diagnostic Interventions

Excessive venipuncture increases the risk of anemia, infection, phlebitis, and venous thrombotic embolism (VTE). Another common complication, especially in persons with diabetes mellitus and preexisting renal insufficiency, is nephropathy induced by the use of contrast dye. When a radiocontrast dye is deemed necessary, a low-osmolar agent should be used at the lowest dose possible, with careful hydration before and after the procedure. Medical procedures, such as thoracentesis and cardiac catheterization, have also been linked to preventable adverse effects, such as cardiac arrhythmias, bleeding, infection, and pneumothorax. Aspiration deaths due to barium, emollient laxatives, and contrast medium; colonic perforations due to endoscopy or enema; and complications associated with percutaneous endoscopic gastrostomy tubes are not uncommon.

Older adults in any setting, but particularly the acute care hospital, are at risk for "geriatric syndromes" which can be caused by inadequate care processes and environmental factors. Functional decline, delirium, falls with injury, and pressure ulcers are associated with the use of restrictive devices and immobility, prolonged use of invasive lines, lack of attention to nutrition, sensory deprivation, inappropriate medication use, inadequate pain control, and environments that do not accommodate age-related changes. Bed rest, in and of itself, can have serious negative effects on older patients and is associated with greater risk for venous thrombotic embolism (VTE), orthostatic hypotension, pneumonia, anorexia, constipation, and fecal impaction (Creditor, 1993).

Iatrogenesis can also occur due to misdiagnosis. For example, the older woman who presents to the emergency department with shortness of breath and abdominal discomfort may not receive appropriate evaluation for a myocardial infarction because of her "atypical presentation." She suffers from underdiagnosis due to the provider's lack of knowledge of aging pathophysiology. Similarly, psychological conditions may be underdiagnosed as well as overdiagnosed. For example, a normal bereavement reaction can be pathologized and described as major depression, leading to inappropriate pharmacologic therapy rather than grief counseling.

Nosocomial Infections

Common nosocomial or facility-acquired infections include those of the urinary tract, those

related to intravascular devices, and pneumonia. Potentially preventable and antibiotic-resistant infections include those of the skin (e.g., methicillin-resistant *Staphylococcus aureus*), urinary tract (e.g., vancomycin-resistant *Enterococci*), oropharyngeal (e.g., *Candida* species), and gastrointestinal (e.g., *Clostridium difficile*). Antibiotic resistance is associated with overprescribing of antibiotics, cross-colonization due to poor hand-washing techniques of health-care workers, and the transfer of colonized patients between institutions.

The use of indwelling urinary catheters is associated with urinary tract infections. In addition to aseptic technique during insertion and careful catheter hygiene, providers are advised to avoid their use whenever possible. Also, implementing systems that prompt providers to review the need for the catheter and encourage early removal are warranted (CDC Guidelines for Prevention of Catheter-Associated Urinary Tract Infections, 2009).

Intravascular infections are related to central intravenous or intra-arterial lines. Organizational practices to limit their occurrence include: the use of antimicrobial catheters, proper disinfection of the skin, and prompt removal of the catheter as soon as possible and whenever there is clinical evidence of infection. Factors associated with nosocomial pneumonia include gastric aspiration, fecal-oral spread of pathogens, spread from the hands of health-care personnel, and cross-contamination from other patients. Nosocomial pneumonia is most common in critically ill, ventilated patients. Preventive measures include proper hand-washing and cleaning of respiratory and medical (including stethoscopes) equipment and maintaining the patient in an upright position to prevent aspiration of gastric contents (CDC (Center for Disease Control) Guidelines for Preventing Health-Care–Associated Pneumonia, 2003).

Environmental Factors

The physical environment of health-care facilities, especially hospitals, may pose fall hazards and limit patient mobility and physical activity. Thus, they may actually contribute to iatrogenic outcomes such as fall-related injuries, skin breakdown, and functional decline, especially in older patients or those who are physically or cognitively challenged. Cluttered walkways, hallway glare, beds and toilets not at the proper height to promote safe transfers, noise levels that interfere with sleep, and lack of access to assistive equipment are frequent culprits. Thus, comfort, enablement of physical activity, and safety are important attributes of a health-care environment.

The social environment, particularly the dynamic between the patient and the health-care worker can also contribute to functional and other outcomes. Communication that is encouraging and insightful can motivate patients and facilitate involvement in function-focused care, thus mitigating risk for falls, pressure ulcers, and functional decline. Additionally, collaboration among the interdisciplinary team and inclusion of patients in decision-making is critical. A collaborative relationship between clinicians, health-care workers, and the patient to attain mutually agreed upon goals can foster more patient control, self-care, and autonomy and prevent iatrogenesis (Berntsen, 2006).

References and Readings

Agency for Healthcare Research and Quality. *Medical errors and patient safety*. htpp://www.ahrq.gov/qual/errorsix.htm Accessed Mar 30, 2012

Berntsen, K. J. (2006). Implementation of patient centeredness to enhance patient safety. *Journal of Nursing Care Quality, 21*(1), 15–19.

CDC Guidelines for Prevention of Catheter-Associated Urinary Tract Infections. (2009). http://www.cdc.gov/hicpac/cauti/001_cauti.html Accessed Mar 30, 2012

CDC (Center for Disease Control) Guidelines for Preventing Health-Care–Associated Pneumonia, 2003. http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5303a1.htm Accessed Mar 30, 2012

Creditor, M. C. (1993). Hazards of hospitalization of the elderly. *Annals of Internal Medicine, 118*(3), 219–223.

Goodman, J. C., Villarreal, P., & Jones, B. (2011). The Social cost of adverse medical events, and what we can do about it. *Health Affairs, 30*(4), 590–595.

Institute of Medicine. (2001). *Crossing the quality chasm: A new health system for the 21st century*. Washington, DC: National Academy Press.

Kohn, L., Corrigan, J., & Donaldson, M. (1999). *To err is human: Building a safer health system*. Washington, DC: National Academy Press.
National Quality Forum. (2005). *Improving patient safety through informed consent for patients with limited health literacy*. Washington, DC: Author.
Swagerty, D., & Brickley, R. (2005). American Medical Directors Association and American Society of Consultant Pharmacists joint position statement on the Beers list of potentially inappropriate medications in older adults. *Journal of the American Medical Directors Association, 6*(1), 80–86.
The Institute for Safe Medication Practices Guidelines. http://www.ismp.org/Tools/guidelines/default.asp Accessed Mar 30, 2012

ICSI

► In Vitro Fertilization, Assisted Reproductive Technology

Ideas

► Cognitions

Identical Twins

► Monozygotic Twins

Idiopathic Raynaud's Phenomenon

► Raynaud's Disease and Stress
► Raynaud's Disease: Behavioral Treatment

Ileitis Terminalis

► Crohn's Disease (CD)

Illness Behavior

Holly Rau and Paula Williams
Department of Psychology, University of Utah, Salt Lake City, UT, USA

Synonyms

Health behaviors; Sickness behavior

Definition

Illness behavior refers to any actions or reactions of an individual who feels unwell for the purpose of defining their state of health and obtaining physical or emotional relief from perceived or actual illness. These behaviors include how individuals monitor and interpret bodily sensations, utilize healthcare resources, discuss illness or symptoms with providers, and adhere to prescribed medical regimens.

Illness behaviors can be organized into two broad categories:

- *Self-care behavior* – Any action taken to manage or improve a health condition in the absence of direct medical attention, which includes managing symptoms and caring for minor injuries. Research indicates that a majority of health problems are managed via self-care behaviors and that most individuals will attempt self-treatment prior to seeking medical attention.
- *Healthcare utilization behavior* – Any action that involves direct use of healthcare services (e.g., medical appointments, medical procedures).

Variation in illness behavior is often attributed to psychological and sociocultural factors and can be characterized as a continuum with both ends constituting abnormal illness behavior. On one end of the continuum, illness perceptions and behaviors are disproportionate to objective pathology (e.g., hypochondriasis) and often involve debilitating psychological distress and overuse of health services. Less debilitating

presentations have been termed *hypochondriacal tendencies* or *health anxiety* and can also include overuse of healthcare services, unnecessary medical tests, missed work, and subjective distress. Overuse of healthcare resources has often been attributed to secondary gains (i.e., external rewards associated with illness, such as missed work). On the other end of the continuum, *delay* in seeking medical care or underuse of health services can have life-threatening consequences, particularly when medical conditions require early detection and treatment (e.g., stroke).

Context is important in determining if an illness behavior is functional or potentially harmful. That is, illness behaviors that might be considered maladaptive in one illness context may serve an adaptive function in another context. For example, denial regarding injury severity may be adaptive during early stages of spinal cord injury (SCI) recovery because it may serve to lower anxiety and allow for active participation in therapies necessary to achieve functional gains. In later stages of SCI recovery, however, denial may interfere with long-term adjustment to disability. Therefore, providers are encouraged to consider the context of behaviors with respect to disease characteristics when maladaptive illness behaviors are suspected.

Illness behavior also includes behavioral presentation and communication with healthcare providers. The nature of patient-provider interactions is determined, in part, by the patient's cultural background and psychological characteristics (e.g., personality, anxiety) and can influence subsequent delivery of services, as well as patient satisfaction with medical care. For example, research suggests that ethnic minorities may be less verbally expressive and assertive compared to Caucasians, and physicians may provide more biomedical information to low-anxious patients. Treatment adherence, which includes taking medication as prescribed as well as making recommended lifestyle behavior changes (e.g., weight loss), is another form of illness behavior with obvious consequences for health outcomes. Beliefs in the *cause* of illness and the *effectiveness* of the prescribed treatment are both associated with adherence.

Cross-References

- ▶ Common-Sense Model of Self-Regulation
- ▶ Health Anxiety
- ▶ Health Assessment Questionnaire
- ▶ Health Beliefs/Health Belief Model
- ▶ Health Risk (Behavior)
- ▶ Hypochondriasis
- ▶ Illness Cognitions and Perceptions
- ▶ Self-care
- ▶ Sickness Behavior
- ▶ Somatic Symptoms
- ▶ Somatization
- ▶ Somatoform Disorders

References and Readings

Christensen, A. J. (2004). *Patient adherence with treatment regimens: Bridging the gap between behavioral science and biomedicine*. New Haven, CT: Yale University Press.

Mechanic, D. (1982). *Symptoms, illness behavior, and help-seeking*. New Brunswick, NJ: Rutgers University Press.

Pilowsky, I. (1997). *Abnormal illness behaviour*. Chichester, UK: Wiley.

Schouten, B. C., & Meeuwesen, L. (2006). Cultural differences in medical communication: A review of the literature. *Patient Education and Counseling, 64*, 21–34.

Williams, P. G., Smith, T. W., & Jordan, K. D. (2010). Health anxiety and hypochondriasis: Interpersonal extensions of the cognitive-behavioral perspective. In G. Beck (Ed.), *Interpersonal processes in the anxiety disorders: Implications for understanding psychopathology and treatment* (pp. 261–284). Washington, DC: American Psychological Association.

Illness Cognitions and Perceptions

Holly Rau and Paula Williams
Department of Psychology, University of Utah, Salt Lake City, UT, USA

Synonyms

Health beliefs; Illness representations

Definition

The terms *illness perceptions* and *illness cognitions* are used to describe a range of cognitive processes underlying attention, interpretation, and behavior in response to illness-related information. Although often studied in the context of various disease populations (e.g., diabetes, cancer), illness cognitions and perceptions are also relevant in the absence of a diagnosed health condition and have important implications for subsequent illness behavior (e.g., self-care, healthcare utilization, treatment adherence).

Description

Self-assessment of health, which includes symptom perception, interpretation, and reporting, is central to illness-related self-regulatory behavior (i.e., self-care decision making, communication with healthcare providers, commitment to treatment regimens, etc.). Underestimation or overestimation of illness status can lead to inappropriate illness behavior, such as overuse or avoidance of healthcare services. Health self-assessment may have better predictive utility than some objective indicators of health with respect to health outcomes. For example, global health ratings have been found to predict mortality above and beyond objective indicators of health (e.g., documented medical conditions, such as hypertension).

Treatment adherence and health outcomes research often focuses on cognitive and perceptual factors contributing to illness behaviors, examining how individuals define illness and monitor illness status. Various cognitive models have attempted to account for variations in symptom perception and interpretation, though most emphasize information processing.

Information-Processing Models

From a cognitive-perceptual standpoint, input received about physical health status, whether from internal (e.g., body sensations) or external (e.g., healthcare professionals) sources, must be processed and cognitively organized. This organization will then affect how illness is managed. Therefore, information-processing models attempt to understand how subjective interpretations of illness exert influence on illness behavior relative to the objective disease characteristics.

- *Bottom-up Information Processing:* Somatic sensations serve as indicators of potential disease or health risks; thus, interpretations of illness are determined, in part, from somatosensory experiences. In support of this supposition, research suggests that functional difficulties (e.g., shortness of breath) are likely to figure more prominently than medical indicators (e.g., prescribed medication) in subjective estimates of illness severity. Although bottom-up models emphasize somatic experience early in the temporal sequence of unfolding illness perceptions, the importance of previous illness exposure and/or illness beliefs is not entirely discounted.
- *Top-down Information Processing:* Schema-driven, or top-down, information-processing models propose that previously formed expectations influence the perception and interpretation of illness-related information. From this perspective, illness status is evaluated by integrating somatosensory experiences into an underlying cognitive framework. This model presumes that stable mental representations of health-relevant information influence the processing and reporting of physical symptoms. Experimental cognitive research supports this notion, although very little is known about the accuracy of illness-related information processing.

The Common-Sense Model of Self-regulation (CSM)

This prominent model of lay representations of health and illness hypothesizes that illness cognitions function as a filter and interpretive schema for illness information, which subsequently guides illness-related actions and behaviors. *Illness information* is posited to come from three main sources: (1) socio-cultural knowledge and communication related to the illness,

(2) esteemed members of the person's social environment (e.g., doctor, spouse, parent), and (3) direct personal experience. Qualitative, anthropological, and psychological research utilizing patients' accounts of illness has identified five logical themes or dimensions that characterize illness representations:

- *Cause* – beliefs regarding factors responsible for causing and perpetuating the illness or disease, including biological, emotional, environmental, psychological causes
- *Consequences* – beliefs regarding the impact of the illness on functional capacity and overall quality of life
- *Identity* – beliefs about the illness label (e.g., diabetes) and attribution of symptoms to the illness (e.g., extreme fatigue)
- *Timeline* – beliefs about the course of the illness (i.e., chronicity) and timescale of illness symptoms (e.g., acute, chronic, episodic)
- *Cure/controllability* – beliefs about the effectiveness of coping behaviors or treatment on the course of illness

These dimensions are thought to interact to affect illness behavior. For example, research indicates that individuals for whom the illness identity involves high threat and attribution of many symptoms to the identity label are more likely to view their illness as uncontrollable, chronic, and as having serious consequences for their lifestyle. Conversely, patients who view themselves as having greater control over their illness appear to be more likely to view their illness as less chronic and with fewer consequences. In addition, research suggests that the cure/control-consequence relationship appears to be dependent on the timeline dimension, and the identity-timeline relationship appears dependent on the consequence dimension.

Illness Cognitions and Illness Management

The Common-Sense Model explicitly links illness cognitions to coping behaviors, which in turn affect outcomes. Research supports the notion that active self-care behaviors are more likely to occur if they are perceived to be efficacious, and a disease seen as controllable is likely to result in cognitive reappraisal of the illness. Conversely, perceiving the illness as uncontrollable, chronic, and highly symptomatic appears to be associated with coping strategies characterized by denial and avoidance. Cognitive constructs from the *Health Belief Model* have also been examined in the context of illness management behavior. In particular, the perceived threat of a health problem, along with cost-benefit appraisal of available strategies, influences the likelihood that any given illness management strategy will be adopted. Research has also demonstrated in a variety of patient populations that non-adherence is related to doubts about the necessity of prescribed treatment, as well as to concerns about the potential adverse effects of the treatment. For example, an individual who views his or her illness as acute with limited long-term consequences is less likely to adhere to prescribed medical treatment, even if the treatment is viewed as effective and with limited adverse effects.

Abnormal Illness Cognitions and Perceptions

For some individuals, self-assessments of health become a source of preoccupation, emotional distress, and chronic disability. In clinical populations, this is referred to as *hypochondriasis*, which is defined as a preoccupation with the belief that one has a serious disease, based on misinterpretation of bodily symptoms. This belief occurs in the absence of known organic pathology and persists despite appropriate medical evaluation and reassurance. Less debilitating presentations of these characteristics have been termed *hypochondriacal tendencies* or *health anxiety*, which suggests that hypochondriasis may be best conceptualized along the spectrum of anxiety disorders.

Similar to the development of other anxiety-related conditions, health anxiety and hypochondriasis are thought to result from an underlying sensitivity to threatening stimuli (e.g., bodily sensations, illness status). Individuals with health anxiety are more likely to interpret physical sensations as signs of physical

illness, as well as over-attend to health-relevant information (with a bias toward illness-confirming information). Frequent medical consultations or seeking out medical information often follow, which can potentially perpetuate illness concerns.

Individuals with hypochondriacal tendencies have also been found to have catastrophic *interpretations* of somatic information. Consistent with these findings, individuals high in health anxiety have been found to rate themselves at greater risk of medical complications than those lower in health anxiety. Patients high in health anxiety are also more likely to recall medical reassurance as less certain in ruling out serious health problems.

Survey of the extant literature suggests that a combination of risk factors, including temperament, personality, prior experience with illness during childhood, and social development, contributes to likelihood of health anxiety, hypochondriacal tendencies, or hypochondriasis. For example, those most at risk may be high in the personality factor neuroticism and have a history involving significant illness-related events and with parent modeling of overconcern or overattention to bodily symptoms.

Treatment Implications

Understanding illness beliefs from the perspective of the individual is essential for changing maladaptive illness behaviors. For this reason, application of the Common-Sense Model has important implications for healthcare providers. Appreciating an individual's illness representations can introduce outlets for patient education and physician intervention, and these types of discussions may be important for preventing functional disability. Tailoring interventions to specific illness cognitions or perceptions is likely to increase the effectiveness. For example, interventions targeting unrealistic perceived consequences are likely to differ from those targeting unrealistic perception of illness timeline. Effective psychological interventions, such as Cognitive-Behavioral Therapy (CBT), focus on changing illness perceptions, reducing unhelpful perceptions, and improving coping skills, and may lead to enhanced functional ability (e.g., improved health or work outcomes).

Cross-References

- ▶ Cognitions
- ▶ Cognitive Appraisal
- ▶ Cognitive Behavioral Therapy (CBT)
- ▶ Cognitive Distortions
- ▶ Common-Sense Model of Self-Regulation
- ▶ Efficacy Cognitions
- ▶ Health Anxiety
- ▶ Health Assessment Questionnaire
- ▶ Health Behaviors
- ▶ Health Beliefs/Health Belief Model
- ▶ Health Outcomes Research
- ▶ Hypochondriasis
- ▶ Illness Perceptions Questionnaire (IPQ-R)
- ▶ Self-care
- ▶ Sickness behavior
- ▶ Somatic Symptoms
- ▶ Somatization
- ▶ Somatoform Disorders

References and Readings

Cameron, L. D., & Leventhal, H. (Eds.). (2003). *The self-regulation of health and illness behaviour*. New York: Routledge.

Leventhal, H., Breland, J. Y., Mora, P. A., & Leventhal, E. A. (2010). Lay representations of illness and treatment: A framework for action. In A. Steptoe (Ed.), *Handbook of behavioral medicine: methods and applications* (pp. 137–154). New York: Springer.

Petersen, S., van den Berg, R. A., Janssens, T., & Van den Bergh, O. (2011). Illness and symptom perception: A theoretical approach towards an integrative measurement model. *Clinical Psychology Review, 31*(3), 428–439.

Williams, P. G. (2004). The psychopathology of self-assessed health: A cognitive approach to health anxiety and hypochondriasis. *Cognitive Therapy and Research, 28*(5), 629–644.

Illness Fatigue

- ▶ Fatigue

Illness Perceptions Questionnaire (IPQ-R)

Jane Upton
School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Definition

The illness perceptions questionnaire measures an individual's beliefs and feelings about their illness.

The Revised Illness Perception Questionnaire (IPQ-R) is a widely used quantitative measure of the five components of illness representations in Leventhal's self-regulatory model (Moss-Morris et al., 2002). It has been validated for a range of chronic conditions in a wide variety of languages. The five components include:

- Identity in which patients rate the number of symptoms they perceive to be part of the illness
- The cause of the illness
- The timeline of the illness, how long they think it will last
- The consequences of the illness, including whether they perceive it to be a serious condition
- Whether they think the illness can be cured or controlled

These components showed good test-retest reliability, with correlations ranging from .46 to .88 (Moss-Morris et al., 2002).

References and Readings

Moss-Morris, R., Weinman, J., Petrie, K., Horne, R., Cameron, L., Buick, D., et al. (2002). The revised Illness Perception Questionnaire (IPQ-R). *Psychology and Health, 17*, 1–16.

Illness Representation Model

► Common-Sense Model of Self-regulation

Illness Representations

► Illness Cognitions and Perceptions

Imaging

► Brain, Imaging
► Neuroimaging

Immune Function

Jos A. Bosch
Department of Clinical Psychology, Faculty of Social and Behavioral Sciences, University of Amsterdam, The Netherlands

Synonyms

Immunity

Definition

Immune function describes the body's response to infection (i.e., the invasion by microorganisms), neoplasms (cancers), and tissue damage.

Description

Successful immune function establishes a state of immunity (Latin: *immunitas*, "freedom from") against infection and disease. Immunity relies on a multifaceted and flexible defense system, the immune system that protects against microorganisms, like bacteria and viruses, and other foreign invaders, like toxins. The immune system also regulates repair processes in response to tissue damage, and it protects against neoplasms (cancers). The immune system does not operate autonomously. It is, among others, influenced by central nervous system processes and their

neurohormonal outflows, which, in turn, are shaped by social and psychological factors. Likewise, activities of the immune system have profound effects on those in the central nervous system, influencing mood, behavior, and cognition.

Immune protection may come at price; the immune system responds in a vigorous manner to perceived threats, which sometimes causes substantial collateral damage to healthy cells and tissues. We experience this as disease symptoms. Damage inflicted by the immune system is also the problem in autoimmune diseases, whereby, for reasons not completely understood, the immune system selects and destroys healthy bodily cells. Hence, the paradoxical observation is that medical treatment often aims to suppress immune system responses, instead of enhancing.

This section provides a brief description of the immune system and its main components in nonjargon terms. The end of this section lists a number of references that provide a more technical and detailed exposition.

The White Blood Cells

Immune function is mediated by an enormous variety of cells and molecules that act together in a dynamic network, whose complexity rivals that of the nervous system. The white blood cells, or leukocytes, are the main effectors of immune responses and are as a collective also described as "immune cells." These cells detect, intercept, and destroy bodily threats. The leukocytes are generated in the bone marrow by so-called hematopoietic progenitor cells and come in three main subtypes: the neutrophils, which make up 70% of leukocytes in the blood; the monocytes (10%); and the lymphocytes (20%). These numbers are somewhat misleading in that the majority of leukocytes (98%) are not in the blood but dispersed in the various tissues, where numbers and proportions of various leukocytes greatly vary from one anatomical location to another and from those in the blood.

The lymphocytes comprise B lymphocytes, T helper lymphocytes, cytotoxic T lymphocytes, and natural killer (NK) cells. The B lymphocytes produce soluble receptors, called antibodies or immunoglobulins, which bind to targets of the immune system, flagging them for destruction. Targets of immune cells and molecules are subsumed under the generic term "antigens," which is an abbreviation of *anti*body *gen*erator. The main function of T helper cells is to orchestrate immune responses via the release cytokines. Finally, the main function of cytotoxic T lymphocytes and NK cells is to destroy infected cells, in particular, cells infected by viruses. They also play an important role in destroying cancerous cells. They do so via the release of toxic substances which induce programmed cell death, or apoptosis. Apoptosis is a process of controlled cellular self-destruction whereby the cell's DNA disintegrates. One of the benefits of this type of cell killing is that the cell dies from the inside, i.e., without rupture (lysis) of its cell membrane, so that infectious viral particles will not leak out and infect other cells.

Innate and Adaptive Immunity

Whereas the immune system consists of many different cell types, each with unique functions in maintaining immunity, these cells are essentially grouped into two classes based on the mechanisms used to detect antigens, i.e., (1) innate immune cells (also denoted as nonspecific or nonadaptive immunity) and (2) adaptive immune cells. The former group of cells utilizes antigen receptors that detect general molecular structures that are common to many microorganisms, the so-called pattern recognition receptors. Examples are receptors that detect double-stranded RNA (ribonucleic acid, which is not produced by human cells and must therefore come from foreign organisms) or receptors that detect lipopolysaccharide (molecules which are only produced by certain bacteria).

By far, most immune cells utilize the above innate immune detection mechanisms, and only the T and B lymphocytes are classed under the adaptive immune system. In contrast to cells of the innate immune system, adaptive immune cells use highly specialized antigen receptors that are capable of recognizing only a single molecular structure. This high specificity, and thus diversity, implies that the number of immune cells expressing a particular antigen receptor

must be low. To generate sufficient numbers of antigen-specific cells needed to eliminate an infection, these adaptive cells divide ("proliferate") upon encountering their cognate antigen, a process that can take up several days. As a result, adaptive immune responses are initially slow, but this disadvantage is compensated by their unique capacity to learn from previous encounters. This immunological memory is established by retaining a reserve of so-called memory cells which perform immune surveillance after the antigen is eliminated. These memory cells respond more swiftly and in greater numbers upon a subsequent reencounter with the antigen, resulting in rapid and effective antigen elimination which prevents the development of disease. This ability to develop immunological memory is exploited by vaccines, whereby small amounts of antigen are injected to produce memory cells against that antigen.

Cytokines: The Messenger Molecules of the Immune System

Cytokines can be regarded as the neurotransmitter and hormones of the immune system and regulate virtually all cellular activities in the immune system and communication among immune cells. Like neurotransmitters, these protein molecules can act in an autocrine or paracrine fashion, or like hormones, act on longer distances (endocrine) via release in to blood stream. Significantly, these molecules also have important regulatory roles outside the immune system. Indeed, many nonimmune cells and tissues have receptors for cytokines which can substantially affect their activities. For example, the effects of cytokines, especially the so-called inflammatory cytokines (see section on The Immune Response and Inflammation below) on the central nervous system is responsible for so-called sickness behavior: a depression-like syndrome characterized by reduced physical and social activity, altered sleep, and negative affect. Likewise, the effect of cytokines on liver, fat, and muscle cells has pronounced metabolic implications, which, depending on the type of cytokine, involves altered glycolysis, fat storage, and insulin resistance. Moreover, many cells and tissues that do not belong to the immune system are themselves capable of producing cytokines. This fact has significant implications for the interpretation immunological data in behavioral medicine research. For example, a short bout of intense physical exercise can elevate the levels of the cytokine IL-6 (interleukin 6) up to 1,000%, which is produced by muscle cells and not by immune cells. Likewise, the oft reported association between depression and elevated cytokine levels in the blood is at least in part accounted for by cytokines released by fat tissue.

The Blood, the Lymphatic System, and the Lymphoid Organs

Most leukocytes continuously traffic into and out of the various tissues in search of antigen and in order to present their finds to other immune cells. The lymphatic vessels and the blood are the transport routes for this trafficking. These routes are also employed to distribute soluble molecules, such as antibodies and cytokines, throughout the body. The lymphatic vessels primarily act as a drainage system, which collects tissue fluid and transfers it into the blood circulation. The lymphatic system does not have a pump, and the drained tissue fluid (lymph) is circulated mainly as a result of muscle activity. Lymphatic vessels pass through lymphoid organs, which are small nodular organs where leukocytes congregate. These local leukocytes interact to exchange antigen that was captured in the upstream tissues and to sample the passing lymph for the presence of antigens. Leukocytes evade the lymph nodes via downstream lymphatic vessels that terminate into the blood circulation; from there, they migrate into the tissues to eliminate infections, cancerous or damaged cells, or they migrate to other lymphoid tissues in further search of antigen. The spleen is a large lymphoid organ that specifically filters the blood, in a manner similar as described above.

The Immune Response and Inflammation

To attract immune cells to sites of infection or tissue damage, the immune system initiates an inflammatory response. This essential part of most immune responses starts with the local

release of cytokines (categorized as inflammatory cytokines, such as TNF-a (tumor necrosis factor-alpha), IL-1b, IL-8) and other chemicals. These messengers are released by tissue cells and local immune cells that have detected molecular signals of infection and tissue damage. The release of these messenger substances has several effects as follows: (1) they attract circulating immune cells into the tissue (a process known as chemotaxis); (2) they affect the local blood vessels by making the vessels leaky, so protective substances from the blood can leak in the tissue, and by making the surrounding blood vessels sticky, via the expression of adhesion molecules that facilitate immune cells to migrate out the blood; (3) they cause local warming of the tissues (pyrogenic effects). These immunological mechanisms underlie the defining symptoms of inflammation: swelling, redness, pain, heat, and diminished tissue function.

Further consequences of the inflammatory response is an enhanced migration of resident immune cells to the draining lymphoid organs (see section above), where they alert other immune cells and hereby initiate larger involvement of the immune system, and the release of inflammatory cytokines into the blood stream. The latter provides a signal for systemic adaptation to inflammation, which involves the release of anti-inflammatory substances that help to keep the inflammation localized (e.g., cortisol from the adrenal cortex and C-reactive protein (CRP) from the liver), metabolic adaptations (e.g., via effects on fat and glycogen stores), and sensory and behavioral adaption (via effects on the nervous system).

Cross-References

- ▶ Behavioral Immunology
- ▶ HIV Infection
- ▶ Immune Responses to Stress
- ▶ Immunoglobulins
- ▶ Infectious Diseases
- ▶ Inflammation
- ▶ Psychoneuroimmunology

References and Readings

Abbas, K. A., Lichtman, A. L., & Pillai, S. (2012). *Cellular and molecular immunology* (7th ed.). Philadelphia: Elsevier, Saunders.

Bosch, J. A., Engeland, C., & Burns, V. E. (2011). Psychoneuroimmunology in vivo: Methods and principles. In J. Decety & J. T. Cacioppo (Eds.), *The Oxford handbook of social neuroscience*. New York: Oxford University Press.

Dantzer, R., O'Connor, J. C., Freund, G. G., Johnson, R. W., & Kelley, K. W. (2008). From inflammation to sickness and depression: When the immune system subjugates the brain. *Nature Reviews Neuroscience, 9*(1), 46–56.

Murphy, K. (2011). *Janeway's immunobiology* (8th ed.). London: Taylor & Francis.

Tracey, K. J. (2002). The inflammatory reflex. *Nature, 420*(6917), 853–859.

Immune Responses to Stress

Victoria E. Burns[1], Jos A. Bosch[2] and Leila Anane[1]

[1]School of Sport and Exercise Sciences, The University of Birmingham, Birmingham, UK

[2]Department of Clinical Psychology, Faculty of Social and Behavioral Sciences, University of Amsterdam, The Netherlands

Synonyms

Stress response

Definition

Stress can be thought of as a constellation of events comprised of a stimulus, "stressor," that precipitates a reaction in the brain, "stress perception," that activates fight-or-flight mechanisms, "stress response" (Dhabhar & McEwen, 1997). This section will concentrate on what happens in the immune system in response to stress.

Description

Introduction

In 1884, the editor of the *British Medical Journal* noted that at funerals, "the depression of spirits under which the chief mourners labour at these melancholy occasions peculiarly predisposes them to some of the worst effects of the chill." Despite many such anecdotes suggesting a link between psychological factors and immune function, it is only relatively recently that these associations have received widespread acceptance in the scientific and medical communities. In fact, as recently as 1984, an editorial piece in *Nature* proclaimed the persistence of a "stout band of near skeptics" who, while acknowledging that "there is probably a link between the central nervous system and the immune system," still questioned whether "enough is yet known to sustain people's hopes of explanation" (Maddox, 1984). The prodigious development of the field of psychoneuroimmunology (PNI) over recent decades is more thoroughly reviewed elsewhere (Ader, 2000; Fleshner & Laudenslager, 2004); these articles clearly illustrate that, although many mechanisms still require further delineation, the progress of PNI, in terms of the quality, quantity, and impact of the research, has exceeded the expectations of all but its most optimistic forefathers. These endeavors have revealed the complex intertwining of the nervous, endocrine, and immune systems that underpin the effects of stress on immunological health.

What is Stress?

Walter Cannon first coined the term "stress" in this context in 1929, describing it as an emergency mechanism that mobilizes energy for fight-or-flight responses. Richard Lazarus proposed that the initiation of this stress response, be it an emotional (i.e., anger, anxiety), behavioral (i.e., running from threat), or biological response (i.e., cardiovascular changes, immune alterations), is governed by a person's perception of their ability to cope with a particular stimulus. Psychological stress occurs, therefore, when a person's perceived ability to cope with a situation is exceeded by the demands of the perceived stressor (Lazarus & Folkman, 1984). One way of broadly categorizing psychological stress is into chronic and acute forms of stress. Chronic stressors are typically major life experiences, such as bereavement or caregiving for an ill family member, that have a negative impact lasting months or even years. In contrast, acute stressors, such as public speaking or examinations, are short lived, lasting minutes to hours, and typically elicit fight-or-flight responses. In PNI research, chronic stressors can be investigated by comparing the health and/or immune function of a group of participants who have been exposed to a particular stressful life event to a non-stressed matched control group. Alternatively, questionnaires can be used to assess the extent of the chronic stress experienced by an individual, either by indicating how many of a checklist of life events they have experienced in a set period of time or through more subjective measures such as perceived stress. Acute stress exposure can be manipulated experimentally; for example, psychological and immunological measures can be timed to coincide with naturalistic and predictable acute stressors, such as examination periods. In addition, there are a variety of validated laboratory procedures, such as the Trier Social Stress Task and the Paced Auditory Serial Addition Task, which can be used to induce acute stress in order to examine its immunological consequences.

Physiological Effects of Stress

Although a multitude of factors are likely to underpin the effects of stress on health, stress-induced alterations in immune function are clearly an important mechanism. Stress triggers a cascade of physiological responses, which provide a plausible pathway linking psychological factors with immune function and subsequent health. The two neuroendocrine pathways that have received most attention in the PNI literature are the hypothalamic-pituitary-adrenal axis (HPA) and the sympathetic-adrenal-medullary (SAM) axis. HPA axis activation is initiated during stress at the paraventricular nucleus of

the hypothalamus which secretes corticotropin-releasing hormone (CRH). This causes the pituitary gland to release adrenocorticotropic hormone (ACTH), which in turn elicits the production of immunomodulatory glucocorticoids, such as cortisol, from the adrenal glands.

In the SAM axis, activation involves preganglionic sympathetic nervous system (SNS) neurons that descend along the spinal cord from nuclei in the brain stem. The release of the neurotransmitter acetylcholine from these neurons in the adrenal medulla stimulates chromaffin cells to secrete the catecholamine epinephrine. In contrast, postganglionic SNS fibers predominantly secrete the catecholamine norepinephrine upon activation. Therefore, activation of the SAM leads to the release of both epinephrine and norepinephrine. These catecholamines can influence immune cells as all leukocytes express adrenergic surface receptors. In addition, postganglionic nerve fibers directly innervate lymphoid organs including the thymus, spleen, lymph nodes, and bone marrow and therefore come into close proximity with immune cells (Glaser & Kiecolt-Glaser, 2005).

It should also be noted that immune cells can also influence the SAM and HPA axis and subsequently impact brain function and psychological responses. Inflammatory cytokines, released during infection, can access the central nervous system through leaky regions in the blood-brain barrier, via specific transport molecules on the brain epithelium, and through the activation of vagal afferent fibers. These cytokines influence neurotransmitter and CRH function and can induce symptoms of "sickness behavior," including anorexia, anhedonia, and reduced locomotor activity. It has been proposed that these changes in behavior reflect a reallocation of available resources, away from metabolically expensive activities such as foraging, toward behaviors likely to promote recovery from infection (Kelley et al., 2003).

Stress, Immune Function, and Disease

Psychological stress is implicated in the pathogenesis and exacerbation of many diseases and pathologies. For example, there is now evidence that stress can promote tumor growth and progression in a variety of cancers. These effects are likely to be mediated at a number of levels, including changes to local inflammatory signaling supporting initial tumor cell growth and proliferation and stress hormone-induced changes to cancer cell-matrix attachments, cell movement and invasion, angiogenesis, and sensitivity to apoptosis (Armaiz-Pena, Lutgendorf, Cole, & Sood, 2009).

There is also a substantial body of evidence linking psychological factors such as chronic stress, depression, and coping strategies with various markers of HIV progression. Further, behavioral interventions designed to improve psychological functioning have had some success in modifying disease-relevant markers of immune function (Antoni, 2003). It has been proposed that these associations between stress and HIV progression are likely mediated predominantly by changes in sympathetic nervous system activity, leading to alterations in cellular vulnerability to infection and innate antiviral responses (Cole, 2008).

The PNI models of cancer and HIV are both consistent with the model in which stress is associated with decrements in immune function which leave the host vulnerable to tumor growth or infection. However, there is also evidence that stress exacerbates diseases in which the overactivity, rather than underactivity, of the immune system is implicated. For example, stress has been proposed to be an "aggravating factor" in asthma, where those people with asthma exhibit worsened symptoms during periods of psychological stress (Chen & Miller, 2007). These observations are contrary to the intuitive perspective that a stress-induced reduction in immune activity would be beneficial for patients with conditions characterized by excessive inflammation. Instead, research has shown that in asthma, stress accentuates the inflammatory response in the airway induced by allergens and irritants, leading to more severe symptoms (Chen & Miller, 2007). Taken together, there is clear evidence that psychological stress impacts the prognosis in a variety of different diseases via neuroendocrine-immune mechanisms.

Stress and Immune Function in Healthy Populations

Psychological stress can also affect the immune function of otherwise healthy individuals. One of the most dramatic demonstrations of this phenomenon was a series of studies conducted by Sheldon Cohen in which he gave 394 healthy participants nasal drops containing live respiratory viruses. Psychological stress, measured by questionnaire, was associated in a dose-response manner with an increased risk of acute infectious respiratory illness. As the extent and timing of the exposure to the pathogen was controlled, these studies provide compelling evidence that stress impacts the individual's ability to protect themselves against infection. Later studies suggest that this effect is mediated by stress-induced disruption of the regulation of proinflammatory cytokines (Cohen, 2005).

However, a key challenge for scientists investigating stress and immune function in healthy people is choosing an appropriate outcome measure in the absence of the disease-specific indicators. The immune system comprises a complex array of different cell types that are required to orchestrate precise and appropriate responses to a wide range of pathogens, all within the context of a variable neuroendocrine milieu. Not only are these cells required to work together in order to produce an effective immune response, what constitutes "effective" may change depending on the nature of the pathogen. For example, different immunological responses are required for intracellular viruses, compared to bacteria or parasites. As such, it is difficult to establish meaningful measures of "effective immune function" in healthy participants. The wide range of psychological and immunological measures employed by researchers to date is demonstrated in an excellent meta-analysis by Segerstrom and Miller, which examines the results of over 300 PNI studies (Segerstrom & Miller, 2004).

This entry will focus on two in vivo measures of immune function, wound healing and antibody response to vaccination, in which an immunological "challenge" is administered and the subsequent immune response is assessed. These approaches assess the functionality of an orchestrated immune response, generated within the neuroendocrine milieu, and yield clinically relevant outcome measures. For example, the wound-healing process involves a complex series of processes, including inflammation, cellular migration and replication, and connective tissue deposition and remodeling; each stage is regulated by the cellular immune system. Wound healing can be assessed using naturalistic wounds, such as those experienced by patients with venous disease or by examining incisions administered during surgery. A more controlled method that also allows greater investigation of the mechanisms underpinning any stress effects involves administering an experimental wound and assessing the rate of healing over time. A recent systematic review and meta-analysis found that higher levels of psychological stress were consistently associated with impaired wound healing across a variety of different wound types (Walburn, Vedhara, Hankins, Rixon, & Weinman, 2009).

Antibody response to vaccination is another in vivo measure of immune function that has clear clinical implications. The controlled dose of an inactivated antigen induces a complex immune cascade that culminates in the production of specific antibodies; the extent of this response can be used as a measure of the functional status of the humoral immune system (Burns & Gallagher, 2010). There is now considerable evidence that chronic psychological stress is associated with a poorer antibody response to many different vaccinations (Cohen, Miller, & Rabin, 2001). However, more recent research has demonstrated that acute stress can, in contrast, augment the immune response to vaccination (Edwards, Burns, Carroll, Drayson, & Ring, 2007), potentially due to the rapid mobilization of immune cells during acute stress (Segerstrom & Miller, 2004).

Models of PNI

Although early PNI research focused on the immunosuppressive effects of stress, more recently, it has become apparent that the precise implications for the immune system are

dependent on the nature of the stressor. For example, the recent meta-analysis revealed that although chronic stressors were consistently associated with suppression of immune function, acute stressors often upregulated some parameters of immunity. Further, as chronic stress has been shown to affect diseases characterized by both reduced and excessive immune function, it is apparent that it may be the balance of immunological parameters that is critical (Segerstrom & Miller, 2004). As an added layer of complexity, there is also evidence that the effects of stress on immunity are not consistent across individuals, even when the stressor is identical; differences in personality traits and cognitive and affective responses may also account for some variation in stress-induced immune changes (Kemeny, 2009).

In terms of the implications of such changes, it is likely that stress-induced immune alterations are part of an adaptive response to threat that serves to enhance immunoprotection. This model postulates that situations of acute stress are likely, from an evolutionary perspective, to be associated with an increased risk of wounding or infection and, therefore, upregulation of immune function in such circumstances would be beneficial. If, however, this upregulation is directed against harmless allergens or self-antigens, or indeed it is dysregulated through chronic activation, it is likely to contribute to immunopathology (Dhabhar, 2002). An alternative, ecological model of human PNI has recently been proposed, which instead suggests that even chronic stress-induced immunosuppression is part of a coordinated effort to efficiently conserve resources, rather than a sign of dysregulation (Segerstrom, 2010). This ecological perspective emphasizes "phenotypic plasticity," in which the value of a trait, such as robust immune activity, varies according to the environmental circumstances. Thus, immunosuppression during times of chronic stress may simply reflect a change in organism priorities toward the pursuit or protection of resources; the cost of reduced immunoprotection may, in these circumstances, be offset by the longer term health benefits of having these resources.

Conclusion

An array of compelling evidence now indicates that stress is associated with changes in immune function in both healthy and patient populations. Plausible physiological pathways through which psychological factors can influence cells of the immune system have been identified and explored, although there is much still to determine. It is now important to more fully elucidate the clinical implications of these changes and to translate these findings into effective biopsychosocial interventions.

Cross-References

▶ Psychoneuroimmunology

References and Readings

Ader, R. (2000). On the development of psychoneuroimmunology. *European Journal of Pharmacology, 405*, 167–176.

Antoni, M. H. (2003). Stress management and psychoneuroimmunology in HIV infection. *CNS Spectrums, 8*, 40–51.

Armaiz-Pena, G. N., Lutgendorf, S. K., Cole, S. W., & Sood, A. K. (2009). Neuroendocrine modulation of cancer progression. *Brain, Behavior, and Immunity, 23*, 10–15.

Burns, V. E., & Gallagher, S. (2010). Antibody response to vaccination as a marker of in vivo immune function in psychophysiological research. *Neuroscience and Biobehavioral Reviews, 35*, 122–126.

Chen, E., & Miller, G. E. (2007). Stress and inflammation in exacerbations of asthma. *Brain, Behavior, and Immunity, 21*, 993–999.

Cohen, S. (2005). Keynote presentation at the Eight International Congress of behavioral medicine: The Pittsburgh common cold studies: Psychosocial predictors of susceptibility to respiratory infectious illness. *International Journal of Behavioral Medicine, 12*, 123–131.

Cohen, S., Miller, G. E., & Rabin, B. S. (2001). Psychological stress and antibody response to immunization: A critical review of the human literature. *Psychosomatic Medicine, 63*, 7–18.

Cole, S. W. (2008). Psychosocial influences on HIV-1 disease progression: Neural, endocrine, and virologic mechanisms. *Psychosomatic Medicine, 70*, 562–568.

Dhabhar, F. S., & McEwen, B. S. (1997). Acute stress enhances while chronic stress suppresses cell-mediated immunity in vivo: A potential role for

leukocyte trafficking. *Brain, Behavior, and Immunity*, *11*, 286–306.
Dhabhar, F. S. (2002). Stress-induced augmentation of immune function – The role of stress hormones, leukocyte trafficking, and cytokines. *Brain, Behavior, and Immunity*, *16*, 785–798.
Edwards, K. M., Burns, V. E., Carroll, D., Drayson, M., & Ring, C. (2007). The acute stress-induced immunoenhancement hypothesis. *Exercise and Sport Science Reviews, 35*, 150–155.
Fleshner, M., & Laudenslager, M. L. (2004). Psychoneuroimmunology: Then and now. *Behavior, Cognition, and Neuroscience Reviews, 3*, 114–130.
Glaser, R., & Kiecolt-Glaser, J. K. (2005). Stress-induced immune dysfunction: Implications for health. *Nature Reviews Immunology, 5*, 243–251.
Kelley, K. W., Bluthe, R. M., Dantzer, R., Zhou, J. H., Shen, W. H., Johnson, R. W., et al. (2003). Cytokine-induced sickness behavior. *Brain, Behavior, and Immunity, 17*(Suppl. 1), S112–S118.
Kemeny, M. E. (2009). Psychobiological responses to social threat: Evolution of a psychological model in psychoneuroimmunology. *Brain, Behavior, and Immunity, 23*, 1–9.
Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.
Maddox, J. (1984). Psychoimmunology before its time. *Nature, 309*, 400.
Segerstrom, S. C. (2010). Resources, stress, and immunity: An ecological perspective on human psychoneuroimmunology. *Annals of Behavioral Medicine, 40*, 114–125.
Segerstrom, S. C., & Miller, G. E. (2004). Psychological stress and the human immune system: A meta-analytic study of 30 years of inquiry. *Psychological Bulletin, 130*, 601–630.
Walburn, J., Vedhara, K., Hankins, M., Rixon, L., & Weinman, J. (2009). Psychological stress and wound healing in humans: A systematic review and meta-analysis. *Journal of Psychosomatic Research, 67*, 253–271.

Immunity

Jos A. Bosch
Department of Clinical Psychology, Faculty of Social and Behavioral Sciences, University of Amsterdam, The Netherlands

Synonyms

Immune function

Definition

Immunity (Latin: *immunitas*, "freedom from") describes a state of adequate defense against infection, i.e., bodily invasion by microorganisms. Immunity is established by the activities of the immune system (see entry on "▶ Immune Function"). Immunity also used to describe a state of adequate defense against neoplasms (cancers) insofar the immune system is involved. The entry on "▶ Immune Function" provides further details on the main types of immunity (innate and adaptive) and mechanisms by which the immune system establishes a state of immunity. Further information can be found in Abbas, Lichtman, and Pillai (2012) and Murphy (2011).

Cross-References

▶ Immune Responses to Stress
▶ Immunoglobulins

References and Readings

Abbas, K. A., Lichtman, A. L., & Pillai, S. (2012). *Cellular and molecular immunology* (7th ed.). Philadelphia: Elsevier, Saunders.
Murphy, K. (2011). *Janeway's immunobiology* (8th ed.). London: Taylor & Francis.

Immunoglobulins

Joanna Long
School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Antibodies

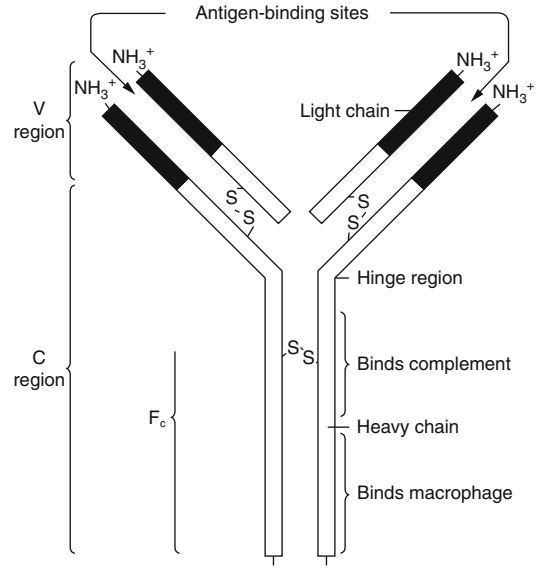
Definition

Immunoglobulins (Igs) are a subclass of antibodies which are produced in the early stages of

B-cell development (Abbas & Lichtman, 2006). The structure of Igs can differ between class types; however, they are built from a number of the same basic units known as immunoglobulin domains (Fig. 1). These are formed from 110 similar, but not identical, amino acid sequences which fold to form a globular structure. Each Ig is composed of two identical myosin heavy chain and two identical myosin light chain units, connected by a number of disulfide bonds. The Fab region is where different antigens bind to depending on the Ig subtype and is known as the antigen binding site. The first immunoglobulin domain possesses a large variability of amino acid sequences which determine the antigen-binding specificity of the antibody molecule, thus allowing great diversity of antigen recognition. The hinge region allows some flexibility of the molecule when binding with other cells. The Fc region is known as the constant region which aids modulation of immune cell activity.

Igs are a part of the immune system known as humoral immunity, which involves immunity conveyed by extracellular fluid such as plasma, lymph, and saliva. Basic functions of antibodies include neutralization, opsonization, and complement activation. The myosin heavy chain formation results in the subclass of antibody produced, of which there are 5 major classes: IgA, IgG, IgM, IgE, and IgD. Each isotype is selectively distributed throughout the body and differs in its effector functions. For example, IgG and IgM dominate in the circulation, IgA dominates within epithelial secretions (e.g., mucosa), and IgE is mostly found near the epithelial surfaces (e.g., skin).

Production of Igs within the immune system begins with the naïve B cell. As these B cells mature, they begin to express both IgM and IgD, and after antigenic activation, these Igs often switch to express a different heavy chain constant region. This is known as class switching and results in a change in effector function of the antibody produced. For example, a mature B cell may alter expression from IgM and IgD to IgG or IgE after antigen activation. IgE is formed by attachment to mast cells which are involved during allergic reactions.



Immunoglobulins, Fig. 1 Immunoglobulin structure (Newsholme & Leech, 2010) (Reprinted with permission)

During a primary response to infection, the major Ig subtype used is IgM, as the B cells are immature and not yet specific to a particular antigen. However, during this initial response, memory cells specific to that antigen are created. During the secondary antibody response, these memory cells are activated and secrete primarily IgG, IgE, or IgA. IgG is also secreted earlier during the secondary response, therefore creating a shorter lag period. This occurs for all T-dependent antigens due to B cells requiring costimulation with an activated antigen-presenting cell in order to produce Igs. T-independent antigens do not require the presence of T cells in order to produce an antibody response. However, due to the lack of cell signaling from the T cell, no isotype switching or generation of B-cell memory can occur. Thus, antibody response to T-independent antigens is almost exclusively IgM, and no secondary response occurs (Abbas & Lichtman, 2006).

Cross-References

► Antigens

References and Readings

Abbas, A. K., & Lichtman, A. H. (2006). *Basic immunology: Functions and disorders of the immune system* (2nd ed.). Philadelphia: Elsevier.

Newsholme, E., & Leech, T. (2010). *Functional biochemistry in health and disease* (2nd ed.). Chichester: Wiley-Blackwell.

Impaired Glucose Tolerance

Barbara Mullan
Centre for Medical Psychology & Evidence-based Decision-making, University of Sydney, Sydney, NSW, Australia

Synonyms

Prediabetes

Definition

The US National Diabetes Data group (1979) first introduced the term impaired glucose tolerance (IGT) to indicate a state of increased risk of progressing to diabetes. It was defined to reduce the stigma of being labeled with diabetes and was also noted that many individuals with IGT could revert back to normal.

According to the World Health Organization and the International Diabetes Foundation (World Health Organization, 2006), IGT is diagnosed if 2-h plasma glucose levels are between 7.8 and 11.1 mmol/l or 140 mg/dl and 200 mg/dl as measured by the oral glucose tolerance test, or if fasting glucose levels are less than 7.0 mmol/l. Two-hour plasma glucose is the level of glucose or the venous plasma glucose 2 h after ingestion of 75 g of oral glucose load. Blood glucose levels normally rise after eating a meal and then gradually fall as the meal is digested. However, in people with impaired glucose tolerance, these levels remain elevated.

In comparison, the definition of type II diabetes mellitus is 2-h plasma glucose levels more that 11.1 mmol/l (200 mg/dl). Therefore, IGT is not a clinical entity but is a risk factor for future diabetes and/or adverse outcomes and denotes individuals in the range between "normal" and diabetes.

Description

Characteristics

IGT is associated with muscle insulin resistance and defective insulin secretion, resulting in less efficient disposal of the glucose load (World Health Organization, 2006). It has been shown that carrying extra body fat inhibits the effectiveness of insulin therefore any glucose that does reach the muscle will have its journey delayed. If this continues untreated, then blood glucose levels will eventually increase into the diabetes range.

It has also been recognized that IGT is associated with increased risk for cardiovascular disease (Fuller, Shipley, Rose, Jarrett, & Keen, 1980). The Whitehall study found that coronary heart disease mortality was approximately double for individuals with IGT in a population of 18,403 London civil servants aged 40–64 (Fuller et al.).

Prevalence of IGT

The prevalence of IGT varies between populations and across different age groups. For example, in the United States, 15.2% of men and 16.4% of women have impaired glucose tolerance (Harris et al., 1998), and in Australia, the rates were found to be 12% in men and 14% in women (Dunstan et al., 2002). A WHO study found that rates were lowest among some Chinese, traditional American Indian, and Pacific island populations (<3%) and the highest among female Muslim Asian Indians in Tanzania (32%) and male Micronesians in Kiribati (28%) (King & Rewers, 1993).

Prevalence rates of approximately 10% or more are common, and it is typically more common in women than in men. The increasing prevalence with age was illustrated in the DECODE study which showed the prevalence of isolated IGT increasing from 2.9% in 30–39-year-old men to 15.1% in 70–79-year-old men

and from 4.5% in 30–39-year-old women to 16.9% in 70–79-year-old women.

Risk Factors

The prevalence of IGT and diabetes appears to have increased in the last 20 years, and this has been linked to the rise of obesity in many countries. Further, health-risk behaviors such as physical inactivity, smoking, and alcohol ingestion are likely to be important and closely interrelated with IGT. As aforementioned, the risk of IGT is also age related.

Women with polycystic ovary syndrome are also more at risk of IGT as they have profound insulin resistance independent of obesity. Insulin resistance is a major risk factor in developing diabetes mellitus type II.

The American Diabetes Association also listed other risk factors for IGT and diabetes which include having a parent or sibling with diabetes; having a family background that is African American, Alaska Native, American Indian, Asian American, Hispanic/Latino, or Pacific Islander; giving birth to a baby weighing more than 9 lb or being diagnosed with gestational diabetes (diabetes first found during pregnancy); having high blood pressure (140/90 or above or being treated for high blood pressure); having a high density lipo-protein (HDL), or "good," cholesterol level below 35 mg/dL or a triglyceride level above 250 mg/dL; having other conditions associated with insulin resistance, such as severe obesity or Acanthosis nigricans; and having a history of cardiovascular disease.

Prevention and Treatment

People with IGT have a one in three chance of developing type II diabetes within 10 years, but this can be minimized through healthy eating and physical activity as this helps cells respond better to insulin. The Diabetes Prevention Program (Orchard et al., 2005) carried out a randomized control trial to compare an intensive lifestyle modification intervention and the drug metformin. The study found that losing just 5–7% of body weight through a healthy low-calorie, low-fat diet and to engage in physical activity of moderate intensity reduced the incidence of metabolic syndrome (three or more of the following characteristics: waist circumference, blood pressure, levels of high-density lipoprotein cholesterol, triglycerides, and fasting plasma glucose) by 41%. Many participants in the lifestyle intervention group returned to normal blood glucose levels and lowered their risk for developing heart disease and other problems associated with diabetes. In comparison, the diabetes drug metformin reduced the risk of developing diabetes by 17%.

References and Readings

Dunstan, D., Zimmet, P., Welborn, T., de Courten, M., Cameron, A., Colagiuri, S., et al. (2002). The rising prevalence of diabetes and impaired glucose tolerance: The Australian diabetes, obesity and lifestyle study. *American Diabetes Association, 25*(5), 829–834.

Fuller, J. H., Shipley, M. J., Rose, G., Jarrett, R. J., & Keen, H. (1980). Coronary-heart-disease risk and impaired glucose tolerance The Whitehall Study. *The Lancet, 315*(8183), 1373–1376.

Harris, M. I., Flegal, K. M., Cowie, C. C., Eberhardt, M. S., Goldstein, D. E., Little, R. R., et al. (1998). Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in US adults. The third national health and nutrition examination survey, 1988–1994. *Diabetes Care, 21*(4), 518.

King, H., & Rewers, M. (1993). Global estimates for prevalence of diabetes mellitus and impaired glucose tolerance in adults. WHO Ad Hoc Diabetes Reporting Group. *Diabetes Care, 16*(1), 157.

National Diabetes Data Group. (1979). Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. *Diabetes, 28*, 1039–1057.

Orchard, T. J., Temprosa, M., Goldberg, R., Haffner, S., Ratner, R., Marcovina, S., et al. (2005). The effect of metformin and intensive lifestyle intervention on the metabolic syndrome: The diabetes prevention program randomized trial. *Annals of Internal Medicine, 142*(8), 611.

World Health Organization. (2006). *Definition and diagnosis of diabetes mellitus and intermediate hyperglycemia*. Report of a WHO/IDF consultation. Retrieved November 1, 2010, from http://www.who.int/diabetes/publications/en/

Impairment

► Disability

Implementation

▶ Dissemination
▶ Translational Behavioral Medicine

Implementation Intentions

Peter M. Gollwitzer and Gabriele Oettingen
Department of Psychology, New York University, New York, NY, USA

Definition

Implementation intentions are if-then plans that spell out in advance how one wants to strive for a set goal. For the if-component, a critical cue is selected (e.g., a good opportunity, an anticipated obstacle) that is linked to a goal-directed response in the then-component. Implementation intentions are known to enhance the rate of goal attainment. They do so by delegating action control to situational cues thus endowing action control with features of automaticity.

Description

Successful goal pursuit requires solving both of two subsequent tasks: first, strongly committing to goals, and then, effectively implementing them. Accordingly, strongly committing to a goal is a necessary but not sufficient step towards goal attainment. Indeed, effective goal pursuit may be hampered by various problems such as failing to get started and to stay on track as well as overextending oneself. Finally, people may fail to disengage from futile means and unattainable goals. Meta-analytic findings suggest that goals (also referred to as goal intentions) account for no more than 28% of variance in goal-directed behavior (Sheeran, 2002). One remedy to impaired goal pursuit is – after one has strongly committed to a goal – to plan out in advance how one wants to deal with potential critical situations (i.e., by adding implementation intentions to one's goal intentions).

Gollwitzer (1999) highlighted the importance of furnishing goal intentions with implementation intentions. While goal intentions (goals) have the structure "I intend to reach Z!" with Z relating to a desired future behavior or outcome, implementation intentions have the structure "If situation X is encountered, then I will perform the goal-directed response Y!" Thus, implementation intentions define when, where, and how one wants to act on one's goal intentions. In order to form an implementation intention, individuals need to identify a goal-relevant situational cue (such as a good opportunity to act, or an obstacle to goal striving) and link it to an instrumental goal-directed response. Goal intentions merely specify a desired future behavior or outcome. On the contrary, the if-component of an implementation intention specifies when and where one wants to act on this goal, and the then-component of the implementation intention specifies how this will be done. For instance, a person with the goal to reduce alcohol consumption might form the following implementation intention: "And whenever a waiter suggests ordering a second drink, then I'll ask for mineral water!" Empirical data supports the assumption that implementation intentions help close the gap between holding goals and attaining them. A meta-analysis based on close to a hundred studies shows a medium to large effect on increased rate of goal attainment ($d = .61$; Gollwitzer & Sheeran, 2006).

Implementation intentions facilitate goal attainment on the basis of *psychological mechanisms* that pertain to the specified situation in the if-part and to the mental link forged between the if-part and the specified goal-directed response in the then-part of the plan (Gollwitzer & Oettingen, 2011). Because forming an implementation intention implies the selection of a critical future situation, the mental representation of this situation becomes highly activated and hence more accessible. This heightened accessibility of the if-part of the plan has been observed in several studies using different experimental tasks (e.g., cue detection, dichotic listening, cued recall, lexical decision, flanker).

However, forming implementation intentions not only heightens the activation (and thus the accessibility) of the mental presentation of the situational cue specified in the if-component but it also forges a strong associative link between the mental representation of this cue and the mental representation of the specified response. These associative links seem to be quite stable over time, and they allow for activation of the mental representation of the specified response (the then-component) by subliminal presentation of the specified critical situational cue (if-component). Moreover, mediation analyses suggest that both cue accessibility and the strength of the cue-response link together mediate the impact of implementation intentions on goal attainment.

Gollwitzer (1999) suggested that the upshot of the strong associative links between the if-part (situational cue) and the then-part (goal-directed response) created by forming implementation intentions is that – once the critical cue is encountered – the initiation of the goal-directed response exhibits features of automaticity. These features include immediacy, efficiency, and redundancy of conscious intent. As a consequence, having formed an implementation intention allows individuals to act in situ without having to deliberate on whether to act or not. Indeed, there is vast empirical evidence that if-then planners act more quickly, deal more effectively with cognitive demands (i.e., speed-up effects still evidence under high cognitive load), and do not need to consciously intend to act in the critical moment. Consistent with this last assumption, implementation intention effects are observed even when the critical cue is presented subliminally or when the respective goal is activated outside of awareness.

The processes underlying implementation intention effects (enhanced cue accessibility, strong cue-response links, automation of responding) help if-then planners to readily see and to seize good opportunities to move toward their goals. Forming an if-then plan thus strategically automates goal striving. People can intentionally make if-then plans thus delegating control of goal-directed responses to preselected situational cues. This strategic automation hypothesis has recently been supported by studies that collected brain data using either electroencephalography (EEG) or functional magnetic resonance imaging (fMRI), suggesting that by forming implementation intentions, people can switch from top-down control of their actions via goals to bottom-up control via specified situational stimuli. Research on mediating processes has also supported the strategic automation hypothesis, albeit in an indirect way. Numerous studies indicated that neither an increase in goal commitment nor an increase in self-efficacy qualified as potential alternative mediators of implementation intention effects.

But what about *potential moderators* of implementation intention effects on goal striving and goal attainment? First, implementation intentions only benefit goal attainment when goal commitment is high; the same is true with respect to people's commitment to executing the formed implementation intention. In addition, self-efficacy was found to moderate implementation intention effects. Prompting participants to form an implementation intention as to when, where, and how to pursue their most important New Year's resolution (e.g., to engage in regular physical exercise) and in addition reflect on past mastery experiences (i.e., situations in which they achieved a similar goal) led to significantly higher levels of self-reported goal progress compared to a mere implementation intention condition. In a recent study where high versus low self-efficacy was manipulated (by asking participants to solve low- or high-difficulty goal-relevant tasks), it was observed that high-self-efficacy participants showed stronger implementation intention effects than low-self-efficacy participants, especially when the tasks to be solved were difficult rather than easy.

Finally, certain personal attributes have been found to moderate implementation intention effects. For instance, socially prescribed perfectionists (i.e., people who are known to try to conform to standards and expectations of others) show weaker implementation intention effects. Possibly social perfectionists may fail to commit to implementation intentions because they feel that social

expectations and standards will change quickly and unpredictably; flexible responding to such circumstances may be impeded by strong commitments to the preplanned course of action as specified in implementation intentions. Moreover, conscientiousness moderates implementation intention effects. Increases in goal attainment are only found for low conscientious individuals, whereas high conscientious individuals often show perfect goal attainment to begin with and thus goal attainment cannot be enhanced. The moderation of implementation intention effects by conscientiousness is in line with the common finding (Gollwitzer & Sheeran, 2006) that implementation intention effects are generally observed to be stronger for difficult than for easy goals.

Which aspects of goal striving have been found to benefit from forming implementation intentions? The effects of implementation intentions have been demonstrated with respect to getting started, staying on track, disengaging from faulty goals and means, as well as avoiding resource depletion (Gollwitzer & Oettingen, 2011). Implementation intentions were found to help individuals to get started with goal striving in terms of remembering to act (e.g., regarding taking vitamin pills, contraceptive pills, influenza vaccination), not missing opportunities to act (e.g., regarding obtaining a mammography), and overcoming an initial reluctance to act (e.g., regarding undertaking a testicular self-examination). Moreover, goals to perform regular breast examinations or cervical cancer screening and to resume activity after joint replacement surgery were all found to be more readily acted upon by individuals who previously had formed implementation intentions.

However, many health goals (e.g., eating a healthy diet, regular physical exercise, reducing alcohol consumption or smoking, downregulating anxiety) cannot be accomplished by a simple, discrete, one-shot action, because they require that people keep striving over an extended period of time. Staying on track may then become very difficult when certain internal stimuli (e.g., being tired, stressed out) or external stimuli (e.g., temptations, distractions) interfere with the desired goal pursuit. Implementation intentions can be used to protect started goal strivings from interferences stemming from both inside and outside the person. Such implementation intentions may use very different formats. For instance, if a person with the goal to eat healthy foods wants to stay firm with respect to seductive offers of unhealthy snacks, she can form suppression-oriented implementation intentions, such as "And if my colleague approaches me offering a snack, then I will not take the snack!" The then-component of such suppression-oriented implementation intentions does not have to be worded, however, as not showing the critical behavior (in the present example "not taking the snack"); it may alternatively specify a replacement behavior ("..., then I will ask for an apple!"), or focus on ignoring the critical cue ("..., then I'll ignore his offer!"). Recent research suggests that mere negation implementation intentions are less effective than the latter two types of implementation intentions (i.e., replacement and ignore implementation intentions).

Two further types of implementation intentions have been proven effective to master temptations and disruptions. The first one specifies the temptation as a situational cue and links it to thinking of the goal as the response in the then-component. The second one specifies an ongoing activity – that is independent of the temptation – as a situational cue and links it to continuing this activity as the response in the then-component. Using, again, the example of a person who has to cope with a seductive offer from a colleague, let us assume that the person already anticipated receiving the tempting offer during an upcoming encounter with this colleague; she therefore formed an implementation intention stipulating in advance what she will converse about when she runs into him. The interaction with the colleague can then come off as planned as the seductive offer will not have a chance to disrupt the course of action (i.e., the conversation).

Goal striving that is no longer promising may require individuals to disengage from a chosen means or the goal altogether. Such disengagement can free up resources and minimize negative

affect. However, individuals often stick to a chosen goal or means too long thus hurting themselves (e.g., setting a too demanding exercise goal, choosing improper means to reach the goal). Implementation intentions can be used to promote adaptive disengagement by (1) specifying negative feedback as a critical cue and (2) linking this cue to switching to a more promising alternative goal or means. Indeed, when research participants were asked to form implementation intentions that linked negative feedback on the ongoing goal striving to immediately switching to a different goal or means, or to reflecting on the quality of the received failure feedback on the ongoing goal striving, adaptive disengagement from goals and means was found to occur more frequently than for participants who had only formed respective goal intentions or had formed no intentions at all.

Finally, forming implementation intentions can help prevent resource depletion as it enables individuals to engage in automated goal striving and behavior control that does not require effortful deliberation (e.g., forming implementation intentions to ask for available vegetarian dishes when a waiter takes one's order). As a consequence, the self should not become depleted when goal striving is regulated by implementation intentions. Indeed, in studies using different ego-depletion paradigms, research participants who used implementation intentions to self-regulate performance on a difficult first task did not show reduced self-regulatory capacity in a subsequent task.

But how much willpower is actually afforded by forming implementation intentions? Any self-regulation strategy that claims to facilitate goal striving has to prove itself under conditions in which people commonly fail to demonstrate willpower. Such conditions are manifold (e.g., when one's competencies are challenged, opponents interfere with one's goal striving), but self-regulation of goal striving becomes particularly difficult when habitual responses are in conflict with initiating and executing the needed goal-directed responses that are instrumental to goal attainment. Can the self-regulation strategy of forming if-then plans help people to let their goals win out over their habitual responses? By assuming that action control by implementation intentions is immediate and efficient and adopting a simple horserace model of action control, people might be able to break habitual responses by forming implementation intentions (e.g., if-then plans that spell out a response contrary to the habitual response to the critical situation). Still, if the habitual response is based on strong habits (e.g., smoking) and the if-then guided response is based on weak implementation intentions, the habitual response should win over the if-then planned response. However, when weak habits are in conflict with strong implementation intentions, the reverse should be true. This implies that controlling behavior based on strong habits by forming implementation intentions requires that these if-then plans are very strong as well.

The strengthening of if-then plans can be achieved in various ways: One pertains to creating particularly strong links between situational cues (if-component) and goal-directed responses (then-component), for instance, by asking participants to use mental imagery. Alternatively, one may tailor the critical cue specified in the if-part of an implementation intention to personally relevant reasons for the habitual behavior one wants to overcome, and then link this cue to an antagonistic response (e.g., if I feel lonely, then I will put on the music in the living room rather than snack in the kitchen). Also, certain formats of implementation intentions (i.e., replacement and ignore implementation intentions) seem to be more effective in fighting strong habits than other if-then plans (e.g., negation implementation intentions). And finally, stronger implementation intention effects are observed when the respective goals are framed as approach rather than avoidance goals and when goals and plans match in their self-regulatory orientation (i.e., either promotion or prevention). Pertaining to the discussion of whether strong habits can be broken by implementation intentions, one should keep in mind that behavior change is possible without changing bad habits; one may also focus on - building new habits in new situational contexts. With respect to this latter approach,

implementation intentions can guide goal striving without having to outrun habitual responses. The delegation of control to situational cues principle, on which implementation intention effects are based, can then unfold its facilitative effects on goal striving in an undisturbed manner.

Trying to achieve behavior change by solely forming implementation intentions however forgets that effective behavior change demands a change in terms of both setting new goals and preparing the respective goal striving by forming implementation intentions. But how can people best select and commit to new goals? Oettingen (2012) has developed a self-regulation strategy of goal setting, called mental contrasting of future and reality that allows people to strongly commit to achieving desired and feasible future outcomes. Specifically, in mental contrasting, people imagine the attainment of a desired future (e.g., regular exercise) and then reflect on obstacles of present reality that stand in the way of attaining the desired future (e.g., not setting aside enough time). Given that the perceived chances of success (expectations of success) are high, people will actively commit to and strive toward reaching the desired future.

One recent behavior change intervention (called MCII; summary by Oettingen & Gollwitzer, 2010) combines mental contrasting (MC) with forming implementation intentions (II). To unfold their beneficial effects, implementation intentions require that strong goal commitments are in place and mental contrasting creates such strong commitments. Implementation intentions are also found to show enhanced benefits when the specification of the if-component is personalized, and mental contrasting guarantees the identification of personally relevant obstacles that can then be specified as the critical cue in the if-component of an implementation intention. Finally, mental contrasting has been found to create a readiness for making plans that link obstacles of present reality to instrumental goal-directed behaviors.

In recent intervention studies with middle-aged women, participants were taught the cognitive principles and individual steps of the MCII self-regulation strategy. Specifically, in one study, participants were asked to apply MCII by themselves to the wish of exercising more. Participants were free to choose whatever form of exercising they wished to engage in, and they were encouraged to anticipate exactly those obstacles that were personally most relevant. Finally, they had to link these obstacles to exactly those goal-directed responses that personally appeared to be most instrumental. Teaching the MCII technique enhanced exercise more than only providing relevant health-related information (i.e., information-only control intervention). Participants in the MCII group exercised nearly twice as much: an average of 1 h more per week than participants in the information-only control group. This effect showed up immediately after the intervention, and it stayed stable throughout the entire period of the study (16 weeks after the intervention). Conducting the same MCII intervention was also effective for promoting healthy eating in middle-aged women (i.e., eating more fruits and vegetables). The achieved behavior change persisted even over a period of 2 years. Follow-up research targeting the eating habit of unhealthy snacking was conducted with college students. It was observed that MCII worked for both students with weak and strong such habits, and it was more effective than either mental contrasting or forming implementation intentions alone. Moreover, MCII was observed to benefit chronic back pain patients in increasing their mobility over a period of 3 months, whereby physical mobility was measured by objective measures (e.g., bicycle ergometer test) as well as self-reported physical functioning.

In sum, MCII qualifies as a cost- and time-effective self-regulation intervention to enhance healthy and to prevent unhealthy behaviors. It helps to solve the two central tasks of goal pursuit: forming strong goal commitments on the one hand and following up on these commitments by effective goal implementation on the other. Not surprisingly, then, combining mental contrasting with implementation intentions offers additional advantages compared to each strategy alone.

Cross-References

- ▶ Behavior Change
- ▶ Habit Strength
- ▶ Intention

References and Readings

Gollwitzer, P. M. (1999). Implementation intentions: Strong effects of simple plans. *American Psychologist, 54*, 493–503.

Gollwitzer, P. M., & Oettingen, G. (2011). Planning promotes goal striving. In K. D. Vohs & R. F. Baumeister (Eds.), *Handbook of self-regulation: Research, theory, and applications* (2nd ed., pp. 162–185). New York \London: Guilford Press.

Gollwitzer, P. M., & Sheeran, P. (2006). Implementation intentions and goal achievement: A meta-analysis of effects and processes. *Advances in Experimental Social Psychology, 38*, 69–119.

Oettingen, G. (2012). Future thought and behavior change. *European Review of Social Psychology*, *23*, 1–63.

Oettingen, G., & Gollwitzer, P. M. (2010). Strategies of setting and implementing goals: Mental contrasting and implementation intentions. In J. E. Maddux & J. P. Tangney (Eds.), *Social psychological foundations of clinical psychology* (pp. 114–135). New York: Guilford Press.

Sheeran, P. (2002). Intention-behavior relations: A conceptual and empirical review. *European Review of Social Psychology, 12*, 1–30.

Stadler, G., Oettingen, G., & Gollwitzer, P. M. (2009). Physical activity in women. Effects of a self-regulation intervention. *American Journal of Preventive Medicine, 36*, 29–34.

Stadler, G., Oettingen, G., & Gollwitzer, P. M. (2010). Intervention effects of information and self-regulation on eating fruits and vegetables over two years. *Health Psychology, 29*, 274–283.

Impotence

- ▶ Erectile Dysfunction

Impulsive Behavior

- ▶ Impulsivity

Impulsivity

Kelly Winter
Epidemiology, Florida International University, Miami, FL, USA

Synonyms

Delay discounting; Disinhibition; Impulsive behavior

Definition

The meaning of this complex construct is widely debated, including whether it is a stable aspect of personality (trait) or a behavior (state). Most descriptions center on negative aspects and include a reference to behavior executed rapidly without forethought and/or self-control, failure of attention, delay discounting, or probability discounting. There are four main types of impulsivity measures: observer-rated scales (e.g., diagnostic interviews), self-report questionnaires (e.g., Barratt Impulsiveness Scale), behavioral laboratory measures (e.g., reward-choice paradigms), and biological measures (e.g., event-related potentials).

Description

Reacting quickly without forethought can be prudent. For instance, a race-car driver whose split-second decision results in victory is exhibiting functional impulsivity, characterized by rapid response time. However, particularly in psychology, emphasis is placed on the causes and negative outcomes of impulsive behavior. For example, if the same driver skips a race to visit friends, he or she is engaging in dysfunctional impulsivity, characterized by an inability to sustain attention.

The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV TR) defines clinically significant impulsivity in terms of interference in daily functioning. In the description

of attention deficit/hyperactivity disorder (ADHD), impulsivity is referred to as "impatience, difficulty delaying responses,... and frequently interrupting... to the point of causing difficulties in social, academic, or occupational settings" (American Psychiatric Association, 2000, p. 86). Great overlap and confusion exists between the concepts of impulsive behavior (motivated by a desire to release tension and/or feel pleasure) and compulsive behavior (motivated by a desire to alleviate anxiety).

Delay and Probability Discounting

Devaluing an outcome (positive or negative) in relation to the length of time that will elapse before it occurs is known as delay discounting. Research has found that children prone to delay discounting – e.g., preschoolers who opt to have one marshmallow now instead of two after a short delay – are more likely to struggle socially and academically in later years (Madden & Bickel, 2009). Devaluing an outcome (reward or punishment) according to the likelihood of its occurrence is known as probability discounting. Both constructs are hypothesized to play key roles in impulsive behavior.

Evolutionary psychologists point out that these tendencies, which favor present rewards, likely benefited early humans, whose lives were often short and unpredictable. Missing out on a meal or a mate in the short term in hopes of finding a better option later could result in ruin under such circumstances. Research suggests that modern humans in economically disadvantaged or dangerous environments are more likely to engage in impulsive behavior, including delay and probability discounting (Grant & Potenza, 2011; Madden & Bickel, 2009).

Related Health Outcomes

Impulsivity is a key feature in many mental disorders (e.g., ADHD, addictions, mood disorders) and unhealthy behaviors (e.g., drunken driving, overeating). Along with sensation seeking, impulsivity plays a role in risky sexual behavior (e.g., unprotected sex, many partners) and alcohol and drug use, all of which increase HIV risk. It is also theorized that impulsivity may partially account for increased HIV prevalence among those with personality disorders, particularly of the antisocial and borderline varieties (Millon, Grossman, Millon, Meagher, & Ramnath, 2004).

Cross-References

- ▶ Addictive Behaviors
- ▶ Alcohol Abuse and Dependence
- ▶ Alcohol Consumption
- ▶ HIV Prevention
- ▶ Personality
- ▶ Risky Behavior
- ▶ Substance Abuse

References and Readings

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders (Revised* (4th ed.). Washington, DC: Author.

Dickman, S. J. (1996). Adverse (and beneficial) consequences of impulsive behavior. In R. S. Feldman (Ed.), *Psychology of adversity* (pp. 197–214). Amherst, MA: The University of Massachusetts Press.

First, M. B., Frances, A., & Pincus, H. A. (2000). Chapter 2: Differential diagnosis by the trees. In *DSM-IV-TR handbook of differential diagnosis*. Washington, DC: American Psychiatric Press. Retrieved from http://www.psychiatryonline.com/content.aspx?aID=119073;.

Grant, G. E., & Potenza, M. N. (Eds.). (2011). *The Oxford handbook of impulse control disorders*. New York: Oxford University Press.

Madden, G. J., & Bickel, W. K. (Eds.). (2009). *Impulsivity: The behavioral and neurological science of discounting*. Washington, DC: American Psychological Association.

Millon, T., Grossman, S., Millon, C., Meagher, S., & Ramnath, R. (2004). *Personality disorders in modern life* (2nd ed.). Hoboken, NJ: Wiley.

Moeller, F. G., Barratt, E. S., Dougherty, D. M., Schmitz, J. M., & Swann, A. C. (2001). Psychiatric aspects of impulsivity. *American Journal of Psychiatry, 158*, 1783–1793.

Stanford, M. S., Mathias, C. W., Dougherty, D. M., Lake, S. L., Anderson, N. E., & Patton, J. H. (2009). Fifty years of the Barratt impulsiveness scale: An update and review. *Personality and Individual Differences, 47*, 385–395.

Webster, C. D., & Jackson, M. A. (Eds.). (1997). *Impulsivity: Theory, assessment, and treatment*. New York: Guilford Press.
Zuckerman, M. (1994). *Behavioral expressions and biosocial bases of sensation seeking*. New York: Cambridge University Press.
http://www.impulsivity.org. Accessed Feb 11, 2011.

In Vitro Fertilization

▶ Infertility and Assisted Reproduction: Psychosocial Aspects
▶ In Vitro Fertilization, Assisted Reproductive Technology

In Vitro Fertilization, Assisted Reproductive Technology

Jason S. Yeh[1] and Susannah D. Copland[2]
[1]Obstetrics and Gynecology, Division of Reproductive Endocrinology and Fertility, Duke University Medical Center, Durham, NC, USA
[2]Obstetrics and Gynecology, Division of Reproductive Endocrinology and Fertility, Duke Fertility Center, Durham, NC, USA

Synonyms

ART; Assisted reproductive technology; Egg donation; Egg donor; Embryo donation; Gestational carrier; ICSI; In vitro fertilization; Intracytoplasmic sperm injection; IVF; PGD; Preimplantation genetic diagnosis; Sperm donation; Sperm donor; Surrogacy; Surrogate

Definition

In vitro fertilization (IVF) is a method of assisted reproductive technology (ART) in which eggs are exposed to sperm in a laboratory, and the resulting embryos are placed into the uterus to achieve a pregnancy.

Description

In vitro fertilization (IVF) is a method of assisted reproductive technology (ART) in which eggs are exposed to sperm in a laboratory, and the resulting embryos are placed into the uterus to achieve a pregnancy. In order to obtain eggs, female patients take medications to stimulate available eggs to move forward toward ovulation. Ultrasound and blood testing monitor the ovarian response. Ultrasound-guided egg retrieval is performed when the eggs are thought to be mature. Once removed, the eggs are exposed to sperm in the laboratory, or in vitro (from the Latin root 'within glass'). Eggs can either be placed with sperm and incubated overnight (conventional in vitro fertilization) or a single sperm can be injected into each egg (in vitro fertilization with intracytoplasmic sperm injection or ICSI). ICSI is usually used to compensate for low levels or poorly functioning sperm. After incubating overnight, embryos are examined to see if fertilization occurred. The fertilized embryos are returned to the incubator for an additional 1–5 days, until they are either transferred into the uterine cavity or frozen for future use. The number of embryos transferred depends largely on the age of the recipient woman and the family planning goals of the intended parents. The American Society of Reproductive Medicine has published guidelines to limit the number of transferred embryos to reduce the likelihood of multifetal gestations. Some countries legislate the maximum numbers of embryos to transfer to reduce the risk of multiple births, instead favoring additional cycles using previously frozen eggs or embryos. Beginning with ovarian stimulation and ending with embryo transfer, one IVF cycle can be accomplished in an approximately 2-week time period; a pregnancy test occurs 2 weeks after the embryo transfer.

Pregnancy rates per IVF cycle vary widely, and clinics have published success rates that range from 10% to 70%. These rates depend, among other factors, on the woman's age, ovarian reserve, medical comorbidities, capabilities of the laboratory facility, and the technical expertise of the laboratory staff. Pregnancy rates can be reported in many ways: pregnancy or live birth per IVF cycle, per egg retrieval or per embryo transfer.

The percentage of IVF cycles that result in the delivery of a viable fetus is called the live birth rate.

Frozen embryos can be thawed for future pregnancies. Women take medications to prepare their uterus for embryo transfer, but do not need to repeat ovarian stimulation. Once a patient or couple completes their family, unused frozen embryos can be donated (to other people or to research) or discarded.

Third Party Reproduction

In vitro fertilization has created opportunities for third party reproduction where another party provides sperm, eggs, embryos, or the use of their uterus so a person or couple can become parents. There are four distinct entities of third party reproduction; some patients require the combination of multiple entities.

Gestational Carrier: A gestational carrier is a woman who agrees to carry, labor, and deliver a child for another person or couple. Gestational carriers assist intended parents who do not have a uterus or in whom pregnancy is medically unsafe. Carriers are usually financially compensated for their services.

Egg Donation: Egg donors undergo ovarian stimulation and egg retrieval to provide an egg for fertilization in the IVF process. The egg can be fertilized with either donated or the intended father's sperm. The resulting embryo is then transferred into the intended mother or gestational carrier. Egg donors can be known or anonymous. Anonymous donors and some known donors are usually financially compensated for services.

Sperm Donation: Sperm donors provide sperm that can be placed inside a uterus to inseminate a woman (intrauterine insemination or IUI) or used to create an embryo by in vitro fertilization that can then be transferred into the uterus of an intended mother or gestational carrier. Generally, sperm donors are financially compensated for their services.

Embryo Donation: Unused embryos from previous IVF cycles can be donated to another patient or couple hoping to become parents. These embryos can be donated in fresh or frozen form and can be transferred into the intended mother or gestational carrier.

The American Society of Reproductive Medicine recommends the routine use of legal and psychological counseling when planning third party reproduction.

Expansions, Controversies, and Disadvantages of In Vitro Fertilization

As cancer treatments have become more successful, IVF has found a role in trying to preserve a woman or man's fertility prior to undergoing cancer treatment. Since radiation and chemotherapy are toxic to egg and sperm, men and women with cancer are often unable to reproduce after undergoing cancer treatments. Eggs, sperm, or embryos can be frozen prior to chemotherapy to facilitate pregnancy after they complete their cancer treatments. This emerging field of science within infertility is called oncofertililty.

Another adjunct to the IVF process is preimplantation genetic diagnosis (PGD) where genetic testing is used to screen embryos for a genetic disease carried by one or both of the parents. Embryos that are continuing to grow in the laboratory are biopsied on the third or the fifth day after egg retrieval. The embryo biopsy is then tested for the genetic difference the couple carries (e.g., mutations that would cause cystic fibrosis or sickle cell disease). Selection of embryos after PGD can prevent delivery of a child affected with a genetic disease. Use of PGD for gender selection or family balancing is highly controversial.

The combination of assisted reproductive technology and third party reproduction has allowed postmenopausal women to become pregnant at any age. The main concerns with pregnancy at greater age are the increased health risks to the mother during pregnancy and shorter time to parent because of her advanced age. Proponents contend that more important than a parent's age is the commitment to a child's well-being. Some countries do not legally allow postmenopausal women to become pregnant which has resulted in some patients traveling abroad to pursue IVF.

Frequently reported disadvantages of IVF and ART include high cost, increased rate of multiple gestations (twins, triplets, etc.), inability to guarantee success, and medications and procedures

that impart medical risk for the female patient. A single IVF cycle can cost upward of US $8,000–$15,000. In the United States, insurance coverage varies between states. A few states have mandated coverage in which insurers providing pregnancy-related benefits must also provide for the diagnosis and treatment of infertility including IVF. This discrepancy between states within one country exemplifies the ongoing debate as to whether or not parenthood is a right and fertility treatments should therefore be paid for with government funding, or a privilege that only the medically insured and/or affluent can afford.

Cross-References

► Infertility and Assisted Reproduction: Psychosocial Aspects

References and Readings

National Collaborating Centre for Women's and Children's Health. (2004). *Fertility: Assessment and treatment for people with fertility problems* (pp. 1–216). London: RCOG Press.

Speroff, L., & Fritz, M. A. (2010). Chapter 32: Assisted reproductive technologies. In L. Speroff & M. A. Fritz (Eds.), *Clinical gynecologic endocrinology and infertility* (8th ed., pp. 1331–1382). Philadelphia: Lippincott Williams and Wilkins.

Steptoe, P. C., & Edwards, R. G. (1978). Birth after the reimplantation of a human embryo. *Lancet, 2*, 366.

Inactivity

► Lifestyle, Sedentary

Incarcerated Youths

► Williams LifeSkills Program

Incidence Study

► Follow-up Study

Income Distribution

► Income Inequality and Health

Income Inequality and Health

Douglas Carroll
School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Income distribution

Definition

Income inequality is usually defined as the proportion of national income and benefit enjoyed by a specified proportion, say 50%, of the least well-off in society. Occasionally, it has been measured using something called the Gini coefficient, which is a statistical measure of dispersal. Applied to national or state income distribution, a Gini coefficient of 0 would reflect total equality, whereas 1 reflects complete inequality. Egalitarian countries such as Japan and Sweden have Gini coefficients ca. 0.25, whereas more unequal countries like the UK and USA have coefficients ca. 0.36 and 0.41, respectively.

Description

That health varies with socioeconomic position in Western societies is now commonplace. Irrespective of how socioeconomic position is measured, by occupational status, income, level of education, or neighborhood deprivation, it shows a consistent negative association with most measures of ill health, all-cause mortality, cardiovascular and cancer (with the exception of breast cancer in women) mortality, various major

morbidities, and subjective ill-being. The association, however, is not a matter of the very poorest in society suffering from very poor health while the rest enjoy uniformly good health. Rather, there is a continuous gradient linking health outcomes to fine gradations in socioeconomic position. The intimate relationship between sociodemographics and health is nowhere better illustrated than by studies showing that as societies becomes less egalitarian, i.e., the socioeconomic ladder gets steeper, so the contingent health inequalities increase.

Recently, this link between the socioeconomic inequalities and health has taken a step further, as a result of studies examining overall health indices in societies and how egalitarian or unequal those societies are. Studies have used two sorts of comparisons: income inequalities and health outcomes in Western countries (including Japan), such as life expectancy and mortality rate, and income inequalities and health outcomes in the USA. The leading academic players in these two enterprises are Richard Wilkinson in the UK and George Kaplan in the USA. Income inequality is variously measured in these studies, but the outcome is the same regardless.

Whatever the measure used, income distribution has been shown to be strongly associated with differences in population life expectancy and mortality rates among Western countries and states in the USA. Put simply, the populations of more egalitarian countries and states live longer. Egalitarian countries, such as Japan and the Scandinavian countries, also enjoy a host other benefits such as less obesity, lower teenage pregnancy rates, less violent crime, and higher childhood literacy rates; more equal countries even recycle more. Egalitarian states in the USA, such as New Hampshire, Vermont, and Utah, experience less violent crime and have lower rates of smoking and sedentary behavior than more unequal states, such as Alabama, Louisiana, and Mississippi.

Why might more equal countries and states apparently do so much better than unequal ones on a range of health and behavioral outcomes? Two notions have attracted consideration. The first is the idea of social capital, i.e., that more equal countries and states invest more and/or invest more inclusively and effectively in those aspects of the social infrastructure that are health promoting, such as education, health care, safe roads and transport, and so on. The other idea is that of social cohesion. It would appear that less egalitarian societies are less cohesive, engendering among their populations poorer social support and greater psychological distress, factors that have been linked to poorer physical health outcomes. Whatever the case, it is clear that not only does income inequality have a powerful effect on population health, but as income equality changes over time, so too does population health,; i.e., there are relative health benefits in a society becoming more egalitarian. Undoubtedly, the best summary of the recent research on income inequality and its consequences is by Wilkinson and Pickett (2009).

References and Readings

Wilkinson, R. G., & Pickett, K. (2009). *The spirit level: Why more equal societies almost always do better*. London: Allen Lane.

Incremental Cost-Effectiveness Ratio (ICER)

▶ Cost-Effectiveness

Independent Living

▶ Assisted Living

Independent Treatments Group Design

▶ Parallel Group Design

Indicators

► Symptoms

Individual Difference Factors

► Personality

Individual Differences

Austin S. Baldwin and Valerie G. Loehr
Department of Psychology, Southern Methodist University, Dallas, TX, USA

Synonyms

Differential psychology

Definition

Individual differences in behavioral medicine include demographic, psychological, and/or behavioral dimensions that are shared by all people, but upon which individuals differ in ways that influence their health. For example, evidence suggests that five personality traits (openness to experience, conscientiousness, extraversion, agreeableness, and neuroticism) are shared among all people, but differences people exhibit on some of these traits (e.g., conscientiousness) account for differences in healthy behavioral practices, and even mortality rates.

Description

Overview

Individual differences that are known to influence health include demographic factors (e.g., race, gender, SES), psychological traits (e.g., conscientiousness, neuroticism), and behavioral dispositions (e.g., anger/hostility, coping styles). These types of individual differences influence health outcomes in a variety of ways. For example, individual differences are known to moderate how people respond to stress, account for differences in the prevalence rates of chronic illness (e.g., coronary heart disease), account for differences in healthy behavioral practices (e.g., dietary habits, regular medical screenings), and even for differences in mortality rates.

The value of considering the role of individual differences in behavioral medicine is twofold. First, because individual differences are often associated with different patterns of behavioral and physiological responses (e.g., how people respond to stress), individual differences provide a useful framework through which researchers and practitioners can understand how different behavioral and physiological responses influence health. Second, individual differences allow researchers and practitioners to identify dimensions on which different people might benefit from different interventions and treatments.

Associations Between Individual Differences and Health

The sections below include descriptions of how selected individual differences are associated with health. Descriptions of differences in demographic factors, psychological traits, and behavioral dispositions are used as examples.

Demographic Factors

Race/Ethnicity. A variety of health differences exist among different minority race/ethnic groups in the United States, including African Americans, Asian Americans and Pacific Islanders, Latinos, and Native Americans (Whitfield, Weidner, Clark, & Anderson, 2002). Brief descriptions of the differences that exist among each of these groups are provided.

African Americans have higher mortality rates compared to Caucasians, and this difference may be due primarily to higher rates of heart disease and stroke (Potts & Thomas, 1999). Related to these observations, African Americans also show greater cardiovascular reactivity to stress (Clark, Moore, & Adams, 1998). Moreover, there is

evidence that African Americans also manifest higher rates of smoking, obesity (at least among women), risky sexual behavior, and lower rates of physical activity, all of which contribute to the mortality rate.

Compared to non-Latinos, Latinos have higher rates of obesity, diabetes, and hypertension, all of which are risk factors for cardiovascular disease. These higher rates are likely due to diets low in fruits and vegetables and high smoking rates. Latinos also have higher rates of alcohol use compared to non-Latinos, but report less risky sexual behavior. In addition, acculturation among Latino populations in the United States plays an important role in health outcomes. For example, Latinos who are acculturated in the United States (e.g., English is the primary language in the household) are more likely to smoke and to do so at greater rates (Navarro, 1996). More acculturated Latinos are also at greater risk of having stroke (Ontiveros, Miller, Markides, & Espino, 1999).

Asian Americans and Pacific Islanders have the best health profile among ethnic groups in the United States, and the limited evidence suggests that this is due, in part, to lower rates of tobacco and alcohol use. Acculturation among this population is also important to health. For example, new immigrants to the United States often experience stress due to language and employment difficulties (Nwadiora & McAddo, 1996).

Native Americans have higher mortality rates relative to other groups, due to higher smoking rates, poorer diets that lack sufficient fruits and vegetables, higher alcohol use, and lower rates of physical activity. Stress due to acculturation may also contribute to higher suicide rates (Lester, 1999).

Gender. Mortality rates are higher for men than women, and men are more prone than women to nearly all of the leading causes of death (e.g., cardiovascular disease, accidents). There is not strong evidence for biological differences, but there is evidence that men have higher rates of smoking, alcohol use, and accidents. Men also have a higher prevalence of hostility, making them more prone to cardiovascular disease (Weidner & Mueller, 2000), and men cope less effectively with stress than women. Moreover, compared to women, men tend to receive less social support that can act as a buffer against psychosocial stressors.

Socioeconomic status (*SES*). Across racial and ethnic groups, gender, and age, there is an inverse relationship between SES and disease morbidity and mortality. Although it has previously been speculated that SES-related differences in health may be due to the overlap between SES groups and racial and ethnic groups (e.g., African Americans, Latinos), SES has an independent effect on health. This effect is due, in part, to higher rates of smoking, obesity, poorer dietary habits, and lower usage rates of preventive health services.

Psychological Traits

Conscientiousness is one of the five factors in the "Big Five model" of personality and is characterized by the tendency to be dutiful, organized, and industrious. Evidence suggests that conscientiousness is associated with differences in mortality rates. In one study, children who were high in conscientiousness were shown to be more likely to live to old age than those low in conscientiousness (Friedman et al., 1995). There are a number of explanations for this association with mortality rates. Conscientious people are more successful in avoiding situations that could potentially harm them, tend to have high fitness levels and healthy dietary habits (Bogg & Roberts, 2004), and are more likely to follow medical recommendations and guidelines. In contrast, individuals low in conscientiousness are more likely to use alcohol, tobacco, and drugs, and engage in risky driving and sex. (Hampson, Goldberg, Vogt, & Dubanoski, 2006).

Neuroticism is another of the five factors in the "Big Five model" of personality. It is characterized by the tendency to experience negative emotions, and is associated with fearfulness, irritability, low self-esteem, social anxiety, poor inhibition of impulses, and helplessness. Elevated levels of neuroticism are associated with an increased risk for illness, psychological distress, and an increased mortality risk in patients with chronic medical conditions

(Christensen, Moran, Wiebe, Ehlers, & Lawton, 2002). Neuroticism also affects the perception of physical symptoms. Individuals high in neuroticism tend to recognize and report their symptoms more quickly, and often present an exaggerated picture of their symptoms (Ellington & Wiebe, 1999) that can comprise a physician's ability to effectively gage a patient's condition. They are also more likely to erroneously believe they have a serious disease. For example, up to 30% of those who did not have detectable heart disease after being referred for coronary angiography were high on neuroticism (Pryor, Harrell, Lee, Califf, & Rosati, 1983).

Hardiness is a collection of personality traits that moderate the negative effects of stress. Hardiness includes the tendency (1) for involvement in, as opposed to alienation from, whatever one encounters (commitment), (2) to not feel or act helpless in the face of adversity (control), and (3) to accept change as normal in life and an opportunity for growth rather than a threat to security (challenge). There is evidence that under stressful conditions, hardy individuals are less likely to get sick than non-hardy individuals, more likely to perceive negative situations positively, and show less physiological strain than non-hardy individuals (Contrada, 1989; Maddi, 1998). However, evidence for the associations between hardiness and objective markers of physical health has been inconsistent.

Dispositional optimism is the tendency to believe that good things are likely to happen in one's life. People who are high in optimism have lower blood pressure, less risk of coronary heart disease, higher levels of pulmonary function in older men, less cancer mortality among the elderly, and less illness-related disruption of social and recreational activities among breast cancer patients compared to their pessimistic counterparts. Evidence suggests that optimism leads people to cope more effectively with stress, that in turn reduces their risk for illness and benefits their recovery from serious medical procedures. This may happen in different ways. First, the positive mood associated with optimism may lead to a state of physiological resilience. For example, the tendency to experience positive emotional states has been tied to greater resistance to the common cold (Cohen, Doyle, Turner, Alper, & Skoner, 2003). Second, an optimistic disposition may improve long-term prospects for psychological adjustment and health by promoting more active and persistent coping efforts (Segerstrom, Castañeda, & Spencer, 2003).

Behavioral Dispositions

Hostility is defined as a set of negative attitudes, beliefs, and appraisals of others that emerged from studies of Type A behavior patterns. Individual differences in hostility also include aggressive tendencies and anger. Evidence has found that individuals high in hostility are at a higher risk for coronary heart disease (CHD) and higher mortality rates than people lower in the disposition. Hostility is associated with heightened inflammatory reactions to stress and to metabolic syndrome, likely explaining its link to CHD (Niaura et al., 2002). Differences in hostility are also associated with increased rates of hypertension, stroke, and diabetes. Finally, evidence suggests that individuals high on hostility may take longer to physiologically recover from stress (Suarez et al., 1997).

Coping Styles. Individuals tend to cope with stress differently, through either avoidant or vigilant coping styles. Avoidant strategies tend to minimize the impact of the stressor by denying that a threat exists, suppressing unpleasant thoughts, or refusing to seek or attend to threatening information. In contrast, vigilant strategies involve actively seeking information about the stressor and how to deal with it. Each style has advantages and disadvantages in managing stress and its effects. Avoiding threatening events works effectively with short-term threats; for example, it may be beneficial in some medical procedures, such as blood donation or dental surgery. However, avoidance may not be useful for managing long-term or persisting threats. Evidence shows that a vigilant style allows individuals to engage in cognitive and emotional efforts needed to deal with long-term problems, but they may experience anxiety and heightened physiological reactivity as a result (Smith, Ruiz, & Uchino, 2000).

In contrast, the chronic use of avoidance may heighten the risk of the adverse effects of stressful life circumstances.

Pathways Through Which Individual Differences Influence Health

Various theoretical models suggest that individual differences, such as personality traits, influence health indirectly through their effect on people's appraisal of and coping with stressful circumstances. The differences in stress appraisal and coping then influence people's physiological and/or behavioral responses. It is the physiological and behavioral responses that then have a direct effect on illness and mortality. In other words, personality traits and behavioral dispositions are thought to indirectly effect health through their influence on stress appraisal, coping, and subsequent responses. Although there is some evidence for the individual behavioral and physiological mechanisms through which individual differences affect health outcomes, evidence for the entirety of these causal pathways is incomplete (Smith, 2006).

Evidence for Individual Difference–Based Interventions

One of the benefits of considering the role of individual differences in behavioral medicine is the ability to tailor interventions and treatments to different types of people. In the sections below, evidence in support of this approach is briefly described.

Matching Interventions to Coping Styles. Evidence suggests that patients indicate less distress during and after stress-inducing surgical procedures when they are given information and use coping strategies that match their preferred styles. For people who prefer a vigilant coping style, this includes detailed information about the procedure and directions to engage in direct forms of coping. For patients with a preference for avoidant coping, this includes providing minimal information about the procedure and directions to engage in avoidant forms of coping (Martelli, Auerbach, Alexander, & Mercuri, 1987; Miller & Mangan, 1983). Evidence from an intervention targeting mammograms suggests that women were more likely to obtain a mammogram when the message advocating mammography matched their preferred coping style – vigilant messages contained detailed information about the benefits of mammography, whereas avoidant messages contained just main points – than when the messages were not matched to coping style (Williams-Piehota, Pizarro, Schneider, Mowad, & Salovey, 2005).

Hostility-Reduction Interventions. There is evidence that patients with CHD who are also high on hostility benefit from a hostility-reduction intervention such that they reported less hostility and had lower resting blood pressure as a result (Gidron, Davidson, & Bata, 1999). There is also evidence that interventions aimed at reducing hostility and other CHD risk factors can have beneficial effects on heart rate reactivity to anger-inducing situations (Bishop et al., 2005).

A few important conclusions can be drawn from this intervention evidence. First, behavioral and physiological responses that stem from individual differences (e.g., anger/hostility) are mutable and can be changed via intervention. This highlights the importance of understanding the processes through which individual differences influence health, in order to screen for risk factors and target those that may be amenable to change. Second, behavioral interventions can be matched to individual differences in ways that facilitate optimal adjustment (e.g., matching to coping styles). It is not clear, however, whether all types of individual differences (e.g., neuroticism) would be amenable to targeted interventions.

Cross-References

- ▶ Coping
- ▶ Ethnic Differences
- ▶ Gender Differences
- ▶ Hardiness and Health
- ▶ Hostility, Cynical
- ▶ Neuroticism
- ▶ Personality
- ▶ Sex Differences

References and Readings

Bishop, G. D., Kaur, D., Tan, V. L. M., Chua, Y., Liew, S., & Mak, K. (2005). Effects of a psychosocial skills training workship on psychophysiological and psychosocial risk in patients undergoing coronary artery bypass grafting. *American Heart Journal, 150*, 602–609.

Bogg, T., & Roberts, B. W. (2004). Conscientiousness and health-related behaviors: A meta-analysis of the leading contributors to mortality. *Psychological Bulletin, 130*, 887–919.

Christensen, A. J., Moran, P. J., Wiebe, J. S., Ehlers, S. L., & Lawton, W. J. (2002). Effect of a behavioral self-regulation intervention on patient adherence in hemodialysis. *Health Psychology, 18*, 169–176.

Clark, V. R., Moore, C. L., & Adams, J. H. (1998). Cholesterol concentrations and cardiovascular reactivity to stress in African American college volunteers. *Journal of Behavioral Medicine, 21*, 505–515.

Cohen, S., Doyle, W. J., Turner, R. B., Alper, C. M., & Skoner, D. P. (2003). Emotional style and susceptibility to the common cold. *Psychosomatic Medicine, 65*, 652–657.

Contrada, R. J. (1989). Type A behavior, personality hardiness, and cardiovascular responses to stress. *Journal of Personality and Social Psychology, 57*, 895–903.

Ellington, L., & Wiebe, D. (1999). Neuroticism, symptom presentation, and medical decision making. *Health Psychology, 18*, 634–643.

Friedman, H. S., Tucker, J. S., Schwartz, J. E., Tomlison-Keasey, C., Wingard, D. L., & Criqui, M. H. (1995). Psychosocial and behavioral predictors of longevity. The aging and death of the "Termites.". *American Psychologist, 50*, 69–78.

Gidron, Y., Davidson, K., & Bata, I. (1999). Short-term effects of a hostility-reduction intervention on male coronary heart disease patients. *Health Psychology, 18*, 416–420.

Hampson, S. E., Goldberg, L. R., Vogt, T. M., & Dubanoski, J. P. (2006). Forty years on: Teachers' assessments of children's personality traits predict self-reported behaviors and outcomes at midlife. *Health Psychology, 25*, 57–64.

Lester, D. (1999). Native American suicide rates, acculturation stress and traditional integration. *Psychological Reports, 84*, 398.

Maddi, S. R. (1998). Hardiness. In E. A. Blechman & K. D. Brownell (Eds.), *Behavioral medicine and women: A comprehensive handbook* (pp. 152–155). New York: Guilford.

Martelli, M. F., Auerbach, S. M., Alexander, J., & Mercuri, L. G. (1987). Stress management in the health care setting: Matching interventions with patient coping styles. *Journal of Consulting and Clinical Psychology, 55*, 201–207.

Miller, S. M., & Mangan, C. E. (1983). Interacting effects of information and coping styles in adapting to gynecological stress: Should the doctor tell all? *Journal of Personality and Social Psychology, 45*, 223–236.

Navarro, A. M. (1996). Cigarette smoking among adult Latinos: The California Tobacco Baseline Survey. *Annals of Behavioral Medicine, 18*, 238–245.

Niaura, R., Todaro, J. F., Stroud, L., Spiro, A., III, Ward, K. D., & Weiss, S. (2002). Hostility, the metabolic syndrome and incident coronary heart disease. *Health Psychology, 21*, 588–593.

Nwadiora, E., & McAddo, H. (1996). Acculturative stress among Amerasian refugees: Gender and racial differences. *Adolescence, 31*, 477–487.

Ontiveros, J., Miller, T. Q., Markides, K. S., & Espino, D. V. (1999). Physical and psychosocial consequences of stroke in elderly Mexican Americans. *Ethnicity & Disease, 9*, 212–217.

Potts, J. L., & Thomas, J. (1999). Traditional coronary risk factors in African Americans. *The American Journal of the Medical Sciences, 317*, 189–192.

Pryor, D. B., Harrell, F. E., Lee, K. L., Califf, R. M., & Rosati, R. A. (1983). Estimating the likelihood of significant coronary artery disease. *American Journal of Medicine, 75*, 771–780.

Segerstrom, S. C., Castañeda, J. O., & Spencer, T. E. (2003). Optimism effects on cellular immunity: Testing the affective and persistence models. *Personality and Indvidiaul Differences, 35*, 1615–1624.

Smith, T. W. (2006). Personality as risk and resilience in physical health. *Current Directions in Psychological Science, 15*, 227–231.

Smith, T. W., Ruiz, J. M., & Uchino, B. N. (2000). Vigilance, active coping, and cardiovascular reactivity during social interaction in young men. *Health Psychology, 19*, 382–392.

Suarez, E. C., Shiller, A. D., Kuhn, C. M., Schanberg, S., Williams, R. B., Jr., & Zimmermann, E. A. (1997). The relationship between hostility and B-adrenergic receptor physiology in health young males. *Psychosomatic Medicine, 59*, 481–487.

Weidner, G., & Mueller, H. (2000). Emotions and heart disease. In M. B. Goldman & M. C. Hatch (Eds.), *Women and health* (pp. 789–796). San Diego, CA: Academic.

Whitfield, K. E., Weidner, G., Clark, R., & Anderson, N. B. (2002). Sociodemographic diversity and behavioral medicine. *Journal of Consulting and Clinical Psychology, 70*, 463–481.

Williams-Piehota, P., Pizarro, J., Schneider, T. R., Mowad, L., & Salovey, P. (2005). Matching health messages to monitor-blunting coping styles to motivate screening mammography. *Health Psychology, 24*, 58–67.

Inducible Nitric Oxide Synthase (iNOS)

► Nitric Oxide Synthase (NOS)

Infant Mortality

Linda C. Baumann and Alyssa Karel
School of Nursing, University of Wisconsin-Madison, Madison, WI, USA

Definition

Infant mortality rate is the number of infant deaths, defined as children under the age of 1 year old, per 1,000 live births in a given year. Infant mortality is used as a major health indicator globally because it is associated with numerous factors that determine a country's overall level of health that include maternal health, prenatal care access, healthcare system quality and access, public health practices, and socioeconomic conditions.

Description

Another indicator used to measure the health status of a country is the under five mortality rate. The under five mortality rate is the probability of a child born in a specific year or period dying before the age of 5, expressed as a number per 1,000 live births. The World Health Organization (WHO) Millennium Development Goals (MDG) has identified the reduction of under five mortality rate by two thirds as goal number 4 to be achieved by 2015.

Variations in infant mortality rates and under five mortality rates are monitored by global regions. Child mortality rates have fallen since 1990 in all country-income groups – with the rate of decline generally faster in high-income and middle-income countries than in low-income countries. Median child mortality fell by almost 50% between 1990 and 2008 in lower middle-income countries, but by only 31% in low-income countries. In 2010, all regions of the world reported infant mortality rates below 200 per 1,000 live births. These numbers reflect a global improvement from 1970, when 40 countries had rates that exceeded this level.

Globally, infant mortality and under five mortality rates are estimated from death registration data that is reported annually to the World Health Organization. Other sources of data come from household surveys and census, especially in countries for which death registration data is not readily available. World regions with the highest infant mortality and under five mortality rates are sub-Saharan Africa and south Asia. Uganda, an example of a country in the sub-Saharan Africa region, had an under five mortality rate of 135 and an infant mortality rate of 84 according to 2008 WHO data. Regions of the world with the lowest rates are western Europe, Australia, high-income areas of Asia-Pacific, and high-income areas of North America. The UK, New Zealand, and South Korea are other high-income countries that lag behind other countries of high-income levels. In 2008, the infant mortality rate in the USA was 7 per 1,000, and the under five mortality rate was 8 per 1,000. The country with the lowest rate was San Marino, a small country located on the European continent, with a rate of 1 death per 1,000 live births. For a list of infant and under five mortality rates by country: http://www.who.int/whosis/whostat/EN_WHS10_Full.pdf.

Reaching MDG 4 will require key interventions, which include access to prenatal and perinatal care, improved child nutrition, vaccines, prevention and management of diarrhea, pneumonia and sepsis, malaria control, and prevention and care of HIV/AIDS. These interventions will lessen the impact of the six conditions for which 90% of worldwide children deaths are attributed to: neonatal causes, pneumonia, diarrhea, malaria, measles, and HIV/AIDS. According to the World Health Statistics 2010, the key to reducing infant mortality rates is the reduction in neonatal mortality, defined as the number of deaths to infants under 28 days of age in a given year. Globally, 40% of deaths in children are attributed to neonatal mortality.

Variations in mortality rates can be attributed to a country's region, but also it is largely related to the level of income of a country, with poorer countries having higher rates. Least developed countries have infant mortality rates 10 times higher and under five mortality is 25 times higher compared to higher income countries. Not only between, but also within a country, infant mortality rates reflect disparities of factors such as race,

socioeconomic status, education, and housing. For example in the USA, the gap between infant mortality rates of white and African American babies is of concern since the rate for African Americans is twice that of the rate for whites.

References and Readings

Denburg, A., & Daneman, D. (2010). The link between social inequality and child health outcomes. *Healthcare Quarterly, 14*, 21–31.

Eliminating Disparities in Infant Mortality. (2007). *Office of Minority health and disparities*. Retrieved August 14, 2010, from http://www.cdc.gov/omhd/amh/factsheets/infant.htm

Rajaratnam, J. K., Marcus, J. R., Flaxman, A. D., Wang, H., Levin-Rector, A., Dwyer, L., et al. (2010). Neonatal, postneonatal, childhood, and under-5 mortality for 187 countries, 1970-2010: a systematic analysis of progress towards Millennium Development Goal 4. *The Lancet, 375*, 1988–2008.

Wong, D., Hockenberry, M., Wilson, D., Perry, S., & Lowdermilk, D. (2006). *Maternal child nursing care* (3rd ed.). St Louis, MO: Mosby Elsevier.

World Healh Organization. (2010). *MDG 4: Reduce child mortality*. Retrieved October 28, 2010, from http://www.who.int/topics/millennium_development_goals/child_mortality/en/

World Health Organization. (2010). *World health statistics 2010*. Geneva: Author.

Infection

► Infectious Diseases

Infectious Diseases

Mark T. Drayson[1] and Anna C. Phillips[2]
[1]College of Medical and Dental Sciences, University of Birmingham, Edgbaston, Birmingham, UK
[2]Sport & Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Infection

Definition

Infectious diseases are illnesses caused by a diverse range of microorganisms. The immune system fights against such infection. 10% of all deaths in the UK, approximately 50,000 people a year, are due to infections, overwhelmingly in the elderly and worldwide the proportion of people dying from infection is 25%. In impoverished tropical environments many children have a 1:8 chance of dying before their 5th birthday.

Description

Microorganisms which cause infection include DNA viruses, RNA viruses, bacteria, fungi, protozoa, and worms. They can also be classified according to whether they replicate inside or outside of cells. Differences between infectious agents and their sites of replication necessitate different immune mechanisms for their control. Table 1 shows the class of microorganism, where it replicates, examples, and the diseases they cause.

Mechanical Barriers to Infection

The skin provides a barrier against infection; when this is intact it provides a relatively effective protection – but it is easily breached by cuts, burns, insect bites and skin diseases. The far larger absorptive surfaces of the intestine and lungs are inevitably more fragile. Moist mucus is effective at trapping small dust particles and droplets containing bacteria or the spores of yeast and fungi. There is a markedly increased susceptibility to infection associated with defects in the mucus, as in cystic fibrosis. Congenital defects in the cilia are also associated with repeated lung infections; cilia waft mucus towards the main bronchi and trachea where it is removed by coughing.

The mouth and pharynx and upper part of the esophagus are protected by stratified squamous epithelium but the epithelium of the rest of the gut is a single layer thick. Mucus again serves to protect the intestinal wall and injury is rapidly repaired.

Infectious Diseases, Table 1 Microorganism types and the diseases they cause

Class and site of replication	Examples	Disease
DNA viruses (intracellular)	Varicella zoster	Chicken pox
	Epstein-Barr virus	Glandular fever
RNA viruses (intracellular)	Rhinoviruses	Common cold
	Rotaviruses	Gastroenteritis
Bacteria (extracellular) (Intracellular)	Staphylococci	Skin infections
	Streptococci	Tonsillitis, pneumonia, impetigo
	Salmonella	Gastroenteritis, enteric fever
	Mycobacteria	Tuberculosis, leprosy
Fungi (extracellular) (Intracellular)	Candida	Thrush
	Pneumocystis	Pneumonia
Protozoa (extracellular) (Intracellular)	Entamoeba	Colitis, liver abscess
	Plasmodium	Malaria
Worms (extracellular) (bowel)	Filarioidea	Filariasis
	Cestodes	Tapeworm

Drainage and Irrigation

Irrigation is an important means by which infection is inhibited. For example, the constant flow of urine from the kidneys helps prevent infection of the urinary tract. Blockage to the outflow of urine impairs renal function and there is a considerable danger that static urine will become infected. Tears, urine, saliva, bile, pancreatic, mucus, and even sebaceous secretions help protect the surfaces they flow over.

It follows that free drainage of secretions is important to avoid infection; blockage of the pharyngo-tympanic tube predisposes to middle ear infection – the clogged opening to a hair follicle may result in a boil.

Chemical Protection

Acid secretions of the stomach help to sterilise the partially-digested food that enters the small intestine. Many secretions contain substances that inhibit the growth or survival of microorganisms; lysozyme in tears is bactericidal and bile acids inhibit the growth of some microorganisms.

Normal Bacterial Flora

There are large numbers of bacteria on the skin, in the mouth and in the large intestine. Normally these are non-invasive commensal microorganisms that do not cause disease. The presence of this normal bacterial flora helps to inhibit the invasion of pathogenic bacteria. Loss of the normal flora associated with the use of antibiotics or excessive use of antiseptic solutions can provide an opportunity for pathogenic bacteria to colonise the vacated tissues. Staphylococcal enteritis is commonly associated with the use of antibiotics. Ingestion of large amounts of non-pathogenic bacteria, such as the lactobacilli found in yogurt, can help protect against this unwanted overgrowth of pathogens during antibiotic therapy.

Sensory Nervous System

This system is vital in protecting the body, for it warns of potential tissue damage. Impaired sensation, for example occurring secondary to lepromatous leprosy or diabetes can result in patients unknowingly damaging their skin and other tissues; the damage is compounded by secondary infections.

Good Public Health and Hygiene

Clean water supply, good nutrition and reasonable standards of hygiene in preparing food have made an immense contribution to the reduced incidence of infection and increased good health and longevity enjoyed by developed countries compared with parts of the world where these, at best, are available only erratically.

Factors Associated with Increased Susceptibility to Infection

Impairment of any of the protective systems listed above increases susceptibility to infection. Certain other factors should be considered.

The immune system cannot gain access to dead, avascular tissue; foreign bodies in the tissues can have the same effect. Examples of sites where infection can gain hold are: infarcts

(tissues that have died because their blood supply has been interrupted, e.g., by a blood clot); accumulations of fluid (e.g., bruises, abscesses, hydronephrosis) – it is important that these are drained; scar tissue such as fibrous adhesions in the peritoneal cavity following abdominal surgery; dead bone caused by lifting of the periosteum or trauma; collapsed lung tissue; other dead tissue produced by burns, trauma and chemical injury; blocked secretions and drainage e.g., middle ear infections, boils, and pyelonephritis.

Innate Immunity

The surfaces of the body (skin, mucous membranes) provide a barrier against infection. These physical barriers are reinforced by secretions. If these barriers are penetrated a wide variety of immune mechanisms utilising proteins and cells act to control and eradicate infection. These immune mechanisms are targeted by molecular recognition of the micro-organism. Micro-organisms are sufficiently different from mammalian tissues that a small range of molecules is sufficient to recognise almost any micro-organism and not interact with normal self. This finite range of molecules that recognise microrganisms is encoded in the mammalian genome and accounts for <100 of the 25,000 or so total genes. The immune mechanisms that are targeted by these molecules are classified as non-adaptive (or innate) immunity.

Adaptive (Specific) Immunity

There is a second category of immune mechanisms that is not effective when a new micro-organism is first encountered but which over a period of a week or two adapts to provide specific defence against that particular micro-organism. This specific defence system employs a variety of effector mechanisms which alone and more usually by enhancing non-adaptive immune mechanisms, efficiently control almost all infections. This is called adaptive (or specific) immunity and relies on cells called T and B, lymphocytes. These lymphocytes recognise micro-organisms by the T cell receptor and the B cell receptor respectively. The molecular part of the micro-organism that these receptors bind to is called antigen and so they are sometimes called antigen specific receptors.

Main Sources of Infectious Disease

Many bacteria have medical importance, and they can cause devastation in countries like Africa, such as *Salmonella*, but have only minor clinical importance in the West. Not all bacteria are bad in fact pure pathogens make up only a tiny number of bacterial species, although many bacteria can act as pathogens if the host becomes immunocompromised. Bacteria are essential for life and of all the cells in the body only 10% belong to the body, the overwhelming majority of the rest are bacteria, residing on surfaces such as skin, gut, mouth etc. In fact the mouth can have a greater concentration of bacteria than the gut or in faeces. This normal flora helps by, amongst other things, occupying sites pathogens would if they got the chance and there is competition between different bacterial species to reside on you as much as there is competition between bacteria and the host.

What is a Bacterium?

Bacteria are living organisms that are called prokaryotes because they lack a cell nucleus. They all contain DNA and RNA but the lack of a distinct nucleus means transcription and translation can occur concomitantly. Medically, it is important to understand the structure of a bacterium, particularly its external structure because this is the part of the organism most in contact with the host so it protects the bacterium (e.g., capsules can prevent some white blood cells called phagocytes from engulfing the bacterium) but it can also warn the host of an "intruder" (e.g., lipopolysaccharide is extremely pro-inflammatory at even tiny concentrations). Lastly, bacterial structure, particularly the cell wall, can reveal a mountain of information on what genus and species the bacterium is. Another key feature is that bacteria generally divide very rapidly, in test-tubes this generation time can be

once every 20 min (somewhat slower in vivo), although there are exceptions such as some mycobacterial species such as *M. tuberculosis* divide once a day. This compares with a division rate of about once every 6 h for a B cell and a generation for humans is 25 years. This means bacteria can evolve much more quickly than the host and it can also acquire resistance against drugs very quickly (sometimes through taking up extra bits of DNA called plasmids). This is why effective, controlled use of antibiotics is so important. To put this in context this means, in theory, 1 bacterium can become 10,000,000,000 after 12 h and every hour of delay means that there could be 10× more bacteria than before. This is why rapid treatment of aggressive infections is essential.

Bacteria come in many shapes and their shape can be used to help identify them. In 1884 a Danish boffin called Hans Christian Gram developed a technique using dyes that could differentiate between certain types of bacteria, this is the Gram's stain and historically has been very important in medicine. This is because it is speedy and economical. Although not infallible, it is very important medically because it can reveal what the bacterial species is, frequently without culturing the bug, by directly staining the biopsy or smear etc. and is able to differentiate at least four different types of bacteria: *Gram positive or Gram negative rods or cocci (round)*. It works by first adding crystal violet as a dye. This is then fixed by iodine. Addition of 95% ethanol or acetone allows the crysal violet-iodine complexes to wash away from Gram-negative bacteria but they get trapped in the thick, dehydrated peptidoglycan of the Gram-positive bacteria. Lastly a counterstain is added to visualise the Gram-negative bacteria. The whole process takes less than 5 min. Some bacteria fail to be detected by Gram staining, this may be because for instance they *lack peptidoglycan (Mycoplasma)* or have a complex cell wall (Mycobacteria). Under these circumstances different staining is required e.g., Ziehls-Neelsen stain for acid-fast mycobacteria.

What Infections are Caused by Bacteria?

Many previously destructive diseases such as syphilis, tuberculosis, tetanus and diphtheria are now well controlled in the West due to improvements in public health, vaccination and antimicrobial treatment. Elsewhere in the world they are a tragic burden that can stifle economic and social development. Some infections likely to be encountered in the West include those caused by: *Staphylococci, Staph. Aureus,* β haemolytic group A streptococci (*Strep. pyogenes* is the beast) cause many of the most severe infections, group B are usually problematic in new-born babies. *Mycobacterium tuberculosis* causes tuberculosis, Clostridia, which include: gram positive spore-forming anaerobic rods. *C. perfringens* – gas gangrene: *C. difficile* – pseudomembranous colitis. *Pseudomonas aeruginosa* – aerobic Gram-negative opportunist particularly in infections in burns. The list goes on and on: *E. coli* (Urinary Tract Infection, gut); *Campylobacter jejuni* (gut); *Salmonella* etc.

What is a Virus?

Viruses are submicroscopic, obligate intracellular parasites. They have no metabolic activity outside the host cell, and thus cannot be considered to be "alive." At the most basic level, a virus particle (virion) consists of a nucleic acid genome surrounded by a protein shell, which may also be surrounded by a membranous envelope. Virions cannot themselves "grow" or undergo division (as bacteria can) but are produced by assembly of preformed components.

Classification of Viruses

Viruses can be classified according to their genomes. These genomes can be more varied than those seen in the entire bacterial, plant or animal kingdom. Viral nucleic acid can be RNA or DNA; single stranded (ss) or double stranded (ds); negative (−), positive (+) or ambisense (+/−); linear circular or segmented. Examples of diseases caused by viruses include: Herpes Simplex Virus, adenovirus, Parvovirus, Reoviruses, Bluetongue, Hepatitis C, Polio, Yellow

Fever, Influenza, Ebola, Measles, Human Immunodeficiency Virus, human T-cell lymphotropic virus type 1, and Hepatitis B.

Virus Structure

The viral genome is wrapped up in a coat made up of multiple copies of a small number of species of protein molecules. Virus particles often form regular geometric shapes, such as helices or icosahedra. There are hundreds of virus structures.

Replication of Viruses

Viruses can replicate only when they infect a susceptible host cell. Following attachment and entry into the cell, the components of the capsid are partially disassembled and the virus begins to direct the cell to synthesise viral components. During this "eclipse" phase, where these new components are being synthesised, infectious virus cannot be recovered. Once the viral genome has been replicated and the viral proteins synthesized, new virus can be assembled. These new viruses may be released from the cell by budding or by cell lysis. The time taken for release of new infectious virus can range from minutes in the case of some viruses (foot and mouth) to several days.

For more detailed information, see Janeway, Travers, Walport, and Shlomchik, (2005).

Cross-References

► Immune Function

References and Readings

Janeway, C. A., Travers, P., Walport, M., & Shlomchik, M. J. (2005). *Immunobiology: The immune system in health and disease* (6th ed.). New York: Garland Science.

Inferential Statistical Testing

► Hypothesis Testing

Inferential Statistics

► Statistical Inference

Infertility and Assisted Reproduction: Psychosocial Aspects

Pamela A. Geller[1] and Alexandra Nelson[2]
[1]Department of Psychology, Drexel University Drexel University College of Medicine, Philadelphia, PA, USA
[2]Drexel University Department of Psychology UNC – Chapel Hill Department of Psychiatry, Chapel Hill, NC, USA

Synonyms

ART; Assisted reproductive technology; In vitro fertilization; Infertility-related stress; Intrauterine insemination; Involuntary childlessness; IUI; IVF

Definition

Infertility is defined as the inability to become pregnant with regular intercourse without the use of contraception within 1 year for women up to age 35 and within 6 months for women older than 35. The term infertility is not synonymous with the term sterility (i.e., the physically or medically diagnosed inability to become pregnant). Couples with diagnosed infertility may become pregnant through natural intercourse or through the assistance of fertility treatments. Such treatments range from administration of medication to more advanced techniques such as intrauterine insemination (IUI) or assisted reproductive technologies (ART) involving the handling of both sperm and egg, such as in vitro fertilization (IVF). Fertility treatments also may involve third-party reproductive options including the use of donor gametes or embryos as well as surrogates. The term involuntary childlessness may be used in

reference to individuals or couples with infertility that are unsuccessful in achieving pregnancy or live-birth delivery. Psychosocial aspects refer to the stressors (i.e., psychological, social, and structural) and consequences associated with infertility and assisted reproduction.

Description

Background and Epidemiology

Infertility is a fairly common condition that affects 72.4 million individuals worldwide. In the USA, 7.4% of married couples of reproductive age experience infertility and 12% of couples experience more broadly defined "subfecundity," which includes difficulties becoming pregnant or carrying a pregnancy to term. Risk for infertility increases with age, particularly among women over 35 years. Rates vary with sociodemographic characteristics, with greater risk of infertility among Hispanic and non-Hispanic black women and women who have not completed college (Chandra, Martinez, Mosher, Abma, & Jones, 2005). Couples may experience "primary infertility" during their first attempt to conceive or "secondary infertility" during attempts to conceive subsequent to prior successful conception. Secondary infertility is more common, with 35% of couples unable to have a second child and approximately 70% of women with infertility having been pregnant at least once. Secondary infertility disproportionately affects African American women compared to Caucasian women.

Infertility has numerous causes, which may be attributed to either the female or male partner, as well as a combination of factors, or may be medically unexplained or idiopathic. Common causes of female-factor infertility include polycystic ovary syndrome (PCOS), anovulation, tubal damage, endometriosis, uterine fibroids, pelvic adhesions, and history of cancer or sexually transmitted infection (STI). Male-factor causes of infertility can include low sperm count or motility, varicocele, and history of STI. A number of lifestyle factors can contribute to infertility, including obesity, smoking, and alcohol use. Being underweight can also underlie infertility, as in the case of anovulation secondary to anorexia nervosa in women. Psychological stress has been implicated in infertility, although research attempting to elucidate this hypothesized relationship has produced equivocal results.

Numerous medical interventions have been developed to assist couples with infertility to achieve pregnancy. Worldwide, approximately 40 million individuals receive fertility-related medical care. In the USA, about 12% of women receive at least one type of fertility-related medical services in their lifetimes and about 8% receive medical help to become pregnant in their lifetimes. Available treatments vary by specific cause of infertility and include surgeries to repair tubal damage, hormonal treatments to stimulate ovulation, IUI, and ART procedures such as IVF and intracytoplasmic sperm injection (ICSI). Third-party reproductive treatments, including gamete donation or use of gestational carriers (i.e., surrogates), also are available. These treatments vary considerably in their procedures, costs, time commitments, and many other aspects. Appropriate treatments vary by case, with some initiating fertility treatment with a less intrusive method (e.g., Clomid to simulate ovulation stimulation) before advancing to more invasive techniques (e.g., IVF) if early treatments are not successful. These treatment methods primarily involve the female partner as the patient, regardless of a couple's cause of infertility. Of note, these treatments also may be utilized by women who do not have infertility but who require medical assistance to become pregnant due to absence of a male reproductive partner.

Psychosocial Stressors

The experiences of infertility and fertility treatment are fraught with psychosocial stressors that contribute to distress. Because many women with infertility pursue medical intervention, and because research studies utilize varying sampling and assessment methodologies at widely varying time points during a couple's infertility experience, it is challenging to parse out distress caused

by infertility from that caused by fertility treatment. Below, the psychosocial stressors and consequences of both infertility and its treatment are discussed.

The experience of infertility is often a devastating disappointment to individuals and couples. The vast majority of young adults, when surveyed, report intentions to have at least one child in their lifetime. Receiving a diagnosis of infertility therefore significantly threatens hopes and expectations regarding parenthood and social roles. Reactions may include initial shock and disappointment; anger at oneself, one's partner, or others with children; hopelessness; loss of control; and guilt or self-blame (e.g., over attributions of infertility, such as contraceptive choices, prior elective abortions, or STIs). Further, individuals may confront challenging decisions regarding resolution of their infertility, including whether to pursue (or continue) fertility treatments, consider adoption, or remain childless. Additionally, during the course of fertility treatments, couples may confront difficult decisions such as whether to attempt subsequent treatment cycles in the event of treatment failure, to invest greater funds for more advanced medical procedures in hopes of increasing potential for success, or to undergo a selective reduction to reduce risk in a multiple pregnancy, which has a higher incidence in those receiving fertility treatment. Cultural and religious factors may influence and complicate these decisions.

The medical and procedural aspects of fertility treatments present myriad challenges. Many treatment options are time-consuming and physically invasive. For example, medications often are administered in injectable formats, which can be painful and inconvenient, and many women report unpleasant side effects of hormonal medications such as mood swings. Women receiving ART treatments may need to visit their physician several times per week and routinely undergo blood work and uncomfortable vaginal ultrasounds. ART protocols require surgical procedures to retrieve eggs and implant embryos. Even when pursuing less technologically complex fertility treatments, couples may be required to adhere to carefully timed intercourse, which can be difficult and emotionally taxing for both partners. Awaiting results of pregnancy testing following fertility treatments can be extremely stressful and anxiety-provoking. The rate of treatment failure is high, with only about 35% of ART treatments resulting in pregnancy. Even after achieving pregnancy, individuals may experience stress related to the threat of or actual miscarriage, which occurs in about 17% of ART pregnancies. Miscarriage is more likely to occur in women with a history of prior miscarriage, further compounding grief and distress for a subset of women. Treatment success is impacted by patient factors including patient age, number of follicles, and use of ovarian stimulation medications.

Fertility treatments, particularly ART, are extremely expensive, and low success rates often necessitate multiple treatment cycles to achieve a pregnancy. In general, costs for fertility treatment range from roughly $1,000 for medication treatments to tens of thousands of dollars for more advanced treatments such as IVF (~$40,000) and IVF with donor gametes (~$70,000); costs per successful outcome are much higher due to adjustments for low success rates. Some nations subsidize the cost of fertility treatments, but in the USA, only 15 states currently require possible coverage for fertility-related medical appointments or treatments, and Medicaid insurance does not provide any such coverage.

Although non-Caucasians are most affected by infertility, the majority of individuals receiving fertility treatment in the USA are Caucasian and come from socioeconomically advantaged backgrounds. Factors likely to explain this disparity include prohibitive treatment costs and lack of insurance, as well as culture-specific concerns about using reproductive technologies, spiritual beliefs, and mistrust of or difficulties communicating with health-care providers.

Infertility is considered stigmatizing in cultures across the world, including in the USA. Parenthood often is viewed as a primary adult role, and many women may internalize negative

social appraisals of childless women as selfish, unnatural, unfeminine, and inadequate, even when childlessness is involuntary. Women with infertility may believe that their bodies are abnormal or "handicapped" and may feel alienated and isolated from their fertile peers. Perceived stigma can produce perceptions of low social support, which in turn corresponds to increased anxiety or depression (Slade, O'Neill, Simpson, & Lashen, 2007). Women may encounter unsupportive social interactions marked by insensitive comments by family, friends, or acquaintances, which may be perceived as intrusive, blameful, or critical, impacting self-esteem and causing significant distress (Mindes, Ingram, Kliewer, & James, 2003). Many women choose to keep their infertility private and experience difficulties discussing their infertility experiences with others, including close friends and family.

Among women seeking fertility treatment for reasons other than infertility, other stressors arise. These individuals may include single women, women in same-sex couples, and women who are not yet experiencing infertility but anticipate future threats to their fertility (e.g., due to cancer treatments or older age), who may choose to freeze eggs or embryos. Medical interventions to help these individuals to achieve pregnancy are increasingly common, but stigma continues to surround the choice to raise children in nontraditional family arrangements such as single-parent or same-sex parent households. Additionally, due to the absence of a male reproductive partner, single and lesbian women utilize donated sperm, a reproductive option that introduces decision-making regarding which sperm donor to select, whether to select a known or anonymous donor, and whether and how to disclose the child's paternity to family, friends, acquaintances, and to the child. Less information is available about the psychosocial experiences of these populations, because the majority of studies examine heterosexual samples with infertility. As these populations are likely to encounter uniquely stressful experiences, careful attention from both the research and clinical communities is warranted.

Psychosocial Consequences of Infertility and Fertility Treatment

Individuals with infertility are at increased risk for emotional stress and psychiatric morbidity. Infertility-related stress is pervasive within this population. Such stress may relate to many of the stressors discussed above and is also thought to derive from intimate relationship strain, impact of infertility on sexual functioning, social relationship strain, and the profound conflicts surrounding potential childlessness and need for parenthood. While such stress affects both sexes, research has consistently revealed that women typically experience infertility-related stress to a greater degree than their male counterparts. Women consistently place greater emphasis and importance on having children compared to their male counterparts and are less open to the possibility of remaining childless when confronting infertility. This greater emphasis on having children corresponds to higher infertility-related stress among women. Despite the heightened impact of infertility-related stress on women, infertility-related stress appears to covary between both members of the same couple, suggesting that the burden of infertility-related stress is often shared.

Mood and anxiety symptoms and disorders are elevated among individuals with infertility. Research has consistently demonstrated a link between infertility-related stress and psychological adjustment. Women with infertility have higher anxiety symptomatology as well as higher rates of Generalized Anxiety Disorder (GAD) (King, 2003). Anxiety is thought to stem from uncertainty related to fertility status and treatment outcome, which is supported by the lack of association between GAD and sterility (or diagnosed inability to become pregnant). This uncertainty may persist for quite a long time, as couples may pursue fertility treatment or hold hope for a natural pregnancy for many years. Depressive symptomatology and depressive disorders are also elevated among individuals with infertility, with research revealing higher rates of diagnosable depressive disorders among the infertility population than among the general population. Depression may prevent

some women from seeking treatment and may also impact engagement in activities that might combat the stress of infertility. For some, fertility treatment may be implicated in the genesis or exacerbation of depression, with many studies reporting that women were psychologically well adjusted upon entering treatment but became significantly depressed during and after treatment (Eugster & Vingerhoets, 1999). Unsuccessful treatment can increase women's distress, and while most adjust over time, some women may experience more severe or persistent negative emotions (Verhaak et al., 2007). Grief and complicated bereavement may also occur following unsuccessful treatment. The occasional incidence of psychiatric events in response to fertility treatment has been reported, which may be partly due to interactions with hormonal treatments; uncommonly, these can include severe reactions such as psychotic experiences, with greater risk among women with past psychiatric histories.

Infertility can have a profound impact on partner relationships. Stressors relevant to intimate relationships, such as blaming oneself or one's partner for causing the fertility problem, discordant stress responses to infertility, and sexual dissatisfaction and low sexual self-esteem, can produce significant strain. Intimate relationship problems can contribute significantly to general distress associated with infertility. Research has indicated associations of poorer marital adjustment, disapproval from the spouse, and problems in the marital relationship with infertility-related stress and anxiety. Difficulties communicating about the infertility experience are also associated with infertility-related stress. Both women and men may be deeply affected by marital strain associated with infertility.

Infertility can also strain social relationships. Given the private nature of infertility described above, individuals may have difficulty communicating with others about their experiences. Both disclosure and nondisclosure can be distressing; women who have difficulty communicating about their infertility tend to experience greater stress and distress, but communication may also intensify feelings of distress or may be met with stigmatizing or unsupportive responses. Women may feel distanced from friends or family members with children, as it may be difficult to spend time with others who are experiencing parenthood. This may result in women isolating themselves from friends and family, posing barriers to accessing social support that may otherwise buffer against the deleterious effects of infertility-related stress.

Couples who achieve a successful outcome after fertility treatment generally experience remission of distress experienced during infertility and treatment. However, it is well documented that emotional burden, psychological distress, and difficulty coping are among the most common reasons for couples choosing to drop out of fertility treatment without achieving a successful outcome, even when controlling for the influences of treatment prognosis and financial constraints. Roughly half of fertility patients discontinue treatment under these circumstances without achieving a successful pregnancy. For couples making this choice, reported motivation to relieve emotional burdens presented by fertility treatment implies that discontinuing treatment may relieve some anxiety or distress and may enable pursuit of alternative resolutions to infertility; these individuals may feel released from a painful emotional cycle of anxiety, hope, disappointment, and grief. However, this decision may also mark the painful loss of hope to become a biological parent and may be accompanied by grief, anger, and a sense of failure.

Psychological Interventions for Infertility-Related Distress

A number of psychological interventions have been developed for women and couples with infertility, with fairly strong empirical support for their efficacy. Goals for these interventions often are to help women manage the emotional challenges of fertility treatment, although some have evaluated their utility in bolstering pregnancy rates. Efficacious treatments often utilize

cognitive-behavioral and mind/body approaches, incorporating psychoeducation, stress management, communication skills training, cognitive approaches to address attributions for infertility, and other coping strategies. Sexual functioning and adherence to timed sexual intercourse also may be addressed. Many interventions are conducted in group formats, although individual and couples' counseling are also commonly recommended for those struggling with significant distress. General support groups are commonly utilized, having long been considered a foundational support resource for women experiencing the isolating and stigmatizing effects of infertility. Social support and communication with others about infertility-related experiences is associated with reduced stress and emotional distress as well as reduced perceptions of stigma. Psychological interventions have been most efficacious in treating affective variables like anxiety and depression, and have less support for improving relationship functioning (Boivin, 2003).

Psychosocial Support in the Twenty-First Century

Despite the availability and efficacy of these interventions, they are consistently underutilized. This may be due to the perceptions that these treatments are too time-consuming, expensive, or invasive or that distress does not warrant professional intervention (Boivin, 2003). This indicates a need to explore alternative sources of support that may be more consistent with the preferences of the population.

In the past decade, a well-documented trend has emerged regarding women's increasing reliance on Internet resources for emotional and informational support related to infertility. Within the infertility population, websites and interactive communication forums are popular, particularly among women. They provide opportunities to access support at any hour of the day, from the convenience of one's home, and in an anonymous manner without the stigma or discomfort of speaking to others face to face. Reading web content, including dynamic discussions by others on communication forums, shares qualities with bibliotherapy, while contributing to such discussions by writing messages may share some characteristics with written emotional expression as well as engagement with face-to-face social support.

Emerging research regarding psychosocial impact of web-based support reflects several unique and generally positive features, including reduced isolation, normalization of emotional experiences, improved fertility-related knowledge and decision-making, and improvements in the partner relationship due to decreased burden on the spouse for support. However, researchers raise concerns over the potential for harmful effects. For example, the quality of web-based resources is often questionable, and the vast majority of websites addressing fertility-related information do not adhere to proposed quality standards; furthermore, in websites that enable contributions from the public, there is a very real possibility for exposure to misinformation and unsupported, anecdotal information. Research has revealed a small possibility that individuals may experience negative emotions as a result of using web-based resources and communication, including painful reactions to hearing either happy or sad fertility-related news of others, as well as the potential for disinhibited, harsh communication directed at specific group members. More research is needed to explore the effects of Internet-mediated support to inform patients, medical providers, and administrators and guide the development of such resources to best meet the needs of the population in managing the psychosocial aspects of infertility.

Cross-References

- ▶ In Vitro Fertilization
- ▶ Internet-Based Interventions

References and Readings

Abbey, A., Halman, L. J., & Andrews, F. M. (1992). Psychosocial, treatment, and demographic predictors of the stress associated with infertility. *Fertility and Sterility, 57*(1), 122–128.

Boivin, J. (2003). A review of psychosocial interventions in infertility. *Social Science & Medicine, 57*(12), 2325–2341.
Chandra, A., Martinez, G. M., Mosher, W. D., Abma, J. C., & Jones, J. (2005). Fertility, family planning, and reproductive health of U.S. women: Data from the 2002 national survey of family growth. *Vital Health Statistics, 23*(25), 1–160.
Eugster, A., & Vingerhoets, A. J. (1999). Psychological aspects of in vitro fertilization: A review. *Social Science & Medicine, 48*(5), 575–589.
Greil, A. L. (1997). Infertility and psychological distress: A critical review of the literature (Review). *Social Science & Medicine, 45*(11), 1679–1704.
Hammarberg, K., Astbury, J., & Baker, H. (2001). Women's experience of IVF: A follow-up study. *Human Reproduction, 16*(2), 374–383.
Homan, G., Davies, M., & Norman, R. (2007). The impact of lifestyle factors on reproductive performance in the general population and those undergoing infertility treatment: A review. *Human Reproduction Update, 13*(3), 209.
King, R. B. (2003). Subfecundity and anxiety in a nationally representative sample. *Social Science & Medicine, 56*(4), 739–751.
Malik, S. H., & Coulson, N. S. (2008). Computer-mediated infertility support groups: An exploratory study of online experiences. *Patient Education and Counseling, 73*(1), 105–113.
Mindes, E. J., Ingram, K. M., Kliewer, W., & James, C. A. (2003). Longitudinal analyses of the relationship between unsupportive social interactions and psychological adjustment among women with fertility problems. *Social Science & Medicine, 56*(10), 2165–2180.
Resolve. The national infertility association. http://www.resolve.org.
Schmidt, L., Holstein, B. E., Christensen, U., & Boivin, J. (2005). Communication and coping as predictors of fertility problem stress: cohort study of 816 participants who did not achieve a delivery after 12 months of fertility treatment. *Human Reproduction, 20*(11), 3248–3256.
Slade, P., O'Neill, C., Simpson, A. J., & Lashen, H. (2007). The relationship between perceived stigma, disclosure patterns, support and distress in new attendees at an infertility clinic. *Human Reproduction, 22*(8), 2309–2317.
Verhaak, C. M., Smeenk, J. M., Evers, A. W., Kremer, J. A., Kraaimaat, F. W., & Braat, D. D. (2007). Women's emotional adjustment to IVF: A systematic review of 25 years of research (Review). *Human reproduction update, 13*(1), 27–36.
White, L., McQuillan, J., & Greil, A. L. (2006). Explaining disparities in treatment seeking: The case of infertility. *Fertility and Sterility, 85*(4), 853–857. doi:10.1016/j.fertnstert.2005.11.039.

Infertility-Related Stress

▶ Infertility and Assisted Reproduction: Psychosocial Aspects

Inflammation

Nicolas Rohleder and Jutta M. Wolf
Department of Psychology, Brandeis University, Waltham, MA, USA

Definition

The term "inflammation" is derived from the Latin *inflammare* which translates to "to set on fire." Inflammation as a process is a component of the overall immune response against invading pathogens and/or tissue damage. A local inflammatory response at a site of a wound and/or infection is characterized by the classical symptoms *calor* (heat), *rubor* (redness), *dolor* (pain), *tumor* (swelling), and *functio laesa* (loss of function). These symptoms are the result of the inflammatory response system being activated in the affected tissue. More specifically, the inflammatory response is started by local resident cells of the innate immune system, which secrete inflammatory mediators and other effector substances. These substances induce vasodilation (dilation of the blood vessels), which allows increased blood flow to the affected area and also causes rubor and calor. Related to this, an increased permeability of the blood vessel walls allows plasma exudation (transfer of blood plasma) into the affected tissue, which accounts for the swelling. Circulating immune cells are attracted to the affected site through chemotaxis and adhesion molecules (cells are attracted by chemical signals on the surface of cells lining the blood vessels), and leave the blood vessel to enter the affected tissue and help fight infection or contribute to wound healing. Pain is a result of the same

inflammatory cells releasing bradykinin (a substance that lowers pain thresholds), while loss of function is a non-specific consequence of local inflammation.

Description

Different Forms of Inflammation

Depending on the efficiency of the inflammatory machinery in relation to the extent and virulence of the invading pathogen, this response might be contained and resolved, and thus remains local, or it might spread to the rest of the body, thereby initiating a systemic inflammatory response. One form of systemic inflammation is sepsis, which is characterized by hyper amplification of inflammatory processes with deleterious effects on the organism, including organ failure and/or death (Cinel & Opal, 2009).

Another, more recently described form of systemic inflammation is chronic low-grade inflammation (Danesh, 1999). This phenomenon, also described as low-grade systemic inflammation, is characterized by increases in concentrations of the same plasma mediators that signal and contribute to local and other forms of systemic inflammation, that is, mainly interleukin-6 (IL-6) and C-reactive protein (CRP). However, concentrations are markedly lower than those measured, for example, in sepsis, but still increased in comparison to the non-inflamed state of a young and healthy organism.

Age, Lifestyle, and Inflammation

An important characteristic of chronic low-grade inflammation is its association with age. Although it is currently unclear whether increasing concentrations of inflammatory mediators are a consequence of the aging process per se, there is compelling evidence that as organisms age, their plasma concentrations of IL-6 and CRP increase slowly, but steadily (Ershler, 1993). Several lifestyle factors have been found associated with low-grade inflammation, the most important of which appear to be physical inactivity, nutrition, and obesity as a consequence of both. Adipose tissue is an important source of inflammatory cytokines, such as IL-6, and increased presence of adipose tissue is associated with increased plasma concentrations of inflammatory mediators (Yudkin, Kumari, Humphries, & Mohamed-Ali, 2000). Furthermore, certain diets, such as typical high fat, high carbohydrate "western" diets, have been associated with low-grade inflammation, while diets with high contents of omega-3 fatty acids, fruits, vegetables, and nuts, as well as typical "Asian" diets seem to be anti-inflammatory (Giugliano, Ceriello, & Esposito, 2006). Another behavioral or lifestyle factor is tobacco smoking, which is associated with increased plasma concentrations of inflammatory mediators (Bazzano, He, Muntner, Vupputuri, & Whelton, 2003).

Long-Term Consequences of Inflammation

While sepsis is acutely life-threatening, one of the most important and disadvantageous forms of inflammation in terms of long-term consequences is probably chronic low-grade inflammation. There is now strong evidence pointing to a central role of low-grade inflammation in many age-related diseases such as cardiovascular disease, insulin resistance and type 2 diabetes, osteoporosis, and cancer (see Ershler, 1993). Several prospective studies have found that increased concentrations of IL-6 and CRP in older adults are a strong predictor of morbidity and all-cause mortality (Ridker, Cushman, Stampfer, Tracy, & Hennekens, 1997; Ferrucci et al., 1999; Bruunsgaard et al., 2003). Many of the pathways between low-grade inflammation and age-related diseases are becoming better understood now. Atherosclerosis, the pathophysiological process underlying heart disease and strokes, has been shown to be essentially a local inflammatory response in artery walls, and as such can be stimulated more easily and strongly in individuals with higher plasma concentrations of inflammatory mediators. Similarly, the process underlying insulin resistance is becoming more and more understood, and inflammatory mediators seem to play an important role in the ability of target cells to understand insulin signals.

Inflammation in Behavioral Medicine

In summary, chronic low-grade inflammation can be an important process to consider for researchers and practitioners in behavioral medicine, as inflammation is prospectively related with some of the most important diseases of later life, and because inflammation is under the control of the central nervous system. Several pathways link states of the central nervous system with peripheral inflammation. Most notably, recent research has shown that low-grade inflammation is more pronounced in individuals suffering from chronic stress, depression, and posttraumatic stress disorder (e.g., Ford & Erlinger, 2004; Miller & Blackwell, 2006). Similarly, acute stress has been shown to induce short-term increases in plasma inflammatory mediators (Steptoe, Hamer, & Chida, 2007). Recent research indicates that increased inflammatory mediators in depression are in part mediated by negative changes in health behaviors, most notably by reduced physical activity, but the same research also shows that a large proportion of this association must be explained by additional factors (Hamer, Molloy, de Oliveira, & Demakakos, 2009). Typical candidates are the stress systems hypothalamus-pituitary-adrenal (HPA) axis and autonomic nervous system (ANS), which show altered basal activity in many stress-related diseases, including depression and PTSD, and which are important regulators of inflammatory activity. Importantly, the ability of the inflammatory system to understand these stress systems' signaling changes after acute and chronic stress, and alterations in the inflammatory system's sensitivity to these signals, can help explain long-term health effects (for a summary see Raison & Miller, 2003; Rohleder, Wolf, & Wolf, 2010).

In addition to this efferent CNS-to-periphery pathway, inflammatory mediators also send signals to the CNS, leading to a set of symptoms summarized as sickness behavior. These symptoms include affective effects, most notably depressive symptoms and fatigue, but also alterations in sleep and cognition (Dantzer, O'Connor, Freund, Johnson, & Kelley, 2008). Sickness behavior is strongest in the context of acute infection, cytokine therapy, and sepsis, but evidence is accumulating in support of the assumption that low-grade inflammation is sufficient to induce depressive symptoms, cognitive decline, and other components of sickness behavior, albeit less pronounced (see, e.g., Raison, Capuron, & Miller, 2006; Alley, Crimmins, Karlamangla, Hu, & Seeman, 2008).

Cross-References

- ▶ Autonomic Balance
- ▶ Depression
- ▶ Psychoneuroimmunology
- ▶ Stress

References and Readings

Alley, D. E., Crimmins, E. M., Karlamangla, A., Hu, P., & Seeman, T. E. (2008). Inflammation and rate of cognitive change in high-functioning older adults. *The Journals of Gerontology. Series A, Biological Sciences and Medical Sciences, 63*(1), 50–55.

Bazzano, L. A., He, J., Muntner, P., Vupputuri, S., & Whelton, P. K. (2003). Relationship between cigarette smoking and novel risk factors for cardiovascular disease in the United States. *Annals of Internal Medicine, 138*(11), 891–897.

Bruunsgaard, H., Ladelund, S., Pedersen, A. N., Schroll, M., Jorgensen, T., & Pedersen, B. K. (2003). Predicting death from tumour necrosis factor-alpha and interleukin-6 in 80-year-old people. *Clinical and Experimental Immunology, 132*(1), 24–31.

Cinel, I., & Opal, S. M. (2009). Molecular biology of inflammation and sepsis: a primer. *Critical Care Medicine, 37*(1), 291–304.

Danesh, J. (1999). Smoldering arteries? Low-grade inflammation and coronary heart disease. *JAMA: The Journal of the American Medical Association, 282*(22), 2169–2171.

Dantzer, R., O'Connor, J. C., Freund, G. G., Johnson, R. W., & Kelley, K. W. (2008). From inflammation to sickness and depression: When the immune system subjugates the brain. *Nature Reviews Neuroscience, 9*(1), 46–56.

Ershler, W. B. (1993). Interleukin-6: A cytokine for gerontologists. *Journal of the American Geriatrics Society, 41*(2), 176–181.

Ferrucci, L., Harris, T. B., Guralnik, J. M., Tracy, R. P., Corti, M. C., Cohen, H. J., et al. (1999). Serum IL-6 level and the development of disability in older persons. *Journal of the American Geriatrics Society, 47*(6), 639–646.

Ford, D. E., & Erlinger, T. P. (2004). Depression and C-reactive protein in US adults: Data from the Third National Health and Nutrition Examination Survey. *Archives of Internal Medicine, 164*(9), 1010–1014.
Giugliano, D., Ceriello, A., & Esposito, K. (2006). The effects of diet on inflammation: Emphasis on the metabolic syndrome. *Journal of the American College of Cardiology, 48*(4), 677–685.
Hamer, M., Molloy, G. J., de Oliveira, C., & Demakakos, P. (2009). Persistent depressive symptomatology and inflammation: To what extent do health behaviours and weight control mediate this relationship? *Brain, Behavior, and Immunity, 23*(4), 413–418.
Kiecolt-Glaser, J. K., Christian, L., Preston, H., Houts, C. R., Malarkey, W. B., Emery, C. F., et al. (2010). Stress, inflammation, and yoga practice. *Psychosomatic Medicine, 72*(2), 113–121.
Miller, G. E., & Blackwell, E. (2006). Turning up the heat: Inflammation as a mechanism linking chronic stress, depression, and heart disease. *Current Directions in Psychological Science, 15*(6), 269–272.
Pace, T. W., Negi, L. T., Adame, D. D., Cole, S. P., Sivilli, T. I., Brown, T. D., et al. (2009). Effect of compassion meditation on neuroendocrine, innate immune and behavioral responses to psychosocial stress. *Psychoneuroendocrinology, 34*(1), 87–98.
Raison, C. L., Capuron, L., & Miller, A. H. (2006). Cytokines sing the blues: Inflammation and the pathogenesis of depression. *Trends in Immunology, 27*(1), 24–31.
Raison, C. L., & Miller, A. H. (2003). When not enough is too much: The role of insufficient glucocorticoid signaling in the pathophysiology of stress-related disorders. *The American Journal of Psychiatry, 160*(9), 1554–1565.
Ridker, P. M., Cushman, M., Stampfer, M. J., Tracy, R. P., & Hennekens, C. H. (1997). Inflammation, aspirin, and the risk of cardiovascular disease in apparently healthy men. *The New England Journal of Medicine, 336*(14), 973–979.
Rohleder, N., Wolf, J. M., & Wolf, O. T. (2010). Glucocorticoid sensitivity of cognitive and inflammatory processes in depression and posttraumatic stress disorder. *Neuroscience and Biobehavioral Reviews, 35*(1), 104–114.
Steptoe, A., Hamer, M., & Chida, Y. (2007). The effects of acute psychological stress on circulating inflammatory factors in humans: A review and meta-analysis. *Brain, Behavior, and Immunity, 21*(7), 901–912.
Yudkin, J. S., Kumari, M., Humphries, S. E., & Mohamed-Ali, V. (2000). Inflammation, obesity, stress and coronary heart disease: Is interleukin-6 the link? *Atherosclerosis, 148*(2), 209–214.

Inflammation-Associated Depression

► Sickness Behavior

Inflammatory Bowel Disease

Yukari Tanaka and Shin Fukudo
Department of Behavioral Medicine, School of Medicine, Tohoku University Graduate, Aoba-ku, Sendai, Japan

Synonyms

Colitis

Definition

Inflammatory bowel disease (IBD) is a specific group of chronic inflammatory conditions of the intestine. IBD consists of the two major diseases: ulcerative colitis (UC) and Crohn's disease (CD). UC causes inflammation in the rectum, colon, and infrequently the terminal ileum. CD can involve any part of the gastrointestinal tract from the mouth to the anus and particularly affects the ileum and/or the colon. IBD most commonly begins during adolescence and early adulthood.

Description

Causes

The cause of IBD is not clearly known, but these diseases are thought to be due to an autoimmune process that has been triggered by unknown factors or agents, such as genetic predisposition, infection of microorganisms, and/or environmental factors. As a result of the ulcers and inflammatory reaction of the mucosal layer of the small intestine or the colon, the intestinal wall is damaged leading to bloody diarrhea and abdominal pain. In recent studies, psychosocial stress and/or psychiatric comorbidity unlikely cause(s) IBD. However, IBD itself evokes stress, and evoked stress response induces some psychiatric comorbidities including anxiety disorders, depressive disorders, or somatoform disorders.

Signs and Symptoms

Symptoms may range from mild to severe: abdominal pain and tenesmus, bloody diarrhea, abnormal bowel movement, fever, anemia, and appetite and weight loss. IBD is characterized by recurring episodes of inflammation of the bowel; thus, patients will go through periods in which the disease flares up and causes symptoms. These periods should be followed by remission, in which symptoms disappear or decrease. UC often appears as the most severe inflammation in the rectum, which may cause frequent diarrhea and bleeding. Inflammation and ulceration in UC are primarily limited to the mucosa and the submucosa. By contrast, the inflammation of the Crohn's disease is transmural and sometimes results in intestinal obstruction, deep ulcers, microperforations, and fistulas. Long-standing IBD is also a risk factor of developing colorectal cancer.

Diagnosis

Diagnosis can be made endoscopically or radiologically. UC almost always involves the rectum and the lower left colon. Endoscopic findings reveal ulcers, edema, erythema and friability of the mucosa, and loss of the vascular pattern. Crypt abscess is a more typical histologic finding in the colonic mucosa of active UC. Radiological findings are loss of the normal mucosal pattern and, with more extensive colorectal inflammation, absent haustration. Endoscopic findings in Crohn's disease are deeper ulcers involving the entire wall of the colon. The deep liner ulcers result adjacent to normal tissue results in "cobblestoning." These lesions are typically discontinuous, which results in "skip" lesions. Barium enema in Crohn's disease demonstrates aphthous ulcers, cobblestoning pattern, narrowing of the lumen called "string sign," or fistulas and abscess formation. The notable histopathological feature is the presence of epithelioid granulomas.

Treatment

Standard treatment for IBD depends on extent of involvement and disease severity. Medical management attempts to initiate and lead to a long-term remission, which is the period between flare ups where very little to no symptoms should be experienced. Medications used to treat IBD include: 5-aminosalicylic acid (5-ASA) compounds, steroids, and medications to suppress the immune system. 5-ASA is the main anti-inflammatory drug and the standard first-line treatment for mild-to-moderate IBD. 5-ASA is available in a variety of different formulations and different delivery systems that split the active moiety in various regions of the intestine. Sulfasalazine are poorly absorbed from the intestine and act from inside the intestine. Sulfasalazine can be effective, but sometimes induces some side effects. On the other hand, mesalamine and olsalazine have fewer side effects than sulfasalazine has. Steroids are powerful drugs and commonly used, but they often produce a variety types of side effects, particularly, in long-time uses. Immunosuppressive drugs, the antimetabolite 6-mercaptopurine (6-MP), are also powerful and help patients get off steroids but may prohibit natural immune defenses. Anti-TNF antibody treatment of Crohn's disease is one of the important therapeutic strategies recently developed. It is a monoclonal antibody and blocks TNF-alpha, which is involved in inflammation of the bowel. Surgical treatment is necessary in some severe cases with perforation, toxic megacolon, and severe bleeding.

Cross-References

▶ Functional Somatic Syndromes

References and Readings

Bernstein, C. N., Blanchard, J. F., Kliewer, E., & Wajda, A. (2001). Cancer risk in patients with inflammatory bowel disease: A population-based study. *Cancer, 91*, 854–862.

Hanauer, S. B., Feagan, B. G., Lichtenstein, G. R., Mayer, L. F., Schreiber, S., Colombel, J. F., et al. (2002). Maintenance infliximab for Crohn's disease: The ACCENT I randomised trial. *Lancet, 359*, 1541–1549.

Podolsky, D. K. (2002). Inflammatory bowel disease. *New England Journal of Medicine, 347*, 417–429.

Winawer, S., Fletcher, R., Rex, D., Bond, J., Burt, R., Ferrucci, J., et al. (2003). Gastrointestinal consortium panel. Colorectal cancer screening and surveillance: Clinical guidelines and rationale – Update based on new evidence. *Gastroenterology, 124*, 544–560.
Xavier, R. J., & Podolsky, D. K. (2007). Unravelling the pathogenesis of inflammatory bowel disease. *Nature, 448*, 427–434.

Inflammatory Markers

► C-Reactive Protein (CRP)

Informed Consent

Yoshiyuki Takimoto
Department of Stress Science and Psychosomatic Medicine, Graduate School of Medicine, The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Definition

Informed consent is giving consent to receive treatment or to become a subject of research based on adequate disclosure and understanding of relevant facts. The requirement for informed consent is based on the ethical principle *autonomy*. The legal grounding for requirement for informed consent is based on the outcome of litigations of disputes arising in the context of medical practice.

There are at least five necessary elements of informed consent: (1) disclosure of information sufficient to make a reasonably informed decision, (2) understanding by the consenter of information and recommendations disclosed, (3) voluntariness in consenting, (4) competence of the individual to understanding information given and reach a reasoned decision, and (5) assent.

The postulate is that a person gives an informed consent to an intervention if and only if the person receives a thorough disclosure about the procedure, comprehends the disclosed information, acts voluntarily, is competent to act, and consents.

Cross-References

► Institutional Review Board (IRB)

References and Readings

Beauchamp, T. L., & Faden, R. R. (2003). Informed consent. In S. G. Post (Ed.), *Encyclopedia of bioethics* (3rd ed., pp. 1271–1280). New York: Macmillan Reference.
Malek, J. (2005). Informed consent. In C. Mitcham (Ed.), *Encyclopaedia of science, technology, and ethics?* (pp. 1016–1019). Detroit: Macmillan Reference.

Inheritance, Genetic

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

Genetic inheritance addresses how traits are transmitted from one generation to another. While the word "inheritance" alone is typically used and taken to mean genetic inheritance, the term cultural inheritance is also a valid concept and deals with how cultural factors are transmitted.

The laws of inheritance (including dominant inheritance and recessive inheritance) were discovered by Mendel without knowledge of the actual molecular biological mechanism, and his research focused on plants and especially the common pea plant, *Pisum sativum*. Instead of trying to address the appearance of whole plants, and hence all of their characteristics, he focused on the inheritance of single, easily visible, and well-distinguished traits. These included round versus wrinkled seed, yellow

versus green seed, and purple versus white flowers (Britannica, 2009). He also made exact counts of the number of plants bearing each trait. These quantitative data allowed him to formulate the rules governing inheritance, as exemplified by the dominant inheritance and recessive inheritance entries in this encyclopedia.

Cross-References

- ▶ Dominant Inheritance
- ▶ Recessive Inheritance

References and Readings

Britannica. (2009). *The Britannica guide to genetics (Introduction by Steve Jones)*. Philadelphia: Running Press.

Inpatient Treatment

- ▶ Substance Abuse: Treatment

Insertion/Deletion Polymorphism

Laura Rodriguez-Murillo[1] and Rany M. Salem[2]
[1]Department of Psychiatry, Columbia University Medical Center, New York, NY, USA
[2]Broad Institute, Cambridge, MA, USA

Definition

An insertion/deletion polymorphism, commonly abbreviated "indel," is a type of genetic variation in which a specific nucleotide sequence is present (insertion) or absent (deletion). While not as common as SNPs, indels are widely spread across the genome. Indels comprise a total of 3 million of the 15 million known genetic variants (The 1000 Genomes Project Consortium, 2010). An indel in the coding region of a gene that is not a multiple of 3 nucleotides results in a frameshift mutation. Shifting the reading frame and the DNA transcript sequence may now code for an entirely different set of amino acids or result in a premature stop codon, altering the protein structure and function. Indel variants with multiples of 3 nucleotides result in a protein with extra amino acids (insertion) or loss of amino acids (deletion), but the other amino acids are not affected. The site of the indel in the gene is important since it determines which part of the protein is affected, since not all amino acids are necessary for proper protein function. A 250-base-pair indel in the angiotensin converting enzyme (ACE) gene explains 50% of the variance of serum ACE levels and has been shown to have important clinical consequences (Scharplatz, Puhan, Steurer, & Bachmann, 2004).

Cross-References

- ▶ Polymorphism
- ▶ Single Nucleotide Polymorphism (SNP)

References and Readings

Lewis, R. (2005). *Human genetics. Concepts and applications* (7th ed.). Boston: McGraw-Hill Science/Engineering/Math.

Scharplatz, M., Puhan, M., Steurer, J., & Bachmann, L. (2004). What is the impact of the ACE gene insertion/deletion (I/D) polymorphism on the clinical effectiveness and adverse events of ACE inhibitors? – Protocol of a systematic review. *BMC Medical Genetics, 5*(1), 23.

Strachan, T., & Read, A. P. (2003). *Human molecular genetics* (3rd ed.). London/New York: Garland Science/Taylor & Francis Group.

The 1000 Genomes Project Consortium. (2010). A map of human genome variation from population-scale sequencing. *Nature, 467*(7319), 1061–1073. doi:10.1038/nature09534.

Insight Meditation

- ▶ Mindfulness

Insomnia

Wendy Troxel
Psychiatry and Psychology, University of Pittsburgh, Pittsburgh, PA, USA

Definition

The term "insomnia" is commonly used in the vernacular to refer to symptoms of insomnia including complaints of difficulty falling asleep, frequent or prolonged awakenings, inadequate sleep quality, or short overall sleep duration in an individual who has adequate time available for sleep. In contrast, an *insomnia disorder* is a syndrome consisting of the insomnia complaint combined with significant daytime impairment or distress, and the exclusion of other causes. Commonly reported daytime impairments associated with insomnia include complaints of mood disturbances (e.g., irritability, mild dysphoria, or difficulty tolerating stress), impaired cognitive function, and daytime fatigue (Moul et al., 2002). Importantly, insomnia patients commonly report feeling fatigued or exhausted during the day, but rarely report daytime sleepiness, per se (i.e., the propensity to fall asleep). Another important feature that distinguishes the insomnia disorder from sleep deprivation is that symptoms are present despite adequate opportunity for sleep. That is, in insomnia the opportunity for sleep is adequate, but the ability to sleep is compromised. In contrast, sleep deprivation is characterized by a restricted opportunity to sleep (due to lifestyles, shiftwork, etc.) with adequate ability.

Description

Prevalence and Consequences

Insomnia is the most common sleep disorder, with prevalence estimates ranging from 10% to 15% in the general population (Ohayon, 1996; Ohayon & Guilleminault, 1999; Ohayon, 2002) and up to 20–30% in primary care medical settings (Simon & Von Korff, 1997; Shochat, Umphress, Israel, & Ancoli-Israel, 1999). Substantial evidence demonstrates the significant health and functional consequences of insomnia, including reduced quality of life and increased health-care utilization, health-care costs, disability, and risk for psychiatric disorders and cardiovascular disease (Althuis, Fredman, Langenberg, & Magaziner, 1998; Brassington, King, & Bliwise, 2000; Brostrom, Stromberg, Dahlstrom, & Fridlund, 2004; Caap-Ahlgren & Dehlin, 2001; Daley, Morin, LeBlanc, Gregoire, & Savard, 2009; Foley, Ancoli-Israel, Britz, & Walsh, 2004; Ford & Kamerow, 1989).

Assessment

Polysomnography or other laboratory sleep measures are not recommended in the diagnosis of insomnia, except to rule out other occult sleep disorders. Rather, the diagnosis is based on a comprehensive clinical evaluation, including an assessment of sleep disturbances, habits, and clinical course, as well as a complete medical and psychiatric history, including use of prescribed or un-prescribed medications (including timing and dose), as all of these factors may influence insomnia. In addition to the clinical interview, questionnaires and sleep diaries are frequently used to provide a more in-depth analysis of qualitative aspects of sleep as well as the timing and variability of sleep patterns over successive nights.

Risk Factors

According to the "3-P" model of insomnia, risk factors for insomnia can be categorized into predisposing, precipitating, and perpetuating factors (Spielman, Caruso, & Glovinsky, 1987). Predisposing or vulnerability factors include advancing age, female sex, being divorced or separated, unemployment, and comorbid medical and psychiatric illness (Lichstein, Taylor, McCrae, & Ruiter, 2011; Morgan & Clarke, 1997; Ohayon, 2002; Su, Huang, & Chou, 2004). To date, genetic factors have been inadequately studied, though they may represent another possible risk (Drake, Scofield, & Roth, 2008; Hamet & Tremblay, 2006; Watson,

Goldberg, Arguelles, & Buchwald, 2006). Precipitating events that initiate insomnia may include major life transitions or psychosocial stressors such as moves, relationship difficulties, occupational and financial problems, and caregiving responsibilities (Bastien, Vallieres, & Morin, 2004; Kappler & Hohagen, 2003). Finally, perpetuating factors include counterproductive thoughts and behaviors related to sleep that maintain or exacerbate insomnia. Empirically supported behavioral and cognitive behavioral interventions aim to reduce insomnia by modifying or eliminating these maladaptive sleep-related thoughts and behaviors (Bastien et al., 2004; Morin, 2004).

References and Readings

Althuis, M. D., Fredman, L., Langenberg, P. W., & Magaziner, J. (1998). The relationship between insomnia and mortality among community-dwelling older women. *Journal of the American Geriatrics Society, 46*, 1270–1273.

Bastien, C. H., Vallieres, A., & Morin, C. M. (2004). Precipitating factors of insomnia. *Behavioral Sleep Medicine, 2*, 50–62.

Brassington, G. S., King, A. C., & Bliwise, D. L. (2000). Sleep problems as a risk factor for falls in a sample of community-dwelling adults aged 64-99 years. *Journal of the American Geriatrics Society, 48*, 1234–1240.

Brostrom, A., Stromberg, A., Dahlstrom, U., & Fridlund, B. (2004). Sleep difficulties, daytime sleepiness, and health-related quality of life in patients with chronic heart failure. *The Journal of Cardiovascular Nursing, 19*, 234–242.

Caap-Ahlgren, M., & Dehlin, O. (2001). Insomnia and depressive symptoms in patients with Parkinson's disease. Relationship to health-related quality of life. An interview study of patients living at home. *Archives of Gerontology and Geriatrics, 32*, 23–33.

Daley, M., Morin, C. M., LeBlanc, M., Gregoire, J. P., & Savard, J. (2009). The economic burden of insomnia: Direct and indirect costs for individuals with insomnia syndrome, insomnia symptoms, and good sleepers. *Sleep, 32*, 55–64.

Drake, C. L., Scofield, H., & Roth, T. (2008). Vulnerability to insomnia: The role of familial aggregation. *Sleep Medicine, 9*, 297–302.

Foley, D., Ancoli-Israel, S., Britz, P., & Walsh, J. (2004). Sleep disturbances and chronic disease in older adults: Results of the 2003 national sleep foundation sleep in America survey. *Journal of Psychosomatic Research, 56*, 497–502.

Ford, D. E., & Kamerow, D. B. (1989). Epidemiologic study of sleep disturbances and psychiatric disorders. An opportunity for prevention? *Journal of the American Medical Association, 262*, 1479–1484.

Hamet, P., & Tremblay, J. (2006). Genetics of the sleep-wake cycle and its disorders. *Metabolism, Clinical and Experimental, 55*, S7–S12.

Kappler, C., & Hohagen, F. (2003). Psychosocial aspects of insomnia. Results of a study in general practice. *European Archives of Psychiatry and Clinical Neuroscience, 253*, 49–52.

Lichstein, K. L., Taylor, D. J., McCrae, C. S., & Ruiter, M. E. (2011). Insomnia: Epidemiology and risk factors. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practices of sleep medicine* (5th ed., pp. 827–837). St. Louis: Elsevier.

Morgan, K., & Clarke, D. (1997). Risk factors for late-life insomnia in a representative general practice sample. *British Journal of General Practice, 47*, 166–169.

Morin, C. M. (2004). Cognitive-behavioral approaches to the treatment of insomnia. *The Journal of Clinical Psychiatry, 65*(Suppl 16), 33–40.

Moul, D. E., Buysse, D. J., Nofzinger, E. A., Pilkonis, P. A., Houck, P. R., & Miewald, J. M. (2002). Symptoms reports in severe chronic insomnia. *Sleep, 25*, 553–563.

Ohayon, M. M. (1996). Epidemiological study on insomnia in a general population. *Sleep, 19*, S7–S15.

Ohayon, M. M. (2002). Epidemiology of insomnia: What we know and what we still need to learn. *Sleep Medicine Reviews, 6*, 97–111.

Ohayon, M. M., & Guilleminault, C. (1999). Epidemiology of sleep disorders. In S. Chokroverty (Ed.), *Sleep disorders medicine: Basic science, technical considerations and clinical aspects* (2nd ed., pp. 301–316). Boston: Butterworth-Heinemann.

Shochat, T., Umphress, J., Israel, A. G., & Ancoli-Israel, S. (1999). Insomnia in primary care patients. *Sleep, 22* (Suppl 2), S359–S365.

Simon, G. E., & Von Korff, M. (1997). Prevalence, burden, and treatment of insomnia in primary care. *The American Journal of Psychiatry, 154*, 1417–1423.

Spielman, A. J., Caruso, L. S., & Glovinsky, P. B. (1987). A behavioral perspective on insomnia treatment [Review]. *Psychiatric Clinics of North America, 10*, 541–553 [60 refs].

Su, T. P., Huang, S. R., & Chou, P. (2004). Prevalence and risk factors of insomnia in community-dwelling Chinese elderly: A Taiwanese urban area survey. *The Australian and New Zealand Journal of Psychiatry, 38*, 706–713.

Watson, N. F., Goldberg, J., Arguelles, L., & Buchwald, D. (2006). Genetic and environmental influences on insomnia, daytime sleepiness, and obesity in twins. *Sleep, 29*, 645–649.

Institute of Medicine

Martica H. Hall
Department of Psychiatry, University of Pittsburgh, Pittsburgh, PA, USA

Basic Information

The Institute of Medicine is a nonprofit organization whose mission is the advancement of medical and biological sciences in the service of health.

Established in 1970 as a branch the National Academies (National Academy of Sciences, National Academy of Engineering, National Research Council, Institute of Medicine), the IOM is a nonprofit organization committed to the advancement of medical and biological sciences in the service of health. The IOM's underlying principle is that authoritative evidence in the medical and biological sciences expands the capabilities of medicine and health care.

Major Impact on the Field

Members are elected to the IOM based upon their record of scientific achievement. The membership and governing Council interact with other scientists and government officials to identify IOM projects. These projects encompass a variety of formats including establishment of expert committees to facilitate cross-disciplinary discussion and discovery, the conduct of workshops and public forums, the generation and publication of authoritative topical reports and books, and the support of high-impact research studies. These activities are conducted with the express function of advancing medical and biological sciences in the service of health and improved health care.

The IOM recognizes the importance of behavioral and social sciences in the service of health-related medical and biological sciences. This commitment is evidenced in high-profile IOM reports regarding interactions among social, psychological, behavioral, and biological factors that underlie health and functioning. Examples include state-of-science reports on health promotion intervention strategies from social and behavioral research (2000); the influence of early environment on neuronal development (2000); gene-environment interactions in relation to health (2006); the consequences of sleep disorders and sleep deprivation (2006); the physiologic, psychological, and psychosocial consequences of deployment stress in military personnel (2007); and environmental risk factors for breast cancer (2011).

Cross-References

- ▶ Gene-Environment Interaction
- ▶ Sleep
- ▶ Sleep Deprivation

Institutional Care

Elizabeth Galik
School of Nursing, University of Maryland, Baltimore, MD, USA

Synonyms

Institutionalization; Long-term care

Definition

Institutional care is provided within a congregate living environment designed to meet the functional, medical, personal, social, and housing needs of individuals who have physical, mental, and/or developmental disabilities. Vulnerable children, and older adults, individuals with developmental disabilities, mental retardation, chronic mental illness, and physical disabilities are more likely to receive care in institutional settings, such as orphanages, nursing homes, residential facilities, and rehabilitation centers. Care and

services in institutional settings often include, but are not limited to, 24-h supervision/monitoring, assistance with activities of daily living, skilled nursing care, rehabilitation, adaptive aids and equipment, psychological services, therapies, social activities, and room and board. The cost of institutional care varies by the facility and the services that are required. Payment for institutional care includes private financial resources, long-term care insurance, Medicaid, and Medicare. In the United States, federal and state governments oversee institutional care facilities which are inspected annually to ensure that they are in compliance with regulatory requirements that address residents' rights, health, and safety.

Cross-References

► Assisted Living

References and Readings

Centers for Medicare and Medicaid Services. (2008). *Guide to choosing a nursing home*. Baltimore: Department of Health and Human Services.

Challis, D., Mozley, C. G., Sutcliffe, C., et al. (2000). Dependency in older people recently admitted to care homes. *Age & Aging, 29*(3), 255–260.

Jones, A. L., Dwyer, L. L., Bercovitz, A. R., & Strahan, G. W. (2009). The national nursing home survey: 2004 overview. National Center for Health Statistics. *Vital and Health Statistics, 13*(167), 1–155.

Roy, P., Rutter, M., & Pickles, A. (2004). Institutional care: Associations between overactivity and lack of selectivity in social relationships. *Journal of Child Psychology and Psychiatry, 45*(4), 866–873.

Institutional Review Board (IRB)

Yoshiyuki Takimoto
Department of Stress Science and Psychosomatic Medicine, Graduate School of Medicine, The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Synonyms

Research ethics committee

Definition

Institutional Review Board (IRB) is a committee containing members of mixed backgrounds and including both scientist and nonscientist members whose task is to review and monitor biomedical and behavioral research involving human subjects. The World Medical Association's Declaration of Helsinki (2010) and Council for International Organizations of Medical Science's International Ethical Guidelines for Biomedical Research Involving Human Subjects establish the international standard for biomedical research and require IRB as scientific review and ethical review committee, which is called the research ethics committee (REC) in the most of the world. IRB is assigned to the authority and responsibility for approving or disapproving proposals to conduct research involving human subjects. The chief purpose of IRB reviews is to assess the scientific and ethical merit and usefulness of the research and its methods and to ensure that research conforms to ethical standards and protects the right and welfare of the research subjects.

Cross-References

► Clinical Trial
► Human Subjects Committee

References and Readings

Bridge, A. (2005). Institutional review board. In C. Mitcham (Ed.), *Encyclopaedia of science, technology, and ethics?* (pp. 1024–1026). Detroit: Macmillan Reference.

Levine, R. J. (2004). Research ethics committees. In S. G. Post (Ed.), *Encyclopedia of bioethics* (3rd ed., pp. 2311–2316). New York: Macmillan Library Reference.

The World Medical Association. (2010). The world medical association's declaration of Helsinki. Retrieved May 07, 2012, from http://www.wma.net/en/30publications/10policies/b3/

Institutionalization

► Institutional Care

Instrumental Conditioning

► Operant Conditioning

Insulin

Adriana Carrillo
Department of Pediatrics, Miller School of Medicine, University of Miami, Miami, FL, USA

Synonyms

Fasting insulin; Insulin sensitivity

Definition

Insulin

Insulin is an essential hormone for maintaining normal blood glucose. It is the principal regulator of glucose utilization in liver, skeletal muscle, and adipose tissue. Abnormalities of insulin synthesis, secretion, or action can result in hypoglycemia or hyperglycemia, including diabetes mellitus.

Synthesis: Insulin is synthesized from the pancreatic beta cells from a single-chain precursor, preproinsulin. Preproinsulin is cleaved to proinsulin which is further cleaved by proconvertases into three polypeptide chains: alpha, beta, and C-peptide chains. Insulin is formed by linkage of the alpha and beta chains by disulfide bonds. The remaining C-peptide is secreted from the beta cell with insulin in equal molar portions. Insulin and C-peptide levels can be used to evaluate pancreatic function and glucose tolerance (Gardner & Shoback, 2007; Kronenber, Melmed, Polonsky, & Larsen, 2008).

Secretion: Insulin secretion is primarily stimulated by glucose. Glucose enters the beta cell through facilitated diffusion mediated by GLUT2 glucose transporter. Glucose is then phosphorylated to glucose-6-phosphate by glucokinase. Glucokinase is the rate-limiting step of glycolysis and primarily determines the rate of insulin secretion in response to blood glucose levels. The metabolism of glucose increases intracellular ATP/ADP ratio resulting in insulin release. Insulin is secreted in a biphasic pattern, an initial peak followed by a sustained plateau after carbohydrate ingestion. Insulin concentrations increase 8–10 min after ingestion of food and peak by 30–45 min. Blood glucose concentrations return to baseline by 90–120 min after ingestion of food. Oral glucose administration increases insulin response greater than intravenous glucose. This is due to stimulation of gut hormones that promote insulin release, called incretins. Incretins like glucagon-like peptides (GLP) increase the sensitivity of the beta cell to glucose. GLP-1 also inhibits glucagon and delays gastric empting. Glucagon is secreted from the pancreatic alpha cells and counters the effects of insulin in the liver by stimulating glycogenolysis.

Insulin secretion is also stimulated by amino acids by increasing the intracellular ATP/ADP ratio. Acutely, free fatty acids and ketones also stimulate insulin secretion. Long-term beta cell exposure to fatty acids inhibits insulin synthesis and secretion. Somatostatin, secreted from pancreatic delta cells, inhibits insulin release (Lifshitz, 2007).

Insulin secretion is also under the control of the autonomic nervous system. The vagus nerve of the parasympathetic nervous systems directly stimulates insulin secretion. In the sympathetic nervous system, activation of β2 receptors increases insulin secretion, while activation of α-adrenergic receptors inhibits insulin secretion. During stress, epinephrine and norepinephrine activate α-adrenergic receptors, blocking insulin release and resulting in hyperglycemia. Other counter regulatory hormones such as cortisol and other glucocorticoids stimulate insulin secretion while inducing peripheral insulin resistance.

Action: Insulin binds to insulin receptors on the cell membrane of target tissues. The insulin receptor is a heterodimeric glycoprotein. Insulin binds to the extracellular α subunits of the insulin receptor which activates intrinsic tyrosine kinase

on the transmembrane β subunits leading to phosphorylation of cytoplasmic substrates inducing signal transduction. In the liver, insulin stimulates glucose uptake, storage of glucose as glycogen, and synthesis of protein, triglycerides, and very-low-density lipoprotein. Also in the liver, insulin inhibits glycogenolysis and gluconeogenesis. In skeletal muscle, insulin stimulates glucose uptake, glycogen synthesis, and protein synthesis by increasing amino acid transport. In adipose tissue, insulin stimulates glucose uptake and storage of triglyceride. Insulin induces production of circulating lipoprotein lipase which hydrolyzes triglycerides from circulating lipoprotein for uptake by adipose cells. Insulin inhibits intracellular lipoprotein lipase to promote the storage of triglycerides. Counter regulator hormones impair insulin action. Epinephrine, cortisol, and growth hormone antagonize insulin action by promoting glycogenolysis, gluconeogenesis, lipolysis, and ketogenesis, and decreasing glucose utilization and clearance (Lifshitz, 2007).

Pathology: Abnormality of insulin production, secretion, or action results in abnormal blood glucose. Uncontrolled secretion of insulin typically presents with hypoglycemia in the neonatal period. Neonatal hyperinsulinism may be transient due to maternal conditions such as uncontrolled gestational diabetes. Congenital hyperinsulinism due to genetic mutations in the islet cell can cause constitutive secretion of insulin and hypoglycemia. Insulin-secreting tumors of the pancreas, insulinomas, are the most common cause of fasting hypoglycemia in otherwise healthy adults.

Genetic mutations that decrease insulin synthesis or secretion can cause neonatal diabetes. Mild defects in insulin synthesis can present later in childhood or early adulthood; this group of mutations is referred to as maturity onset diabetes of the young (MODY). Mutations associated with MODY are transmitted autosomal dominant.

Abnormality of insulin action may be due to genetic mutations of the insulin receptor or due to type 2 diabetes. In type 2 diabetes, target tissues have decreased sensitivity to insulin. The etiology of type 2 diabetes is unclear. It is clear that obesity and consumption of a high-carbohydrate diet may unmask diabetes in a person who is genetically susceptible. The pancreas compensates for insulin resistance by elevated insulin levels. Beta cells begin to lose insulin response to glucose intake, and glucose intolerance develops.

Type 1 diabetes mellitus results from autoimmune destruction of pancreatic beta cells and consequently insulinopenia. Type 1 diabetes typically presents in childhood. Peak age of presentation is bimodal, at 5–7 years of age and at the time of puberty. Symptoms of diabetes usually do not present until greater than 80% of the islet cells have been destroyed (Lifshitz, 2007).

Synthetic Insulin: Recombinant human insulin is necessary for the treatment of type 1 diabetes and neonatal diabetes and may be adjunctive in type 2 diabetes. Insulin analogues are administered subcutaneously several times per day for optimal control. Rapid acting insulin is administered just prior to carbohydrate consumption. Intermediate and long acting insulin can be administered once or twice a day for basal insulin. An insulin pump provides continuous insulin infusion of rapid acting insulin to provide more physiologic insulin therapy. Blood glucose must be monitored closely with any insulin regimen to avoid hypoglycemia (Lifshitz, 2007; Sperling, 2008).

CNS: Insulin and insulin receptors have been found throughout the central nervous system. In the hypothalamus, insulin is associated with regulation of body energy homeostasis. In the hippocampus and cerebral cortex, insulin has been shown to be involved in cognitive function. For example, in animals, changes in insulin signaling in the hippocampus and temporal cortex are associated with memory and learning. In humans, acute increase in insulin is associated with increased memory capacity. However, chronically elevated insulin levels can cause neural damage through microvascular damage by oxidative stress and endothelial damage. Epidemiologic data suggests an association between insulin resistance syndrome and dementia which is also attributed to microvascular damage.

Cross-References

- ▶ Fasting Insulin
- ▶ Insulin Resistance
- ▶ Insulin Sensitivity

References and Readings

Gardner, D. G., & Shoback, D. (2007). *Greenspan's basic & clinical endocrinology* (8th ed.). New York: McGraw-Hill.
Kronenber, H., Melmed, S., Polonsky, K., & Larsen, P. R. (2008). *Williams textbook of endocrinology* (11th ed.). Philadelphia: Saunders Elsevier.
Lifshitz, F. (2007). *Pediatric endocrinology* (5th ed.). New York: Informa Healthcare.
Sperling, M. (2008). *Pediatric endocrinology* (3rd ed.). Philadelphia: Suanders Elsevier.

Insulin Effectiveness

- ▶ Insulin Sensitivity

Insulin Pumps

Della Matheson
Diabetes Research Institute, Miller School of Medicine, University of Miami, Miami, FL, USA

Synonyms

Continuous subcutaneous insulin infusion

Definition

Insulin pump therapy, often referred to as continuous subcutaneous insulin infusion (CSII), is an alternate method of insulin administration for persons who require multiple daily injections (MDI) for treatment of diabetes. The concept in insulin pump therapy was first introduced by Arnold Kadish in Beverly Hills, CA, in the 1960s. However, it was not until the early 1980s that the first FDA-approved pump became available for use by people with type 1 diabetes.

The basic design of pumps is that a syringe-driven pump uses a subcutaneously placed catheter to deliver insulin that is controlled by electronics within the pump (Skyler, 2010). The insulin used in pumps is rapid-acting insulin (regular insulin or more recently the insulin analogs: Novolog, Humalog, Apidra). Insulin pumps provide the most physiologic method for delivery of insulin. In nondiabetic people, the pancreas secretes background insulin throughout the day and night that maintains normal blood glucose levels in the non-fed state. The pancreas also secretes insulin in larger quantities in rapid response to blood glucose excursions after food is ingested. Unlike MDI, which employs 1–2 injections of long acting insulin to mimic the background insulin requirement, and 3–4 injections per day of short acting insulin injected before each meal, CSII allows the use of only rapid-acting insulin which is infused continuously throughout the day to provide a background of insulin (basal rate) and allows the wearer to press a button prior to each meal to deliver a larger amount of insulin to accommodate the glucose content of the meal (bolus), thus mimicking the normal insulin secretion patterns. Modern pumps are small, discreet, and fashionable in appearance. They are also often referred to as "smart pumps" because they integrate concepts of intensive insulin therapy and principles of good diabetes self-management into their programs. The features that incorporate these concepts include insulin:carbohydrate ratio calculator for meals, insulin sensitivity factor and target blood glucose to calculate proper correction doses for high blood sugars, insulin-on-board feature which helps to calculate the amount of circulating insulin still active in the blood stream to help prevent overdosage of insulin, and multiple basal rates to customize background insulin to individual variations in need for background insulin. It should be understood that insulin pumps do not automatically provide insulin in response to glucose; the person wearing the pump must program the pump and make decisions about how much insulin should be taken in

collaboration with the health-care team. The future may well usher in more advances in insulin pump therapy that lead us closer to a pump with automatic insulin delivery, aka a "closed loop system." With the advent of continuous glucose monitoring (CGM) and sensor-augmented pumps, research is underway to achieve this goal.

It is well established through the Diabetes Control and Complications Trials (DCCT) that good glycemic control (HbA1c <7.4%) prevents long-term microvascular and macrovascular complications (The Diabetes Control and Complications Trial Research Group, 1993). It is generally accepted that intensive insulin therapy through MDI or CSII is the best method for achieving the targets set forth by the DCCT. However, there appears to be no firm evidence to suggest that CSII is better than MDI to achieve these targets. The main benefits of CSII appear to be enhanced flexibility of lifestyle and quality of life, both of which may impact patient adherence to the strenuous demands of intensive insulin therapy. The risks of insulin pumps have minimized greatly with improved technology over the past 20 years. The greatest risk with use of CSII is diabetic ketoacidosis (DKA); since only rapid-acting insulin is used in the device, if the flow of insulin is disrupted, deterioration to DKA may occur rapidly as there is no long acting insulin to help sustain the individual. With education and proper training, this has been greatly diminished in pump users. Disruption in the flow of insulin most often occurs due to an occlusion in the catheter or bent catheter, pump running out of insulin and user not refilling on a timely basis, a dislodged catheter, and infrequently a mechanical failure of the pump (Ponder, Skyler, Kruger, Matheson, & Brown, 2008).

Currently available on the market in the USA are the following pumps: Animas (Johnson & Johnson), MiniMed (Medtronic), OmniPod (Insulet Corp.), and Amigo (Nipro).

Cross-References

- ▶ Diabetes
- ▶ Insulin
- ▶ Insulin-Dependent Diabetes Mellitus (IDDM)
- ▶ Type 1 Diabetes

References and Readings

Ponder, S. W., Skyler, J. S., Kruger, D. F., Matheson, D., & Brown, B. W. (2008). Unexplained hyperglycemia in continuous subcutaneous insulin infusion: Evaluation and treatment. *The Diabetes Educator, 34*, 327–333.

Skyler, J. S. (2010). Continuous subcutaneous insulin infusion – An historical perspective. *Diabetes Technology & Therapeutics, 12*(Suppl. 1), S5–S7.

Skyler, J. S., Ponder, S. W., Kruger, D. F., Matheson, D., & Parkin, C. G. (2007). Is there a place for insulin pump therapy in your practice? *Clinical Diabetes, 2*, 50–56.

The Diabetes Control and Complications Trial Research Group. (1993). The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. The Diabetes Control and Complications Trial Research Group. *New England Journal of Medicine, 329*, 977–986.

Insulin Resistance

- ▶ Hyperinsulinemia

Insulin Resistance (IR) Syndrome

Alan M. Delamater
Department of Pediatrics, University of Miami Miller School of Medicine, Miami, FL, USA

Synonyms

Metabolic syndrome

Definition

The insulin resistance syndrome is a term that refers to the clustering of four health risk factors: obesity, hyperinsulinemia and insulin resistance, high blood pressure, and dyslipidemia (Reaven, 1988). This syndrome was previously known as syndrome X and more recently as the metabolic syndrome.

References and Readings

Reaven, G. M. (1988). Pathophysiology of insulin resistance in human disease. *Physiology Review, 75*, 473–486.

Insulin Sensitivity

Janine Sanchez
Department of Pediatrics, University of Miami, Miami, FL, USA

Synonyms

Insulin effectiveness

Definition

Insulin sensitivity is the ability of insulin to stimulate glucose uptake, promote peripheral glucose disposal, and suppress hepatic glucose production. The primary site of glucose dispose is skeletal muscle. The gold standard method for the determination of insulin sensitivity is the hyperinsulinemic-euglycemic insulin clamp. However, the clamp technique is expensive and complex. Surrogate methods to determine insulin sensitivity include fasting insulin and glucose, oral glucose tolerance test, and intravenous glucose tolerance test. The most common calculations using fasting insulin and glucose are fasting glucose to insulin ratio, homeostasis model assessment of insulin resistance (HOMA), and quantitative insulin sensitivity check index (QUICKI). The HOMA and QUICKI models are mathematical estimates of beta cell function and insulin resistance.

Impaired insulin sensitivity or insulin resistance precedes glucose intolerance in the development of type 2 diabetes. As insulin sensitivity decreases, insulin levels will become elevated in attempt to maintain effectiveness and euglycemia. If insulin sensitivity continues to decline, patients may develop glucose intolerance and then type 2 diabetes. Insulin resistance is associated with a metabolic and cardiovascular cluster of disorders including type 2 diabetes, glucose intolerance, dyslipidemia, fatty liver, hypertension, obesity, polycystic ovarian syndrome, and endothelial dysfunction. Insulin resistance can also occur in patients with poorly controlled type 1 diabetes. This leads to increased insulin requirements to maintain euglycemia.

Insulin resistance can also be associated with rare syndromes due to insulin receptor abnormalities or insulin antibodies. In addition, insulin resistance increases during puberty and decreases when puberty is complete. Clinical signs associated with insulin resistance include acanthosis nigricans (darkening of skin around neck and axilla), obesity, and hypertension.

Exercise, healthy diet, and weight loss can improve insulin sensitivity. The metabolic and cardiovascular diseases associated with insulin resistance also improve as insulin sensitivity improves.

Cross-References

- Fasting Insulin
- Hyperinsulinemia
- Insulin
- Insulin Resistance
- Insulin Resistance (IR) Syndrome

References and Readings

Bloomgarden, Z. T. (2006). Measures of insulin sensitivity. *Clinics in Laboratory Medicine, 26*(3), 611–633.

Borai, A., Livingstone, C., & Ferns, G. A. (2007). The biochemical assessment of insulin resistance. *Annals of Clinical Biochemistry, 44*, 324–342.

Levy-Marchal, C., & Arslanian, S. (2010). Insulin resistance in children: Consensus, perspective, and future directions. *The Journal of Clinical Endocrinology and Metabolism, 95*(12), 5189–5198.

Sperling, M. A. (2009). *Pediatric endocrinology* (3rd ed.). Philadelphia: WB Saunders.

Wilson, J. D. (2008). *Williams textbook of endocrinology* (11th ed.). Philadelphia: WB Saunders.

Insulin Shock

► Hypoglycemia

Insulin-Dependent Diabetes Mellitus (IDDM)

Luigi Meneghini
Diabetes Research Institute, University of Miami, Miami, FL, USA

Synonyms

Autoimmune diabetes; Juvenile diabetes; Type 1 diabetes

Definition

Insulin-dependent diabetes mellitus (IDDM) is now referred to as type 1 diabetes (T1DM) and designates an elevation in blood glucose due to insufficient production of insulin thought to result from cell-mediated autoimmune destruction of insulin-producing pancreatic beta cells. Autoimmunity is manifested by mononuclear cell invasion of islets (insulitis) and the production of islet-specific antibodies, such as GAD (glutamic acid decarboxylase), IA-2 autoantibodies, and ICA (islet cell antibodies), detectable in ~85% of newly diagnosed patients. Genetic, environmental, and possibly other unknown factors contribute to disease susceptibility, which is most commonly manifested in the childhood or teenage years (hence the former designation of juvenile diabetes).

If not replaced, absolute insulin deficiency in T1DM results in severe hyperglycemia and diabetic ketoacidosis (DKA), which if left untreated can prove fatal (hence the designation IDDM). Insulin replacement ideally takes the form of basal/bolus insulin therapy, delivered either via multiple daily insulin injections or an insulin pump infusion. Poorly controlled type 1 diabetes can lead to, among other complications, nephropathy (kidney damage manifested by excess protein in the urine) and end-stage kidney damage, retinopathy potentially leading to decreased visual acuity, and neuropathy and the risk of diabetic foot infections or ulcerations. Long-standing diabetes is also associated with increased risk for heart disease, stroke, and peripheral vascular disease. Maintaining good glycemic control, as deflected by an HbA1c of 7% or less, will prevent many of the chronic complications of the disease.

Cross-References

► Insulin
► Type 1 Diabetes Mellitus

References and Readings

Joslin, E. P., & Kahn, C. R. (2005). *Joslin's diabetes mellitus* (14th ed.). Philadelphia: Lippincott Williams & Willkins.

Insulin-Producing Cell

► Beta Cells

Integrated Behavioral Medicine Research, Practice, Policy

► Translational Behavioral Medicine

Integrated Care

► Clinical Settings

Integrated Health Care

► Disease Management

Integrative Medicine

Margaret A. Chesney[1] and David E. Anderson[2]
[1]Department of Medicine & Center for Integrative Medicine, University of California, San Francisco, CA, USA
[2]Division of Nephrology, Department of Medicine, University of California, San Francisco, CA, USA

Synonyms

Holistic medicine

Definition

Integrative medicine is a term for an emerging field of study and clinical practice that combines the best of complementary, alternative, and conventional medicine. Complementary and alternative medicine is defined by the National Institutes of Health as a broad range of healing approaches, therapies, and philosophies that conventional Western medicine does not commonly use, study, or make available. Complementary medicine refers to the use of these therapies in addition to conventional medicine; alternative medicine refers to their use in place of conventional medicine. Integrative medicine also incorporates lifestyle interventions that have emerged as important aspects of health promotion and disease prevention within mainstream Western medicine (Institute of Medicine [IOM], 2009a).

Initially driven by consumer demand, integrative medicine is a rapidly growing field that may offer a pathway toward better health care and the potential for significant decreases in national health care costs. It emphasizes the human capacity for healing, the centrality of the relationship between patient and clinician, a focus on the whole person, and use of all appropriate therapeutic approaches, disciplines, and health-care professionals (Consortium of Academic Health Centers in Integrative Medicine (CAHCIM)) (http://www.imconsortium.org). This holistic orientation contrasts with the stereotypical approach of conventional medicine based on specialization and reliance on biotechnology in diagnosis and treatment.

Description

Background

The integrative medicine movement has emerged from dissatisfaction with conventional biomedicine. Some 20 years ago, it was observed that public confidence in the medical establishment was eroding and that the fundamental relationship between patient and physician was disintegrating (Snyderman and Weil, 2002). This state of affairs was attributed to the fact that the historical role of the physician as a comprehensive caregiver had diminished due to increasing reliance on costly and impersonal technologies rather than careful histories and physical examinations. The biomedical approach conceptualized the body as a machine, the doctor's task as that of repairing the machine, and presumed that healing of the whole could be effected via treatment of its component parts. This approach resulted in spectacular successes in some areas of medicine, but a different perspective was seen to be needed for chronic diseases.

By 1993, more than a third of all adults in the United States reported using some form of "unconventional" medicine, such as various relaxation techniques, chiropractic, and massage (Astin, 1998; Eisenberg et al., 1993). In 1995, the National Institutes of Health (NIH) established an Office of Alternative Medicine, which grew by 1998 into the National Center for Complementary and Alternative Medicine (NCCAM). The stated goal of NCCAM is to define, through rigorous scientific investigation, the usefulness and safety of complementary and alternative medicine interventions and their roles in improving health and health care. Alternative and complementary medicine practices fall into broad clusters of approaches including natural products (e.g., special diets, probiotics, dietary supplements, and botanical medicine), mind-body practices (e.g., meditation, deep breathing, cognitive

therapies, yoga, tai chi, and qi gong), and manipulative and body-based practices (manual medicine, chiropractic, massage, special forms of exercise and movement therapies, etc.). NCCAM also supports studies of whole medical systems such as traditional Chinese medicine and Ayurvedic (Indian) medicine. Acupuncture, one of the oldest healing practices in the world, is an important form of complementary medicine.

Academic centers devoted to integrative medicine began to appear that offered programs in research, professional education, and clinical practice. A proliferation of these centers led to the formation of umbrella organizations that provided support for this growing field, such as CAHCIM, founded in 1988, and the Bravewell Collaborative, founded in 2002. The Bravewell group, a philanthropic collaborative, supported the efforts of CAHCIM, which by 2011 included more than 50 academic health centers and associated health-care systems (CAHCIM, http://www.imconsortium.org).

Philosophy of Integrative Medicine

Integrative medicine reflects a systems approach to understanding health and healing. It recognizes that humans are complex entities, and the most important influences on health, for individuals and society, are not the factors at play within any single domain – environmental, behavioral, or genetic – but the dynamics, synergies, and reciprocal interactions among these domains. In other words, the human body functions like an orchestrated network of interconnected systems, whose overall condition is sensitive to environmental context, dietary intake, and other lifestyle factors which can increase susceptibility to other pathogenic variables (IOM, 2009a).

The goal of integrative medicine is to provide patient-centered, evidence-based care that honors an integrated systems approach to health across the life span. Patient-centered care means treating the patient as an integral part of the care team and putting responsibility for important aspects of health monitoring and care in the patient's hands. Evidence-based health care involves increased reliance on a foundational science that takes an empirical approach to treatment efficacy, as well as the investigation of biological mechanisms by which environmental, psychosocial, behavioral, and genetic factors influence health and disease.

Seven of the most common chronic diseases – heart disease, hypertension, stroke, cancer, diabetes, pulmonary conditions, and mental disorders – account for more than half of all health-care costs, and more than 70% of deaths (Centers for Disease Control and Prevention, www.cdc.gov). The traditional view was that these disorders are primarily genetic in origin and largely unpreventable. The integrative medicine community recognizes that factors such as where we live, conditions of our environments, income and education levels, and relationships with friends and family all interact with genetic predispositions to influence health and potentiate the development of acute and chronic diseases. Evidence is accumulating that many disorders can be prevented or their onset delayed via healthy lifestyles characterized by attention to dietary intake, increased physical activity, stress management by various mind-body approaches, adequate sleep, and decreased tobacco and alcohol use. Some integrative medicine centers offer "coaching" in nutrition, physical fitness, stress management, and emotional and spiritual health as parts of a broad-spectrum approach. Such comprehensive interventions have been shown, for example, to slow the progress of atherosclerosis and are now covered in the United States by Medicare (Frieden, 2005). The development of cost-effective programs for maintaining health-promoting behavior over the long term without compromise of individual freedom remains an important goal if the integrative medicine movement is to realize its full potential.

Evidence Base for Integrative Medicine

The Cochrane Library lists thousands of studies of complementary medicine and hundreds of reviews of these studies. Reviews of the literature regarding some approaches, such as meditation, yoga, and therapeutic massage, have often been favorable but typically note that sample sizes tend to be small and the designs contain methodological weaknesses. Many of these studies have been conducted in the form of randomized

clinical trials (RCTs) that are based on well-defined interventions and carefully selected patient populations. As in pharmacological research, however, the results of these RCTs are presented in terms of average treatment effects, compared with a control group, often placebo or "usual care." Thus, the relevance of these findings to decision making in the individual case is limited (Greenfield et al., 2007).

Moreover, integrative medicine interventions sometimes lend themselves poorly to classic RCT methodology because they often involve potentially synergistic, multimodal, and complex interactions that are delivered in the context of a patient-practitioner relationship and influenced by patient preferences, expectations, and motivations. It is known that patients vary in their expectations regarding the perceived efficacy of treatments, and these expectations can influence treatment outcome. For example, the impact of expectations on therapeutic outcomes was analyzed in a series of four RCTs of acupuncture effects on chronic low back pain, osteoarthritis of the knee, migraine headache, and tension headache (Linde et al., 2007). Positive attitudes toward acupuncture and high personal expectations for treatment benefits were consistently associated with better outcomes at the end of treatment and at 4-month follow-up.

Integrative medicine encourages the patient to take an active role in his or her own health, and is sensitive to nuances in the patient-practitioner interaction. The placebo effect is of particular relevance to integrative medicine because of the importance of the patient-practitioner relationship. It has been found, for example, that the therapeutic power of analgesic drugs is markedly greater when administered by a clinician than when administered automatically by an infusion pump (Benedetti et al., 2005).

Progress in understanding mechanism of action of the placebo effect has been slow, however, because of complexities in the definition of the term placebo (Miller and Kaptchuk, 2008). In conventional medicine, where attention to healing by technological intervention has overshadowed healing via practitioner-patient interaction, the role of the placebo effect in treatment effects has often been minimized. Nevertheless, some conventional treatments, including knee surgery (Moseley et al., 2002) and spinal injections (Buchbinder et al., 2009: Kallmes et al., 2009) that are superior to no treatment (wait list) conditions have been matched in efficacy by placebo "controls." Indeed, one focus of research in integrative medicine is on the role of contextual factors in healing, such as the environment of the clinical setting, the cognitive and affective communication of clinicians, and the ritual of administering treatment (Kaptchuk et al., 2008).

Because of such complexities, integrative medicine advocates have called for a broadening of the range of strategies for evaluating the effectiveness of integrative medicine interventions beyond standardized RCTs (Fonnebo et al., 2007). Among these is comparative effectiveness research (CER), which was defined by the IOM (IOM, 2009a) as "the generation and synthesis of evidence that compares the benefits and harms of alternative methods to prevent, diagnose, treat and monitor a clinical condition, or to improve the delivery of care (p. 13)" with the goal of improving health care at the individual as well as the population level. In CER, research designs permit the study of complex interventions as they are applied in clinical practice (FCC, http://www.hhs.gov) and encourage analysis of subgroups within studies to meet the growing interest in personalized or patient-centered medicine (Goodman, 2009; Institute of Medicine [IOM], 2011).

While RCTs, CER, and other strategies examine efficacy, integrative medicine research is also beginning to explore the biological pathways and mechanisms by which interventions exercise their influence. This research is needed to establish biological plausibility and reassure conventional practitioners about the value of integrative medicine.

Relevance to Preventive Medicine

The integrative medicine movement has the potential to reduce health-care costs via attention to strategies for the prevention of chronic disease associated with the maintenance of health and improved quality of life (IOM, 2009a). Evidence for this can be found in corporate programs that focus on prevention and wellness. While not

explicitly conceptualized as integrative medicine programs, these efforts are consistent with the integrative medicine philosophy that advocates a culture of health, including modifications to cafeteria and vending machine options, physical environments that promote exercise, and the use of quiet areas for relaxation and stress reduction. For example, a review of the long-term impact of the health and wellness program at Johnson and Johnson reported significant reductions in medical expenditures over a 4-year period together with decreased outpatient and mental health visits (Ozminkowski et al., 2002). Another program, consisting of work site health education, nutritional counseling, smoking cessation counseling, physical activity promotion, selected physician referral, and health counseling, reported improvements in quality of life and scores on risk factors, together with significant reductions in health care and inpatient expenses (Richard et al., 2009).

In summary, integrative medicine is a rapidly growing field that merges the best of complementary and alternative medicine with conventional medicine. Initially driven by consumer dissatisfaction with aspects of biomedicine, it reasserts the importance of the patient-practitioner relationship and the need to consider health and disease within a holistic orientation. It is developing relevant methodology for systematic investigation of the validity and utility of complex interventions and for investigation of the importance of health and wellness within the context of the interactions of the individual with the environment. Within this framework, the placebo effect is redefined as contextual healing and thereby merits independent investigation. The ultimate success of integrative medicine will depend upon the evidence of relative efficacy of a variety of interventions in a variety of disorders and with an eventual understanding of the mechanisms by which salutary treatment effects are achieved.

Cross-References

- ▶ Acupuncture
- ▶ Alternative Medicine
- ▶ Chronic Disease Management
- ▶ Chronic Disease or Illness
- ▶ Comparative Effectiveness Research
- ▶ Complementary and Alternative Medicine
- ▶ Cost-Effectiveness
- ▶ Exercise, Benefits of
- ▶ Health Care Costs
- ▶ Patient-Centered Care
- ▶ Physical Activity and Health
- ▶ Placebo and Placebo Effect
- ▶ Randomized Clinical Trial
- ▶ Self-care
- ▶ Self-management
- ▶ Sleep
- ▶ Stress
- ▶ Stress Management
- ▶ Yoga

References and Readings

Astin, J. (1998). Why patients use alternative medicine results of a national study. *Journal of the American Medical Association, 279*(19), 1548–1553.

Benedetti, F., Mayberg, H. S., Wager, T. D., Stohler, C. S., & Zubieta, J. K. (2005). Neurobiological mechanisms of the placebo effect. *Journal of Neuroscience, 25*, 10390–10402.

Buchbinder, R., Osborne, R. H., Ebeling, P. R., Wark, J. D., Mitchell, P., & Wriedt, C., et al. (2009). A randomized trial of vertebroplasty for painful osteoporotic. *The New England Journal of Medicine, 361*(6), 557–568.

Centers for Disease Control and Prevention. (2010). *Chronic disease prevention and health promotion: Costs of chronic disease*. Retrieved December 22, 2010, from http://www.cdc.gov/chronicdisease/overview/index.htm

Consortium of Academic Health Centers for Integrative Medicine. (2009). *Definition of integrative medicine*. Retrieved August 28, 2011, from http://www.imconsortium.org/about/home.html

Consortium of Academic Health Centers for Integrative Medicine. (2009). *Definition of integrative medicine*. Retrieved August 1, 2011, from http://www.imconsortium.org/about/history/home.html

Eisenberg, D. M., Kessler, R. C., Foster, C., Norlock, F. E., Calkins, D. R., & Delbanco, T. L. (1993). Unconventional medicine in the United States – prevalence, costs & patterns of use. *The New England Journal of Medicine, 328*, 246–252.

FCC (Federal Coordinating Council for Comparative Effectiveness Research). (2009). *Report to the President and Congress*. Accessed October 11, 2010, from http://www.hhs.gov/recovery/programs/cer/cerannualrpt.pdf

Fonnebo, V., Grimsgaard, S., Walach, H., Ritenbaugh, C., Norheim, A. J., MacPherson, H., et al. (2007).

Researching complementary and alternative treatments: The gate keepers are not at home. *BMC Medical Research Methodology, 7*(7), 1–6.
Frieden, J. (2005). CMS panel backs coverage for lifestyle programs. *Clinical Psychiatry News, 1*(2005), 72.
Goodman, C. (2009). *Comparative effectiveness research and personalized medicine: From contradiction to synergy*. Accessed October 14, 2010, from http://www.lewin.com/content/publications/Lewin_CER-PM.pdf
Greenfield, S. R., Kravita, N., & Kaplan, S. H. (2007). Heterogeneity of treatment effects: Implications for guidelines, payment, and quality assessment. *The American Journal of Medicine, 120*(4A), 53–59.
IOM (Institute of Medicine). (2009a). *Integrative medicine and the health of the public: A summary of the February 2009 summit*. Washington, DC: The National Academies Press.
IOM (Institute of Medicine). (2009b). *Initial national priorities for comparative effectiveness research*. Washington, DC: The National Academies Press.
IOM (Institute of Medicine) (2011) *Patients charting the course: Citizen engagement in the learning health system*. Washington, DC: The National Academies Press.
Kallmes, D. F., Comstock, B. A., Heagerty, P. J., Turner, J. A., Wilson, D. J., Diamond, T. H., et al. (2009). A randomized trial of vertebroplasty for osteoporotic spinal fractures. *The New England Journal of Medicine, 361*(6), 569–579.
Kaptchuk, T. J., Kelley, J. M., Conboy, L. A., Davis, R. B., Kerr, C. E., Jacobson, E. E., et al. (2008). Components of placebo effect: Randomised controlled trial in patients with irritable bowel syndrome. *British Medical Journal, 336*, 999–1003.
Linde, K., Witt, C. M., Streng, A., Weidenhammer, W., Wagenpfeil, S., Brinkhaus, B., et al. (2007). The impact of patient expectations on outcomes in four randomized controlled trials of acupuncture in patients with chronic pain. *Pain, 128*, 264–271.
Miller, F. G., & Kaptchuk, T. J. (2008). The power of context: Reconceptualizing the placebo effect. *Journal of the Royal Society of Medicine, 101*, 222–225.
Moseley, J. B., O'Malley, K., Petersen, N. J., Menke, T. J., Brody, B. A., Kuykendall, D. H., et al. (2002). A controlled trial of arthroscopic surgery for osteoarthritis of the knee. *The New England Journal of Medicine, 347*(2), 81–88.
Ozminkoski, R. J., Ling, D., Goetzel, R. Z., Bruno, J. A., Rutter, K. R., Isaac, F., et al. (2002). Long-term impact of Johnson and Johnson's Health and Awareness Program on health care utilization and expenditures. *Journal of Occupational and Environmental Medicine, 44*(1), 21–29.
Richard, V., Milani, M. D., & Lavie, C. J. (2009). Impact of worksite wellness intervention on cardiac risk factors and one-year health care costs. *Preventive Cardiology, 104*(10), 1389–1392.
Snyderman, R., & Weil, A. T. (2002). Integrative medicine. Bringing medicine back to its roots. *Archives of Internal Medicine, 162*, 395–397.

Intellectual Disability

▶ Developmental Disabilities

Intellectual Testing

▶ Assessment

Intention

Peter A. Hall
Faculty of Applied Health Sciences, University of Waterloo, Waterloo, ON, Canada

I

Definition

Intention strength can be defined as the quantity of personal resources that an individual is prepared to invest in executing a behavior. Intention strength is closely akin to the concept of "motivation," with high levels of intention strength understood to represent strong motivation to perform a behavior. Intentions play a prominent role in several theories of health behavior, including the *Theory of Reasoned Action* (Fishbein & Ajzen, 1975), the *Theory of Planned Behavior* (Ajzen & Madden, 1986), the *Health Action Process Approach* (Schwarzer, 2001), and *Temporal Self-regulation Theory* (Hall & Fong, 2007). From an empirical perspective, intentions are among the strongest predictors of health behavior performance. However, a number of factors are known to moderate intention-behavior relations, including perceived/actual controllability of the behavior, as well as habit strength (Webb & Sheeran, 2006).

Cross-References

▶ Cognitive Mediators
▶ Theory of Planned Behavior
▶ Theory of Reasoned Action

References and Readings

Ajzen, I., & Madden, T. J. (1986). Prediction of goal-directed behavior: Attitudes, intentions, and perceived behavioral control. *Journal of Experimental Social Psychology, 22*, 453–474.

Fishbein, M., & Ajzen, I. (1975). *Belief, attitude, intention and behavior: An introduction to theory and research*. Reading, MA: Addison-Wesley.

Hall, P. A., & Fong, G. T. (2007). Temporal self-regulation theory: A model for individual health behavior. *Health Psychology Review, 1*, 6–52.

Schwarzer, R. (2001). Social-cognitive factors in changing health-related behaviors. *Current Directions in Psychological Science, 10*, 47–51.

Webb, T. L., & Sheeran, P. (2006). Does changing behavioral intentions engender behavior change? A meta-analysis of the experimental evidence. *Psychological Bulletin, 132*, 249–268.

Intention Strength

▶ Intention

Interest Testing

▶ Functional Versus Vocational Assessment

Interleukins

▶ Cytokines

Interleukins, -1 (IL-1), -6 (IL-6), -18 (IL-18)

Nicolas Rohleder
Department of Psychology, Brandeis University, Waltham, MA, USA

Synonyms

B-cell stimulatory factor 2; Cytotoxic T cell differentiation factor; Hepatocyte stimulating factor; Hybridoma growth factor; Hybridoma plasmacytoma growth factor; Lymphocyte-activating factor (LAF)

Definition

Interleukins (IL) are chemical messenger molecules of the immune system, and therefore fall into the category of cytokines. The term "interleukin" denotes their primarily ascribed function, i.e., the communication between (inter) white blood cells (leukocytes). IL-1, IL-6, and IL-18 are pro-inflammatory cytokines due to their predominant function to stimulate inflammatory processes in target cells.

Interleukin-1 is one of the first discovered cytokines, and consists of a family of three different molecules: IL-1alpha and IL-1beta exert their actions through binding to different members of the IL-1 receptor family, while the third molecule IL-1 receptor antagonist (IL-1ra) blocks these effects. The IL-1 family of cytokines was first cloned in 1984 (for a summary, see Dinarello, 1994). IL-1 has a wide range of effects, which include activation of interleukin-6 gene expression and other inflammatory mechanisms, thereby making it a central pro-inflammatory cytokine. In addition to inflammatory stimuli, IL-1 can also be activated by psychosocial factors: For example, IL-1beta gene expression has been shown to be upregulated following an acute laboratory stress paradigm (Brydon et al., 2005), and the same was reported for plasma concentrations of IL-1ra (Rohleder et al., 2006).

Interleukin-6 was first described as a T lymphocyte derived factor that was required for antibody production by B lymphocytes, and was consequently called B-cell stimulatory factor-2 (summarized, e.g., in Kishimoto, 2010). Additional functions were described in parallel, and led to the existence of different names for the same substance (e.g., hybridoma/plasmacytoma growth factor, or hepatocyte stimulating factor, etc.), and only the cloning of the molecule made clear that it was indeed the same protein, which was then named IL-6 (Poupart et al., 1987). IL-6 effects are not limited to the immune system, but include many tissues throughout the body. IL-6 is

therefore frequently referred to as a pleiotropic or endocrine cytokine (Papanicolaou & Vgontzas, 2000). While originally thought to be secreted only from immune cells upon inflammatory stimulation, it is now clear that IL-6 is secreted from a variety of cells, e.g., adipocytes and endothelial cells (e.g., Hoch et al., 2008; Kobayashi et al., 2003). IL-6 acts by interaction with a complex of the IL-6 receptor (IL-6R) and an additional protein gp130 (Heinrich, Behrmann, Muller-Newen, Schaper, & Graeve, 1998) and subsequent activation of the JAK-Stat pathway, ultimately inducing transcription of inflammatory gene products (Kishimoto, 2010).

Interleukin-18 was first cloned in 1995 and referred to as interferon-gamma inducing factor (IGIF) (Okamura et al., 1995). IL-18 shares some similarities in origin, receptors, and signaling pathways with the inflammatory cytokine IL-1, which implies overlaps in function, and underscores the role of IL-18 in inflammatory signaling (Arend, Palmer & Gabay, 2008). In contrast to IL-6, IL-18 seems to be produced mainly by cells of the immune system, such as monocytes/macrophages and dendritic cells. Similar to IL-6, however, IL-18 has the potential to affect more than immune cells, because the IL-18 receptor complex is present on endothelial cells, smooth muscle cells, as well as cells involved in bone and cartilage formation, in addition to immune cells. IL-18 signals through a receptor complex that bears similarity with the IL-1 receptor system, and as such activates the transcription of inflammatory gene products (Arend et al., 2008).

Inflammatory cytokines are interesting molecules in biobehavioral research, because they serve as a link between central nervous system states with pathophysiological factors that are central in a variety of human diseases. Inflammatory cytokine production is modulated by the sympathetic nervous system and the hypothalamus-pituitary-adrenal axis, and pro-inflammatory cytokines in blood are sensitive to acute and chronic stress, and found to be increased in depression and posttraumatic stress disorder (e.g., Steptoe, Hamer, & Chida, 2007; Rohleder, Marin, Ma, & Miller, 2009; Rohleder, Wolf, & Wolf, 2010). Inflammatory cytokines also increase with age, and have been found to predict later life morbidity and mortality (e.g., Bruunsgaard et al., 2003).

Cross-References

- Depression
- Inflammation
- Posttraumatic Stress Disorder
- Psychoneuroimmunology
- Stress

References and Readings

Arend, W. P., Palmer, G., & Gabay, C. (2008). IL-1, IL-18, and IL-33 families of cytokines. *Immunological Reviews, 223*, 20–38.

Bruunsgaard, H., Ladelund, S., Pedersen, A. N., Schroll, M., Jorgensen, T., & Pedersen, B. K. (2003). Predicting death from tumour necrosis factor-alpha and interleukin-6 in 80-year-old people. *Clinical and Experimental Immunology, 132*(1), 24–31.

Brydon, L., Edwards, S., Jia, H., Mohamed-Ali, V., Zachary, I., Martin, J. F., et al. (2005). Psychological stress activates interleukin-1beta gene expression in human mononuclear cells. *Brain, Behavior, and Immunity, 19*(6), 540–546.

Dinarello, C. A. (1994). The interleukin-1 family: 10 years of discovery. *The FASEB Journal, 8*(15), 1314–1325.

Heinrich, P. C., Behrmann, I., Muller-Newen, G., Schaper, F., & Graeve, L. (1998). Interleukin-6-type cytokine signalling through the Gp130/Jak/Stat pathway. *Biochemical Journal, 334*(Pt 2), 297–314.

Hoch, M., Eberle, A. N., Peterli, R., Peters, T., Seboek, D., Keller, U., et al. (2008). LPS induces interleukin-6 and interleukin-8 but not tumor necrosis factor-alpha in human adipocytes. *Cytokine, 41*(1), 29–37.

Kishimoto, T. (2010). IL-6: From its discovery to clinical applications. *International Immunology, 22*(5), 347–352.

Kobayashi, S., Nagino, M., Komatsu, S., Naruse, K., Nimura, Y., Nakanishi, M., et al. (2003). Stretch-induced IL-6 secretion from endothelial cells requires NF-kappaB activation. *Biochemical and Biophysical Research Communications, 308*(2), 306–312.

Okamura, H., Tsutsi, H., Komatsu, T., Yutsudo, M., Hakura, A., Tanimoto, T., et al. (1995). Cloning of a new cytokine that induces IFN-gamma production by T cells. *Nature, 378*(6552), 88–91.

Papanicolaou, D. A., & Vgontzas, A. N. (2000). Interleukin-6: The endocrine cytokine. *The Journal of Clinical Endocrinology and Metabolism, 85*(3), 1331–1333.

Poupart, P., Vandenabeele, P., Cayphas, S., Van Snick, J., Haegeman, G., Kruys, V., et al. (1987). B cell growth modulating and differentiating activity of recombinant

I

human 26-kd protein (BSF-2, HuIFN-beta 2, HPGF). *The EMBO Journal, 6*(5), 1219–1224.
Rohleder, N., Marin, T. J., Ma, R., & Miller, G. E. (2009). Biologic cost of caring for a cancer patient: Dysregulation of pro- and anti-inflammatory signaling pathways. *Journal of Clinical Oncology, 27*(18), 2909–2915.
Rohleder, N., Wolf, J. M., Herpfer, I., Fiebich, B. L., Kirschbaum, C., & Lieb, K. (2006). No response of plasma substance P, but delayed increase of interleukin-1 receptor antagonist to acute psychosocial stress. *Life Sciences, 78*(26), 3082–3089.
Rohleder, N., Wolf, J. M., & Wolf, O. T. (2010). Glucocorticoid sensitivity of cognitive and inflammatory processes in depression and posttraumatic stress disorder. *Neuroscience and Biobehavioral Reviews, 35*(1), 104–114.
Steptoe, A., Hamer, M., & Chida, Y. (2007). The effects of acute psychological stress on circulating inflammatory factors in humans: A review and meta-analysis. *Brain, Behavior, and Immunity, 21*(7), 901–912.

Intermediate Variable

► Mediators

Intermittent Claudication

► Peripheral Arterial Disease (PAD)/Vascular Disease

Internal and External Validity Issues

► RE-AIM Guidelines

International Society of Behavioral Medicine

Neil Schneiderman
Department of Psychology, Behavioral Medicine Research Center, University of Miami, Coral Gables, FL, USA

Basic Information

The International Society of Behavioral Medicine (ISBM) is a federation of 26 national, regional, and more specialized scientific societies, whose goal is to serve the needs of all health-related disciplines concerned with issues relevant to behavioral medicine. Behavioral medicine is an interdisciplinary field concerned with the development and integration of sociocultural, psychosocial, behavioral, and biomedical knowledge relevant to health and illness and the application of this knowledge to disease prevention, health promotion, etiology, diagnosis, treatment, and rehabilitation. Each full member constituent society of the ISBM includes both biomedical and behavioral scientists. Societies with interests relevant to behavioral medicine, but which do not meet all criteria for membership (e.g., having both biomedical and behavioral scientists as individual members), may apply to become affiliate members. The Division of Health Psychology of the American Psychological Association and the Association of Pediatric Psychology are affiliate members. The ISBM was formally founded in June 1990 in conjunction with the First International Congress of Behavioral Medicine, held in Uppsala, Sweden.

One major goal of the ISBM has been to encourage the formation of national and regional societies of behavioral medicine. Beginning with six founding organizations (Academy of Behavioral Medicine Research, USA; Czechoslovakia Medical Association Section on Neurohumoral Integration and Behavioral Medicine and Modification; German Association of Behavioral Medicine and Modification; Dutch Behavioral Medicine Federation; Society of Behavioral Medicine, USA; and the Swedish Society of Behavioral Medicine), by 2010 there were 24 national societies and 2 regional societies (An Australasian Society constituted primarily of members from Australia and New Zealand; and a Central and Eastern European Society with membership from 11 central and eastern European countries). A change in the ISBM bylaws in 2011 now allows specialized scientific societies besides national and regional scientific societies, who otherwise meet ISBM membership criteria, to apply for ISBM membership. It is important to note that societies rather than individual persons belong to the ISBM. Individuals

living in a country that does not have a society belonging to ISBM can derive ISBM benefits (e.g., reduced registration fees for the international congress; journal access) by joining an existing society belonging to ISBM. Listing of the constituent societies of the ISBM as well as the bylaws, charter, membership information, activities of the society, newsletter and members of the Executive Committee, Board and Governing Council can be found at the ISBM website http://www.isbm.info.

A second major goal of the ISBM is to encourage and coordinate communications and interactions among various health professionals including biomedical and behavioral science researchers, educators, clinicians and public health workers without regard to specific discipline loyalties. One major vehicle for promoting interdisciplinary, international scientific communication has been via the International Congresses of Behavioral Medicine, staged by the ISBM in conjunction with host societies every two years since 1990. The Congresses feature keynote addresses master lectures, symposia, oral presentations, posters, and preconference workshops. A unique feature of each symposium is that it usually features representation from more than one country with participation from more than one discipline.

The scientific program features approximately 25 distinct tracks covering a range of biomedical, behavioral, and sociocultural behavioral medicine topics. Symposia and oral presentations are presented in approximately eight simultaneous, parallel sessions. Poster sessions are prominently featured so that they are not in conflict with the oral presentations. About 800 individuals, most formally presenting their research, attend the Congresses. A listing of the ISBM presidents, congresses, and program chairs is presented below.

President	Term	Congress	Date	ISBM Program Chair (s)
		Uppsala	1990	Arne Öhman/ Andrew Steptoe
Stephen Weiss	1990–1992	Hamburg	1992	Kristina Orth-Gomér
Kristina Orth-Gomér	1992–1994	Amsterdam	1994	Neil Schneiderman
Andrew Steptoe	1994–1996	Washington	1996	*Ad Appels/ Craig Ewart* (SBM)
Johannes Siegrist	1996–1998	Copenhagen	1998	Jane Wardle
Neil Schneiderman	1998–2000	Brisbane	2000	Margaret Chesney
Brian Oldenberg	2000–2002	Helsinki	2002	Christina Lee
Gunilla Burell	2002–2004	Mainz	2004	Neil Schneiderman
Antti Uutela	2004–2006	Bangkok	2006	Brian Oldenburg
Redford Williams	2006–2008	Tokyo	2008	Theresa Marteau
Hege Eriksen	2008–2010	Washington	2010	Linda Baumann
Norito Kawakami	2010–2012	Budapest	2012	Frank Penedo
Joost Dekker	2012–2014	Groningen	2014	

Besides the congresses, a second major vehicle for promoting interdisciplinary, international scientific communication by the ISBM has been through the *International Journal of Behavioral Medicine* (IJBM). The IJBM, which is the official publication of the ISBM, presents original research and integrative reviews on interactions among behavioral, psychosocial, environmental, genetic, and biomedical factors relevant to health and illness. The scope of the IJBM extends from research on biobehavioral mechanisms and clinical studies on diagnosis, treatment, and rehabilitation to research on public health, including health promotion and prevention. The IJBM was originally published in 1994 with Lawrence Erlbaum Associates as the publisher and Neil Schneiderman (1994–1998) as founding editor. Ulf Lundberg succeeded Schneiderman as editor from 1999 to 2005 and was himself succeeded by Joost Dekker (2006–2011). Starting with Volume 16 in 2009, under the editorship of Dekker, Springer became the publisher of IJBM. Beginning in 2012, the editorship of the IJBM passed to Christina Lee. Throughout its existence the IJBM has had a strong, scholarly

group of associate editors, balanced in terms of discipline, scientific expertise, geography, and gender. They, in turn, have been supported by an effective editorial board. Within the ISBM the reach of the journal is extensive as all members of ISBM member societies receive free access to the online version of the IJBM. The IJBM offers a free table of contents alert e-mail service that notifies individuals as soon as a new issue of IJBM has been published online.

A third vehicle for promoting interdisciplinary, international scientific communication within the ISBM has been the ISBM Newsletter. Published twice per year, the newsletter provides an outlet for communication to and from the membership of the constituent societies of the ISBM including reports from the ISBM president, newsletter editor, IJBM editor, ISBM committee chairs (e.g., program committee), information of interest from member societies, and interviews with various people who have contributed to the development of the ISBM.

Still a fourth vehicle for promoting interdisciplinary, international scientific communication has been through education and training activities including workshops and teaching seminars that have featured collaborations between prominent members of the ISBM and potential, emerging, or established member societies. These have led to the founding or strengthening of member societies of the ISBM and to published volumes (e.g., Orth-Gomér & Schneiderman, 1996). The Education and Training Committee also has interest in monitoring, developing, and publicizing curricula relevant to behavioral medicine.

The governance of the ISBM occurs through its Governing Council, Executive Committee and Board. Supervising the control and direction of the affairs of the ISBM occurs through the Council. This Council comprises one voting representative from each member society of the ISBM, who is designated by that society. The Board, which conducts the normal deliberative business of the ISBM between Council meetings, reports to the Council. In instances in which the ISBM President finds that there is need for immediate action by a group smaller than the Board, the President will call upon an Executive Committee consisting of the President, Past-President, President-Elect, Secretary, and Treasurer. The Board consists of the members of the Executive Committee, the chairs of the Communications, Education and Training, Finance, International Collaborative Studies, Membership, Nominating, Organizational Liaison, Program, and Strategic Planning committees as well as the editors of the IJBM and newsletter and any elected member(s)-at-large. Although the outcome of elections is based upon the votes of the Governing Council, the ISBM has attempted to consider balance in terms of discipline, scientific stature, gender, and age.

Major Impact on the Field

The International Society of Behavioral Medicine (ISBM) is the largest Behavioral Medicine Organization in the world. Its constituent 26 national and regional societies represent the vast majority of self-identified behavioral medicine researchers in the world. Through its website, Congresses, journals, newsletters, and teaching seminars, the ISBM facilitates communications among behavioral medicine scientists and other professionals throughout the world.

References and Readings

Orth-Gomér, K., & Schneiderman, N. (1996). *Behavioral medicine approaches to cardiovascular disease prevention*. Mahwah, NJ: Erlbaum.

Steptoe, A. (2010). *Handbook of behavioral medicine: Methods and applications*. New York: Springer.

Internet Science

▶ Internet-Based Studies

Internet-Based Interventions

▶ eHealth and Behavioral Intervention Technologies

Internet-Based Studies

Ulf-Dietrich Reips
Faculty of Engineering; Faculty of Education and Psychology, Universidad de Deusto, Bilbao, Spain
IKERBASQUE, Basque Foundation for Science, Bilbao, Spain

Synonyms

Internet science; iScience; Internet-mediated studies; Web-based studies

Definition

Internet-based studies are roughly systematized in four categories (Reips, 2006): Internet-based experiments, web surveys and questionnaires, Internet-based assessment, and nonreactive data collection on the Internet. In a wider sense, studies about human activities on the Internet can also be defined as Internet-based studies.

Description

Beginning

The first Internet-based studies were conducted in the mid-1990s, shortly after the World Wide Web had been invented at CERN in Geneva (Musch & Reips, 2000; Reips, 2006). Conducting studies via the Internet is considered a second revolution in behavioral research, after the computer revolution in the late 1960s and early 1970s that brought about many advantages over widely used paper-and-pencil procedures (e.g., automated processes, heightened precision). The Internet revolution in behavioral research added the dimension of interactivity via a worldwide network that resulted in several mostly advantageous characteristics of Internet-based studies (see below).

Examples for studies conducted on the WWW, current and archived, can be viewed at sites like the *Web experiment list* (http://wexlist.net, Reips & Lengler, 2005), see Fig. 1, and the *Psychological Research on the Net* list that is maintained by John Krantz (http://psych.hanover.edu/research/exponnet.html).

Characteristics

Although Internet-based studies have some inherent limitations due to a lack of control and observation of conditions, they also have a number of advantages over lab research (Birnbaum, 2004; Kraut et al., 2004; Reips, 2002; Schmidt, 1997). Some of the chief advantages are that (1) researchers can recruit and study large numbers of participants very quickly; (2) it is possible to collect and access large behavioral data sets, recruit large heterogeneous samples and people with rare characteristics (e.g., people who have had biofeedback training in more than one country or persons suffering from sexsomnia and their peers, Mangan & Reips, 2007) from locations far away; and (3) the method is more cost-effective in time, space, and labor in comparison with lab research. Compared to paper-and-pencil research, most of the advantages of computer-mediated research apply, for example, process variables ("paradata") that can be recorded (Stieger & Reips, 2010).

Types

Internet-based studies are roughly systematized in four categories (Reips, 2006, see Fig. 2): Internet-based experiments (Reips, 2002), web surveys and questionnaires (Dillman & Bowker, 2001; Dillman, Smyth, & Christian, 2009), Internet-based assessments (Buchanan, 2001; Buffardi & Campbell, 2008), and nonreactive data collection on the Internet (Reips & Garaizar, 2011). Within psychology, most Internet-based research is conducted in the fields of social psychology and cognition (Musch & Reips, 2000; Reips & Lengler, 2005). Within behavioral medicine, as judged from an analysis of the articles published in the *Journal of Medical Internet Research*, the most frequented fields seem to be e-health and e-mental health, in particular depression, obesity/weight management, and smoking cessation.

Internet-Based Studies, Fig. 1 The web experiment list and web survey list

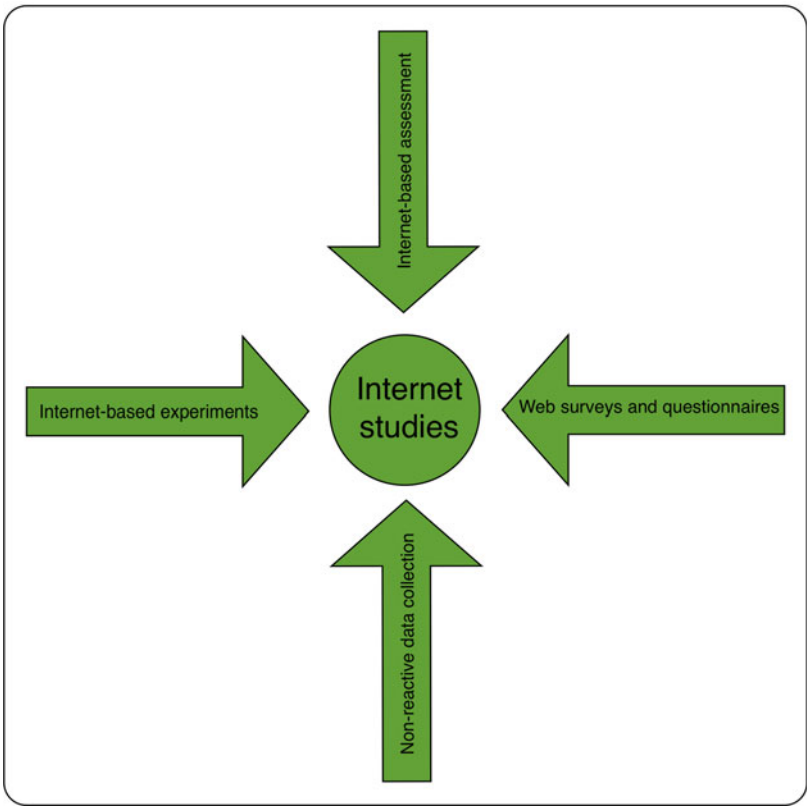
In a wider sense, studies about human activities on the Internet (e.g., accessing Internet support groups, online dating) can also be defined as Internet-based studies (e.g., Eysenbach, Powell, Englesakis, Rizo, & Stern, 2004; Whitty & Buchanan, 2010).

Recently, the advent of social media, that is, highly interactive Internet-based communication platforms, has spurred the interest of researchers. Social networking services like Facebook, Tuenti, Orkut, LinkedIn, Twitter, and Student VZ are seen as vast resources for detailed descriptions of human behavior (Reips & Garaizar, 2011).

Methods

For anyone planning to conduct studies via the Internet, it is important to realize that there is a growing body of literature on theoretical insights, empirical results, and practical guidelines that needs to be taken into account when setting up Internet-based data collection.

Reips (2002) and Reips and Birnbaum (2011) provide guidelines and overviews of techniques, methods, and tools for Internet-based studies. They summarize challenges and solutions in design, security, recruitment, sampling, self-selection, multiple submissions, reactance-free question



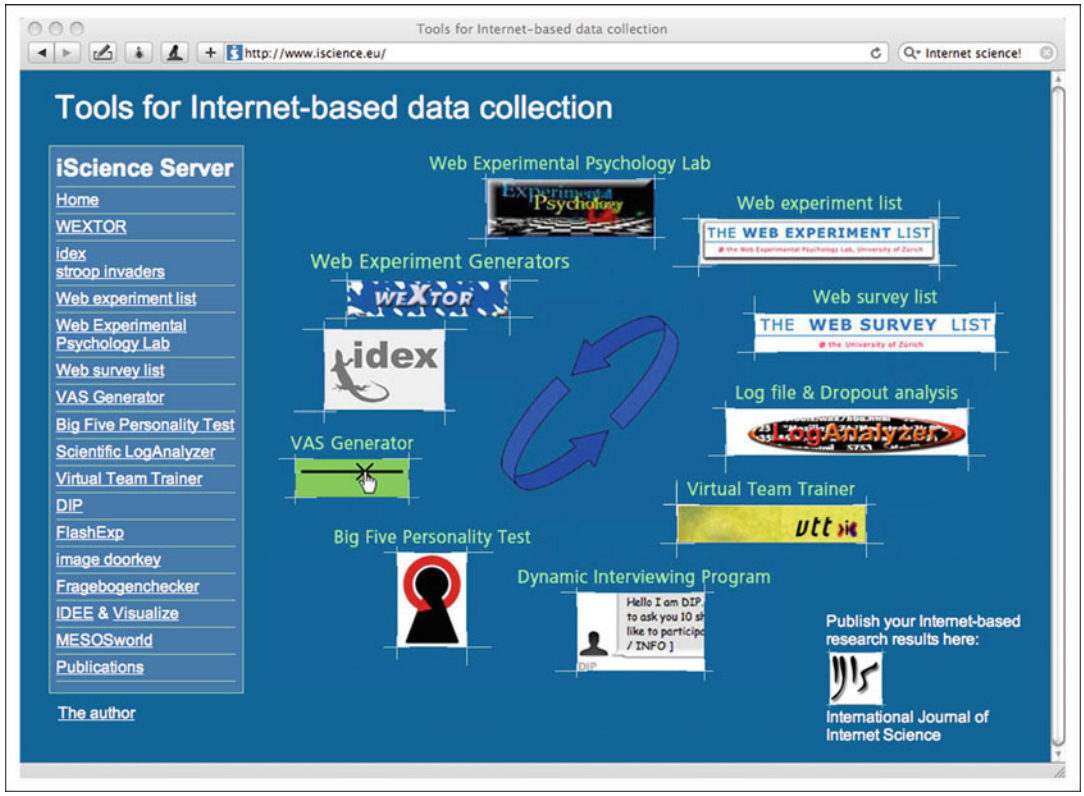
Internet-Based Studies, Fig. 2 Types of Internet-based studies

design, response time measurement, dropout, error estimation, data handling, and data quality. For example, in creating items for a Web questionnaire it is often forgotten to add a neutral option ("please choose") in drop-down menus. Thus, one of the answer options is automatically selected, even if the respondent skips the item. Another pitfall is the higher likelihood for attrition on the web, compared to lab-based studies. Among other methods that have been developed in web methodology and Internet science, Reips (2010) explains the one-item-one-screen (OIOS) design, the seriousness check, subsampling procedures, multiple site entry, and the high hurdle technique and discusses empirical results from investigations into the validity of several of the techniques.

Implications

Most authors agree that the Internet is a viable option for conducting studies in the behavioral and social sciences (Birnbaum, 2004; Joinson, McKenna, Postmes, & Reips, 2007; Kraut et al., 2004; Musch & Reips, 2000; Reips, 2002, 2006). Care should be taken to properly implement an adequate methodology suited for Internet-based studies (Reips, 2002) and consider potentially biasing effects of technology (Schmidt, 2007).

Two important issues for much of human interaction on and with the Internet are: real and perceived privacy as well as trust, for example, in accessing health-related information via the Internet (Buchanan, Joinson, Paine, & Reips, 2007; Eysenbach et al., 2004). Such information can be accessed more easily via the Internet, in part because it can be found more privately. Threats to privacy on the Internet reduce the willingness to look for information online. Furthermore, people are less likely to search for healthcare information through mechanisms or agencies that they do not trust



Internet-Based Studies, Fig. 3 A portal for researchers that links to many tools for Internet-based studies: the iScience Server at http://iscience.eu

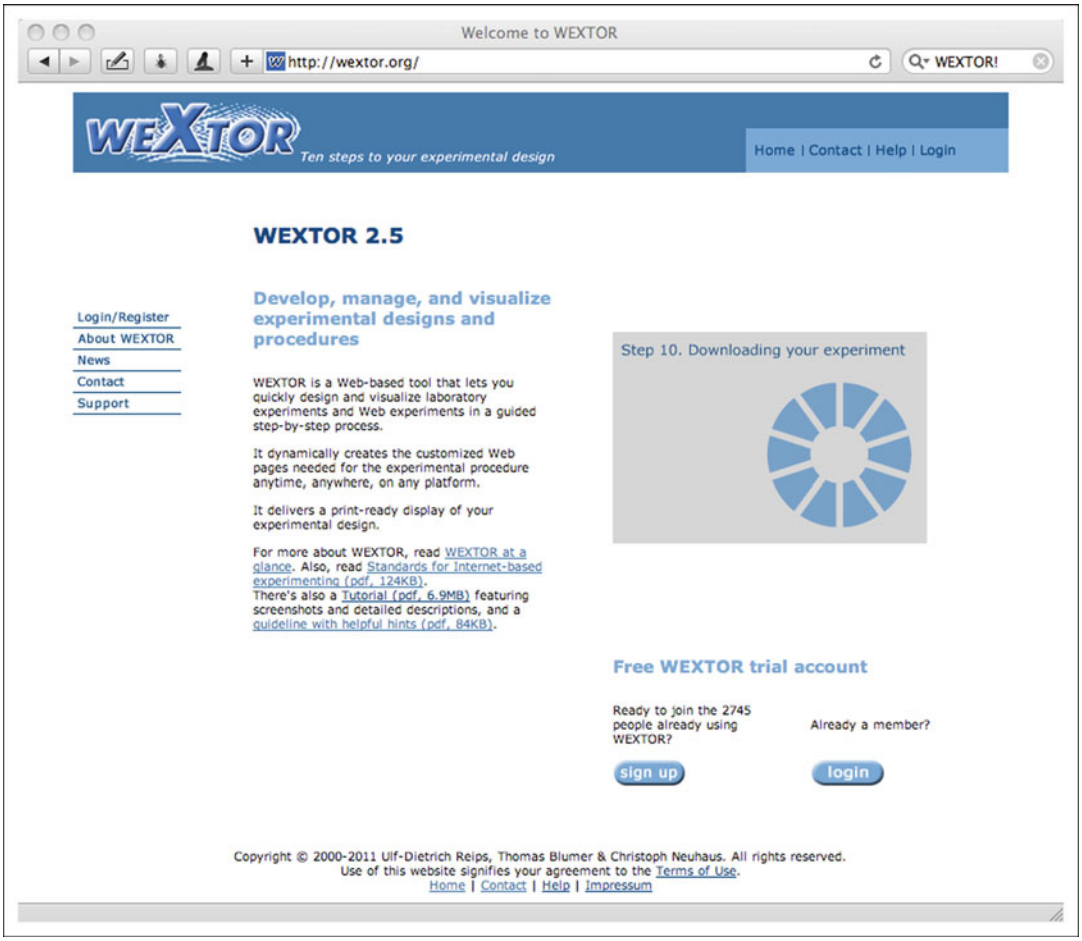
(Buchanan et al., 2007). For the data quality in Internet-based studies that usually involve self-disclosure perceived, privacy and trust are essential as well (Joinson, Reips, Buchanan, & Paine Schofield, 2010).

It seems important that authors, reviewers, and editors of articles reporting results from Internet-based studies follow the guidelines that were developed in this new field. Scientists in behavioral medicine considering to conduct research via the Internet will thus need to prepare for challenges like more heterogeneous samples, higher nonresponse, and the effects of mode and technologies. Benefits like wider reach, better quality of data from truly voluntary respondents, and a reduced tendency for socially desirable responding, reduced costs, and validated tools for Internet-based research will further increase the use of the new methodology.

Resources

The following resources are available for researchers who want to conduct Internet-based studies:

- iScience Server (http://iscience.eu/, Fig. 3): A portal for the methodology of Internet-based research
- iScience Maps (http://tweetminer.eu/, Reips & Garaizar, 2011): A data mining tool for the social media service Twitter
- Questionnaire evaluator (http://iscience.eu/fbchecker/): An interactive web tool to examine questions and questionnaires (in German and English)
- Big 5 (http://webscience.deusto.es/big5/): A self-scoring Big Five personality test ready for use by simply linking it to another online study (in English, German, and Spanish)



Internet-Based Studies, Fig. 4 WEXTOR, a web service to generate and conduct Internet-based experiments

- VAS Generator (http://www.vasgenerator.net/, Reips & Funke, 2008): A tool to create visual analogue scales (VAS) for online studies
- WEXTOR (http://wextor.org, Reips & Neuhaus, 2002, Fig. 4): A system for conducting web-based experiments

References and Readings

Birnbaum, M. H. (2004). Human research and data collection via the Internet. *Annual Review of Psychology, 55*, 803–832.

Buchanan, T. (2001). Online personality assessment. In U.-D. Reips & M. Bosnjak (Eds.), *Dimensions of Internet science* (pp. 57–74). Lengerich, Germany: Pabst Science.

Buchanan, T., Joinson, A. N., Paine, C., & Reips, U.-D. (2007). Looking for medical information on the Internet: Self-disclosure, privacy and trust. *He@lth Information on the Internet, 58*, 8–9.

Buffardi, L. E., & Campbell, W. K. (2008). Narcissism and social networking web sites. *Personality and Social Psychology Bulletin, 34*, 1303–1314.

Dillman, D. A., & Bowker, D. (2001). The web questionnaire challenge to survey methodologists. In U.-D. Reips & M. Bosnjak (Eds.), *Dimensions of Internet science* (pp. 159–177). Lengerich, Germany: Pabst Science.

Dillman, D. A., Smyth, J. D., & Christian, L. M. (2009). *Internet, mail and mixed-mode surveys: The tailored design method* (3rd ed.). Hoboken, NJ: Wiley.

Eysenbach, G., Powell, J., Englesakis, M., Rizo, C., & Stern, A. (2004). Health-related virtual communities and electronic support groups: Systematic review of the effects of online peer-to-peer interactions. *British Medical Journal, 328*, 1166–1170.

Joinson, A. N., McKenna, K., Postmes, T., & Reips, U.-D. (Eds.). (2007). *The Oxford handbook of Internet psychology*. Oxford: Oxford University Press.

Joinson, A. N., Reips, U.-D., Buchanan, T., & Paine Schofield, C. (2010). Privacy, trust, and self-disclosure online. *Human Computer Interaction, 25*, 1–24.

Kraut, R., Olson, J., Banaji, M., Bruckman, A., Cohen, J., & Couper, M. (2004). Psychological research online: Report of board of scientific affairs' advisory group on the conduct of research on the Internet. *American Psychologist, 59*, 105–117.

Mangan, M., & Reips, U.-D. (2007). Sleep, sex, and the Web: Surveying the difficult-to-reach clinical population suffering from sexsomnia. *Behavior Research Methods, 39*, 233–236.

Musch, J., & Reips, U.-D. (2000). A brief history of web experimenting. In M. H. Birnbaum (Ed.), *Psychological experiments on the Internet* (pp. 61–88). San Diego, CA: Academic Press.

Reips, U.-D. (2002). Standards for Internet-based experimenting. *Experimental Psychology, 49*, 243–256.

Reips, U.-D. (2006). Web-based methods. In M. Eid & E. Diener (Eds.), *Handbook of multimethod measurement in psychology* (pp. 73–85). Washington, DC: American Psychological Association.

Reips, U.-D. (2007). The methodology of Internet-based experiments. In A. Joinson, K. McKenna, T. Postmes, & U.-D. Reips (Eds.), *The Oxford handbook of Internet psychology* (pp. 373–390). Oxford: Oxford University Press.

Reips, U.-D. (2010). Design and formatting in Internet-based research. In S. Gosling & J. Johnson (Eds.), *Advanced Internet methods in the behavioral sciences* (pp. 29–43). Washington, DC: American Psychological Association.

Reips, U.-D., & Birnbaum, M. H. (2011). Behavioral research and data collection via the Internet. In R. W. Proctor & K.-P. L. Vu (Eds.), *The handbook of human factors in web design* (2nd ed.). Mahwah, NJ: Erlbaum.

Reips, U.-D., & Bosnjak, M. (Eds.). (2001). *Dimensions of Internet science*. Lengerich, Germany: Pabst Science.

Reips, U.-D., & Garaizar, P. (2011). Mining Twitter: Microblogging as a source for psychological wisdom of the crowds. *Behavior Research Methods, 43*, 635–642.

Reips, U.-D., & Funke, F. (2008). Interval level measurement with visual analogue scales in Internet-based research: VAS Generator. *Behavior Research Methods, 40*, 699–704.

Reips, U.-D., & Lengler, R. (2005). The web experiment list: A web service for the recruitment of participants and archiving of Internet-based experiments. *Behavior Research Methods, 37*, 287–292.

Reips, U.-D., & Neuhaus, C. (2002). WEXTOR: A web-based tool for generating and visualizing experimental designs and procedures. *Behavior Research Methods, Instruments, & Computers, 34*, 234–240.

Schmidt, W. C. (1997). World-Wide Web survey research: Benefits, potential problems, and solutions. *Behavioral Research Methods, Instruments, & Computers, 29*, 274–279.

Schmidt, W. C. (2007). Technical considerations when implementing online research. In A. Joinson, K. McKenna, T. Postmes, & U.-D. Reips (Eds.), *The Oxford handbook of Internet psychology* (pp. 461–472). Oxford: Oxford University Press.

Stieger, S., & Reips, U.-D. (2010). What are participants doing while filling in an online questionnaire: A paradata collection tool and an empirical study. *Computers in Human Behavior, 26*, 1488–1495.

Whitty, M. T., & Buchanan, T. (2010). "What's in a 'Screen Name'?" Attractiveness of different types of screen names used by online daters. *International Journal of Internet Science, 5*, 5–19.

Internet-Mediated Studies

▶ Internet-Based Studies

Interpersonal Circumplex

Timothy W. Smith
Department of Psychology, University of Utah, Salt Lake City, UT, USA

Synonyms

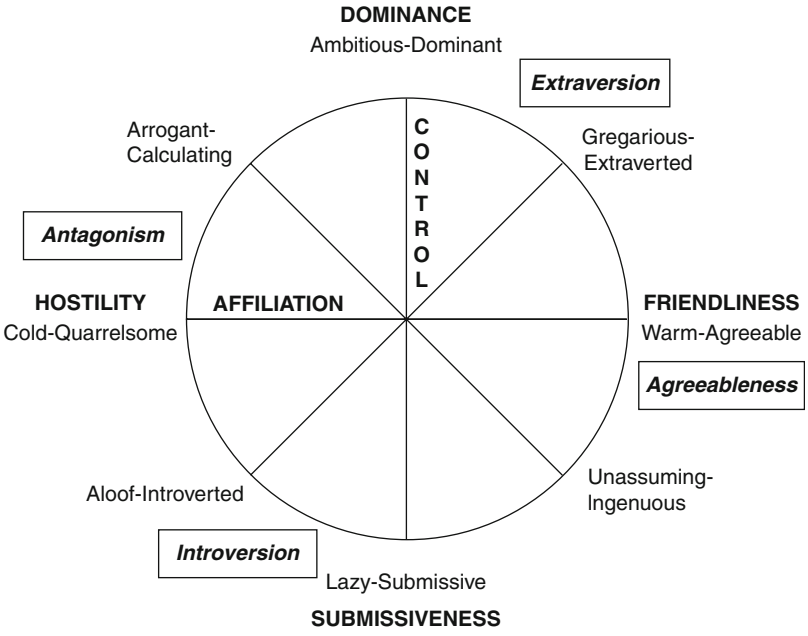
Affiliation; Control; Dominance; Hostility; Social behavior; Submissiveness; Warmth

Definition

The interpersonal circumplex (IPC; see Fig. 1) is a conceptual model of social behavior, comprising two main dimensions: *affiliation* (i.e., warmth, friendliness vs. hostility, quarrelsomeness) and *control* (i.e., dominance, directiveness vs. submissiveness, deference).

Description

The IPC has a long history, dating to the work of Sullivan, Leary, and other mid-twentieth century behavioral scientists (for a review, see Fornier,



Interpersonal Circumplex, Fig. 1 The interpersonal circumplex

Moskowitz, & Zuroff, 2011). This structural model is a cornerstone of the interpersonal tradition in personality, clinical, and social psychology (Horowitz & Strack, 2011) and can be used to describe momentary social behavior, more stable individual differences in social behavior (i.e., personality traits), and characteristics of social relationships and social contexts. The IPC describes specific actions as blends of control and affiliation, and similarly describes personality characteristics and aspects of social relationships as varying in dominance versus submissiveness and warmth versus hostility. In terms of personality traits, as seen in Fig. 1, affiliation and control are rotational equivalents of the Five Factor Model traits of agreeableness versus antagonism and extraversion versus introversion. Agreeableness corresponds to somewhat submissive warmth in the IPC, whereas extraversion corresponds to somewhat warm dominance.

The interpersonal circumplex can also be used to describe social motives. In interpersonal theory, social interactions involve the exchange of two broad and primary social resources: status (i.e., esteem or regard from others) and love (i.e., liking, acceptance, or inclusion by others). These resources are the focus of two similarly broad social motives; *agency* refers to striving for status, achievement, or power, whereas *communion* refers to striving for connection with others and the maintenance of relationships. Hence, agency and communion are the motivational equivalents of the IPC control and affiliation dimensions, respectively. Dominant behavior reflects an attempt to gain or assert status, whereas deference or submissiveness reflects granting status to an interaction partner. Warm behavior reflects seeking affection, acceptance, or inclusion from interaction partners, or granting such connection to them. Hostile behavior, in contrast, reflects withholding or revoking such connection.

The IPC is the conceptual foundation for another major tenet of the interpersonal tradition – the principle of complementarity (for a review, see Sadler, Etheir, & Woody, 2011). In this view, social behavior of one individual tends to invite or evoke responses from an interaction partner that are similar in affiliation (i.e., warmth invites warmth; hostility invites hostility) and opposite in control (i.e., dominance invites deference). Empirical evidence regarding complementarity for the affiliation dimension of the IPC is stronger than for the control dimension (Sadler et al., 2011).

In behavioral medicine, psychosomatic medicine, and health psychology, the IPC has been used primarily in the study of psychosocial risk factors for disease and the psychophysiological mechanisms underlying those associations. Specifically, the IPC and the related concept of complementarity are useful in the development of an integrated understanding of psychosocial influences on disease (Smith & Cundiff, 2011; Smith, Glazer, Ruiz, & Uchino, 2004). A variety of personality characteristics, symptoms of emotional distress, and emotional disorders have been studied as potential risk factors for serious illnesses (e.g., coronary heart disease), all-cause mortality, and reduced longevity. Similarly, a variety of aspects of social relationships (e.g., social isolation, low social support, conflict or strain in personal relationships) and social contexts (low socioeconomic status of individuals and neighborhoods, job stress) have been examined as risk factors for these same outcomes. However, these risk factors are usually studied individually, with little concern about their potentially overlapping associations with disease (Smith, 2010; Suls & Bunde, 2005). Further, risk factors that are conceptualized as aspects of individuals (e.g., personality, emotional distress) are typically seen as a class of influences on disease that is distinct from characteristics of social relationships and contexts also examined as risk factors (e.g., low social support; Lett et al., 2005). This piecemeal approach to risk factors impedes an integrated view of psychosocial risk as it ignores overlapping influences on disease and naturally occurring patterns or aggregations of risk factors (Smith & Cundiff, 2011). To address this problem, measures of multiple psychosocial risk factors can be correlated with independent measures of the two IPC dimensions. Such analyses identify the associated interpersonal style or characteristics associated with multiple risk factors and can thereby identify broader or common dimensions of risk.

For example, anger and hostility predict the development and course of coronary heart disease (CHD), as do depression and anxiety (Chida & Steptoe, 2009; Nicholson, Kuper, & Hemingway, 2006). Each of these individual differences in negative affect is associated with low affiliation in the IPC (Gallo & Smith, 1998; Smith, Traupman, Uchino, & Berg, 2010). The substantial interrelationship among these negative affective characteristics and the fact that they also share a common interpersonal style characterized by low warmth and high quarrelsomeness suggest that they might have overlapping associations with subsequent disease. The complementarity principle suggests that given this specific interpersonal style, these diverse risk factors should also be associated with low warmth and greater conflict in personal relationships, a prediction supported by a growing body of research (for a review, see Smith & Cundiff, 2011). Hence, these diverse risk factors may be linked to adverse health outcomes in part by common interpersonal processes – low levels of social support and high levels of interpersonal conflict.

Risk factors that are conceptualized as aspects of the social environment (e.g., social isolation, low social support, conflict in close relationships) can also be located in the IPC. For example, individuals who report low levels of social support describe their relationships as low in warmth and high in hostility, and reports of conflict and strain in close relationships are associated with seeing one's partner as expressing little warmth and high levels of both hostility and control (Smith et al., 2010; Trobst, 2000). Low socioeconomic status is associated with a variety of negative health outcomes and is also associated with exposure to lower warmth from others and higher levels of both hostility and control (Gallo, Smith & Cox, 2006). That is, low SES individuals often feel both "put off" and "put down" during interactions with others.

The control dimension of the IPC has also been directly related to health outcomes as the personality trait of dominance is associated with greater risk of coronary heart disease (Smith et al., 2008; for a review, see Smith & Cundiff, 2011). Further, high levels of control during marital interactions is associated with increased risk of atherosclerosis among men, whereas low levels of warmth are associated with increased risk among women (Smith et al., 2011).

The IPC also provides a framework for examining the psychophysiological mechanisms linking psychosocial risk factors and disease, such as cardiovascular, neuroendocrine, inflammatory, and immunologic responses to stressors. As noted previously, the IPC can be used to describe individual differences in social behavior (i.e., personality traits), stable aspects of social relationships or contexts, momentary social behavior, and social stimuli. With the IPC, these four classes of influences on physiological stress responses can be examined in a single, integrative framework (Smith, Gallo, & Ruiz, 2003). In general, the IPC framework and related research calls attention to the importance of recurring patterns of interpersonal behavior as a common process underlying many psychosocial influences on health. The related research suggests that recurring interactions involving low affiliation (i.e., low levels of warmth, high levels of hostility), effortful expression of control toward others (i.e., high dominance), or exposure to unwelcome control by others may be common elements of psychosocial risk (Smith et al., 2010).

Cross-References

- ▶ Interpersonal Relationships
- ▶ Personality
- ▶ Social Relationships
- ▶ Social Stress
- ▶ Social Support

References and Readings

Chida, Y., & Steptoe, A. (2009). The association of anger and hostility with future coronary heart disease: A meta-analytic review of prospective evidence. *Journal of the American College of Cardiology, 53*, 774–778.

Fournier, M. A., Moskowitz, D. S., & Zuroff, D. C. (2011). Origins and applications of the interpersonal circumplex. In L. M. Horowitz & S. Strack (Eds.), *Hanbook of interpersonal psychology: Theory, research, assessment, and therapeutic interventions* (pp. 57–73). Hoeboken, NJ: Wiley.

Gallo, L. C., & Smith, T. W. (1998). Construct validation of health-relevant personality traits: Interpersonal circumplex and five-factor model analyses of the aggression questionnaire. *International Journal of Behavioral Medicine, 5*, 129–147.

Gallo, L. C., Smith, T. W., & Cox, C. (2006). Socioeconomic status, psychosocial processes, and perceived health: An interpersonal perspective. *Annals of Behavioral Medicine, 31*, 109–119.

Horowitz, L. M., & Strack, S. (2011). *Handbook of interpersonal psychology: Theory, research, assessment, and therapeutic intervention*. Hoboken, NJ: Wiley.

Lett, H. S., Blumenthal, J. A., Babyak, M., Strauman, T., Robbins, C., & Sherwood, A. (2005). Social support and coronary heart disease: Epidemiologic evidence and implications for treatment. *Psychosomatic Medicine, 67*, 869–878.

Nicholson, A., Kuper, H., & Hemingway, H. (2006). Depression as an aetiologic and prognostic factor in coronary heart disease: A meta-analysis of 6,362 events among 146,538 participants in 54 observational studies. *European Heart Journal, 27*, 2763–2774.

Sadler, P., Ethier, N., & Woody, E. (2011). Interpersonal complementarity. In L. M. Horowitz & S. Strack (Eds.), *Hanbook of interpersonal psychology: Theory, research, assessment, and therapeutic interventions* (pp. 123–142). Hoeboken, NJ: Wiley.

Smith, T. W. (2010). Conceptualization, measurement, and analysis of negative affective risk factors. In A. Steptoe (Ed.), *Handbook of behavioral medicine research: Methods and applications* (pp. 155–168). New York: Springer.

Smith, T. W., & Cundiff, J. M. (2011). An interpersonal perspective on risk for coronary heart disease. In L. M. Horowitz & S. Strack (Eds.), *Hanbook of interpersonal psychology: Theory, research, assessment, and therapeutic interventions* (pp. 471–489). Hoeboken, NJ: Wiley.

Smith, T. W., Gallo, L. C., & Ruiz, J. M. (2003). Toward a social psychophysiology of cardiovascular reactivity: Interpersonal concepts and methods in the study of stress and cardiovascular disease. In J. Suls & K. Wallston (Eds.), *Social psychological foundations of health and illness* (pp. 335–366). Oxford, UK: Blackwell.

Smith, T. W., Glazer, K., Ruiz, J. M., & Gallo, L. C. (2004). Hostility, anger, aggressiveness, and coronary heart disease: An interpersonal perspective on personality, emotion, and health. *Journal of Personality, 72*, 1217–1270.

Smith, T. W., Traupman, E., Uchino, B. N., & Berg, C. A. (2010). Interpersonal circumplex descriptions of psychosocial risk factors for physical illness: Application to hostility, neuroticism, and marital adjustment. *Journal of Personality, 78*, 1011–1036.

Smith, T. W., Uchino, B. N., Berg, C. A., Florsheim, P., Pearce, G., Hawkins, M., et al. (2008). Self-reports and spouse ratings of negative affectivity, dominance and affiliation in coronary artery disease: Where should we look and who should we ask when studying personality and health? *Health Psychology, 27*, 676–684.

Smith, T. W., Uchino, B. N., Florsheim, P., Berg, C. A., Butner, J., Hawkins, M., et al. (2011). Affiliation and control during marital disagreement, history of divorce, and asymptomatic coronary artery calcification in older couples. *Psychosomatic Medicine, 73*, 350–357.

Suls, J., & Bunde, J. (2005). Anger, anxiety, and depression as risk factors for cardiovascular disease: The problems and implications of overlapping affective dispositions. *Psychological Bulletin, 131*, 260–300.

Trobst, K. K. (2000). An interpersonal conceptualization and quantification of social support transactions. *Personality and Social Psychology Bulletin, 26*, 971–986.

Interpersonal Conflict

► Social Conflict

Interpersonal Relationships

Bert N. Uchino[1], Kimberly Bowen[1], McKenzie Carlisle[1], Maija Reblin[2] and Rebecca Campo[3]
[1]Department of Psychology and Health Psychology Program, University of Utah, Salt Lake City, UT, USA
[2]College of Nursing, University of Utah, Salt Lake City, UT, USA
[3]Huntsman Cancer Institute, University of Utah, Salt Lake City, UT, USA

Synonyms

Psychosocial factors and traumatic events; Relationship processes; Social processes

Definition

Interpersonal processes refer broadly to actual or perceived elements of the social world. These processes can be generally positive (e.g., support) or negative (e.g., conflict) and of more specific types such as emotional support or insensitivity to others. It is also used to refer to the broader social context (e.g., social networks) in which such processes are embedded.

Description

Introduction

Interpersonal processes such as social support and social negativity have long been suspected as contributors to physical health outcomes. However, most biomedical research aimed at understanding disease has focused on biological processes (e.g., physiology, pathogens). There is now strong evidence linking interpersonal processes to biological pathways. This provides a bridge that can connect these perspectives to gain a more integrative understanding of the complex, multiply determined nature of health.

Interpersonal perspectives on health have typically been examined at different levels of analysis. At a broad level, sociologists have examined social networks and how they might influence health-relevant processes. For instance, these studies often examine the number or amount of contact with family or friends and/or the interconnections among them. More recently, sophisticated social network analyses have examined how proximal linkages among social ties might influence health-relevant outcomes such as obesity perhaps via unhealthy social norms. Distinctions are also often made in this literature between different relationship types (e.g., strong, weak ties). One important distinction is the extent to which a social tie is voluntary or obligatory. Voluntary ties, such as a recreational buddy, should be better for health because a person can choose to engage in more rewarding social experiences or sever such ties if they become nonrewarding. On the other hand, obligatory ties, such as relatives, are hard to sever even during difficult circumstances (e.g., familial conflict).

Psychological approaches often focus on the more specific interpersonal functions served by social network members, especially social support. Social support can be differentiated in terms of whether it is perceived or received. Perceived support refers to the belief that support would be available if needed, whereas received support refers to support that is obtained. These dimensions of support are often not highly correlated as individuals may perceive support to be available

but not actually seek it for various reasons (e.g., concerns about how others might react to such needs). As will be covered below, the distinction between perceived and received support is an important one because they often have different links to health outcomes. Perceived and received social support are further differentiated by more specific components including emotional (i.e., esteem enhancing), instrumental/informational (i.e., direct material or informational aid), and belonging (i.e., availability of others to do things with) support. Work in this area has also drawn the distinction between receiving support and being a support provider as the two are often correlated but may also have different links to health. However, the most common distinction in health-related research is related to perceived and received support.

More recently, researchers have also been focusing attention on the negative aspects of interpersonal processes. Although relationships may be sources of joy and support, there is also a "darker side" to interpersonal relationships that includes conflict, hindrance, and undermining. Social negativity is not simply the opposite of support, and research suggests that these are separable dimensions and hence are worthy of study in their own right or in combination. More generally, an examination of social negativity is important because there are theoretical reasons to believe that it may have stronger links to health outcomes compared to positive aspects of relationships. For instance, research on the negativity bias suggests that negative information or experiences have stronger links to psychological and physiological outcomes and hence may be more consequential for health. Similar to the support literature, researchers are also beginning to define more specific components of social negativity. Brooks and Dunkel-Schetter (2011) have recently argued that the main components of social negativity can be defined at the behavioral level and include conflict, insensitivity, and interference.

What is the Evidence Linking Interpersonal Processes to Health?

In a recent meta-analysis of 148 studies, Holt-Lunstad, Smith, and Layton (2010) found strong evidence linking both social networks and perceived social support to lower mortality rates. Overall, there was a 50% reduction in mortality that was attributable to relationships which remained even when considering variables such as age, gender, initial health status, and type of disease. These links were strongest for composite measures of social networks that typically indexed a variety of social ties (e.g., marriage, social activities). However, perceptions of support were also strong predictors of lower mortality rates. These results are consistent with other reviews in which relationships predict lower mortality from more specific causes such as cardiovascular disease and cancer. The one measure in the Holt-Lunstad et al. review that was not significantly related to mortality was received support. These data parallel studies indicating that received support also has inconsistent links with mental health outcomes.

In contrast to receiving support, being a support provider may be associated with beneficial health outcomes. Stephanie Brown and her colleagues have shown that providing support to family and friends is associated with decreased mortality even after considering how much support is received. This literature is sometimes seen as at odds with the caregiver literature which also involves being a support provider but has been consistently linked to negative health outcomes. However, there are important differences between these social contexts as caregiving involves considerable more interpersonal stress (e.g., anxiety over the slow loss of a family member) compared to being an everyday support provider in which there may also be more choice in the matter. Future research will be needed to disentangle the critical interpersonal elements that contribute to when being a support provider confers health costs or benefits.

Finally, a small but growing epidemiological literature also suggests that social negativity is related to higher rates of morbidity and mortality. For instance, a prospective study by De Vogli, Chandola, and Marmot (2007) of over 9,000 individuals found that social negativity predicted greater incident cardiovascular disease. Consistent with a negativity bias, these researchers

found that these effects were stronger than observed for social support. These data highlight the importance of not just considering positive interpersonal processes (i.e., support) but also negative aspects that appear to be powerful contributors to health risks.

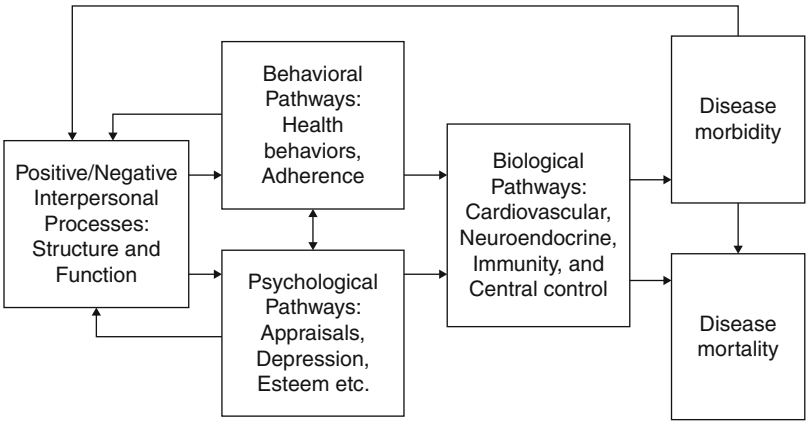
What Are the Pathways Responsible for Such Links?

As shown in Fig. 1, there are several plausible pathways linking positive and negative interpersonal processes to health. One possibility is that interpersonal processes influence health behaviors (for better or worse) which in turn influence health. For instance, social control theorists argue that being socially integrated can influence health in direct and indirect ways. Direct social control refers to active attempts by social network members to influence health (e.g., a parent setting a healthy family diet). Indirect social control refers to the increased motivation to engage in self-care because of meaningful relationships (e.g., wanting to be healthy to see a child grow up). Of course, social networks can also negatively influence health behaviors via unhealthy norms and social influence (e.g., substance abuse, obesity). There is consistent evidence that health behaviors can explain part of the link between interpersonal processes such as social support and health. However, many studies also find that statistically controlling for health behaviors such as exercise and smoking does not eliminate the link between relationships and health. These findings highlight the need to consider additional theoretical pathways.

Contemporary models of relationships and health have postulated psychological processes such as stress, depression, and positive emotional states as important additional pathways (see Fig. 1). For instance, the stress-buffering hypothesis predicts that support may prevent or reduce stress appraisals with corresponding influences on emotion-linked physiological responses. Associations between social support and health that do not involve stress processes have been postulated to be mediated by positive emotions, predictability, and a sense of self-worth. Although there is strong evidence linking interpersonal processes to these psychological processes, currently there is little evidence that these pathways directly explain links between social support and health. Future research will be needed to elucidate the issues that may be responsible for the current state of the literature (e.g., weaker tests of mediation, also see below).

One issue that is important in examining psychological pathways is the need to consider an expanded set of such mechanisms made salient by recent theoretical analyses. As noted earlier, the meta-analysis by Holt-Lunstad, Smith, and Layton (2010) did not find evidence that received support was related to mortality rates. Recent research is elaborating on the key contextual processes that determine whether receiving support has beneficial or harmful influences. For instance, receiving unsolicited support may increase distress because it conveys the message that the recipient is incapable of managing difficulties, thereby threatening a person's sense of esteem or independence. However, receiving support that is viewed as responsive to the recipient is linked to better well-being. This research has not been applied to the health domain but suggests a more specific set of psychological mediators to model in future work.

Finally, it is clear that interpersonal processes have effects on health-relevant physiological pathways. The three most studied biological pathways include those related to cardiovascular, neuroendocrine, and immune function. There is relatively strong evidence linking supportive relationships to lower cardiovascular reactivity during stress, as well as lower ambulatory blood pressure (ABP) during daily life. Likewise, negative relationship processes such as interpersonal conflict can exacerbate cardiovascular reactions and heighten ABP. The literature linking social relationships to immune processes is also strong. Social support has been linked to better immune function as indexed by natural killer cell activity and responses to vaccines. More recently, social support has been related to lower immune-mediated inflammation as indexed by IL-6, although less consistent findings are apparent as a function of other cytokines. Social negativity has also been linked to poorer immune system



Interpersonal Relationships, Fig. 1 Broad model highlighting salient pathways linking interpersonal processes to health

function in laboratory settings, and more recently to greater inflammation.

Neuroendocrine processes are particularly important to model because they may mediate both cardiovascular and immune responses. For instance, immune cells have receptors for many basic neuroendocrine hormones including catecholamines and cortisol. There is evidence linking social support to lower cortisol levels, although this tends to be an area in need of more research. However, researchers interested in social negativity have found consistent links between threats to the social self (e.g., evaluation) and increases in cortisol which might be health relevant.

One promising recent line of research links interpersonal processes to neural activity as indexed by imaging techniques. These studies are important because they are beginning to highlight the brain mechanisms that might coordinate the complex peripheral physiological pathways reviewed above. For instance, Naomi Eisenberger and colleagues have shown that social exclusion or "social pain" activates similar neural pathways involved in physical pain (i.e., dorsal anterior cingulate cortex). Consistent with the ability of supportive ties to buffer physiological reactivity, preliminary work also links positive or supportive interactions to lower activity in brain areas linked to autonomic and neuroendocrine changes (e.g., anterior cingulate cortex).

As shown in Fig. 1, it is also important to consider more complex pathways (e.g., reciprocal links). For instance, disease morbidity is associated with interpersonal processes and highlights the fact that health problems occur within a dyadic (e.g., marital) or broader family context and hence can influence interpersonal functioning in positive or negative ways. The diagnosis of a chronic condition may foster an appreciation for relationships (e.g., an emphasis on what is important in life). In contrast, some chronic conditions can place considerable stress on the family and decrease effective social support as might be the case for a person caring for a family member with Alzheimer's disease. These reciprocal pathways will need increased attention as researchers seek to design optimal interventions that capture the complexity of these interpersonal processes.

Intervention and Policy Implications

As researchers learn more about the links between interpersonal processes and health, it becomes appealing to use this information to inform relevant interventions in clinical populations. There are different types of interventions being implemented, many of which include elements of education and understanding, such as couple-oriented or larger support groups. A recent meta-analysis by Martire, Schulz, Helgeson, Small, and Saghafi (2010) found that couple-oriented interventions (e.g., disease education, role of relationships in illness management) were associated with lower depression and pain, along with better relationship functioning. Support groups also appear to foster psychological adjustment in many cases and can serve

I

unique functions (e.g., personal insight into the disease context). Such support groups are also particularly useful if there are gaps in the support needs of patients or if family members are overburdened. In addition to support groups, some broad interventions such as cognitive behavioral therapy focus on teaching general psychosocial skills and capitalizing on support within existing networks. These interventions appear to be effective in augmenting coping skills, as well as lowering disease-related distress and improving health outcomes.

Of course, the practicality and cost-effectiveness of an intervention are also important to consider. Recent research is examining this issue by focusing on telephone and internet-based support interventions. Preliminary studies have documented some positive influences on mental health outcomes and suggest the potential usefulness of alternative support interventions that may be especially important for individuals with practical (e.g., transportation), physical (e.g., disability), or social (e.g., anxiety) barriers. Future work will be needed to fully evaluate their potential benefits across a wider range of health outcomes.

It is also clear that most interventions focus on individuals who are most at risk or who already have medical problems. An alternative way of thinking about interpersonal interventions is as a form of primary prevention that focuses on healthy individuals. Given that many chronic diseases have a long-term etiology and develop over decades (e.g., coronary artery disease), primary prevention efforts for interpersonal processes may be particularly important to consider. For instance, Repetti, Taylor, and Seeman (2002) have argued that early familial interventions are an important starting point and may pay large dividends in the long term. A focus on primary prevention also raises the interesting possibility that social skills interventions aimed at improving relationship functioning may be useful if applied early on with children and adolescents to place them on healthier trajectories.

Finally, research on interpersonal processes and health may carry important policy implications (Umberson & Montez, 2010). Many of society's most vexing problems have clear interpersonal components (e.g., aggression, abuse, conflict). Given the research evidence to date, public policies can be aimed at increasing funding for understanding the more specific nature of such links or at fostering better interpersonal functioning. For instance, in order to encourage stronger marriages, the Deficit Reduction Act of 2005 devoted significant funds to the Healthy Marriage Initiative (HMI). The HMI focuses on research and demonstration projects regarding relationship education and skill building (e.g., listening, problem-solving). Recent evaluations of these programs have yielded promising effects on relationship quality and communication patterns (Hawkins, Blanchard, Baldwin, & Fawcett, 2008). More recently, the importance of the social determinants of health was acknowledged explicitly in Healthy People 2020, a nationwide health promotion plan. Such policy promises to shine a stronger spotlight on health-relevant interpersonal factors that may elevate funding priorities, public perception, and dialogue on future policy implementation.

Conclusions

Interpersonal processes are clearly related to significant health outcomes. Research is now being evaluated to test more specific models and pathways such as social control, health behaviors, and psychological and biological pathways. Contemporary research is also positioned to better capture the complex nature of interpersonal processes by increasing its focus on dyads, families, and larger social networks. The explicit goal of Healthy People 2020 to understand and harness the social determinants of health confers greater prominence and resources to this area but also signifies higher expectations. Interdisciplinary work that focuses on integrative multilevel analyses using diverse approaches (e.g., laboratory, daily experience, interventions) is likely to facilitate progress toward the long-standing goal of translating basic research into effective interventions and the integration of interpersonal processes into any and all discussions of public health.

Cross-References

- ▶ Family, Relationships
- ▶ Social Conflict
- ▶ Social Relationships
- ▶ Social Stress
- ▶ Social Support

References and Readings

Berkman, L. F., Glass, T., Brissette, I., & Seeman, T. E. (2000). From social integration to health: Durkheim in the new millennium. *Social Science and Medicine, 51*, 843–857.

Brooks, K., & Dunkel-Schetter, C. (2011). Social negativity and health: Conceptual and measurement issues. *Social and Personality Psychology Compass, 5*(11), 904–918.

Brown, S. L., Nesse, R. M., Vinokur, A. D., & Smith, D. M. (2003). Providing social support may be more beneficial than receiving it: Results from a prospective study of mortality. *Psychological Science, 14*, 320–327.

Cohen, S. (2004). Social relationships and health. *American Psychologist, 59*, 676–684.

De Vogli, R., Chandola, T., & Marmot, M. G. (2007). Negative aspects of close relationships and heart disease. *Archives of Internal Medicine, 167*, 1951–1957.

Dickerson, S. S. (2008). Emotional and physiological responses to social-evaluative threat. *Social and Personality Psychology Compass, 2*, 1362–1368.

Eisenberger, N. I. (2010). The neural basis of social pain: Findings and implications. In G. MacDonald & L. A. Jensen-Campbell (Eds.), *Social pain: Neuropsychological and health implications of loss and exclusion* (pp. 53–78). Washington, DC: American Psychological Association.

Hawkins, A. J., Blanchard, V. L., Baldwin, S. A., & Fawcett, E. B. (2008). Does marriage and relationship education work? A meta-analytic study. *Journal of Consulting and Clinical Psychology, 76*, 723–734.

Holt-Lunstad, J., Smith, T. B., & Layton, B. (2010). Social relationships and mortality: A meta-analysis. *PLoS Medicine, 7*, 1–20.

Martire, L. M., Schulz, R., Helgeson, V. S., Small, B. J., & Saghafi, E. M. (2010). Review and meta-analysis of couple-oriented interventions for chronic illness. *Annals of Behavioral Medicine, 40*, 325–342.

Repetti, R. L., Taylor, S. E., & Seeman, T. E. (2002). Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin, 128*, 330–366.

Smith, K. P., & Christakis, N. A. (2008). Social networks and health. *Annual Review of Sociology, 34*, 405–429.

Thoits, P. (2011). Mechanisms linking social ties and support to physical and mental health. *Journal of Health and Social Behavior, 52*(2), 145–161.

Uchino, B. N. (2006). Social support and health: A review of physiological processes potentially underlying links to disease outcomes. *Journal of Behavioral Medicine, 29*, 377–387.

Uchino, B. N. (2009). Understanding the links between social support and physical health: A lifespan perspective with emphasis on the separability of perceived and received support. *Perspectives in Psychological Science, 4*, 236–255.

Umberson, D., & Montez, J. K. (2010). Social relationships and health: A flashpoint for health policy. *Journal of Health and Social Behavior, 51*, S54–S66.

Wills, T. A., & Shinar, O. (2000). Measuring perceived and received social support. In S. Cohen, L. Gordon, & B. Gottlieb (Eds.), *Social support measurement and intervention: A guide for health and social scientists* (pp. 86–135). New York: Oxford University Press.

Interpersonal Stress or Conflict

- ▶ Social Stress

Intervention Theories

Nelli Hankonen
Department of Lifestyle and Participation, National Institute for Health and Welfare
University of Helsinki, Helsinki, Finland

Synonyms

Theories of behavior change

Definition

Health-promoting interventions aim to reduce health-risk behaviors and/or to promote behaviors and support environments conducive to health. In this pursuit, use of theory will serve several important purposes in various phases of interventions: In the beginning of intervention planning, theories can be used to *describe the target group* as well as *contribute to understanding of the health-promoting behaviors and environmental conditions*. Theories may also

inform the design of the intervention by helping to *identify possible determinants of both risk and healthful behaviors and environments* and *to select methods to promote change (intervention techniques/behavior change techniques)*. Finally, theories are useful in *evaluating the intervention (e.g., choice of measures)* and *retrieving evidence from interventions for the refinement of existing theories*. (Bartholomew & Mullen, 2011; Bartholomew, Parcel, Kok, Gottlieb, & Fernández, 2011; Michie & Prestwich, 2010; Nutbeam & Harris, 2004). Theories used to these ends may be called intervention theories. More than one theory may be used in an intervention.

Description

Planning of complex interventions should be based on a sound theoretical understanding of how the change is expected to take place (Campbell et al., 2000; Craig et al., 2008). It has been shown that interventions that have a theoretical basis are more successful than those that have not (Peters, Kok, Ten Dam, Buijs, & Paulussen, 2009; Webb, Joseph, Yardley, & Michie, 2010). Theories may focus on determinants that predict or explain outcomes or means of engendering change in the determinants. The first mentioned characterize the problem and are called *conceptual theories*, as they suggest what to change. The latter are *action theories*, as they suggest how the change is achievable.

Given the plethora of theories with potential use in health-promotion interventions, only few can be mentioned below for an overview of theories. A recent systematic review across scientific disciplines (Hobbs et al., 2011) identified several theories – altogether close to 100 – that could be used in interventions to change behavior. Intervention theories can be classified by the ecological level they target.

Frequently used theories are those that focus on the *individual* level, focusing mainly on direct changes in *behaviors* of a person. A well-known example in this category is the theory of planned behavior (Ajzen, 1991) that identifies subjective norms, attitudes toward behavior, and perceived behavioral control as predictors of intention. The latter in turn is considered as the main factor influencing behavior. Behavior change is suggested to occur when salient behavioral, normative, and/or control beliefs are successfully changed. Reflecting growing consensus of central determinants of behavior change, the integrated social cognition model (Fishbein et al., 2001) incorporates eight variables as key determinants of behavior: environment, skills, and intention, and (as predictors of intention), identity, self-efficacy, emotional reaction, perceived advantages and disadvantages of the target behavior, and social influences. Communication-persuasion matrix (McGuire, 2001) outlines seven phases ("RASMICE") of constructing a persuasive health campaign: reviewing the realities, axiological analysis, surveying the sociocultural situation, mapping the mental matrix, teasing out the target themes, constructing the communication, and evaluating the effectiveness. The model includes 12 output persuasion steps that describe the process of being persuaded, e.g., receiver's attention and comprehension, acquiring skills, changes in attitudes, memory storage, and behavior. The matrix includes five input communication factors: message source, message content and style, communication channel, receiver (audience), and destination. Each step requires certain choices related to the communication variables.

Health behaviors are not affected by individual psychological factors only, but also by social and structural factors of environments people live in. Accordingly, some theories focus on changing *environments* for better health. Theories of social networks and social support use concepts of the *interpersonal* level, while there are others that address the *organizational* level. Schein's theory of organizational culture (2004) identifies leaders as key players in changing organizations. According to Schein, organization's design and structure; stories, legends, and myths, as well as formal statements of the organizational philosophy and values are only secondary sources of bringing about change. Schein proposes that these secondary mechanisms are effective only if they are in concordance with primary mechanisms – leaders' behaviors. Organizational

change is influenced by these primary mechanisms, such as what leaders pay attention to, measure and control; how leaders react to critical incidents and organizational crises; what criteria they set for allocating scarce resources and recruiting and promoting personnel; and whether they teach, coach, and model the target behavior.

A renowned example of *community* level theories is the diffusion of innovation theory (Rogers, 2002). It outlines four elements in the diffusion of new ideas in communities: innovation, communication channels, time, and the social system. Innovations are more likely to be successfully adopted when they are, e.g., compatible with existing socioeconomic and cultural values, perceived easy to understand and use, and have observable results in the community.

Theories on the *societal or policy* level may also be used as intervention theories. Milio (1987) has proposed that the development of healthy public policy is not simply the production of policy statements but, instead, is a dynamic process, continuously passing through stages of initiation, adoption, implementation, evaluation, and reformulation. Four major players (public policy makers, interested parties, the public, and the mass media) are important in the policy development. Longest (2010) suggests that the health policy making process is complex, interactive, and cyclical, and the process entails three intertwined phases: policy formulation (agenda setting and development of legislation), policy implementation (rulemaking and operation), and policy modification. Health policies are made within the contest of the political marketplace where demanders of policies and suppliers of policy interact. Preferences and feedback of various interest groups, political circumstances, and formal legislation processes influence the process. Finally, some theories, such as theories related to systems, power, and empowerment can be conceptualized as *multilevel theories* as they are applicable at more than one environmental level.

Frameworks exist to help in selecting appropriate theories for an intervention for a given behavior in a specific population and translating an abstract theory into a practical intervention program. A popular one is intervention mapping (Bartholomew et al., 2011) that outlines six steps during which a theory- and evidence-based program and its implementation and evaluation are systematically planned. The intervention mapping steps include (1) needs assessment, (2) preparing matrices of change objectives, (3) selecting theory-based intervention methods and practical applications, (4) producing the intervention program components and materials, (5) planning program adoption and implementation, and (6) planning for evaluation.

Cross-References

- ▶ Adherence
- ▶ Attitudes
- ▶ Attribution Theory
- ▶ Behavior
- ▶ Behavior Change
- ▶ Behavior Change Techniques
- ▶ Cancer Prevention
- ▶ Cancer Treatment and Management
- ▶ Clinical Trial
- ▶ Community Coalitions
- ▶ Community-Based Health Programs
- ▶ Diabetes Education
- ▶ Dissemination
- ▶ Eating Behavior
- ▶ Effectiveness
- ▶ Efficacy
- ▶ Empowerment
- ▶ Evidence-Based Behavioral Medicine (EBBM)
- ▶ Group Therapy/Intervention
- ▶ Health
- ▶ Health Behaviors
- ▶ Health Behavior Change
- ▶ Health Beliefs/Health Belief Model
- ▶ Healthy Eating
- ▶ Health Education
- ▶ Health Psychology
- ▶ Internet-Based Interventions
- ▶ Lifestyle Changes
- ▶ Multilevel Intervention
- ▶ Obesity: Prevention and Treatment
- ▶ Physical Activity Interventions
- ▶ Population Health

- ▶ Preventive Care
- ▶ Psychological Factors
- ▶ Public Health
- ▶ Risky Behavior
- ▶ Screening
- ▶ Sedentary Behaviors
- ▶ Self-efficacy
- ▶ Self-regulation Model
- ▶ Smoking Behavior
- ▶ Smoking Cessation
- ▶ Smoking Prevention Policies and Programs
- ▶ Social Factors
- ▶ Social Marketing
- ▶ Social Support
- ▶ Sociocultural
- ▶ Tailored Communications
- ▶ Theory
- ▶ Tobacco Cessation
- ▶ Transtheoretical Model of Behavior Change

References and Readings

Ajzen, I. (1991). The theory of planned behavior. *Organizational Behavior and Human Decision Processes, 50*, 179–211.

Bartholomew, L. K., & Mullen, P. D. (2011). Five roles for using theory and evidence in the design and testing of behavior change interventions. *Journal of Public Health Dentistry, 71*, S20–S33.

Bartholomew, L. K., Parcel, G. S., Kok, G., Gottlieb, N. H., & Fernández, M. E. (2011). *Planning health promotion programs. An intervention mapping approach* (3rd ed.). San Francisco: Jossey-Bass.

Campbell, M., Fitzpatrick, R., Haines, A., Kinmonth, A. L., Sandercock, P., Spiegelhalter, D., et al. (2000). Framework for design and evaluation of complex interventions to improve health. *BMJ, 321*(7262), 694–696.

Craig, P., Dieppe, P., Macintyre, S., Michie, S., Nazareth, I., & Petticrew, M. (2008). Developing and evaluating complex interventions: the new Medical Research Council guidance. *BMJ, 337*, a1655. doi:10.1136/bmj.a1655.

Fishbein, M., Triandis, H. C., Kanfer, F. H., Becker, M. H., Middlestadt, S. E., & Eichler, A. (2001). Factors influencing behavior and behavior change. In A. Baum, T. A. Revenson, & J. E. Singer (Eds.), *Handbook of health psychology*. Mahwah, NJ: Lawrence Erlbaum Associates.

Hobbs, L., Michie, S., Campbell, R., & Hildon, Z. (2011). Behaviour change theories across psychology, sociology, anthropology and economics: A systematic review. *Psychology and Health, 26*(Suppl. 2), 31.

Longest, B. B. (2010). *Healthy policymaking in the United States* (5th ed.). Chicago: Health Administration Press.

McGuire, W. J. (2001). Input and output variables currently promising for constructing persuasive communications. In R. E. Rice & C. K. Atkin (Eds.), *Public communication campaigns* (3rd ed., pp. 22–48). Thousand Oaks, CA: Sage.

Michie, S., & Prestwich, A. (2010). Are interventions theory-based? Development of a theory coding scheme. *Health Psychology, 29*(1), 1–8.

Milio, N. (1987). Making healthy public policy: developing the science by learning the art: An ecological framework for policy studies. *Health Promotion, 2*(3), 263–274.

Nutbeam, D., & Harris, E. (2004). *Theory in a nutshell. A practical guide to health promotion theories* (2nd ed.). Australia: McGraw-Hill.

Peters, L., Kok, G., Ten Dam, G., Buijs, G., & Paulussen, T. (2009). Effective elements of school health promotion across behavioral domains: A systematic review of reviews. *BMC Public Health, 9*(1), 182.

Rogers, R. W. (2002). Diffusion of preventive interventions. *Addictive Behaviors, 27*, 989–993.

Schein, E. H. (2004). *Organizational culture and leadership* (3rd ed.). San Francisco: Jossey-Bass.

Webb, L. T., Joseph, J., Yardley, L., & Michie, S. (2010). Using the internet to promote health behavior change: A systematic review and meta-analysis of the impact of theoretical basis, use of behavior change techniques, and mode of delivery on efficacy. *Journal of Medical Internet Research, 12*(1), e4.

Interventions Therapy

- ▶ Therapy, Family and Marital

Interview

Nikola Stenzel[1] and Stefan Krumm[2]
[1]Department of Clinical Psychology and Psychotherapy, Philipps University of Marburg, Marburg, Germany
[2]University of Muenster, Muenster, Germany

Synonyms

Diagnostic interview

Definition

Interviews are among the most valuable and most frequently used tools in clinical practice. They can be defined as dyadic face-to-face interactions between two people (interviewer and interviewee) that are conducted for specific purposes. Clinical interviews cover a wide range of purposes, for example, rapport building, problem exploration, or establishing a psychiatric diagnosis according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV).

Typically, the interaction is asymmetric in the sense that the interviewer poses questions and the interviewee provides answers. Interview questions can be categorized according to their function and/or their form. Interview questions may have the following functions: introductory questions, gathering information, rapport building, transition from one topic of the interview to another, and reassuring that information was understood correctly. Depending on the specific purpose within an interview, the questions may have the following forms: open or closed, direct or indirect, suggestive. Closed interview questions can be answered with a single word or short phrase, whereas open interview questions allow unrestricted answers. Direct questions directly address the topic at hand; indirect questions address broader or related topics to gather information about the topic at hand. Suggestive interview questions imply a certain answer and, thus, should be avoided in an unbiased assessment. The appropriateness of different forms of interview questions depends on the interview phase and information to be assessed.

Interviews may be conducted in a more or less structured way. Structured interviews include a predefined interview schedule. Such a schedule specifies crucial elements of the interview, such as the introduction and the subtopics to be addressed. Furthermore, it defines the interview questions, provides details on sequence of questions, etc. The more elements are specified in advance, the more structured the interview is. The degree of structure should be chosen depending on the interview purpose (e.g., exploration, decision making).

Structured interviews show several advantages, in particular for diagnostic decision making according to DSM-IV. Several established interview schedules are available. For example, the Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1996) is an established interview system that is frequently used by clinicians. Another frequently used highly structured interview is the Diagnostic Interview Schedule for DSM-IV (DIS-IV; Robins et al., 2000).

Interviews provide multiple advantages in comparison to other assessment tools such as tests and self-reports. First, interviews can be conducted in an interactive way (e.g., the interviewer can rephrase questions if necessary). Second, interviews are often considered the more natural form of an assessment (as compared to test with multiple choice answers). Third, expert judgment can be made throughout the interview (e.g., based on behavior observations), which may provide further information in addition to the answers given by the interviewee. However, interviews also show disadvantages compared to tests and self-reports with regard to psychometric properties and costs of administration time (as interviews are mostly conducted in dyadic face-to-face situations). Hence, the applicability of interviews needs to be considered on a case-by-case basis.

Cross-References

- ▶ Assessment
- ▶ Diagnostic Interview Schedule
- ▶ Psychiatric Diagnosis
- ▶ Structured Clinical Interview for DSM-IV (SCID)

References and Readings

Barbour, K. A., & Davison, G. C. (2004). Clinical interviewing. In S. N. Haynes & E. M. Heiby (Eds.), *Comprehensive handbook of psychological assessment* (Behavioral Assessment, Vol. 3, pp. 181–193). Hoboken, NJ: Wiley.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1996). *Structured clinical interview for DSM-IV axis I disorders, clinician version (SCID-CV)*. Washington, DC: American Psychiatric Press.
Morrison, J. (2008). *The first interview: A guide for clinicians*. New York: Guilford Press.
Robins, L. N., Cottler, L. B., Buchholz, K. K., Compton, W. M., North, C. S., & Rourke, K. M. (2000). *Diagnostic interview schedule for the DSM-IV (DIS-IV)*. St Louis, MO: Washington University School of Medicine.

Intima-Media Thickness (IMT)

Jonathan Newman
Columbia University, New York, NY, USA

Definition

Intima-media thickness (IMT) is defined by the measurement of a component of the walls of muscular arteries (intima) by specific modalities of ultrasound, at defined locations in the human body. Intima-media thickness of the carotid artery has been the location most consistently associated with risk of atherosclerotic risk. Because ultrasounds of the carotid artery can be performed safely, at low risk (no ionizing radiation) and with a relatively high degree of reproducibility, IMT has been a common surrogate endpoint in epidemiologic studies and in clinic trials.

Carotid IMT (CIMT) is correlated with other vascular risk factors and is considered an "intermediate phenotype" of early atherosclerosis that independently predicts vascular events. In general, greater CIMT values are associated with greater cardiovascular risk. However, an absolute definition of abnormal CIMT has been problematic due to the effects of age and hypertension on CIMT, independent of their atherosclerotic risk. Despite evidence suggesting intrareader differences in CIMT measurement are quite small (0.04 mm), the clinical utility of serial CIMT measurement (e.g., to assess response to therapy) has not been demonstrated, and the routine use of CIMT has been hindered by the lack of standard assessment techniques in major epidemiologic studies. As a result, there is neither universal consensus on what constitutes an abnormal test result nor guidelines on how CIMT informs risk stratification of individual cardiovascular risk.

References and Readings

Bots, M. L., Baldassarre, D., Simon, A., de Groot, E., O'Leary, D. H., Riley, W., et al. (2007). Carotid intima-media thickness and coronary atherosclerosis: Weak or strong relations? *European Heart Journal, 28*(4), 398–406.
Chambless, L. E., Heiss, G., Folsom, A. R., Rosamond, W., Szklo, M., Sharrett, A. R., et al. (1997). Association of coronary heart disease incidence with carotid arterial wall thickness and major risk factors: The Atherosclerosis Risk in Communities (ARIC) study, 1987–1993. *American Journal of Epidemiology, 146*, 483–494.
Mark Helfand, D. I., Buckley, M. F., Rongwei, Fu, Rogers, K., Fleming, C., & Humphrey, L. L. (2009). Emerging risk factors for coronary heart disease: A summary of systematic reviews conducted for the U.S. Preventive Services Task Force. *Annals of Internal Medicine, 151*, 496–507.
Redberg, R. F., Vogel, R. A., Criqui, M. H., Herrington, D. M., Lima, J. A. C., & Roman, M. J. (2003). Task force #3-what is the spectrum of current and emerging techniques for the noninvasive measurement of atherosclerosis? *Journal of the American College of Cardiology, 41*, 1886–1898.

Intimate Partner Violence

▶ Family Violence

Intoxication

▶ Binge Drinking

Intracytoplasmic Sperm Injection

▶ In Vitro Fertilization, Assisted Reproductive Technology

Intrauterine Insemination

▶ Infertility and Assisted Reproduction: Psychosocial Aspects

Intrinsic Religiousness (Religiosity)

Kevin S. Masters
Department of Psychology, University of Colorado, Denver, CO, USA

Definition

Intrinsic religiousness (initially and still sometimes referred to as intrinsic religiosity) is characterized as religion that is an end in itself, a master motive. Thus, individuals described by intrinsic religiousness view their religion as the framework for their lives, and they try to consistently live the religion they believe. A prototypic intrinsic religiousness test item is "My whole approach to life is based upon my religion."

Description

Intrinsic religiousness was first described by Gordon Allport and colleagues in the 1960s (see Allport & Ross, 1967) when investigating the possible reasons for discrepant findings in the area of religiousness and prejudice. At that time, some studies demonstrated that religiousness was positively associated with prejudice whereas other studies found the opposite. Allport hypothesized that one's religious orientation, or sentiment, may provide guidance in sorting out these findings. The construct of religious orientation was later clarified by Gorsuch (1994) to be a motivational variable.

Intrinsic religiousness has often been measured by the Religious Orientation Scale (Allport & Ross, 1967). More recently, Gorsuch and McPherson (1989) developed the I/E-Revised scale as a more psychometrically sound instrument. Internal consistency measures for intrinsic religiousness scales are excellent, typically in the mid.80s (Donahue, 1985). Scholars in the psychology of religion have debated the relative strengths and weaknesses of the religious orientation construct (e.g., Kirkpatrick & Hood, 1990; Masters, 1991), but it remains the most empirically investigated and heuristic construct in this area of work.

Investigators attempting to determine the relations between religion and health have also turned to religious orientation. Smith, McCullough, and Poll (2003), in a meta-analytic study, found that intrinsic religiousness was the only measure of "positive" religiousness to statistically differentiate itself from other measures of religious attitudes and beliefs in that studies with measures of intrinsic religiousness had stronger negative correlations with symptoms of depression than studies with measures of religious attitudes and beliefs. Similarly, Masters and Bergin (1992) provided a narrative review of the literature and found intrinsic religiousness to be related negatively with depression, anxiety, and obsessive-compulsive symptoms and positively with empathic concern, self-consciousness, internal awareness, flexible approach to life, tolerance, self-control, active coping, self-esteem, efficacy, and ego strength. McCullough and Willoughby (2009) suggest that intrinsic religiousness may be part of a personality core that forms the substrates for conscientiousness, agreeableness, and self-control. They also note that intrinsic religiousness may provide a basis for meaning in life. A recent investigation found intrinsic religiousness related to dampened laboratory-induced cardiovascular reactivity in older adults (Masters, Hill, Kircher, Lensegrav Benson, & Fallon, 2004) wherein the older intrinsically religious adults reacted to stress similarly to individuals 50 years younger and demonstrated significantly less reactivity than their extrinsically religious same-age counterparts. Masters and Knestel (2011) found that among a random sample of community-dwelling adults, those characterized by intrinsic religiousness were

less likely to be divorced, reported overall better health status, lower body mass index, less cigarette use, and fewer number of daily drinks of alcohol than did those characterized as extrinsically religious. There were no differences in the percentages of individuals who were classified as intrinsically religious based on religious denomination. Nevertheless, it is not entirely clear how intrinsic religiousness may interact with religious denomination, and some have suggested that the construct, as currently conceptualized and measured, is more congruent with Protestant notions of religiosity and perhaps most appropriately applied to this religious group (Cohen et al., 2005).

Cross-References

► Extrinsic Religiousness (Religiosity)

References and Readings

Allport, G. W., & Ross, J. M. (1967). Personal religious orientation and prejudice. *Journal of Personality and Social Psychology, 5*, 432–443.

Cohen, A. B., Pierce, J. D., Jr., Chambers, J., Meade, R., Gorvine, B. J., & Koenig, H. G. (2005). Intrinsic and extrinsic religiosity, belief in the afterlife, death anxiety, and life satisfaction in young Catholics and Protestants. *Journal of Research in Personality, 39*, 307–324.

Donahue, M. J. (1985). Intrinsic and extrinsic religiousness: Review and meta-analysis. *Journal of Personality and Social Psychology, 48*, 400–419.

Gorcush, R. L., & McPherson, S. E. (1989). Intrinsic/extrinsic measurement: I/E-revised and single-item scales. *Journal for the Scientific Study of Religion, 28*, 348–354.

Gorsuch, R. L. (1994). Toward motivational theories of intrinsic religious commitment. *Journal for the Scientific Study of Religion, 33*, 315–325.

Kirkpatrick, L. A., & Hood, J. (1990). Intrinsic-extrinsic religious orientation: The boon or bane of contemporary psychology of religion? *Journal for the Scientific Study of Religion, 29*, 442–462.

Masters, K. S. (1991). Of boons, banes, babies, and bath water: A reply to the Kirkpatrick and Hood discussion of intrinsic-extrinsic religious orientation. *Journal for the Scientific Study of Religion, 30*, 312–317.

Masters, K. S., & Bergin, A. E. (1992). Religious orientation and mental health. In J. F. Schumaker (Ed.), *Religion and mental health* (pp. 221–232). New York: Oxford University Press.

Masters, K. S., Hill, R. D., Kircher, J. C., Lensegrav-Benson, T. L., & Fallon, J. A. (2004). Religious orientation, aging, and blood pressure reactivity to interpersonal and cognitive stressors. *Annals of Behavioral Medicine, 28*, 171–178.

Masters, K. S., & Knestel, A. (2011). Religious orientation among a random sample of community dwelling adults: Relations with health status and health relevant behaviors. *The International Journal for the Psychology of Religion, 21*, 63–76.

McCullough, M. E., & Willoughby, B. L. B. (2009). Religion, self-regulation, and self-control: Associations, explanations, and implications. *Psychological Bulletin, 135*, 69–93.

Smith, T. B., McCullough, M. E., & Poll, J. (2003). Religiousness and depression: Evidence for a main effect and the moderating influence of stressful life events. *Psychological Bulletin, 129*, 614–636.

Intrusive Thoughts

► Perseverative Cognition
► Worry

Intrusive Thoughts, Intrusiveness

Bart Verkuil and J. F. Brosschot
Clinical, Health and Neuro Psychology, Leiden University, Leiden, Netherlands

Definition

An intrusive thought has been defined as “any distinct, identifiable cognitive event that is unwanted, unintended and recurrent. It interrupts the flow of thought, interferes in task performance, is associated with negative affect, and is difficult to control” (Clark, 2005, p. 4). Intrusive thoughts are particularly present in psychopathological states such as obsessive compulsive disorders (then often referred to as “obsessions”) and

post-traumatic stress disorder (thoughts or images related to the experienced trauma). Yet, they are also frequently observed in the nonclinical population, where 80–99% has reported experiencing unwanted intrusive thoughts once in a while (Clark & Purdon, 1995). They appear highly similar to worrisome thoughts, but it has been suggested that intrusive thoughts are shorter in duration and are appraised as more ego-dystonic than worries (Clark, 2005).

Intrusive thoughts can be considered a form of so-called perseverative cognition (see ▶ Perseverative Cognition), which is defined as "the repeated or chronic activation of the cognitive representation of one or more psychological stressors (threat)" (Brosschot, Gerin, & Thayer, 2006). Perseverative cognition is believed to be responsible for a large part of the health impact of psychological stressors, because it later prolongs the physiological responses to these stressors.

Cross-References

▶ Perseverative Cognition
▶ Rumination
▶ Worry

References and Readings

Brosschot, J. F., Gerin, W., & Thayer, J. F. (2006). The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *Journal of Psychosomatic Research, 60*, 113–124.

Clark, D. A. (2005). *Intrusive thoughts in clinical disorders: Theory, research, and treatment*. New York: The Guilford Press.

Clark, D. A., & Purdon, C. L. (1995). The assessment of unwanted intrusive thoughts: A review and critique of the literature. *Behaviour Research and Therapy, 33*, 967–976.

Invasive Cervical Cancer

▶ Cancer, Cervical

Inverse Relationship

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Negative relationship

Definition

An inverse relationship is one in which the value of one parameter tends to decrease as the value of the other parameter in the relationship increases. It is often described as a negative relationship.

Imagine the age of a car and its value. In general terms, as a car increases in age its value will decrease (the amount of miles driven per unit of time also factors in to this, but we will leave that aside here). This relationship is not perfect since, for two identical cars, the degree to and manner in which they are driven and maintained will impact the value, and the cars may differ considerably in value 10 years after their contemporaneous purchase. Nonetheless, the general association can be described as an inverse one.

In many cases, such a relationship can be captured meaningfully by a correlation coefficient. These coefficients range from −1 to +1 (typically written as just 1, without the + sign). A value of −1 represents a perfect inverse linear association: As one parameter increases, the other decreases in a perfect linear relationship. Values between −1 and zero represent less perfect associations.

In contrast, a positive relationship is one in which both parameters change in the same direction: as one increases so the other increases. Such a relationship would be described by a correlation coefficient between zero and 1, with 1 representing a perfect linear relationship. (The technique of correlation cannot meaningfully describe nonlinear patterns of association between two variables, such as curvilinear and exponential relationships.)

Involuntary Childlessness

▶ Infertility and Assisted Reproduction: Psychosocial Aspects

Involuntary Exposure to Tobacco Smoke

▶ Secondhand Smoke

Irritable Bowel Syndrome (IBS): Psychological Treatment

Tomohiko Muratsubaki and Shin Fukudo
Department of Behavioral Medicine, School of Medicine, Tohoku University Graduate, Aoba-ku, Sendai, Japan

Synonyms

Psychotherapy for IBS

Definition

Psychological treatments are non-pharmacological therapies that mainly modify higher neural function including emotion, cognition, motivation, learning, and memories for IBS patients.

Description

Irritable bowel syndrome (IBS) is a very common gastrointestinal disorder characterized by recurrent abdominal pain and altered bowel habits without major organic diseases as assessed by routine gastroenterological examination (Drossman, 2006). The pathogenesis of IBS is affected or exacerbated by combination of factors including psychosocial states, dysmotility of the gut, and visceral hypersensitivity. The concept of biopsychosocial model best fits to pathophysiology and treatment strategy for IBS. Psychosocial stress likely plays major role in the exacerbation of all of these factors. In fact, IBS patients have symptoms which are more exaggerated by psychosocial stress than healthy controls (Whitehead, Crowell, Robinson, Heller, & Schuster, 1992), show more response in colonic motility to psychosocial stress (Fukudo, Nomura, Muranaka, & Taguchi, 1993), and have the corticotrophin-releasing hormone as a major mediator of stress response (Fukudo, Nomura, & Hongo, 1998).

Brain-gut interaction plays in an important role in the pathogenesis of IBS. Brain-gut interactions mainly consist of reciprocal interactions between the central nervous system (CNS) and the enteric nervous system (ENS) as well as influence of endocrine system, immune system, and gut microbiota. Brain imaging studies have clarified the roles of the anterior cingulate cortex, amygdala, insula, and the brain stem in response to visceral stimulation (Fukudo et al., 2009). These structures produce both visceral pain and negative emotion that are typical symptoms of IBS patients. Psychological stress, anticipatory anxiety, and mental tension in daily life exert a negative influence on CNS, which influences ENS. Gastrointestinal dysmotility and perception lead to symptoms of IBS, i.e., abdominal pain, abdominal discomfort, diarrhea, or constipation. The gut signals which produce these symptoms also influence the CNS. From the psychosocial viewpoint, IBS has a negative impact on subjects' daily activity and quality of life (Drossman et al., 1993). IBS patients frequently show gastrointestinal dysmotility under stress (Fukudo et al., 1993) and have psychiatric comorbidities, especially depressive disorders, anxiety disorders, and somatoform disorders (Drossman, 2006). Note that the present stress alone does not exacerbate symptoms of IBS. Past traumatic life event such as sexual/physical abuse in childhood also exacerbate symptoms of IBS by low stress or environmental changes and can be a factor of intractable IBS. In addition, physician needs to

understand psychosocial risk factors including abuse, economical loss, and social withdrawal behind symptoms of IBS.

A wide range of psychotherapeutic interventions are used for the treatment of IBS, including cognitive behavioral therapy (CBT), hypnotherapy, and relaxation techniques. There are some critical reviews on the effect of the psychotherapy for IBS (Talley, Owen, Boyce, & Paterson, 1996). Therefore, it is still difficult to say that effective psychotherapy for IBS has already been established. However, the evidence level rises in the recent research. Although several pharmacological agents for IBS have been used, some patients do not respond to pharmacotherapy (Levy et al., 2006). Psychotherapy might be useful for these patients. A key issue in effective management of IBS is establishing a doctor-patient relationship. Good psychological and therapeutic effects for IBS patients are produced by clinical practices such as explanation, instruction, and administration accompanying well doctor-patient relationship. Conversely, poor psychological and therapeutic effects such as distrust and negative emotion are produced by clinical practices accompanying negative doctor-patient relationship. Factors that contribute to an effective doctor-patient relationship include empathy toward the patients, patient education, validation of the illness, reassurance, treatment negotiation, and establishment of reasonable limits in time and effort (Grover & Drossman, 2010). A good doctor-patient relationship is associated with a positive prognosis of IBS (Owens, Nelson, & Talley, 1995).

There is evidence from a systematic review and meta-analysis of randomized controlled trials on IBS that psychological treatment in general is effective for IBS (Zijdenbos, de Wit, van der Heijden, Rubin, & Quartero, 2009). The theoretical background of psychotherapies for IBS is as follows. First, IBS patients have deranged lifestyles characterized by more perceived stress, more irregular sleep, and more irregular meals (Shinozaki et al., 2008). Second, there is the close relationship between daily stress or hassles and IBS symptoms (Whitehead et al., 1992). Third, IBS patients have high levels of anxiety, depression, and somatization (Kanazawa et al., 2004). Fourth, intractable IBS patients have often experienced traumatic life events (Drossman et al., 1990). Fifth, the effects of health beliefs and learned behaviors may adversely affect outcome. Sixth, IBS patients are known to respond highly to placebo. Seventh, as described above, doctor-patient relationship plays a role in IBS treatment.

Psychological treatments including a delicate observation and stress management is needed for IBS patients. Treatments compose direct or indirect coping to stress and stress sensitivity. IBS patients often have symptoms characterized by abdominal pains in stressful situation when they cannot secure personal space or relieve symptoms by defecation. Because of these symptoms, IBS patients often avoid transportation, feel insecurity in going to the school or the company, and limit going out. For this situation, behavioral intervention is useful. Behavioral intervention is focused on modifying maladaptive behavior which links to exacerbation or maintenance of IBS symptoms and forming a habit of adaptive behavior. It is composed of setting clear goals on treatment, learning nutrition and lifestyle, brief cognitive restructuring, stimulus control, problem-solving method, and prevention of relapse. IBS patients often have illness behavior which has been formed in the early developmental history. Modification of cognitive error which links to this behavior is needed. The main purpose of CBT is to modify negative automatic thoughts and dysfunctional beliefs and to increase or decrease a particular behavior. Even if individual is in the same environment, how to feel the stress and how to interpret it are greatly variable. Actually, CBT was found to be more effective for IBS than an educational intervention (Drossman et al., 2003). Because IBS is exacerbated by stress, therapeutic modification of the stress sensitivity using stress coping or CBT is effective for IBS symptoms (Kennedy et al., 2005; Moss-Morris, McAlpine, Didsbury, & Spence, 2010).

Relaxation is also useful as direct coping to stress. This coping includes deep breathing, distraction, breaking, slow bathing, and taking holiday. Hypnotherapy is a representative strategy for refractory IBS (Whorwell, Prior, & Faragher, 1984). Autogenic training (AT) is auto-hypnosis

with the goal of being able to self-administer suggestions of relaxation. In brief, traditional AT consists of 6 standard exercises after the formula "I am at peace." The first exercise aims at muscular relaxation by repetition of a verbal formula, "my right arm is heavy" emphasizing heaviness. Subsequent passive concentration is focused on feeling warm, initiated by the instruction "my right arm is warm," followed by cardiac activity using the formula "my heartbeat is calm and regular." Then follows passive concentration on the respiratory mechanism with the formula "it breathes me," then on warmth around the abdominal region with "my solar plexus is warm," and finally on coolness in the cranial region with "my forehead is cool and clear" (Kanji & Ernst, 2000). AT is reported to be effective for intractable IBS patients (Shinozaki et al., 2010).

Cross-References

- ▶ Behavioral Therapy
- ▶ Cognitive Behavioral Therapy (CBT)
- ▶ Relaxation: Techniques/Therapy
- ▶ Stress Management

References and Readings

Drossman, D. A. (2006). The functional gastrointestinal disorders and the Rome III process. *Gastroenterology, 130*, 1377–1390.

Drossman, D. A., Leserman, J., Nachman, G., Li, Z., Gluck, H., Toomey, T. C., et al. (1990). Sexual and physical abuse in women with functional or organic gastrointestinal disorders. *Annals of Internal Medicine, 113*, 828–833.

Drossman, D. A., Li, Z., Andruzzi, E., Temple, R. D., Talley, N. J., Thompson, W. G., et al. (1993). U.S. householder survey of functional gastrointestinal disorders: Prevalence, sociodemography and health impact. *Digestive Diseases & Sciences, 38*, 1569–1580.

Drossman, D. A., Toner, B. B., Whitehead, W. E., Diamant, N. E., Dalton, C. B., Duncan, S., et al. (2003). Cognitive-behavioral therapy vs. education and desipramine vs. placebo for moderate to severe functional bowel disorders. *Gastroenterology, 125*, 19–31.

Fukudo, S., Kanazawa, M., Mizuno, T., Hamaguchi, T., Kano, M., Watanabe, S., et al. (2009). Impact of serotonin transporter gene polymorphism on brain activation by colorectal distention. *Neuroimage, 47*, 946–951.

Fukudo, S., Nomura, T., & Hongo, M. (1998). Impact of corticotropin-releasing hormone on gastrointestinal motility and adrenocorticotropic hormone in normal controls and patients with irritable bowel syndrome. *Gut, 42*, 845–849.

Fukudo, S., Nomura, T., Muranaka, M., & Taguchi, F. (1993). Brain-gut response to stress and cholinergic stimulation in irritable bowel syndrome. A preliminary study. *Journal of Clinical Gastroenterology, 17*, 133–141.

Grover, M., & Drossman, D. A. (2010). Functional abdominal pain. *Current Gastroenterology Reports, 12*, 391–398.

Kanazawa, M., Endo, Y., Whitehead, W. E., Kano, M., Hongo, M., & Fukudo, S. (2004). Patients and nonconsulters with irritable bowel syndrome reporting a parental history of bowel problems have more impaired psychological distress. *Digestive Diseases and Sciences, 49*, 1046–1053.

Kanji, N., & Ernst, E. (2000). Autogenic training for stress and anxiety: A systematic review. *Complementary Therapies in Medicine, 8*, 106–110.

Kennedy, T., Jones, R., Darnley, S., Seed, P., Wessely, S., & Chalder, T. (2005). Cognitive behaviour therapy in addition to antispasmodic treatment for irritable bowel syndrome in primary care: Randomised controlled trial. *British Medical Journal, 331*, 435.

Levy, R. L., Olden, K. W., Naliboff, B. D., Bradley, L. A., Francisconi, C., Drossman, D. A., et al. (2006). Psychosocial aspects of the functional gastrointestinal disorders. *Gastroenterology, 130*, 1447–1458.

Moss-Morris, R., McAlpine, L., Didsbury, L. P., & Spence, M. J. (2010). A randomized controlled trial of a cognitive behavioural therapy-based self-management intervention for irritable bowel syndrome in primary care. *Psychological Medicine, 40*, 85–94.

Owens, D. M., Nelson, D. K., & Talley, N. J. (1995). The irritable bowel syndrome: Long term prognosisand the physician-patient interaction. *Annals of Internal Medicine, 122*, 107–112.

Shinozaki, M., Fukudo, S., Hongo, M., Shimosegawa, T., Sasaki, D., Matsueda, K., et al. (2008). High prevalence of irritable bowel syndrome in medical outpatients in Japan. *Journal of Clinical Gastroenterology, 42*, 1010–1016.

Shinozaki, M., Kanazawa, M., Kano, M., Endo, Y., Nakaya, N., Hongo, M., et al. (2010). Effect of autogenic training on general improvement in patients with irritable bowel syndrome: A randomized controlled trial. *Applied Psychophysiology and Biofeedback, 35*, 189–198.

Talley, N. J., Owen, B. K., Boyce, P., & Paterson, K. (1996). Psychological treatments for irritable bowel syndrome: A critique of controlled treatment trials. *American Journal of Gastroenterology, 91*, 277–283.

Whitehead, W. E., Crowell, M. D., Robinson, J. C., Heller, B. R., & Schuster, M. M. (1992). Effects of stressful

life events on bowel symptoms: Subjects with irritable bowel syndrome compared with subjects without bowel dysfunction. *Gut, 33*, 825–830.
Whorwell, P. J., Prior, A., & Faragher, E. B. (1984). Controlled trial of hypnotherapy in the treatment of severe refractory irritable bowel syndrome. *Lancet, 2*, 1232–1233.
Zijdenbos, I. L., de Wit, N. J., van der Heijden, G. J., Rubin, G., & Quartero, A. O. (2009). Psychological treatments for the management of irritable bowel syndrome. *Cochrane Database Systematic Review, 1*, CD006442.

Ischemic Heart Disease

Siqin Ye
Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Synonyms

Coronary heart disease

Definition

Ischemic heart disease refers to various manifestations of heart disease caused by ischemia, i.e., inadequate blood flow to the myocardium (Morrow & Gersh, 2008).

Description

Ischemic heart disease is typically due to coronary heart disease caused by atherosclerosis (see entry, ▶ Coronary Heart Disease). Patients with ischemic heart disease may have symptoms of angina, or chest pain (see entry, ▶ Angina Pectoris). Sudden rupture of coronary atherosclerotic plaques may cause myocardial infarction, or heart attack (see entry, ▶ Acute Myocardial Infarction). If there is sufficient damage done to the heart, the patient may also develop heart failure or ischemic cardiomyopathy (see entry, ▶ Congestive Heart Failure).

Cross-References

▶ Acute Myocardial Infarction
▶ Angina Pectoris
▶ Congestive Heart Failure
▶ Coronary Heart Disease

References and Readings

Morrow, D. A., & Gersh, B. J. (2008). Chronic coronary artery disease. In P. Libby, R. O. Bonow, D. L. Mann, D. P. Zipes, & E. Braunwald (Eds.), *Braunwald's heart disease: A textbook of cardiovascular medicine* (pp. 1353–1417). Philadelphia: Saunders Elsevier.

iScience

▶ Internet-Based Studies

Isometric/Isotonic Exercise

Anthony Remaud
Elisabeth Bruyere Research Institute, University of Ottawa, Ottawa, ON, Canada
Laboratory "Motricité, Interactions, Performance", University of Nantes, Nantes, France

Synonyms

Exercise-general category; Exercise testing; Exercise, benefits of; Graded exercise; Resistance training; Static Exercise

Definition

An *isometric* exercise refers to any physical activity involving static muscle contractions. The term "isometric" is derived from the Greek roots "isos" (equal) and "metron" (measure), which means that during isometric exercise,

muscle length is broadly unchanged and consequently joint angle remains the same. From a biomechanical point of view, this exercise modality means that the net moment generated by the individual at the joint level is equal to the resistant moment applied to the body segment in an opposing direction. Actually, muscles slightly shorten and involve a stretch at the tendon level but the length of the muscle-tendon complex remains constant. Depending on the amount of resistance applied to the body segment, the level of muscle contraction can be maximal or submaximal. Performing the "wall sit/phantom chair" exercise, holding a free weight in a fixed position or pushing against an unmovable object such as a wall can illustrate different types of isometric exercise. Since it does not require any specific equipment, isometric exercise can be performed anywhere, anytime.

An *isotonic* exercise implies dynamic muscle contractions. The term "isotonic" is derived from the Greek roots "isos" (equal) and "tonos" (tension), which means that during isotonic exercise, muscle tension is supposed to remain constant during the movement. From a biomechanical point of view, this exercise modality means that the net moment generated by the individual at the joint level is unchanged throughout the range of motion whereas movement velocity varies. In the literature, isotonic exercises often designate strength training workouts performed with a constant resistance using bodyweight (e.g., push-up exercise) or free weights (e.g., barbell bench press). These types of exercise can be performed in concentric mode if the net moment generated at the joint level is superior to the resistant moment applied to the body segment (muscle shortening) or in eccentric mode if this net moment is inferior to the resistant moment (muscle lengthening). Actually, the isotonic condition is rarely achieved during strength training workouts notably due to the acceleration and deceleration of the load during the movement which modify the resistant moment. Therefore, the terms "isoinertial" or "isoload" have been proposed to better describe this concept.

Different disciplines relevant to health such as physical medicine and rehabilitation, physiotherapy, or athletic training commonly use isometric and isotonic exercises as a means for functional muscle assessment and/or strength training.

Cross-References

- ▶ Aerobic Exercise
- ▶ Health
- ▶ Physical Activity
- ▶ Physical Activity Interventions

References and Readings

Abernethy, P., Wilson, G., & Logan, P. (1995). Strength and power assessment. Issues, controversies and challenges. *Sports Medicine, 19*(6), 401–417.

Enoka, R. M. (2002). *Neuromechanics of human movement* (3rd ed.). Champaign: Human Kinetics.

Watkins, J. (1999). *Structure and function of the musculoskeletal system* (1st ed.). Champaign: Human Kinetics.

IUI

▶ Infertility and Assisted Reproduction: Psychosocial Aspects

IVF

▶ Infertility and Assisted Reproduction: Psychosocial Aspects

▶ In Vitro Fertilization, Assisted Reproductive Technology

J

Job Analyses

► Job Related to Health

Job Characteristics

► Job Demands

Job Classification

Karen Jacobs[1], Miranda Hellman[2], Jacqueline Markowitz[2] and Ellen Wuest[2]
[1]Occupational Therapy, College of Health and Rehabilitation Science, Sargent College, Boston University, Boston, MA, USA
[2]Boston University, Boston, MA, USA

Synonyms

Employment; Job evaluation; National occupational classification; National statistics socio-economic classification; Occupation; Occupational classification; United States Department of Labor

Definition

Job classification is the defining or categorization of types of employment either by function or activity. Job classification is useful to combine jobs for test validation, wage and salary administration, and for the development of performance appraisal instruments (Sackett, Cornelius, & Carron, 1981). Job classification is also critical for structuring personnel selection, training, and career development (Pearlman, 1980). Successful job classification can also be useful for client-oriented applications, allowing people to be matched to job vacancies that fit their skill set (International Labor Organization, 2010). The specificity of the classification is important depending on the type of work. A general level of classification is sufficient for distinctly different jobs, but more specific classifications are necessary to distinguish jobs within occupational groups, such as management jobs or clerical jobs (Schippmann, Prien, & Hughes, 1991). When a job is classified, it should be consistent and should match all other jobs assigned to that class, it should be clear and each job should only fall under one assignment, and the classifications should be interpretable (Hartman, Mumford, & Mueller, 1992).

Description

International Classification of Jobs

Job classification is a process that occurs internationally, with similarities and differences between countries. The United States Department of Labor has classified all workers into one of 840 occupations. These occupations are grouped together based on similar job duties, skills, education, and training (US Department

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

of Labor, 2010). Canada utilizes similar ways to classify jobs, and created the National Occupational Classification (NOC) that gives statisticians, labor market analysts, career counselors, employers, and individual job seekers a way to understand the nature of that job and collect appropriate data (Human Resources and Skills Development Canada, 2009). The NOC is organized in such a manner that each occupation is assigned a four digit number that provides information about the job; for example, 3113 is for dentists, and the "31" symbolizes that it is a health field requiring a university-level education (Human Resources and Skills Development Canada, 2009). The United Kingdom uses the Standard Occupational Classification (SOC), which has three volumes. The first volume outlines the background, resources, concepts, and processes of how the jobs are classified, the second codes the index for classification, and the third shows the relationship between the SOC and the National Statistics Socio-economic Classification (Office for National Statistics, 2010). The International Standard Classification of Occupations is a model to classify jobs globally, allows for international reporting, and can be used in countries that have not yet developed their own means to classify jobs (2010).

Cross-References

- ▶ Prevention: Primary, Secondary, Tertiary
- ▶ Preventive Care

References and Readings

Hartman, E. A., Mumford, M. D., & Mueller, S. (1992). Validity of job classifications: An examination of alternative indicators. *Human Performance, 5*, 191–211.

Human Resources and Skills Development Canada. (2009). National occupation classification. Retrieved February 23, 2011, from http://www.hrsdc.gc.ca/eng/workplaceskills/noc/index.shtml

International Labor Organization. (2010). International standard classification of organizations. Retrieved February 23, 2011, from http://www.ilo.org/public/english/bureau/stat/isco/index.htm

Office for National Statistics. (2010). Standard occupational classification. Retrieved February 23, 2011, from http://www.ons.gov.uk/about-statostocs/classifications/current/soc2010/index.html

Pearlman, K. (1980). Job families: A review and discussion of their implications for personnel selection. *Psychological Bulletin, 87*, 1–28.

Sackett, P. R., Cornelius, E. T., III, & Carron, T. J. (1981). A comparison of global judgment vs. task oriented approaches to job classification. *Personnel Psychology, 34*, 791–806.

Schippmann, J. S., Prien, E. P., & Hughes, G. L. (1991). The content of management work: Formation of task and job skill composite classifications. *Journal of Business and Psychology, 5*, 325–356.

United States Department of Labor. (2010). Standard occupation classification. Retrieved February 22, 2011, from http://www.bls.gov/soc/

Job Components

- ▶ Job Performance

Job Control

- ▶ Job Demands
- ▶ Job Related to Health

Job Demand/Control/Strain

Töres Theorell
Stress Research Institute, Stockholm University, Stockholm, Sweden

Synonyms

Decision Authority; Decision Latitude; Mental Work Load

Definition

The job demand/control model is used for studying psychosocial stressors in the work environment.

Description

The *demand/control model* was created for the study of psychosocial working conditions. It was published by Robert Karasek in his Ph.D. thesis in 1976 (Massachusetts Institute of Technology) and as a scientific article (Karasek, 1979). While Karasek had already mentioned social support as a potentially important additional dimension, this part of the model – adding up to the *demand/control/support model* – was first published in 1986 (Johns Hopkins University) by Jeffrey V. Johnson in his Ph.D. thesis (Johnson, 1989). Sociopsychological and biological theory underlying the model was further developed in 1990 by Karasek and Theorell (Karasek & Theorell, 1990).

The basic underlying idea behind the creation of the model was that crucial psychosocial stressors (factors inducing adverse long-lasting stress reactions in employees) in the work environment should be assessed. Reduction of such stressors for the improvement of employee health by means of work redesign should then be the ultimate goal. According to the initial demand/control model theory, high psychological demands are endangering employee health particularly when the employees have little control (low decision latitude; the combination labeled "job strain") over their own conditions. On the other hand, when demands are high and decision latitude is also high ("active work") psychological growth, improved coping, and biological regeneration take place. Still according to the theory, the combination of low demands and low decision latitude ("passive work") is of intermediate importance to employee health. However, employees working for a long time in the "passive" work situation may lose competence and "stop growing" psychologically. The low-demand/high-control situation ("relaxed work") is the ideal situation serving as reference in the model. The "relaxed"/"job strain" diagonal of the model is of more importance to the employee health whereas the "passive"/"active" diagonal is more important to productivity.

The demand/control/support model has been tested in many epidemiological studies. The assessment of demand, control, and support has mostly been made by means of standardized questionnaires. The most widely used Job Content Questionnaire (JCQ) has been translated into many languages (Karasek et al., 1998). A shorter version (Demand Control Questionnaire (DCQ)) has been used primarily in the Nordic countries. JCQ contains scales for the assessment of psychological demand, decision latitude, and its two subcomponents (skill discretion and decision authority), and also social support from superiors and coworkers. JCQ also includes questions about physical demands and job insecurity.

Measures of psychological job demands, decision latitude, and social support can also be derived from job titles. The basis of such assessments is the creation from representative samples in the working population of a "job exposure matrix" (JEM) indicating the average demand, decision latitude, and support scores for each job title. The means can also be specified with regard to age group, length of employment, and gender since these factors could influence the means. In most studies, the three-digit version of the international classification of jobs has been used for the psychosocial JEM. This provides a more "objective" way of assessing the work characteristics. However, in the recording of the demand/control/support model it is mainly the decision latitude dimension that provides sufficiently reliable data since both demands and social support assessments vary more between individuals within occupations than they do for decision latitude. Another more objective or "external" way of measuring is to train experts to do collective assessments of job sites with regard to demand, control, and support.

How does the demand/control/support model relate to people's physiological status? In several epidemiological studies, the model has been examined in relation to biological risk factors for cardiovascular disease. Accordingly in some studies, high or increasing job strain has been significantly related to elevated levels of sleep disturbance, blood pressure (particularly when blood pressure is monitored automatically during 24 h), morning excretion of cortisol in saliva and immunological parameters, such as plasma

fibrinogen, all of which regarded as risk factors for cardiovascular disease. High or rising levels of job strain are also related to lowered plasma testosterone concentration, which has regenerative/restorative functions protecting the body against adverse effects of stress (Theorell, 2009). In addition, low levels of decision latitude have been shown in one study to be related to low parasympathetic activity (another protector against adverse stress effects), as it is reflected in heart rate variability measured by means of 24-h electrocardiogram. All these observations indicate that job strain may affect physiology in adverse ways. Poor social support has been shown to add to the associations. However, there are relatively few studies of physiological correlates of Demand Control Support (DCS) and hence, there is no complete scientific consensus in this area – with the exception of fully automated measures of high blood pressure which are consistently related to high job strain.

Epidemiological studies have also been made of the relationship between job strain and adverse health habits such as smoking, physical activity, and alcohol consumption. The findings in these studies are conflicting. The relationships between health habits and the Demand Control Support (DCS) model depend on contexts, such as social class, age group, country, etc. Social class has been discussed extensively since low socioeconomic status is related to low decision latitude and high socioeconomic status to high psychological demands. Whether or not to adjust for social class remains to be resolved. In many studies, the relationship between the DCS and disease outcome is only marginally affected by adjustment for socioeconomic group, whereas other studies show stronger effects of such adjustments.

Several prospective epidemiological studies have been published regarding the relationship between the DCS model and risk of cardiovascular disease (mainly myocardial infarction). Designs, outcome measures, and DCS exposure measures have varied between studies. Several of the published studies have had too small statistical power or too long follow-up periods (for instance, 20 years). Meta-analytical studies, however, indicate that among working men without initial heart disease job strain (in most studies defined in such a way that 20–30% of the study population is defined as being exposed – the remaining participants are mostly used as reference population) is associated with a 40% elevation of the risk of developing a myocardial infarction during a 5-year follow-up. The job strain–related risk among working men seems to be stronger (in the order of 80% excess risk) in analyses confined to those below age 55 at start of follow-up. The combination of job strain and lack of social support (iso-strain) has been less extensively studied but is also related to risk of cardiovascular disease. There are different opinions regarding the role of smoking, physical activity, and eating/drinking habits in these associations. In several studies, adjustments for such risk factors have not affected the association markedly. For women the epidemiological evidence is not clear.

Early signs of atherosclerosis assessed by means of noninvasive methods (Carotid artery Intima Media Thickness, IMT) have been shown to be correlated with degree of job strain in Finnish men in their younger middle age, even after adjustment for accepted risk factors for cardiovascular disease. This is a developing research field like the study of interaction between genetic stress-related genetic allele combinations and exposure to job strain (Kivimäki et al., 2008).

Mental disease has also been examined in several prospective epidemiological studies including demand/control/support which show that the DCS model can predict risk of depression and "burnout." Depression seems to be particularly strongly related prospectively to high psychological demands and lack of social support at work.

Musculoskeletal disorders (in particular neck-shoulder pain) have also been related to the DCS in many studies. There seems to be an interaction between physical demands (such as heavy lifting or awkward working positions with static load) and high psychological demands/low decision latitude/poor social support in the etiology of neck-shoulder pain. The findings in different studies seem to depend on the type of work (white collar/blue collar), gender, age, and other contextual factors. While there is no full scientific

consensus regarding the role of the DCS model in musculoskeletal disorders, researchers agree that psychosocial job factors represented by the DCS are important in rehabilitation after the onset of such disorders.

The theory underlying the demand/control/support model has been used in several other epidemiological contexts, for instance productivity, international labor markets, pregnancy outcome, and use of protective equipment in industry.

Karasek has recently elaborated the DCS model in his stress disequilibrium theory (Karasek, 2008). This postulates that release and storage of energy are crucial features. In the job strain situation, energy is constantly released with insufficient opportunity for energy storage. During periods of energy storage there is also opportunity for regeneration and anabolism.

Cross-References

► Job Performance

References and Readings

Johnson, J. V. (1989). Control, collectivity and the psychosocial work environment. In S. Sauter, J. J. Hurrell, & C. L. Cooper (Eds.), *Job control and worker health*. London: Wiley.

Karasek, R. A. (1979). Job demands, job decision latitude and mental strain: Implications for job redesign. *Administrative Sciences Quarterly, 24*, 285–308.

Karasek, R. A. (2008). Low social control and physiological deregulation – the stress-disequilibrium theory, towards a new demand-control model. *Scandinavian Journal of Work, Environment and Health*, Suppl. (6), 117–135.

Karasek, R. A., Brisson, C., Kawakami, N., Houtman, I., Bongers, P. M., & Amick, B. (1998). The job content questionnaire (JCQ): an instrument for internationally comparative assessments of psychosocial job characteristics. *Journal of Occupational Health Psychology, 3*, 322–355.

Karasek, R. A., & Theorell, T. (1990). *Healthy work: Stress, productivity and the reconstruction of working life*. New York: Basic Books.

Kivimäki, M., Vahtera, J., Elovainio, M., Keltikangas-Järvinen L., Virtanen, M., Hintsanen, M., et al., (2008). What are the next steps for research on work stress and coronary heart disease? *Scandinavian Journal of Work, Environment and Health*, Suppl. (6), 33–40.

Theorell, T. (2009). Anabolism and catabolism. In S. Sonnentag, P. L. Perrewé, & D. C. Ganster (Eds.), *Research in occupational stress and well-being* (Current perspectives on job-stress recovery, Vol. 7, pp. 249–276). Emerald/JAI Press: Bingley.

Job Demands

Karen Jacobs[1], Miranda Hellman[2], Jacqueline Markowitz[2] and Ellen Wuest[2]
[1]Occupational Therapy, College of Health and Rehabilitation Science, Sargent Collage, Boston University, Boston, MA, USA
[2]Boston University, Boston, MA, USA

Synonyms

Job characteristics; Job control; Job strain; Work engagement

Definition

Job demands are aspects of any job that include the costs to the person completing the tasks necessary to the job. While physical demands are often the most obvious job demands, psychological, physiological, social, and organizational costs are also present in the demands of any job (Bakker & Demerouti, 2007). Job demands are dependent on the stress sources present in any environment a job is done and can be measured by the amount of stress caused to the worker participating in the job (Karasek, 1979). The job demands-resources model is a current popular model for exploring job demands by also incorporating job resources and finding a balance between them to reduce job strain (Bakker, Demerouti, Taris, Schaufeli, & Schreurs 2003).

Cross-References

► Job Related to Health

- ▶ Social Relationships
- ▶ Social Strain
- ▶ Social Stress
- ▶ Social Support

References and Readings

Bakker, A. B., & Demerouti, E. (2007). The job demands-resources model: State of the art. *Journal of Managerial Psychology, 22*(3), 309–328.

Bakker, A. B., Demerouti, E., Taris, T., Schaufeli, W. B., & Schreurs, P. (2003). A multi-group analysis of the job demands-resources model in four home care organizations. *International Journal of Stress Management, 10*, 16–38.

Karasek, R. A. (1979). Job demands, job decision latitude, and mental strain: Implications for job redesign. *Administrative Science Quarterly, 24*(2), 285–308.

Job Diagnostic Survey

Marie-Louise Schult
Karolinska Institute, Department of Clinical Sciences, Department of Neurobiology, Care Sciences and Society, The Rehabilitation Medicine, University Clinic Danderyd Hospital, Stockholm, Sweden

Synonyms

Characteristics Study; Work

Definition

The Job Diagnostic Survey Questionnaire (JDS) intends to (a) diagnose existing jobs to determine if and how these might be redesigned to improve employee's motivation, satisfaction, and productivity and (b) evaluate the effects.

Description

The Job Diagnostic Survey (JDS) was developed by Hackman and Oldham in 1975. JDS is based on theoretical frameworks by Turner and Lawrence (1965), Hackman and Lawler (1971), and further developed by Hackman and Oldham (1975, 1980). JDS is based on the theoretical model the "Job Characteristics Model" (Hackman & Oldham, 1975, 1980) which contains three basic psychological critical states that promote high performance, motivation, and satisfaction at work. The critical states are as follows: (a) a person must experience the work as meaningful, valuable, and worthwhile; (b) a person should assume responsibility for the result of the work; and (c) a person should have knowledge of the results from work.

Experienced meaningfulness of a job is enhanced primarily by three core job dimensions: skill variety (a variety of different activities involving the use of different skills), task identity (doing a job from the beginning to end), and task significance (substantial impact).

Experienced responsibility for work outcomes increases when a job has high autonomy (substantial freedom, independence).

Knowledge of the results is increased when a job gives high feedback (the employee gets direct and clear information about the effectiveness of performance).

Affective responses from the work is investigated using questions regarding (a) general satisfaction (the degree to which the employee is satisfied with the job); (b) internal work motivation (the employee experiences positive internal feelings when performing effectively at work); and (c) specific satisfaction of job security, pay, social, supervisory, and opportunity for personal growth and development.

Job Diagnostic Survey Questionnaire

The original *Job Diagnostic Survey Questionnaire* (JDS) (Hackman & Oldham, 1975) is designed to be completed by workers. It contains questions and statements of Likert type, ranging from one to seven with higher scores indicating the greater extent to which the respondent's own work possesses the characteristics being assessed. JDS includes four major parts. The first part "job dimensions" includes skill variety ($n = 3$), task identity ($n = 3$), task significance

($n = 3$), autonomy ($n = 3$), feedback from job itself ($n = 3$), feedback from agents ($n = 3$), and dealing with others ($n = 3$). The second part "psychological states" includes experienced meaningfulness from work ($n = 4$), experienced responsibility for the work ($n = 6$), and knowledge of the results ($n = 4$). Part three "the affective responses to the job" includes general satisfaction ($n = 5$), internal work motivation ($n = 6$), and specific satisfaction with job security ($n = 2$), pay ($n = 2$), social ($n = 3$), supervisory ($n = 3$), and growth ($n = 4$). Part four "growth needs strength" includes questions in "would like" format ($n = 6$) and "job choice" format ($n = 12$).

The Job Characteristics Model suggests that it is possible to generate a summary score reflecting an overall *motivating potential* (MPS) of a job based on the five core job dimensions:

$$\text{MPS} = \frac{(\text{Skill variety} + \text{Task identify} + \text{Task significance})}{3} \times (\text{Autonomy}) \times (\text{Feedback})$$

Reliability

The instrument is not recommended for use at individual level; however, it shows satisfactory reliability when used on groups of five or more, performing the same type of job. The reliability ranges from a high internal consistency of 0.88 to a low of 0.58 (Hackman & Oldham, 1975). Results from test-retest are not available. JDS norm–referenced data are based on 6,930 persons working in 876 different jobs in 56 organizations (Oldham, Hackman, & Stepina, 1978).

Validity

Early work by Hackman and Oldham (1975) showed good face validity, with items being checked and re-checked during the 2-year development period. Fried and Ferris (1987) describe in their meta-analysis modest support for the model, suggesting that the model does not give a complete picture of the motivational effects of job characteristics. A recent study of the empirical evidence of the Job Characteristic Model was investigated in British establishments (DeVaro, Li, & Brookshire, 2007). The study result generally supports the model's prediction that task variety and worker autonomy are positively associated with labor productivity and product quality.

Oldham and Hackman (2010) describe how they under-recognized in their early work (1975, 1980) the importance for work redesign of the broader context, for example, the organization's formal properties and the culture within which the organization operates. They further suggest important areas for future research and discuss concepts such as social sources of motivation, job crafting, and work design for teams.

In several studies published during the years 2008–2011 using the JDS, only parts of the original questionnaire have been used, most frequently the subscales for job control (Tucker et al., 2009), job autonomy (Grandey, Fisk, & Steiner, 2005), and job satisfaction (Huffman, Youngcourt, Payne, & Castro, 2008; Nelson, Johnson, & Bebbington, 2009).

The JDS is generally applicable across industrial sectors and occupations. Investigations using the JDS have among others previously been published for customer-contact employees (Grandey et al., 2005), university employees (Liu, Spector, Liu, & Shi, 2011), army soldiers (Huffman et al., 2008), mental health professionals (Pedrini et al., 2009), psychiatrists (Kumar, Fisher, Robinson, Hatcher, & Bhagat, 2007), and pediatric nursing (Eaton & Thomas, 1997).

Cross-References

- ▶ Job Demand/Control/Strain
- ▶ Job Demands
- ▶ Job Satisfaction/Dissatisfaction

References and Readings

DeVaro, J., Li, R., & Brookshire, D. (2007). Analyzing the job characteristic model: New support from cross-section establishments. *International Journal of Human Resource Management, 18*, 986–2003.

Eaton, N., & Thomas, P. (1997). Job diagnostic surveys on paedric nursing: An evaluation tool. *Journal of Nursing Management, 5*, 167–173.

Fried, Y., & Ferris, G. R. (1987). The validity of the job characteristics model: A review and meta-analysis. *Personnel Psychology, 40*, 287–322.
Grandey, A. A., Fisk, G. M., & Steiner, D. D. (2005). Must "service with a smile" be stressful? The moderating role of personal control for American and French employees. *Journal of Applied Psychology, 90*, 893–904.
Hackman, J. R., & Lawler, E. (1971). Employee reactions to job characteristics. *Journal of Applied Psychology*, *55*, 259–286.
Hackman, J. R., & Oldham, G. R. (1975). Development of the job diagnostic survey. *Journal of Applied Psychology, 60*, 159–170.
Hackman, J. R., & Oldham, G. R. (1980). *Work redesign*. Reading, MA: Addison-Wesley.
Huffman, A. H., Youngcourt, S. S., Payne, S. C., & Castro, C. A. (2008). The importance of construct breadth when examining interrole conflict. *Educational and Psychological Measurement, 68*, 515–530.
Kumar, S., Fisher, J., Robinson, E., Hatcher, S., & Bhagat, R. N. (2007). Burnout and job satisfaction in New Zealand psychiatrists: A national study. *The International Journal of Social Psychiatry, 53*, 306–316.
Liu, C., Spector, P.E., Liu, Y., & Shi, L. (2011). The interaction of job autonomy and conflict with supervisor in China and the United States: A qualitative and quantitative comparison. *International Journal of Stress Management, 18*(3), 222–245.
Nelson, T., Johnson, T., & Bebbington, P. (2009). Satisfaction and burnout among crisis resolution, assertive outreach and community mental health teams. A multicentre cross sectional survey. *Social Psychiatry and Psychiatric Epidemiology, 44*, 541–549.
Oldham, G. R., & Hackman, J. R. (2010). Not what it was and not what it will be: The future of job design research. *Journal of Organizational Behavior, 31*, 463–479.
Oldham, G. R., Hackman, J. R., & Stepina, L. (1978). *Norms for the job diagnostic survey* (Technical Report No. 16), New Haven: School of Organisation and Management, Yale University.
Pedrini, L., Magni, L. R., Giovannini, C., Panetta, V., Zacchi, V., Rossi, G., et al. (2009). Burnout in nonhospital psychiatric residential facilities. *Psychiatric Services, 60*, 1547–1551. Ps.psychiatryonline.org.
Schult, M. L., & Söderback, I. (2000). A method for "Diagnosing" jobs before redesign in chronic-pain patients: Preliminary findings. *Journal of Occupational Rehabilitation, 10*, 295–309.
Tucker, J. S., Sinclair, R. R., Mohr, C. D., Adler, A. B., Thomas, J. L., & Salvi, A. D. (2009). Stress and counterproductive work behaviour: Multiple relationships between demands, control, and soldier indiscipline over time. *Journal of Occupational Health Psychology, 14*, 257–271.
Turner, N., & Lawrence, R. (1965). *Industrial jobs and the worker*. Boston: Harvard Graduate School of Business Administration.

Job Evaluation

▶ Job Classification

Job Performance

Karen Jacobs[1], Miranda Hellman[2], Ellen Wuest[2] and Jacqueline Markowitz[2]
[1]Occupational Therapy, College of Health and Rehabilitation Science, Sargent College, Boston University, Boston, MA, USA
[2]Boston University, Boston, MA, USA

Synonyms

Employee appraisal; Job components; Job performance standards; Knowledge of work; Productivity; Quality of work; Situational responsiveness

Definition

Job performance relates to the act of doing a job. Job performance is a means to reach a goal or set of goals within a job, role, or organization (Campbell, 1990), but not the actual consequences of the acts performed within a job. Campbell (1990) affirms that job performance is not a single action but rather a "complex activity" (p. 704). Performance in a job is strictly a behavior and a separate entity from the outcomes of a particular job which relate to success and productivity.

Cross-References

▶ Stress
▶ Stress Management
▶ Workload
▶ Work-Related Stress

References and Readings

Campbell, J. (1990). Modeling the performance prediction problem in industrial and organizational psychology. In M. Dunnette & L. Hough (Eds.), *Handbook of industrial and organizational psychology* (pp. 686–707). Palo Alto, CA: Consulting Psychologists Press.

Job Performance Standards

► Job Performance

Job Prestige

► Occupational Status

Job Related to Health

Karen Jacobs[1], Miranda Hellman[2], Jacqueline Markowitz[2] and Ellen Wuest[2]
[1]Occupational Therapy, College of Health and Rehabilitation Science, Sargent Collage, Boston University, Boston, MA, USA
[2]Boston University, Boston, MA, USA

Synonyms

Job analyses; Job control; Job demands; Job strain; Work-related stress

Definition

A *job* is "a single position or a group of positions, at one establishment, whose major work activities and objectives are similar in terms of worker actions, methodologies, materials, products, and/or worker characteristics; and whose array of work activities differs significantly from those of other positions" (US Department of Labor, 1982). Employees are reimbursed by working a job, which is how a job differs from a volunteer opportunity. Reimbursement does not have to be monetary but should be something of use and value to the employee.

Occupational health promotes the highest degree of physical, mental, and social well-being of workers in all occupations. It is aimed to prevent workers' ill health caused by working conditions, protect them in their works from risks that include adverse factors to health, and place them in optimum adapted occupational environment.

Related to health, it is therefore important to analyze jobs in order to understand the specific activities and requirements of the people who perform them. According to the US Office of Personnel Management (USOPM), job analysis is a "systematic procedure for gathering, documenting, and analyzing information about the content, context, and requirements of the job. It demonstrates that there is a clear relationship between the tasks performed on the job and the worker's competencies required to perform the tasks" (US Office of Personnel Management [USOPM], 2011). Job analysis requires that certain categories of information be reported in a systematic way (US Department of Labor, 1982). The two major categories of job analysis are Work Performed and Worker Characteristics (US Department of Labor). Together, work performed and worker characteristics define the dimensions of job requirements.

Job related to health thus includes adaption of work to the worker and correspondence between worker's capability and the job requirements.

Description

Some job demands may have an impact on the worker's health and contribute to health problems such as heart disease, depression, and burnout.

Having a defined system to analyze jobs helps people to determine which jobs they are capable of performing that ill health do not occur. For example, if a person acquires an injury that leaves them unable to lift heavy objects, they will have to consult job analyses to determine jobs that they can perform despite their lifting restriction.

Unfortunately, jobs can be a cause of mental, physical, and emotional harm for employees. Because of the health risks sometimes associated with holding a job, there are organizations designed to protect employees. One such organization is the International Labour Organization (ILO). It "is devoted to promoting social justice and internationally recognized human and labor rights, pursuing its founding mission that labor peace is essential to prosperity. Today, the ILO helps advance the creation of decent work and the economic and working conditions that give working people and business people a stake in lasting peace, prosperity and progress. Its main aims are to promote rights at work, encourage decent employment opportunities, enhance social protection and strengthen dialogue on work-related issues" (ILO, 2011).

Cross-References

- ▶ Self-concept
- ▶ Workload
- ▶ Work-Related Stress

References and Readings

International Labour Organization. (2011). *Mission and objectives*. Retrieved February 28, 2011, from http://www.ilo.org/global/about-the-ilo/mission-and-objectives/lang–en/index.htm

US Department of Labor. (1982). *A guide to job analysis: A "how-to" publication for occupational analysts*. Stout, Wisconsin: Stout Vocational Rehabilitation Institute.

US Office of Personnel Management. (2011). *Job analysis tools*. Retrieved February 28, 2011, from http://www.opm.gov/hiringtoolkit/docs/jobanalysis.pdf

Job Satisfaction/Dissatisfaction

Karen Jacobs[1], Miranda Hellman[2], Jacqueline Markowitz[2] and Ellen Wuest[2]
[1]Occupational Therapy, College of Health and Rehabilitation Science, Sargent College, Boston University, Boston, MA, USA
[2]Boston University, Boston, MA, USA

Synonyms

Social support at work; Work autonomy; Work fulfillment/non-fulfillment; Work performance feedback; Work satisfaction/dissatisfaction

Definition

Job satisfaction, that is, intrinsic and extrinsic job satisfaction, deals with the contentment with a particular job, done by an employee. Contentment with a job can be due to the internal qualities of an employee, including personality and personal lenses, and also the external features of the workplace including the environment. Some form of management controlling the quality of job outcomes often influences job satisfaction as well as the amount of power the employee is given.

Description

There are two major forms of job satisfaction: *intrinsic and extrinsic job satisfaction*. Intrinsic job satisfaction is the contentment with the type of work the employee is doing, while extrinsic job satisfaction encompasses the environment that the job is being completed in. Intrinsic job satisfaction is most variable between people because of individual preferences for different types of work. Extrinsic job satisfaction includes environmental factors such as salary, coworkers, and management (Jones, 1992).

Job satisfaction may be improved with varying the different types of tasks, the ability to get promoted, changing environmental factors, and higher employment salary. Although commonly thought to be an essential indicator of job satisfaction, salary level only has a marginal correlation of 15% with job satisfaction (Judge, Piccolo, Podsakoff, Shaw, & Rich, 2010). Moreover, job satisfaction has no direct relationship with job performance (Laffaldano & Muchinsky, 1984) but is strictly dependent on the context and outlook of the individual completing the job.

Job satisfaction can be measured by the amount of stress, the work group characteristics, the physical environment, and organizational rewards/punishments (Brief & Weiss, 2002). The most common way to measure job satisfaction in a traditional work environment is to give employees questionnaires, often constructed using a Likert scale. These assessments allow individuals to rate all aspects of the work satisfaction. Items most commonly given low scores may be improved within a workplace resulting in the job satisfaction of all involved to likely increase.

Job dissatisfaction is the discontentment of the conditions involved in measuring job satisfaction. Although these present as opposing concepts, an employee is able to have fluctuating feelings of both of these concepts as the work environment changes. Although an employee may be dissatisfied with the extrinsic aspects of a job, he or she may conversely be satisfied with the intrinsic features of that same job. Extrinsic factors, in particular, are associated with levels of job dissatisfaction high enough to result in a worker quitting a job (Arnett & Polkinghorne, 2010). A lack of achievement, responsibility, and recognition may result in job dissatisfaction (Dunnette, Campbell, & Hakel, 1966). While an employee may enjoy the tasks they are completing, their appreciation at their facility is an important element in maintaining the employee's satisfaction. Working conditions, company policies, and security are most likely to cause job dissatisfaction (Dunnette et al., 1966) as these are least likely to change if offensive.

Conclusion

Work satisfaction/dissatisfaction can be influenced by job characteristics and have an impact on a worker's well-being. For example, work demands such as a high work pressure, emotional demands, and role ambiguity may lead to sleeping problems, exhaustion, etc. Impaired health work resources such as social support, performance feedback, and autonomy might lead to work-related learning, work engagement, and organizational commitment (www.emeraldinsight.com/0268-3946.htm).

Cross-References

- ▶ Depression: Symptoms
- ▶ Emotions: Positive and Negative
- ▶ Job Demand/Control/Strain
- ▶ Social Strain
- ▶ Social Stress
- ▶ Social Support

References and Readings

Arnett, S. E., & Polkinghorne, F. W. (2010). Job Dissatisfaction: A factor in maintaining a highly-qualified family and consumers sciences teacher workforce. *Online Journal of Workforce Education and Development, IV*(4), 1–13.

Brief, A. P., & Weiss, H. M. (2002). Organizational behavior: Affect in the workplace. *Annual Review of Psychology, 53*, 279–307.

Dunnette, M. D., Campbell, J. P., & Hakel, M. D. (1966). Factors contributing to job satisfaction and job dissatisfaction in six occupational groups. *Organizational Behavior and Human Performance, 2*(2), 143–174.

Iaffaldano, M. T., & Muchinsky, P. M. (1984). Job satisfaction and job performance: A meta-analysis. *Psychological Bulletin, 97*(2), 251–273.

Jones, L. K. (1992). Job satisfaction. In *Encyclopedia of career change and work issues* (pp. 142–143). Phoenix: The Oryx Press.

Judge, T. A., Piccolo, R. F., Podsakoff, N. P., Shaw, J. C., & Rich, B. L. (2010). The relationship between pay and job satisfaction: A meta-analysis of the literature. *Journal of Vocational Behavior, 77*(2), 157–167.

Job Strain

- ▶ Job Demands
- ▶ Job Related to Health

Joint Inflammation

- ▶ Arthritis

Joint Pain

- ▶ Arthritis

Juvenile Diabetes

- ▶ Type 1 Diabetes Mellitus
- ▶ Insulin-Dependent Diabetes Mellitus (IDDM)

K

Kallikrein-3

► Prostate-Specific Antigen (PSA)

Kaposi's Sarcoma

David J. Finitsis
Department of Psychology, University of Connecticut, Storrs, CT, USA

Synonyms

KS

Definition

Kaposi's sarcoma (KS) is a rare disorder typically affecting the skin, mucous membranes, and blood vessels (Kubota, 2000); it was first described by Hungarian physician Moritz Kaposi in 1872 (Safai, 1997). Originally described as a cancer, current research suggests that KS is virally mediated (Hayward, Alcendor, & Arav-Broger, 2010).

Description

KS presents as pink, purple, and/or brownish red maculae or plaques which may hemorrhage (Knipe & Howley, 2007; Kubota, 2000).

Multiple forms of KS have been identified. The classic form is indolent, progressing slowly with localized expression that remains confined to the skin and subcutaneous tissues. This form was originally reported by Kaposi among a circumscribed population of primarily elderly men from Eastern Europe and the Mediterranean. It is rarely life-threatening and may spontaneously remit. A form was subsequently identified in certain regions of sub-Saharan Africa affecting adults and children and may present as either indolent or aggressive. An aggressive form has since been described in immunosuppressed organ transplant recipients (Knipe & Howley, 2007).

The acquired immune deficiency syndrome (AIDS) – related form of KS – is likewise aggressive. Lesions spread rapidly to involve a significant area of the skin and may develop into larger nodules and tumors. Aggressive KS may also extend to the lymphatic and gastrointestinal systems as well as the lungs (Kubota, 2000). With their pervasive involvement of the lymph nodes, mucosal tissue, and solid organs, aggressive forms of KS carry a significant risk of fatal complications (Kubota, 2000; Safai, 1997).

Etiology

Moritz Kaposi originally categorized KS as a small cell sarcoma, but KS presents as multiple lesions with a course that may be indolent in the immunologically intact patient. This differs greatly from cancer which typically manifests as

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

a unifocal lesion that spreads by metastasis. The current theory is that an infectious agent causes KS: researchers have identified a unique herpes-like virus from KS lesions, called Kaposi's sarcoma-associated herpesvirus (KSHV) or human herpesvirus 8 (HHV8). Forms of this virus have since been found present in all cases of Kaposi's sarcoma regardless of human immunodeficiency virus (HIV) serostatus (Hayward, Alcendor, & Arav-Broger, 2010).

Treatment

Historically, Kaposi's sarcoma has been treated as a cancer with surgery, radiation, and chemotherapy. Since aggressive KS is linked to immune compromise, treatments that restore the immune system can indirectly treat KS. In its AIDS-related form, the use of highly active antiretroviral therapy (HAART) is associated with the reduction and remission of KS lesions (Hayward, Alcendor, & Arav-Broger, 2010).

Cross-References

► Immune Function

References and Readings

Hayward, G., Alcendor, D., & Arav-Broger, R. (2010). The role of KSHV in pathogenesis of Kaposi's sarcoma. In K. Khalili (Ed.), *Viral oncology basic science and clinical applications*. Hoboken, NJ: Wiley-Blackwell.

Knipe, D., & Howley, P. (Eds.). (2007). *Field's virology* (5th ed.). Philadelphia: Lippincott, Williams, and Wilkins.

Kubota, M. K. (2000). Human immunodeficiency virus infection and its complications. In R. Rakel (Ed.), *Conn's current therapy*. Philadelphia: W. B. Saunders Company.

Safai, B. (1997). Kaposi's sarcoma and acquired immunodeficiency syndrome. In V. DeVita Jr. (Ed.), *AIDS etiology, diagnosis, treatment, and prevention* (pp. 296–317). PhiladelphiaPA: Lippincott-Raven.

Kaufmann, Peter G.

Peter Kaufmann
Division of Prevention & Population Sciences, National Heart, Lung, and Blood Institute
Clinical Applications and Prevention Branch, Bethesda, MD, USA

Biographical Information

Peter Kaufmann was born in a World War II refugee camp near Lauenburg, Germany, on February 5, 1942. He immigrated to the United States with his parents in 1952, settling in Chicago, Illinois. Kaufmann received his BS and MA degrees in Psychology from Loyola University (Chicago) and his Ph.D. from the University of Chicago. He served briefly as Assistant Professor of Psychology at Emory and Henry College in Virginia, then pursued postdoctoral training in neurosciences in the Department of Physiology and Pharmacology at Duke University, in North Carolina, where he conducted research on the cellular organization of the visual system. He continued research in neuroscience at the Duke Center for Hyperbaric Medicine and Environmental Physiology, where he characterized the nature of central nervous system hyperexcitability arising from extreme barometric pressures such as encountered in deep-sea

diving, with support from the National Heart, Lung, and Blood Institute (NHLBI) and the Office of Naval Research.

In 1983, Kaufmann came to the NHLBI to develop a program of research on the neurophysiologic basis of behavioral risk factors of cardiovascular diseases. He rose to Chief of the Behavioral Medicine Branch in 1991, and Leader of the Behavioral Medicine and Prevention Research Group in 1994. In 1996, he organized the NHLBI Task Force on Behavioral Research in Cardiovascular, Lung, and Blood Health and Disease. Upon reorganization of the NHLBI in 2006, he was appointed Deputy Chief of the Clinical Applications and Prevention Branch. In 2000, he served as Acting Director of the National Institutes of Health (NIH) Office of Behavioral and Social Sciences Research (OBSSR). He served on numerous NIH-wide committees, including the NIH Behavioral and Social Sciences Coordinating Committee, the NIH Prevention Research Coordinating Committee, and as member and chair of the Data and Safety Monitoring Board (DSMB) for trials within the National Institute for Drug Abuse Clinical Trials Network. He testified on matters of behavioral and social sciences research in health before the Senate Committee on Appropriations, Subcommittee on Labor, Health and Human Services, and Education in 2000 and in 2002.

In addition to his work at the NIH, Kaufmann served on the Executive Council of the Academy of Behavioral Medicine Research (2003–2009) and the Executive Committee of the Society of Behavioral Medicine (2006–2009), serving as President from 2007 to 2008. He was Secretary of the International Society of Behavioral Medicine in 2009. He has received numerous awards for his work, including the NIH Director's Award (2011), several NIH Merit awards, the Distinguished Service Award of the Society of Behavioral Medicine (2009), Meritorious Research Service Commendation of the American Psychological Association (2003), the National Leadership Award of the Society of Behavioral Medicine (2002), and the Career Service to Health Psychology Award of the American Psychological Association (2002). He was elected Fellow of the Society of Behavioral Medicine in 1991 and Fellow of the Academy of Behavioral Medicine Research in 1992.

Major Accomplishments

Kaufmann's early work in hyperbaric medicine was to examine the reported rostro-caudal progression of high pressure hyperexcitability symptoms, demonstrating simultaneous effects at all levels of the central and peripheral nervous system. Along the way, he gained understanding of gas exchange, cardiac function, and the circulatory system. His initial focus at NHLBI was to develop research initiatives on the neurobiology of cardiovascular diseases, including the role of neuroactive peptides in response to mental stress, genetic influences in hypertension, and cardiovascular reactivity. Extending the work by David Krantz, James Muller, James Blumenthal, David Sheps and their collaborators, he launched the multisite Psychophysiological Investigations of Myocardial Ischemia (PIMI) study, which delineated the effects of mental stress on cardiac function in healthy men and women as well as in patients with obstructive coronary heart disease (CHD). This study demonstrated that vulnerability to mental stress increased mortality of CHD patients.

As identified behavioral risk factors grew in number and importance, Kaufmann recognized the importance of moving beyond descriptive studies and the study of mechanisms of action to develop clinical interventions that could be implemented in clinical practice. Spurred by the NHLBI's leadership in randomized clinical trials methodology, he turned his attention to identifying suitable targets for behavioral interventions and organized several randomized clinical trials to evaluate interventions for behavioral risk factors in cardiovascular diseases. He organized the Hypertension Intervention Pooling Project with Stephen Weiss, a rigorous examination of the methods and design of clinical trials in non-pharmacological management of hypertension.

He designed the stress management arm of the Trials of Hypertension Prevention with David Batey and collaborators and the Raynaud's Treatment Study (RTS), a multi-site clinical trial comparing behavioral and pharmacological treatments for Raynaud's Phenomenon, with Robin Hill. Inspired by Redford Williams' work on the influence of social support in reducing mortality and Robert Carney's research on depression in myocardial infarction patients, he was instrumental in launching the landmark Enhancing Recovery in Coronary Heart Disease Patients (ENRICHD) randomized trial with Susan Czajkowski. The trial evaluated whether cognitive behavioral therapy (CBT), supplemented by pharmacotherapy for treating depression and low perceived social support in patients after myocardial infarction would reduce the incidence of major acute cardiac events. The trial demonstrated the efficacy of CBT for depressed cardiac patients and led to insights about the role of depression in cardiovascular risk.

Kaufmann's work emphasized rigorous research designs and monitoring, contributing to setting the standard for clinical investigation in behavioral medicine. Along the way, he served as steward of numerous individual and group-randomized clinical trials involving stress management, meditation, relaxation therapy, and behavioral risk factor interventions for patients with hypertension, coronary heart disease, obesity, and tobacco addiction. He was successful in promoting collaborative research in behavioral medicine through Program Project Grants, Research Centers in Mind-Body Medicine, Translation Research, Research Centers for the study of health disparities and public health.

During his tenure as Acting Director of OBSSR in 2000, Kaufmann launched a committee to establish methods and a process for evaluating the efficacy of behavioral interventions. The Evidence-Based Behavioral Medicine project emerged and, under the guidance of Karina Davidson, was eventually adopted as a standing committee of the Society of Behavioral Medicine. At that time he also supported the early planning for the National Children's Study and designed and organized the Summer Institute on Randomized Clinical Trials Involving Behavioral Interventions, whose objective was to build a cadre of well-trained clinical trialists. The Summer Institute has continued as an annual event under Kaufmann's leadership with the collaboration and continued support of the OBSSR, particularly through the efforts and dedicated collaboration of Ronald Abeles. The Summer Institute has trained hundreds of junior investigators in the science of clinical trials.

Kaufmann has also provided leadership for numerous NHLBI and trans-NIH research initiatives and conferences, spanning a broad array of topics such as behavioral genetics, basic and applied research on the physiology and pharmacology of mental stress, pain and cardiovascular regulation, the role of mood disorders in somatic illness, translational science, health literacy, socioeconomic status and health, health disparities, and early life influences on health. With Redford Williams, he engaged in scientific exchange with Soviet scientists on behavioral factors in hypertension and in collaborative ventures with behavioral scientists in Lithuania. Kaufmann served on the editorial boards of Psychosomatic Medicine, Psichologija (Lithuania), and as Guest Editor of the Annals of Behavioral Medicine and the Journal of Cardiovascular Electrophysiology. He has held Adjunct teaching appointments at George Mason University in Virginia and Montgomery College, Maryland.

Cross-References

- ▶ Cognitive Behavioral Therapy (CBT)
- ▶ Evidence-Based Behavioral Medicine (EBBM)
- ▶ Randomized Clinical Trial
- ▶ Society of Behavioral Medicine

References and Readings

Batey, D. M., Kaufmann, P. G., Raczynski, J. M., Hollis, J. F., Murphy, J. K., & Rosner, B., et al. for the Phase I Trials of Hypertension Prevention (TOHP-I) Collaborative Research Group, (2000). Stress management intervention for primary prevention of

hypertension: Detailed results from phase I of trials of hypertension prevention (TOHP). *Annals of Epidemiology* 10, 45–58.
Berkman, L. F., Blumenthal, J., Burg, M., Carney, R. M., Catellier, D., Cowan, M. J., et al. (2003). The effects of treating depression and low perceived social support on clinical events after myocardial infarction: The enhancing recovery in coronary heart disease patients (ENRICHD) randomized trial. *Journal of the American Medical Association, 289*, 3106–3116.
Berntson, G. G., Bigger, J. T., Eckberg, D. L., Grossman, G., Kaufmann, P. G., Malik, M., et al. (1997). Heart rate variability: Origins, methods, and interpretive caveats. *Psychophysiology, 34*, 623–648.2.
Evans, D. L., Charney, D. S., Lewis, L., Golden, R. N., Gorman, J. M., Krishnan, K. R., et al. (2005). Mood disorders in the medically Ill: Scientific review and recommendations. *Biological Psychiatry, 58*, 175–189.
Sheps, D. S., McMahon, R. P., Becker, L., Carney, R. M., Freedland, K. E., Cohen, J. D., et al. (2002). Mental stress-induced ischemia and all-cause mortality in patients with coronary artery disease: Results from the psychophysiological investigations of myocardial ischemia study. *Circulation, 105*, 1780–1784.

Kawakami, Norito

Akihito Shimazu
Department of Mental Health, Graduate School of Medicine, The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Biographical Information

Prof. Norito Kawakami

Norito Kawakami was born on March 2, 1957, in Okayama, Japan. He graduated from Gifu University School of Medicine and acquired his M.D. degree in 1981. He was awarded his D.M. Sc. in 1985 from The University of Tokyo. After receiving his doctorate, Kawakami began his career as an assistant professor at The University of Tokyo. Then, he worked at the University of Texas School of Public Health, Houston, TX (1990–1991), and The University of Tokyo Faculty of Medicine (1991–1992) as a visiting researcher. He returned to Gifu University School of Medicine as an associate professor in the Department of Public Health (1992–2000). He moved to Okayama University Graduate School of Medicine, Dentistry & Pharmaceutical Science as a professor in the Department of Hygiene & Preventive Medicine (2000–2006). He is currently a professor in the Department of Mental Health, Graduate School of Medicine, University of Tokyo (2006).

Major Accomplishments

Kawakami has published more than 150 research papers in international journals concerning a broad range of topics in occupational health and behavioral medicine, such as the epidemiology of job stress (Kawakami, Kobayashi, Araki, Haratani, & Furui, 1995) and its health effects (Inoue et al., 2010; Kawakami et al., 1997, 2000) and workplace stress management interventions (Kawakami, Kobayashi, Takao, & Tsutsumi, 2005; Tsutsumi, Nagami, Yoshikawa, Kogi, & Kawakami, 2009).

He has also published papers regarding the epidemiology of mental disorders in the workplace and community (Kawakami et al., 1996; Kawakami, Shimizu, Haratani, Iwata, & Kitamura, 2004; WHO World Mental Health Survey Consortium, 2004), social inequality in health, smoking (Honjo, Tsutsumi, Kawachi, & Kawakami, 2006; Kawakami, Takatsuka, Shimizu, & Takai, 1998), and diabetes (Kawakami, Takatsuka, Shimizu, & Ishibashi, 1999).

Kawakami has served as the chair of the International Collaboration Studies Committee

(2004–2008), president of the International Society of Behavioral Medicine (2010–2012), chair of the Scientific Committee of Work Organization and Psychosocial Factors at Work (2006–2011), and as a board member for the International Commission on Occupational Health (2009). He has also served as an associate editor of the International Journal of Behavioral Medicine (2005–2008) and the editor-in-chief of the Journal of Occupational Health (2008–2011).

Cross-References

- ▶ International Society of Behavioral Medicine
- ▶ Mental Illness
- ▶ Occupational Health
- ▶ Smoking and Health
- ▶ Social Epidemiology
- ▶ Stress Management

References and Readings

Honjo, K., Tsutsumi, A., Kawachi, I., & Kawakami, N. (2006). What accounts for the relationship between social class and smoking cessation? Results of a path analysis. *Social Science & Medicine, 62*(2), 317–328.

Inoue, A., Kawakami, N., Haratani, T., Kobayashi, F., Ishizaki, M., Hayashi, T., et al. (2010). Job stressors and long-term sick leave due to depressive disorders among Japanese male employees: Findings from the Japan Work Stress and Health Cohort study. *Journal of Epidemiology and Community Health, 64*(3), 229–235.

Kawakami, N., Akachi, K., Shimizu, H., Haratani, T., Kobayashi, F., Ishizaki, M., et al. (2000). Job strain, social support in the workplace, and haemoglobin A1c in Japanese men. *Occupational and Environmental Medicine, 57*(12), 805–809.

Kawakami, N., Iwata, N., Tanigawa, T., Oga, H., Araki, S., Fujihara, S., et al. (1996). Prevalence of mood and anxiety disorders in a working population in Japan. *Journal of Occupational and Environmental Medicine, 38*(9), 899–905.

Kawakami, N., Kobayashi, F., Araki, S., Haratani, T., & Furui, H. (1995). Assessment of job stress dimensions based on the job demands- control model of employees of telecommunication and electric power companies in Japan: Reliability and validity of the Japanese version of the job content questionnaire. *International Journal of Behavioral Medicine, 2*(4), 358–375.

Kawakami, N., Kobayashi, Y., Takao, S., & Tsutsumi, A. (2005). Effects of web-based supervisor training on supervisor support and psychological distress among workers: A randomized controlled trial. *Preventive Medicine, 41*(2), 471–478.

Kawakami, N., Shimizu, H., Haratani, T., Iwata, N., & Kitamura, T. (2004). Lifetime and 6-month? Prevalence of DSM-III-R psychiatric disorders in an urban community in Japan? *Psychiatry Research, 121*(3), 293–301.

Kawakami, N., Takatsuka, N., Shimizu, H., & Ishibashi, H. (1999). Depressive symptoms and occurrence of type 2 diabetes among Japanese men. *Diabetes Care, 22*(7), 1071–1076.

Kawakami, N., Takatsuka, N., Shimizu, H., & Takai, A. (1998). Life-time prevalence and risk factors of tobacco/nicotine dependence in male ever-smokers in Japan. *Addiction, 93*(7), 1023–1032.

Kawakami, N., Tanigawa, T., Araki, S., Nakata, A., Sakurai, S., Yokoyama, K., et al. (1997). Effects of job strain on helper-inducer (CD4+CD29+) and suppressor-inducer (CD4+CD45RA+) T cells in Japanese blue-collar workers. *Psychotherapy and Psychosomatics, 66*(4), 192–198.

Tsutsumi, A., Nagami, M., Yoshikawa, T., Kogi, K., & Kawakami, N. (2009). Participatory intervention for workplace improvements on mental health and job performance among blue-collar workers: A cluster randomized controlled trial. *Journal of Occupational and Environmental Medicine, 51*(5), 554–563.

WHO World Mental Health Survey Consortium. (2004). Prevalence, severity, and unmet need for treatment of mental disorders in the World Health Organization. World Mental Health Surveys. *Journal of the American Medical Association, 291*(21), 2581–2590.

Killer Cell Activity

- ▶ Natural Killer Cell Activity

Kinesics

- ▶ Nonverbal Communication

Kinesiotherapy

- ▶ Physical Therapy

Kissing Disease

▶ Epstein-Barr Virus

Knowledge of Work

▶ Job Performance

Knowledge Translation

▶ Research to Practice Translation

KS

▶ Kaposi's Sarcoma

Kuopio Ischemic Heart Disease Risk Factor Study

Jussi Kauhanen
Institute of Public Health and Clinical Nutrition, University of Eastern Finland, Kuopio, Finland

Definition

The Kuopio Ischemic Heart Disease Risk Factor Study (KIHD) is an extensive epidemiologic research project that was launched in the 1980s. Within 20 years, it has produced over 400 original peer reviewed research papers in health-related international journals. The KIHD is a population-based longitudinal follow-up study, which initially involved nearly 3,000 middle-aged (42–60 years) men from the Kuopio region in Eastern Finland. Ten years later, women of same age ($N > 1,000$) were recruited to the study. A major part of the original cohorts have been reexamined 4, 11, and 20 years after the baseline. Register linkages have been established with national covering registries, i.e., the National Death Registry of Finland, Hospital Discharge Registry, Cancer Registry, and other disease-specific national health registries in Finland, to monitor annual events of early deaths as well as new cases and treatment episodes due to various diseases. Despite its name, the KIHD has not been restricted only to cardiovascular outcomes. Some of the major research lines have been within the domains of chronic disease epidemiology, nutritional epidemiology, alcohol epidemiology, psychosocial epidemiology, as well as molecular and genetic epidemiology. Being a multisdisciplinary project with extensive data collection, the health determinants examined within the KIHD study have involved a wide range of factors from genes to health behavior and environmental risks and from biochemical markers to psychological, psychosocial, and socioeconomic factors. It has been possible to construct some historical cohort designs, to take a lifecourse look at possible early childhood determinants on later health. Some of the key researchers in the project over the years have included authors such as Salonen JT, Kauhanen J, Tuomainen TP, Kurl S, Laukkanen J, Virtanen J (all from Finland), Kaplan G, Krause N (from the USA), and Lynch JW (Australia). The KIHD study is estimated to yield interesting scientific findings well into the 2020s.

Cross-References

▶ Affect
▶ Aging
▶ Alcohol Consumption
▶ Antioxidant
▶ Atherosclerosis
▶ Binge Drinking
▶ Biomarkers
▶ Blood Pressure Reactivity or Responses
▶ Cardiac Death
▶ Chronic Disease or Illness
▶ Cohort Study
▶ Elderly

- Epidemiology
- Exercise Testing
- General Population
- Genetics
- Health Behaviors
- Health Risk
- Homocysteine
- Hopelessness
- Hypertension
- Ischemic Heart Disease
- Job Demand/Control/Strain
- Lifestyle
- Lipid Abnormalities
- Metabolic Syndrome
- Mortality
- Physical Activity and Health
- Population-Based Study
- Psychosocial Factors
- Public Health
- Smoking and Health
- Social Epidemiology
- Sociocultural
- Socioeconomic Status (SES)

References and Readings

IL6R Genetics Consortium Emerging Risk Factors Collaboration (2012). Interleukin-6 receptor pathways in coronary heart disease: a collaborative meta-analysis of 82 studies. *Lancet*, *379*, 1205–1213.

Kauhanen, J., Kaplan, G. A., Goldberg, D. E., Salonen, J. T. (1997). Beer binging and mortality: results from the Kuopio ischaemic heart disease risk factor study, a prospective population based study. *BMJ*, *315*, 846–851.

Kauhanen, L., Lynch, J. W., Lakka, H. M., Kauhanen, J., Smith, G. D. (2010). Association of diarrhoea, poor hygiene and poor social conditions in childhood with blood pressure in adulthood. *J Epidemiol Community Health*, *64*, 394–399.

Laukkanen, J. A., Mäkikallio, T. H., Rauramaa, R., Kiviniemi, V., Ronkainen, K., Kurl, S. (2010). Cardiorespiratory fitness is related to the risk of sudden cardiac death: a population-based follow-up study. *J Am Coll Cardiol*, *56*, 1476–1483.

Lynch, J. W., Kaplan, G. A., Cohen, R. D., Kauhanen, J., Wilson, T. W., Smith, N. L., Salonen, J. T. (1994). Childhood and adult socioeconomic status as predictors of mortality in Finland. *Lancet*, *343*, 524–527.

Salonen, J. T. (1988). Is there a continuing need for longitudinal epidemiologic research? The Kuopio Ischaemic Heart Disease Risk Factor Study. *Annals of Clinical Research, 20*(1–2), 46–50.

L

Laboratory Stress Protocol

► Trier Social Stress Test

Latent Variable

Jamil A. Malik
National Institute of Psychology, Quaid-i-Azam University/VU University Amsterdam, Islamabad, Pakistan

Synonyms

Hidden variable; Hypothetical construct; Hypothetical variable

Definition

Several of the concepts of social sciences extracted from various theories cannot be observed directly, although these concepts might be of interest to social scientists and researchers. For example:

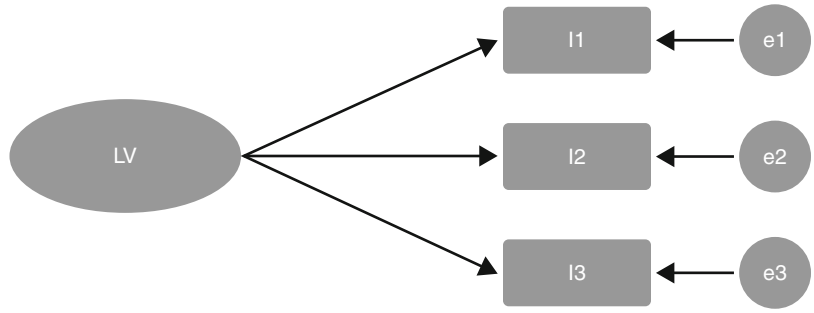
- In health: treatment adherence/compliance and well-being
- In psychology: stress, self-worth, and personality dimensions
- In education: memory, spatial ability, and problem solving
- In sociology: optimism, hostility, socioeconomic status, and attitudes
- In political science: leadership, conservatism, fundamentalism, political competence, etc.

In such cases where a concept is unobservable directly yet researchers are interested in their empirical testing, researchers have to rely on assumptions. In other words, they implicitly or explicitly operationalize these concepts into formal measurement models (constructs). These constructs can then only be measured indirectly via observable indicators/behaviors. Scaling (i.e., collection of relevant information on a questionnaire) is the most common technique to collect data for an unobservable construct. What is relevant is based on the assumptions of the researcher and derived from operational definition of the construct.

Various types of scaling techniques have been developed for deriving information on unobservable constructs of interest from observable indicators. An important family of scaling methods is formed by latent variables. A latent variable model is a possibly nonlinear, path analysis or graphical model. In addition to the manifest variables, the model includes one or more unobserved or latent variables representing the constructs of interest. Latent variable models are developed on two basic assumptions. The first assumption is that the responses on the indicators are the result of an individual's position on the latent variable(s). Secondly, it is assumed that

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

Latent Variable,
Fig. 1 Latent variable



the manifest variables have nothing in common after controlling for the latent variable(s), which is often referred to as the axiom of local independence.

Figure 1 shows a latent variable model, i.e., an unobservable variable LV represented by an oval which is assumed to be the result of commonality measured by three manifest indicators I1, I2, and I3. The ovals e1, e2, and e3 are residual also known as error variances. The concept of latent variables is based on the classical test theory, which assumes that any measure is a function of two variables: (1) the true score and (2) the error variation. This assertion can be written as a formula: $(X = T + E)$ where X represents the observed score on the measure, T is the person's true score, and E is the error variation.

Historically, the idea of latent variables arose primarily from psychometrics, beginning with the g factor of Spearman (1904). Spearman developed factor analytic models for continuous variables in the context of intelligence testing. The tradition continued with other psychologists such as Thomson, Thurstone, and Burt, who were investigating the mental ability of children, as suggested by the correlation and covariance matrices from cognitive tests variables. The technique was further developed with the work of Lawley (1943), Thurstone (1947), and Lawley and Maxwell (1963), and finally, it entered into the conceptual framework of confirmatory factor analysis (CFA) with Jöreskog (1971); Wiley, Schmidt, and Bramble (1973); and Sörbom (1974). In subsequent years, CFA became a very popular technique, largely because of the LISREL program by Jöreskog and Sorbom (1993). At present, there are various developments that emphasize this common framework for latent variables analysis, cases in point being the work of Muthén and Muthén (1998), McDonald (1999), and Moustaki and Knott (2000).

Cross-References

▶ Structural Equation Modeling (SEM)

References and Readings

Bartholomew, D. J., & Knott, M. (1999). *Latent variable models and factor analysis*. London: Arnold.
Borsboom, D., Mellenbergh, G. J., & van Heerden, J. (2003). The theoretical status of latent variables. *Psychological Review*, *110*(2), 203–219.
Jöreskog, K. G. (1971). Statistical analysis of sets of congeneric tests. *Psychometrika*, *36*, 109–133.
Jöreskog, K. G., & Sörbom, D. (1993). *LISREL 8 user's reference guide*. Chicago: Scientific Software International.
Lawley, D. N., & Maxwell, A. E. (1963). *Factor analysis as a statistical method*. London: Butterworth.
Marcoulides, G., & Moustaki, I. (2002). *Latent variable and latent structure models*. London: Lawrence Earlbaum.
McDonald, R. P. (1999). *Test theory: A unified treatment*. Mahwah, NJ: Erlbaum.
Moustaki, I., & Knott, M. (2000). Generalized latent trait models. *Psychometrika*, *65*, 391–411.
Muthén, L. K., & Muthén, B. O. (1998). *Mplus user's guide*. Los Angeles: Muthén & Muthén.
Sörbom, D. (1974). A general method for studying differences in factor means and factor structures between groups. *Psychometrika*, *55*, 229–239.
Spearman, C. (1904). General intelligence, objectively determined and measured. *American Journal of Psychology*, *15*, 201–293.

Thurstone, L. L. (1947). *Multiple factor analysis*. Chicago: University of Chicago Press.
Wiley, D. E., Schmidt, W. H., & Bramble, W. J. (1973). Studies of a class of covariance structure models. *Journal of the American Statistical Association*, *86*, 317–321.

Lateral Mammillary Nucleus

▶ Hypothalamus

Lateral Nucleus

▶ Hypothalamus

Lateral Preoptic Area

▶ Hypothalamus

Latino Health

▶ Hispanic/Latino Health

Lay Health Advisors

▶ Promotoras

Lay Health Advocates

▶ Promotoras

Lazarus Theory

▶ Cognitive Appraisal

Learned Helplessness

Theresa A. Morgan
Alpert Medical School of Brown University, Department of Psychiatry, Brown University, Providence, RI, USA

Synonyms

Locus of control

Definition

Literally, learning to be helpless; a set of motivations, perceptions, and behaviors that conclude that one's behavior does not effect any control over one's surroundings (i.e., that no matter what one does, things will not change). The effect is most notable under aversive conditions where change is easily attained but is not sought due to learned helplessness.

Description

Learned helplessness refers to the condition of an organism (human or animal) wherein behavior is consistent with the perception that actions do not impact outcomes. In humans, this typically manifests as a view of the world as uncontrollable, unpredictable, and insensitive to individual needs or acts. Behaviorally, one might fail to be proactive even when opportunities to effect change exist (Walker, 2001).

The term "learned helplessness" originated with a set of animal studies from the 1960s and 1970s (e.g., Seligman & Maier, 1967; Overmier & Seligman, 1967). In these studies, one group (1) of dogs was given repeated, inescapable shocks while restrained. Later, these same animals appeared to passively accept shocks without seeking escape, even in cases where escape options were readily available (e.g., shock could be escaped by jumping over a small barrier or pushing a button or lever to turn off the shock). The

authors hypothesized that the animals were experiencing "learned helplessness" because previous experience taught them that behavior and the shock were independent; that is, they had learned to behave helplessly in the face of aversive stimuli. Later studies compared animals from this first group to (2) animals that experienced no previous shocks, (3) animals that experienced previous shocks as controllable (yoked to the learned helplessness group), and (4) animals that first received training in escaping shocks but later were given inescapable, uncontrollable shocks. All three later groups (2, 3, 4) learned to avoid shocks, where the first group (1) did not. As such, the critical component to developing learned helplessness is whether an organism first experiences the environment as controllable (see Peterson, Meier, & Seligman, 1995; Walker, 2001).

Since these original studies, learned helplessness has been applied to a wide variety of psychological phenomena, including acceptance of victimization (and abuse), pessimism, psychological well-being, low achievement at work or school, sudden death or illness, and health behaviors such as diet, exercise, and treatment adherence. Much research indicates that learned helplessness becomes a psychological deficit only when it manifests consistently, across multiple situations (Sahoo, 2002). As such, learned helplessness may describe behavior in a discrete situation (e.g., as with the shocks previously described), but it is most linked to problematic life outcomes when it becomes a trait-like, generalized set of perceptions and behaviors.

In humans, learned helplessness is most commonly studied in the context of depression. The "learned helplessness theory" specifically posits that depression occurs after experiencing the world as uncontrollable and then behaving consistent with the perception of helplessness. In this case, it is not actual controllability that is important so much as *perceived* controllability. In depression, learned helplessness theory also includes the perception of future uncontrollability: essentially that bad things happen now and in the future and that nothing one does can have any effect on their occurrence (see Peterson & Seligman, 1985). This hypothesis explains many of the more difficult to treat features of depression, including failure to approach previously enjoyable activities, failure to regulate eating and sleep, and similar self-defeating behaviors. Although research does link learned helplessness and depression, the direction of causality has yet to be firmly established. An alternative hypothesis suggests that a third variable, such as an external locus of control, causes both learned helplessness and depression to manifest (see Walker, 2001).

Cross-References

- ▶ Depression: Measurement
- ▶ Depression: Symptoms
- ▶ Depression: Treatment

References and Readings

Overmier, J. B., & Seligman, M. E. P. (1967). Effects of inescapable shock upon subsequent escape and avoidance responding. *Journal of Comparative and Physiological Psychology, 63*, 28–33.

Peterson, C., Meier, S. F., & Seligman, M. E. P. (1995). *Learned helplessness: A theory for the age of personal control*. New York: Oxford University Press.

Peterson, C., & Seligman, M. E. P. (1984). Causal explanations as a risk factor for depression: Theory and evidence. *Psychological Review, 91*, 347–374.

Peterson, C., & Seligman, M. E. P. (1985). The learned helplessness model of depression: Current status of theory and research. In E. E. Beckman & W. R. Leber (Eds.), *Handbook of depression: Treatment, assessment and research* (pp. 914–939). Homewood, IL: Dorsey.

Sahoo, F. M. (2002). *Dynamics of human helplessness*. New Delhi, India: Concept Publishing.

Seligman, M. E. P., & Maier, S. F. (1967). Failure to escape traumatic shock. *Journal of Experimental Psychology, 74*, 1–9.

Walker, J. (2001). *Control and the psychology of health: Theory, measurement and applications*. Philadelphia: Open University Press.

Learned Symptom Behavior

▶ Symptom Magnification Syndrome

Leisure Physical Activity

► Exercise

Leptin

Falk Kiefer[1] and Mustafa al'Absi[2]
[1]Department of Addictive Behavior and Addiction Medicine, Central Institute of Mental Health, Mannheim, Baden-Württemberg, Germany
[2]University of Minnesota Medical School, University of Minnesota, 235 School of Medicine, Duluth, MN, USA

Synonyms

Appetite; Hormones; Peptide

Definition

Leptin (Greek leptos: thin) is a protein hormone released mainly by white adipocytes. It binds at leptin receptors in brain neurons involved in regulating energy intake and expenditure, mainly in the lateral hypothalamus, where the activity of neurons containing neuropeptide Y (NPY) and agouti-related peptide (AgRP) is inhibited, while the activity of neurons expressing α-melanocyte-stimulating hormone (α-MSH) is increased. The effects of leptin were observed by studying mutant obese mice (*ob* mice) that arose at random at the Jackson Laboratory in 1950. The human *ob* gene is located on chromosome 7 encoding a protein of 167 amino acids.

Even though leptin is also synthesized by other tissues including the placenta, ovaries, skeletal muscle, stomach, mammary epithelial cells, bone marrow, pituitary, and liver, circulating leptin concentration is highly associated with the total amount of body fat and positively correlated with BMI. Despite the fact that leptin is reducing appetite in subjects of normal body weight, it was found to occur in obese people with unusually high concentration, thus indicating a possible leptin resistance. The leptin deficiency here may lead to increased food intake and body weight.

Experiments in animals have shown that leptin levels in circulation provide indication of both energy stores and energy balance. Research has also shown that food restriction is associated with suppression of circulating leptin, and that increased food intake increases leptin levels. Administering leptin was also found to reduce food intake resulting in loss of body weight. Leptin crosses the blood brain barrier, and administering it peripherally alters neuronal activity in several hypothalamic and brain stem regions leading to multiple changes that result in reduced appetite.

In addition to its hypothalamic target, recent results suggest that leptin also affects mesolimbic pathways and might provide a link between energy homeostasis and reward-related behavior. Leptin receptors were detected on dopaminergic neurons in the ventral tegmental area (VTA) inhibiting dopamine signaling in the nucleus accumbens (NAc). Based on these findings, there is evidence that elevated leptin is involved in processing of reward-predicting signals such as seeing, smelling, or tasting food. Important insights into the regulatory role of leptin on central processing of food-related stimuli have been gained by studies in patients with rare genetic defects leading to a leptin deficiency syndrome (LDS). In LDS patients, visual food cues were associated with increased striatal activation; moreover, this effect was neutralized by intravenous application of recombinant leptin. A second study reported marked CNS-activation in LDS patients following visual food stimuli.

Recent studies have demonstrated an important role of leptin in addictive behavior. For example, research has found positive correlations of plasma leptin and alcohol craving in alcohol-dependent subjects, as well as showed a positive correlation of plasma leptin and nicotine craving in nicotine-dependent subjects. It is assumed that leptin mediates a reduction of the basal dopaminergic activity in the mesolimbic system, contributing to a hypodopaminergic functioning associated with reward deficiency. These results suggest that addictive substances as well as food

may hold a greater reward value for subjects showing higher leptin plasma levels.

Cross-References

- ▶ Addiction
- ▶ Appetite
- ▶ Hormones

References and Readings

Farooqi, S., & O'Rahilly, S. (2007). Genetics of obesity in humans. *Endocrine Reviews, 27*(7), 710–718.

Friedman, J. M., & Halaas, J. L. (1998). Leptin and the regulation of body weight in mammals. *Nature, 395*(6704), 763–770.

Margetic, S., Gazzola, C., Pegg, G. G., & Hill, R. A. (2002). Leptin: A review of its peripheral actions and interactions. *International Journal of Obesity and Related Metabolic Disorders, 26*, 1407–1433.

Level of Occupational Performance

- ▶ Activities of Daily Living (ADL)

Levels of Prevention

- ▶ Prevention: Primary, Secondary, Tertiary

Lexapro®

- ▶ Selective Serotonin Reuptake Inhibitors (SSRIs)

Life Course

- ▶ Life Span

Life Cycle

- ▶ Life Span

Life Events

Anna C. Phillips
Sport & Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Stressful events

Definition

Life events are specific occurrences experienced by an individual that are perceived as stressful, e.g., bereavement, divorce, and moving house. Life events are not always negative events but can be something positive which is perceived as stressful due to the amount of change it brings into the individual's life, e.g., getting married and changing job.

Life event measures are often used to identify relationships between stress and health within behavioral medicine research. Life events are usually assessed by standardized interview or questionnaire. Such questionnaires ask the individual to indicate which events have happened to them over a specific time period such as 1 year or one month. Life events are usually limited to several key domains such as health, marriage, relationships, bereavement, work, housing, and finance. Some life event measures are specific to certain populations or age groups such as the Life Events Scale for Students (Linden, 1984). These measures often ask individuals to rate how stressful each event that occurred was on a rating scale. This method is based on the well-established Life Events and Difficulties Schedule (Brown & Harris, 1989). Other questionnaires use pre-identified weightings of the severity of events to determine an overall life events burden score, such as the Undergraduate Stress Questionnaire (Crandall, Preisler, & Aussprung, 1992) in which more severe events such as bereavement are given higher weightings.

Cross-References

► Daily Stress

References and Readings

Brown, G. W., & Harris, T. O. (1989). *Social origins of depression: A study of psychiatric disorder in women*. London: Routledge.

Crandall, C. S., Preisler, J. J., & Aussprung, J. (1992). Measuring life event stress in the lives of college students: The undergraduate stress questionnaire (USQ). *Journal of Behavioral Medicine, 15*, 627–662.

Linden, W. (1984). Development and initial validation of a life event scale for students. *Canadian Counsellor, 18*, 106–110.

Life Expectancy

► Longevity

Life Orientation Test (LOT)

► Optimism and Pessimism: Measurement

Life Skills

► Occupational Therapy

Life Span

Patricia Cristine Heyn[1] and Katherine S. Hall[2]
[1]Department of Physical Medicine and Rehabilitation, University of Colorado Denver Anschutz Medical Campus School of Medicine, Aurora, CO, USA
[2]Durham VA Medical Center Geriatric Research, Education, and Clinical Center, Durham, NC, USA

Synonyms

Life course; Life cycle; Life time

Definition

In its most general sense, life span refers to the length of time between birth and death. Importantly, life span is distinct from life expectancy, which reflects the average number of years that an individual might be expected to live (often reflective of a biological limit on life). Developmental behavioral theories posit that certain factors impact behavior and that these factors, and thus individual behavior, change as the individual ages. Such theories of behavior specify that what happens earlier in life affects what happens later. As such, incorporating a life span approach in behavioral medicine implies the implementation of longitudinal studies that begin early on in the developmental stages (i.e., childhood) and continue into late adulthood. As the cost and feasibility of such life span studies can be prohibitive, many behavioral researchers opt instead to conduct cross-sectional studies of various age cohorts representative of the full age spectrum. Although informative, such study designs do not examine the *process* of aging, and as such, they cannot provide a causal explanation of how the processes of aging impact behavioral outcomes and vice versa. Finally, life span differs from health span (Rowe & Kahn, 1987, 1998), which reflects the length of time prior to the onset of disease or disability. Health span has been adopted in the behavioral medicine literature as an indicator of healthy aging.

References and Readings

Baltes, P. B., Reese, H. W., & Lipsitt, L. P. (1980). Life-span developmental psychology. *Annual Review Psychology, 31*, 65–110.

Rowe, J. W., & Kahn, R. L. (1987). Human aging: Usual and successful. *Science, 237*, 143–149.

Rowe, J. W., & Kahn, R. L. (1998). *Successful aging*. New York: Pantheon/Random House.

Staudinger, U. M., Marsiske, M., & Baltes, P. B. (1995). Resilience and reserve capacity in later adulthood: Potentials and limits of development across the life span. In *Developmental psychology* (Risk, disorder, and adaptation, Vol. 2, pp. 801–847). New York: Wiley.

Life Time

► Life Span

Life Years Lost

Anthony J. Wheeler and Scott DeBerard
Department of Psychology, Utah State University, Logan, UT, USA

Synonyms

Potential years of life lost (PYLL); Years of potential life lost (YPLL)

Definition

Life years lost (LYL) represents the number of years not lived to full potential due to disease or other fatal events. The metric LYL is calculated by subtracting the actual number of years lived from an expected duration, usually drawn from a standard life table. For example, if an individual died from breast cancer at age 49, and was expected to live to age 75, they would have lost 26 life years. The specific definition and calculation of LYL vary, with some researchers weighting quantities of LYL differently to adjust for social and economic losses.

References and Readings

Haomiao, J., & Lubetkin, E. I. (2010). Trends in quality-adjusted life-years lost contributed by smoking and obesity. *American Journal of Preventive Medicine, 38*(2), 138–144.

Mathers, C., Ma Fat, D., Boerma, J. T., & World Health Organization. (2008). *The global burden of disease: 2004 update*. Geneva: World Health Organization.

Muennig, P. (2008). *Cost-effectiveness analyses in health: A practical approach*. San Francisco: Wiley.

Steenland, K., & Armstrong, B. (2006). An overview of methods for calculating the burden of disease due to specific risk factors. *Epidemiology, 17*(5), 512–519.

Lifestyle

Jordan Carlson
Public Health, San Diego State University, University of California San Diego, San Diego, CA, USA

Synonyms

Health behaviors

Definition

The term lifestyle does not have a well-accepted definition but can be understood to be the health behaviors in which a person engages on a daily basis.

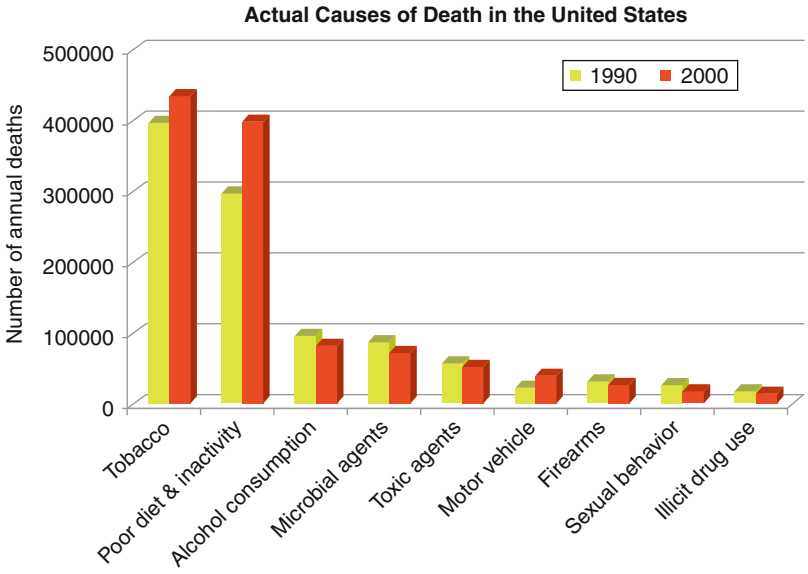
Description

Lifestyle Versus Nonlifestyle Health Behaviors

Health behaviors are actions taken by a person to maintain, attain, or regain good health and to prevent illness. Lifestyle health behaviors are different from other health behaviors in that they are engaged on a daily basis. For example, smoking is a lifestyle health behavior because it typically occurs on a daily basis. Medical care, such as medical appointments or cancer screenings, do not occur on a daily basis and thus can be considered nonlifestyle health behaviors.

Commonly Studied Lifestyle Behaviors

There are many lifestyle behaviors that are related to health outcomes and commonly studied in behavioral medicine (Glanz, Rimer,



Lifestyle, Fig. 1 Actual causes of death in the United States (Data from Mokdad et al., 2004)

Viswanath, & Orleans, 2010). Following are some examples:

- Dental hygiene
- Diet/nutrition
- Driving safety/seat belt use
- Drug use
- Medication adherence
- Physical activity
- Safe sex practices
- Sedentary behavior
- Smoking
- Social behaviors
- Stress management/relaxation
- Sun exposure/protection

Lifestyle Behaviors and Chronic Diseases

The leading causes of death in the USA drastically shifted in the twentieth century. At the beginning of the century, the leading causes of death were pneumonia, influenza, and tuberculosis (Armstrong, Conn, & Pinner, 1999). By 2000, the leading cause of death was heart disease (710, 760 deaths), followed by cancer (553, 091 deaths) and stroke (167, 661 deaths). The "actual causes of death" were estimated based on the behavioral causes of these diseases (Mokdad, Marks, Stroup, & Gerberding, 2004). The leading actual causes of death in 2000 were tobacco use (435,000 deaths; 18.1% of all deaths) and poor diet and physical inactivity (400,000 deaths; 16.6% of all deaths) (Fig. 1). These death rates were drastically higher than those from all other actual causes of death and were much higher in 2000 than in 1990. These actual causes of death are considered preventable and are often the focus of lifestyle behavior interventions.

Cross-References

- ▶ Behavior
- ▶ Health Behavior Change
- ▶ Lifestyle Changes
- ▶ Lifestyle, Modification

References and Readings

Armstrong, G. L., Conn, L. A., & Pinner, R. W. (1999). Trends in infectious disease mortality in the United States during the 20th century. *Journal of the American Medical Association, 281*(1), 61.

Glanz, K., Rimer, B. K., Viswanath, K., & Orleans, C. T. (2010). *Health behavior and health education: Theory, research, and practice*. San Francisco: Josey-Bass.

Mokdad, A. H., Marks, J. S., Stroup, D. F., & Gerberding, J. L. (2004). Actual causes of death in the United States, 2000. *Journal of the American Medical Association, 291*(10), 1238.

Lifestyle Changes

Spencer M. Richard and Scott DeBerard
Department of Psychology, Utah State University, Logan, UT, USA

Synonyms

Alcohol abuse; Management; Obesity treatment; Smoking; Smoking cessation; Substance abuse; Weight; Weight loss

Definition

Lifestyle changes are defined as changes that alter various lifestyle-related behaviors such as diet, physical activity, sexual behavior, smoking, alcohol consumption, substance use, and other behaviors not otherwise defined as psychopathology. Lifestyle changes may often relate to other comorbid mental and/or physical health conditions such as substance abuse disorders, obesity, asthma, sexually transmitted diseases, depression, and anxiety.

Description

Diet/Physical Activity

Obesity and overweight account for a substantial portion of medical care costs in the United States. Current research suggests that approximately two thirds of the population in the United States qualifies as overweight (body mass index between 25.1 and 29.9) or obese (body mass index > 30; World Health Organization, 2010).These rates are often attributed to lifestyle factors including unbalanced diet and lack of physical exercise/activity. Little research has been done to effectively measure the direct health-care cost benefits of modifying these lifestyle factors. However, one recent projection estimated that by altering diet alone (i.e., decreasing caloric intake by 100 kcal/day, saturated fat by 5 g/day, and sodium by 400 mg/day), national health-care costs could potentially be cut by $60 billion to $120 billion per year (Dall et al., 2010).

Interventions on diet and physical activity have traditionally focused on cognitive behavioral approaches intended to produce weight loss and increased physical activity. Overweight and obese individuals experience increased risk of several mental and physical health problems including cardiovascular disease, depression symptoms, sleep problems, diabetes, and others (Centers for Disease Control & Prevention, 2010). In general, empirical research has shown a trend for 5–10% weight loss during the course of treatment, followed by subsequent relapse and weight gain by at least half of participants at or beyond preintervention weight (Wadden & Butryn, 2003). Altering diet and exercise has been shown to significantly reduce weight and lower risk for weight-related illnesses and cardiometabolic disease (Goodpaster, et al., 2010). Recent trials indicate increased efficacy of behaviorally based interventions with values and acceptance components (Forman, Butryn, Hoffman, & Herbert, 2009; Lillis, Hayes, Buntin, & Masuda, 2009). These approaches have been shown to lead to better maintained weight loss and physical activity measurements as well as increased mental health outcomes. Additional evidence suggests that use of internal and externally based incentives may increase adherence to lifestyle changes in diet and exercise.

Smoking

Cigarette smoking has been shown as a significant risk factor for myriad physical health problems including coronary heart disease, stroke, cancers, chronic obstructive lung disease (COPD), and death (Centers for Disease Control & Prevention, 2011). Smoking directly or indirectly causes approximately 440,000 deaths annually in the United States, including 80% of lung cancer deaths in women and 90% of lung cancer deaths in men. In addition, smoking has been implicated in reproductive health problems including infertility, preterm birth, low birth weight, and increased risk of sudden infant death syndrome. Smoking cessation has been linked with a number of positive health outcomes. Individuals that quit smoking before middle age may

significantly reduce risk of developing lung cancer (Peto et al., 2000).

In general, smoking cessation at younger ages increases health benefits exponentially. Smoking cessation treatment is often brief (as little as one targeted session) and can be delivered in many environments including primary care, outpatient counseling, and in home by a number of professional and paraprofessional primary care and/or mental health workers including nurses, doctors, counselors, and psychologists. Treatment methods focusing on motivation enhancement, behavioral commitment, acceptance, and mindfulness have been indicated as recommended treatment modalities by the National Registry of Evidence-based Programs and Practices (NREPP). In addition, smoking cessation may lead to other related lifestyle improvements including decreased long-term substance use and may moderately increase quality of life (Sarna, Bialous, Cooley, Jun, & Feskanich, 2008).

Alcohol/Substance Use

Alcohol and other substance use represent a considerable health risk and account for a substantial health-care and legal system resources. The National Institute on Drug Abuse (NIDA) estimates annual costs of nearly $600 billion in the United States alone including costs of prevention, treatment, legal costs, and lost worker productivity (National Institute on Drug Abuse, 2009). The abuse of alcohol and other drugs are widely varying depending on the nature and frequency of use and type of substance being used. In general, substance use has been linked to several physical and mental health problems including cardiovascular disease, COPD, birth defects, paranoia, psychotic symptoms, depression, and anxiety, among many more.

Alcohol and other drug use are treated in both inpatient and outpatient medical and mental health settings. Similar to smoking cessation treatment, significant health benefits are observed by reducing or eliminating alcohol or other substance use. Depending on the extent of use and type of substance being used, treatment may include any combination of the following aspects recommended by NIDA and NREPP: medications (including antagonists or substitutes for the substance of abuse), individual counseling, group counseling, detoxification, supervision, ongoing case management, and treatment of other comorbid medical and mental health conditions. Effective psychological treatments tend to include multidimensional approaches including cognitive-behavioral intervention, family therapy, motivation enhancement, acceptance, mindfulness, behavior commitments, and contingency management.

Summary

Lifestyle changes can have significant and long-lasting physical and mental health effects. Treating problematic lifestyle behaviors can be a brief, cost-effective, and meaningful strategy by a variety of health-care service providers to improve physical and mental health of their patients and clients.

Cross-References

- Activity Level
- Addictive Behaviors
- Aerobic Exercise
- Alcohol Abuse and Dependence
- Alcohol Consumption
- Behavior
- Behavior Change
- Behavior Change Techniques
- Behavior Modification
- Behavioral Intervention
- Behavioral Therapy
- Body Mass Index
- Caloric Intake
- Cancer and Diet
- Cancer and Physical Activity
- Cancer and Smoking
- Cancer: Psychosocial Treatment
- Cardiovascular Disease Prevention
- Cessation Intervention (Smoking or Tobacco)
- Cognitive Behavioral Therapy (CBT)
- Dependence, Drug
- Disease Management
- Education, Patient
- Exercise-General Category

- Health Behavior Change
- Health Behaviors
- Heart Disease and Smoking
- Heart Disease and Type A Behavior
- HIV Prevention
- Lifestyle, Modification
- Lifestyle, Active
- Lifestyle, Healthy
- Mindfulness
- Motivational Interviewing
- Obesity in Children
- Obesity: Causes and Consequences
- Physical Activity and Health
- Physical Activity Interventions
- Physical Activity, Psychosocial Aspects, Benefits
- Physical Health
- Physical Inactivity
- Preventive Care
- Relapse, Relapse Prevention
- Risky Behavior
- Self-Care
- Smoking Cessation
- Tobacco Cessation
- Tobacco Use
- Weight: Control, Gain/Loss/Reduction, Maintenance, Monitoring

References and Readings

Centers for Disease Control and Prevention. (2010). *Overweight and obesity*. Retrieved on October 30, 2011, from http://www.cdc.gov/obesity/

Centers for Disease Control and Prevention. (2011). *Health effects of cigarette smoking*. Retrieved on October 31, 2011, from http://www.cdc.gov/tobacco/data_statistics/fact_sheets/health_effects/effects_cig_smoking/

Dall, T. M., Zhang, Y., Chen, Y. J., Quick, W. V., Yang, W. G., & Fogli, J. (2010). The economic burden of diabetes. *Health Affairs, 29*(2), 297–303.

Forman, E. M., Butryn, M. L., Hoffman, K. L., & Herbert, J. D. (2009). An open trial f an acceptance-based behavioral intervention for weight loss. *Cognitive and Behavioral Practice, 16*, 223–235.

Goodpaster, B. H., DeLany, J. P., Otto, A. D., Kuller, L., Vockley, J., South-Paul, J. E., et al. (2010). Effects of diet and physical activity interventions on weight loss and cardiometabolic risk factors in severely obese individuals: A randomized trial. *Journal of the American Medical Association, 304*(16), 1795–1802.

Lillis, J., Hayes, S., Bunting, K., & Masuda, A. (2009). Teaching acceptance and mindfulness to improve the lives of the obese: A preliminary test of a theoretical model. *Annals of Behavioral Medicine, 37*(1), 58–69.

National Institute on Drug Abuse (2009). Treatment approaches for drug addiction. *InfoFacts*. Retrieved on October 31, 2011, from http://nida.nih.gov/PDF/InfoFacts/IF_Treatment_Approaches_2009_to_NIDA_92209.pdf

Peto, R., Darby, S., Deo, H., Silcocks, P., Whitley, E., & Doll, R. (2000). Smoking, smoking cessation, and lung cancer in the UK since 1950: Combination of national statistics with two case–control studies. *British Medical Journal, 321*(7257), 323–329.

Sarna, L., Bialous, S. A., Cooley, M. E., Jun, H., & Feskanich, D. (2008). Impact of smoking and smoking cessation on health-related quality of life in women in the Nurses' Health Study. *Quality of Life Research: An International Journal of Quality of Life Aspects of Treatment, Care & Rehabilitation, 17*(10), 1217–1227.

Wadden, T. A., & Butryn, M. L. (2003). Behavioral treatment of obesity. *Endocrinology Metabolism Clinics of North America, 32*(4), 981–1003.

World Health Organization. (2010). *Factsheet on obesity and overweight*. Retrieved October 30, 2011, from http://www.who.int/mediacentre/factsheets/fs311/en/index.html

Lifestyle, Active

Victoria Anne Sublette
School of Public Health, University of Sydney, Sydney, NSW, Australia

Synonyms

Active way of life; Healthy lifestyle

Definition

An active lifestyle is one in which individuals incorporate regular and substantial physical movement into their daily routines to benefit their cardiovascular, muscular, and psychological health.

Description

Engagement in an active lifestyle includes regular physical activity which protects physical and mental

health by reducing individuals' risk of developing diseases and depression and is associated with a significant reduction in premature mortality rates from all causes for men and women (Keim, Blanton, & Kretsch, 2004; Morrison, Bennett, Butow, Mullan, & White, 2008).

Low-impact exercise and weight-bearing exercise such as walking and dancing promotes bone development in the young and preserves bone density during adulthood. Achieving peak bone mass protects individuals against the development of osteoporosis, a disease characterized by a reduction in bone density due to calcium loss, leading to brittle bones and an increased risk of fractures (Department of Health and Ageing, 2010).

Physical Benefits of an Active Lifestyle

- Helps build and maintain healthy bones, muscles and joints
- Builds endurance and muscular strength
- Protects against brain damage caused by stroke
- Lowers risk factors for cardiovascular disease, colon cancer, and type 2 diabetes
- Improves balance, strength, suppleness and mobility
- Instrumental in reaching and maintaining a healthy weight
- Helps control blood pressure
- Increases energy
- Reduces muscle tension
- Improves sleep
- Improves body shape

Psychological Benefits of an Active Lifestyle

- Reduces stress
- Improves mood and self-esteem
- Reduces symptoms of depression and anxiety
- Improves cognitive function
- Improves quality of life

(Centers for Disease Control and Prevention, 2010; Keim et al., 2004; Van Praag, 2009).

An active lifestyle also has profound benefits for brain function. Physical activity improves learning, memory, and cognitive processes such as planning, scheduling, and coordination. An active lifestyle also stimulates the adult and aging brain to generate new neurons preventing cognitive decline in older age and protects the brain against damage caused by stroke, promotes recovery after injury, and is an antidepressant (Van Praag, 2009).

Moderate and vigorous-intensity aerobic or resistance training exercise can reduce symptoms of depression. Research has found inactive persons are approximately twice as likely to have symptoms of mild to moderate depression than individuals who are more active. The antidepressant effect of physical exercise has been shown to be similar in magnitude to antidepressant medications in some studies (Van Praag, 2009).

Guidelines for an Active Lifestyle

For those 5–18 years old, 60 min a day of moderate to vigorous physical activity combined is recommended. Children and adolescents should include muscle-strengthening and bone-strengthening exercises as part of their 60 min a day at least 3 days a week. Examples of moderate activities are a brisk walk, a bike ride, or active play. Examples of vigorous exercise are running, swimming laps, organized sports, and ballet (Centers for Disease Control and Prevention, 2010; Department of Health and Ageing, 2010; U.S. Department of Health & Human Services, 2008).

Recommendations for adults age 18 years and over are at least 150 min (2 h and 30 min) a week of moderate-intensity, or 75 min (1 h and 15 min) a week of vigorous-intensity aerobic physical activity. Vigorous-intensity aerobic exercise moves large muscles in a rhythmic manner for a sustained period of time and includes brisk walking, jogging, biking, dancing, and swimming. Aerobic exercise should be performed in increments of at least 10 min each. Adults should also perform muscle-strengthening activities that involve all major muscle groups at moderate to high intensity two or more days a week.

For optimum health, an active lifestyle that incorporates greater physical health benefits and weight control can be obtained by engaging in physical activity of more vigorous intensity or of longer duration (Centers for Disease Control and Prevention, 2010; Department of Health and Ageing, 2010; U.S. Department of Health & Human Services, 2008).

Cross-References

- ▶ Activity Level
- ▶ Health Behaviors
- ▶ Lifestyle, Healthy

References and Readings

Centers for Disease Control and Prevention. (2010). Physical activity and health. *Physical activity for everyone*. Retrieved November 25, 2010, from http://www.cdc.gov/physicalactivity/everyone/health/index.html

Department of Health and Ageing. (2010). Physical activity guidelines. *Physical activity*. Retrieved November 20, 2010, from http://www.health.gov.au/internet/main/publishing.nsf/Content/health-pubhlth-strateg-phys-act-guidelines#rec_12_18

Keim, N., Blanton, C., & Kretsch, M. J. (2004). America's obesity epidemic: Measuring physical activity to promote an active lifestyle. *Journal of the American Dietetic Association, 104*(9), 1398–1409.

Morrison, V., Bennett, P., Butow, P., Mullan, B., & White, K. (2008). *Introduction to health psychology in Australia* (1st ed.). Frenchs Forest: Pearson Prentice Hall.

U.S. Department of Health & Human Services. (2008). *2008 physical activity guidelines for Americans*. Retrieved from www.health.gov/paguidelines

Van Praag, H. (2009). Exercise and the brain: Something to chew on. *Trends in Neurosciences, 32*(5), 283–290.

Lifestyle, Healthy

Emily Kothe
School of Psychology, University of Sydney, Sydney, NSW, Australia

Definition

The term healthy lifestyle does not have a single widely accepted definition. According to the World Health Organization, lifestyle is a way of living which is based on identifiable patterns of behavior. These patterns of behavior are determined by the interplay between individual, social, and environmental factors.

In this context, healthy lifestyle can be defined as a pattern of behavior which maximizes health protective behaviors while minimizing health risk behaviors.

Description

Lifestyle health behaviors are typically defined as behaviors which are performed on a daily (or almost daily) basis. This is distinct from other health behaviors such as medical screening which is performed less regularly.

While it is important to recognize that healthy lifestyle is not a unitary concept which can be prescribed to all people, a number of key patterns of behavior are included in most descriptions of healthy lifestyle (see Table 1).

These include:

- Active lifestyle
- Healthy diet

Together, poor diet and physical inactivity are among the leading causes of major noncommunicable diseases. These two lifestyle patterns contribute substantially to the global burden of disease. Following a healthy lifestyle pattern, characterized by an active lifestyle and healthy diet, can lead to large reduction in individual risk of disease.

Active Lifestyle

An active lifestyle can be defined as a pattern of behavior which includes regular physical activity and limited sedentary behaviors. Regular engagement in physical activity is known to be health protective and is known to be associated with is associated with improved mental and physical health.

Research shows that regular physical activity is associated with reduction in all-cause mortality and with decreased risk of osteoporosis, cardiovascular disease, diabetes mellitus, and some forms of cancer (Morrison, Bennett, Butow, Mullan, & White, 2008). For more information, see "active lifestyle."

In addition to including physical behavior, an active lifestyle is characterized by limited engagement in sedentary behaviors. Sedentary

Lifestyle, Healthy, Table 1 Physical activity guidelines by age group

Age range	Physical activity guidelines	
5–18 years	60 min (1 h) a day of moderate to vigorous physical activity	Children and adolescents should include muscle-strengthening and bone-strengthening exercises as part of their 60 min a day at least 3 days a week
18–64 years	150 min (2 h and 30 min) a week of moderate-intensity	Adults should also perform muscle-strengthening activities that involve all major muscle groups at moderate to high intensity two or more days a week
	or	
	75 min (1 h and 15 min) a week of vigorous-intensity aerobic physical activity	
65+ years	At least 30 min (half an hour) of moderate intensity physical activity on most, preferably all, days	Older people should be active every day in as many ways as possible, doing a range of physical activities that incorporate fitness, strength, balance, and flexibility

Source: Centers for Disease Control and Prevention (2010), Department of Health and Ageing (2010), U.S. Department of Health & Human Services (2008)

behaviors are those which involve very limited energy expenditure (e.g., computer use, TV viewing).

As a pattern of behavior, sedentary behavior often co-occurs with limited physical activity since it often displaces more physically active behaviors. This pattern of behavior is particularly unhealthy since the influence of sedentary behavior on health appears to be independent of the influence of physical inactivity. Research suggests that combining sedentary behavior and physical inactivity has approximately twice the negative health impact as either behavior alone (Owen, Healy, Matthews, & Dunstan, 2010). For more information, see "► Lifestyle, Sedentary."

Healthy Diet

An individual's diet plays an important part in long-term health. Diet is known to directly influence the risk of a number of life-limiting diseases (e.g., some forms of cancer, heart disease). Diet may also influence risk of disease through its influence on weight and obesity.

As a pattern of behavior, healthy diet is a pattern of eating behavior which maintains a state of well-being while reducing risk of chronic disease.

The World Health Organization recommends that dietary guidelines for populations and individuals should include the following recommendations:

- Achieve energy balance and a healthy weight
- Limit energy intake from total fats and shift fat consumption away from saturated fats to unsaturated fats and towards the elimination of trans-fatty acids
- Increase consumption of fruits and vegetables, and legumes, whole grains, and nuts
- Limit the intake of free sugars
- Limit salt (sodium) consumption from all sources and ensure that salt is iodized

Many countries have dietary guidelines that give more specific guidance in relation to these broad concepts. For example, the Dietary Guidelines for Americans recommend that salt intake be limited to a maximum of sodium intake of 2,300 mg for healthy adults and 1,500 mg for individuals with hypertension.

It is important to recognize that a healthy diet can be accomplished with a range of different dietary patterns. The suitability of each dietary pattern for a given individual will depend on that person's culture, income, family structure, age, health status, and home/work environment.

Cross-References

► Healthy Eating
► Lifestyle
► Lifestyle, Active
► Lifestyle, Sedentary

References and Readings

Centers for Disease Control and Prevention. (2010). Physical activity and health. *Physical activity for everyone*. Retrieved November 25, 2010, from http://www.cdc.gov/physicalactivity/everyone/health/index.html

Department of Health and Ageing. (2010). Physical activity guidelines. *Physical activity*. Retrieved November 20, 2010, from http://www.health.gov.au/internet/main/publishing.nsf/Content/health-pubhlth-strateg-phys-act-guidelines#rec_12_18

Morrison, V., Bennett, P., Butow, P., Mullan, B., & White, K. (2008). *Introduction to health psychology in Australia* (1st ed.). Frenchs Forest: Pearson Prentice Hall.

Owen, N., Healy, G. N., Matthews, C. E., & Dunstan, D. W. (2010). Too much sitting: The population health science of sedentary behavior. *Exercise and Sport Sciences Reviews, 38*(3), 105–113.

U.S. Department of Health and Human Services and U.S. Department of Agriculture. (2005). *Dietary guidelines for Americans, 2005* (6th ed.). Washington, DC: U.S. Government Printing Office. Retrieved November 13, 2010, from http://www.health.gov/dietaryguidelines/dga2005/document/default.htm

U.S. Department of Health & Human Services. (2008). *2008 Physical activity guidelines for Americans*. Retrieved November 12, 2010, from www.health.gov/paguidelines

World Health Organization. (1998). *Health promotion glossary: WHO/HPR/HEP/98.1*. Retrieved November 17, 2010, from http://www.who.int/hpr/NPH/docs/hp_glossary_en.pdf

World Health Organization. (2002). *The World Health Report 2002. Reducing risks, promoting healthy life*. Geneva: Author.

World Health Organization. (2004) *Global strategy on diet, physical activity and health*. Retrieved November 17, 2010, from http://www.who.int/dietphysicalactivity/strategy/eb11344/en/index.html

Lifestyle, Modification

Jordan Carlson
Public Health, San Diego State University, University of California San Diego, San Diego, CA, USA

Synonyms

Behavior change; Behavior modification; Behavioral intervention; Lifestyle changes

Definition

The term lifestyle modification can be used to refer to behavioral interventions that attempt to create change in multiple lifestyle health behaviors.

Description

Lifestyle Versus Nonlifestyle Health Behaviors

Health behaviors are actions taken by a person to maintain, attain, or regain good health and to prevent illness. Lifestyle health behaviors are different from other health behaviors in that they are engaged in on a daily basis (e.g., smoking, dietary behaviors, physical activity). Medical care behaviors (e.g., attendance of medical visits or cancer screenings) are not engaged in on a daily basis and thus can be considered nonlifestyle health behaviors. For more information and examples of commonly studied lifestyle behaviors, see "▶ lifestyle."

Although the term lifestyle can be used to refer to single behaviors in which people engage on a daily basis, this entry will focus on lifestyle modification of multiple health behaviors. For information on single behavior modification, see "▶ behavior change," "▶ behavior modification," and "▶ behavioral intervention."

Rationale for Multiple Versus Single Behavior Modification

The leading causes of death in the USA are heart disease, cancer, and stroke. Each of these diseases has the same underlying cause: poor diet, physical inactivity, and smoking (among others). One rationale for multiple vs. single health behavior modification is that changing multiple behaviors will affect health more than single behavior modification. Another rationale is that lifestyle health behaviors tend to cluster or co-occur. For example, a smoker or drug user is also likely to engage in poor dietary habits and be physically inactive. Thus, targeting multiple vs. single lifestyle behaviors should have the greatest potential to improve physical health outcomes of

primary interest in behavioral medicine, such as heart disease, cancer, and stroke (Prochaska, Spring, & Nigg, 2008; Prochaska, 2008).

Individual Versus Population-Based Approach

There are two main approaches to lifestyle modification: individual and population-based (Prochaska et al., 2008). The individual approach is person-centered and often includes tailored modification strategies for each person. For example, a person who consumes no fruits and vegetables but is physically active may be targeted to improve his/her fruit and vegetable consumption, while a person who consumes no fruits and vegetables and is physically inactive may be targeted to make improvements in both areas. In the individual approach, change/success is measured at the person level. An advantage to this approach is that it increases the potential overall impact on a person's health, while a disadvantage is that it likely increases the behavior change demands and program complexity.

The population-based approach to lifestyle modification typically includes a modification program across an entire population. These programs often create change to community policies and/or environments. Advantages of population-based approaches include the ability to reach large numbers of people and create sustainable changes. Disadvantages are that some people may not be reached by the modification program and environmental and policy changes can be costly and time intensive.

Theories of Lifestyle Modification/Behavior Change

Detailed information on behavior change interventions and theories can be found under the terms "▶ behavioral intervention" and "▶ theory." In brief, individual level theories such as the Transtheoretical Model of Behavior Change, Social Cognitive Theory, and Learning Theory have been useful in designing lifestyle modification interventions. More recently, ecological models (Sallis, Owen, & Fisher, 2010) have been used to inform population-based intervention approaches. Ecological models take into account multiple levels of influence on health behaviors, such as the individual, interpersonal, environment, and policy levels.

Success of Lifestyle Modification Programs

Success of multiple behavior lifestyle interventions has been documented, although this is a new area of research. A recent review found that multiple health behavior interventions resulted in greater weight loss than single behavior interventions (Sweet & Fortier, 2010). However, it is unknown how lifestyle behaviors change together and if intervention efforts should focus on grouping certain behaviors together (e.g., target diet and physical activity together but drug use separately).

Cross-References

- ▶ Behavior Change
- ▶ Behavior Modification
- ▶ Behavioral Inhibition
- ▶ Behavioral Intervention
- ▶ Behavioral Therapy
- ▶ Health Behavior Change
- ▶ Health Promotion
- ▶ Lifestyle Changes

References and Readings

Bandura, A. (2004). Health promotion by social cognitive means. *Health Education & Behavior, 31*, 143–164.

Prochaska, J. O. (2008). Multiple health behavior research represents the future of preventive medicine. *Preventive Medicine, 46*(3), 281–285.

Prochaska, J. J., Spring, B., & Nigg, C. R. (2008). Multiple health behavior change research: An introduction and overview. *Preventive Medicine, 46*(3), 181–188.

Sallis, J., Owen, N., & Fisher, E. (2010). Ecological models of health behavior. In K. Glanz, B. K. Rimer, K. Viswanath, & C. T. Orleans (Eds.), *Health behavior and health education: Theory, research, and practice*. San Francisco: Josey-Bass.

Sweet, S. N., & Fortier, M. S. (2010). Improving physical activity and dietary behaviours with single or multiple health behaviour interventions? A synthesis of meta-analyses and reviews. *International Journal of Environmental Research and Public Health, 7*(4), 1720–1743.

Lifestyle, Sedentary

Jordan Carlson
Public Health, San Diego State University, University of California San Diego, San Diego, CA, USA

Synonyms

Inactivity; Sedentary activity; Sedentary behaviors

Definition

A sedentary lifestyle consists of a daily routine of high amounts of sitting and/or very low energy expenditure activities paired with very low levels of physical activity.

Description

Sedentary Behavior

Sedentary behaviors are those that involve very little or no muscle movement and/or energy expenditure and usually involve sitting. Examples of sedentary activities include TV viewing, computer use, sitting in an automobile, or most other activities that involve sitting. For more information, see "► sedentary behaviors."

Physical Inactivity

A person who engages in little to no physical activity (specifically moderate-to-vigorous intensity physical activity) is said to be physically inactive. For more information, see "► exercise" and "► physical inactivity."

Sedentary Lifestyle Consists of Sedentary Behavior and Physical Inactivity

High amounts of sedentary activity often co-occur with low amounts of physical activity. Sedentary activity competes with physical activity and displaces physical activity when a person chooses sedentary behaviors over physical activity. This type of person typically avoids most or all types of physical activity and spends most of the day engaged in sitting behaviors such as TV watching or other screen time behaviors (e.g., computer use). He or she may be inactive in their occupation and leisure time, travel by automobile rather than active forms of transport, and engage in relatively little household work. This type of person is often referred to as a "precontemplator" as termed in the Transtheoretical Model of Behavior Change (see "► Transtheoretical Model of Behavior Change"), meaning that they have not considered becoming more active and would be difficult to reach with behavioral intervention.

Prevalence of Sedentary Activity and Physical Inactivity

Sedentary prevalence rates were measured objectively in 2004 using electronic activity monitors on a nationally representative sample of US youth and adults (Matthews et al., 2008). Adults were sedentary for an average of 7.5 h per day, with older age being related to more sedentary time. Children spend 6 h per day in sedentary activities, while youth spent 7.5–8 h per day sedentary. Women/girls spent only slightly more time in sedentary activity than men/boys, on average.

According to self-reports, 35.5% of US adults were classified as physically inactive based on the US Department of Health and Human Services physical activity guidelines. Inactivity is more prevalent in women, older adults, people of race/ethnic minority, less educated people, and those who are obese (Carlson et al., 2008). Objective data suggests much higher rates of physical inactivity: 58% of children, 92% of adolescents, and 96% of adults have been classified as inactive (Troiano et al., 2008).

Sedentary Lifestyle and Health

Sedentary behavior has recently been linked with multiple negative health outcomes, independent of the amount of physical activity in which a person engages. Specifically, the amount of time a person spends viewing TV, number of breaks from sitting (i.e., how many times a person stands up), and total time spent sitting were found to be associated with biomarkers (i.e., risk factors and symptoms) of cardiovascular disease, such as obesity, waist

circumference, systolic blood pressure, plasma glucose, triglycerides, HDL cholesterol, and mortality (Owen, Healy, Matthews, & Dunstan, 2010).

Physical inactivity is also related to a myriad of health problems, such as cardiovascular disease, type 2 diabetes, some cancers, and mortality (Blair et al., 1996; Haskell, Blair, & Hill, 2009).

Because the biology underlying the link between sedentary behavior and health is thought to be unique from the biology of inactivity and health, a person who lives a sedentary lifestyle is likely affected negatively by both processes. Thus, a sedentary lifestyle is thought to have around twice the negative health impact as being either sedentary or physically inactive (Owen et al., 2010).

Cross-References

- ▶ Physical Inactivity
- ▶ Sedentary Behaviors

References and Readings

Blair, S. N., Kampert, J. B., Kohl, H. W., 3rd, Barlow, C. E., Macera, C. A., Paffenbarger, R. S., Jr., & Gibbons, L. W. (1996). Influences of cardiorespiratory fitness and other precursors on cardiovascular disease and all-cause mortality in men and women. *Journal of the American Medical Association, 276*(3), 205.

Carlson, S. A., Fulton, J. E., Galuska, D. A., Kruger, J., Lobelo, F., & Loustalot, F. V. (2008). Prevalence of self-reported physically active adults-United states, 2007. *MMWR. Morbidity and Mortality Weekly Report, 57*, 1297–1300.

Haskell, W. L., Blair, S. N., & Hill, J. O. (2009). Physical activity: Health outcomes and importance for public health policy. *Preventive Medicine, 49*(4), 280–282.

Matthews, C. E., Chen, K. Y., Freedson, P. S., Buchowski, M. S., Beech, B. M., Pate, R. R., & Troiano, R. P. (2008). Amount of time spent in sedentary behaviors in the united states, 2003–2004. *American Journal of Epidemiology, 167*(7), 875–881.

Owen, N., Healy, G. N., Matthews, C. E., & Dunstan, D. W. (2010). Too much sitting: The population health science of sedentary behavior. *Exercise and Sport Sciences Reviews, 38*(3), 105–113.

Troiano, R. P., Berrigan, D., Dodd, K. W., Masse, L. C., Tilert, T., & McDowell, M. (2008). Physical activity in the united states measured by accelerometer. *Medicine & Science in Sports & Exercise, 40*(1), 181–188.

Likelihood Judgments

▶ Cancer Risk Perceptions

Limited Resource

▶ Self-Regulatory Fatigue

Linear Mixed-Effects Model

▶ Hierarchical Linear Modeling (HLM)

Linear Regression

▶ Regression Analysis

Lipid

Jonathan Newman
Columbia University, New York, NY, USA

Synonyms

Plasma lipid; Total cholesterol

Definition

Lipid is a general term of broad importance in human health and disease. There are both endogenous (produced by the body for physiologic processes) and exogenous (dietary intake) sources of lipid. Lipids are produced by the human body for many physiologic functions, including the synthesis of cell walls (made from cholesterol, a lipid). Dietary lipid is found most commonly as fatty acids, cholesterol, and cholesterol esters which are

absorbed by the body and used to sustain physiologic processes or stored for future energy use.

In cardiology and cardiovascular disease, lipid is generally considered to be the cholesterol content in the blood. The subcategories of lipid are low-density lipoprotein (LDL), high-density lipoprotein (HDL), very-low-density lipoprotein (VLDL), and triglycerides (TGs). The function of each lipid is outlined in other sections (see ▶ Lipid, Plasma; ▶ Lipoprotein).

Cross-References

▶ Lipid Abnormalities
▶ Lipid Metabolism
▶ Lipid, Plasma
▶ Lipoprotein
▶ Triglyceride

Lipid Abnormalities

Leah Rosenberg
Department of Medicine, School of Medicine, Duke University, Durham, NC, USA

Synonyms

Dyslipidemia

Definition

Lipid abnormalities encompass a variety of pathophysiological states of dysregulated cholesterol metabolism.

Description

Lipid abnormalities are extremely common in industrialized societies and are well known to be linked to adverse cardiovascular events, such as myocardial infarction and stroke. Dyslipidemias such as increased low-density lipoprotein (LDL), decreased high-density lipoprotein (HDL), and/or increased triglycerides have been identified as strongly associated with the risk of incident cardiovascular events. Lipid abnormalities are an excellent target for both primary and secondary prevention efforts, as there are a variety of lifestyle and pharmacologic approaches to correct these levels.

Cholesterol is a type of lipid that is hydrophobic and thus insoluble in human plasma. Lipids are carried with proteins into tissues for their varied cellular functions. Cholesterol and other lipids have enzymatic roles that are integral to human metabolism. When lipids are connected to a protein, they are referred to as lipoproteins. There are five biologically important lipoproteins, two of which will be discussed here: LDL and HDL. Both LDL and HDL ferry cholesterol esters and have distinct receptor types based on their function. LDL in the liver can be converted in secretable bile acids and excreted into the feces. Alternatively, LDL can be brought into extrahepatic cells, causing excessive cholesterol in the intracellular space. HDL cholesterol differs from LDL as it scavenges excess cholesterol from cells for excretion and thus has protective cardiovascular events. In common clinical parlance, LDL is known as "bad cholesterol," whereas HDL is called "good cholesterol."

Triglycerides are another important class of lipids. They are storage molecules that, when hydrolyzed, release free fatty acids into the circulation. They are created from the breakdown of fats and carbohydrates and are stored in adipose tissue. When excess calories are ingested, they are ferried to fat stores by triglycerides in the bloodstream.

When the levels of LDL, HDL, and triglycerides are abnormal, it is referred to as dyslipidemia. One definition of lipid imbalance or dyslipidemia that uses data from a large-scale observational study called NHANES III states that dyslipidemia is defined by being below the 10th percentile for HDL levels or above the 90th percentile for LDL and triglyceride levels nationally. Essentially, dyslipidemia implies high LDL and triglycerides and/or low HDL.

The Adult Treatment Panel (ATP III) guidelines, authored by the National Cholesterol Education Program (NCEP), are guidelines published in

Lipid Abnormalities, Table 1 The Adult Treatment Panel (ATP III) Treatment Guidelines

Risk category	LDL target	LDL threshold to start TLC	LDL threshold to start drug therapy
CHD/CHD risk equivalent	<100 mg/dL	≥100 mg/dL	≥130 mg/dL
			100–129 mg/dL on TLC (optional)
Moderately high	<130 mg/dL	≥130 mg/dL	≥130 mg/dL
Moderate	<130 mg/dL	≥130 mg/dL	≥160 mg/dL
Low	<160 mg/dL	≥160 mg/dL	≥190 mg/dL
			160–189 mg/dL on TLC (optional)

CHD = coronary heart disease, LDL = low-density lipoprotein, TLC = therapeutic lifestyle changes.

Lipid Abnormalities, Table 2 2004 Update to The Adult Treatment Panel (ATP III) Treatment Guidelines

Risk category	LDL target	LDL threshold to start TLC	LDL threshold to start drug therapy
CHD/CHD risk equivalent	<100 mg/dL	≥100 mg/dL	≥100 mg/dL
	<70 mg/dL (optional)		<100 mg/dL (optional)
Moderately high	<130 mg/dL or <100 mg/dL (optional)	≥130 mg/dL	≥130 mg/dL
	<100 mg/dL (optional)		100–129 mg/dL (optional)
Moderate	<130 mg/dL	≥130 mg/dL	≥160 mg/dL
Low	<160 mg/dL	≥160 mg/dL	≥190 mg/dL
			160–189 mg/dL on TLC (optional)

CHD = coronary heart disease, LDL = low-density lipoprotein, TLC = therapeutic lifestyle changes.

2001 for the diagnosis and treatment of dyslipidemia in adult patients. These guidelines provide an evidence-based approach for identifying and treating patients with lipid abnormalities, with a particular focus on reducing LDL by lifestyle modification and lipid-lowering therapy (primarily HMG-CoA reductase inhibitors or statins). The major feature of the ATP III guidelines was a focus on multiple risk factors to determine baseline cardiovascular risk in order to identify risk-based LDL goals (see Table 1) for non-pharmacological therapy (i.e., therapeutic lifestyle changes, TLC) or lipid-lowering drug therapy (i.e., statins). Four groups of baseline cardiovascular risk are defined as follows: high risk (coronary heart disease or coronary heart disease risk equivalents including peripheral artery disease, abdominal aortic aneurysm, symptomatic carotid artery disease, diabetes, or the presence of two or more risk factors with a 10-year Framingham risk score >20%), moderately high risk (two or more risk factors with a 10-year Framingham risk score 10–20%), moderate risk (two or more risk factors with a 10-year Framingham risk score less than or equal to 10%), and low risk (0 or 1 risk factor). The ATP III guidelines also discussed optimal goals for triglycerides (less than 150 mg/dL) and LDL (greater than or equal to 40 mg/dL) and also identified metabolic syndrome as an important at-risk phenotype.

In 2004, another NCEP report was published as an update to the ATP III guidelines. This report was endorsed by the National Heart, Lung, and Blood Institute, American College of Cardiology Foundation, and American Heart Association. Based on several randomized trials that were completed after the ATP III guidelines were published, the LDL cut points for initiating statin therapy (as well as on-treatment goals), especially for patients in the high-risk and moderately high-risk categories, were lowered (Table 2).

In 2008, the JUPITER trial, or Justification for the Use of Statins in Primary Prevention: An Intervention Trial Evaluating Rosuvastatin was published. JUPITER was a landmark randomized double-blinded, controlled trial comparing rosuvastatin (a statin) or placebo in patients,

mostly in the low- to moderate-risk category who had an LDL less than 130 mg/dL, who otherwise would not typically be a candidate for initiating statin therapy. An additional and important inclusion criterion was a C-reactive protein (a cardiovascular biomarker) of 2 mg/dL or higher. Compared with placebo, there was a significant reduction in cardiovascular events associated with rosuvastatin treatment. Despite the importance and clinical implications of the study findings, critics of JUPITER have stated concerns that the study was stopped prematurely (thus overestimating the reduction in cardiovascular events), questioned the magnitude of the absolute risk reduction observed in the trial (i.e., the rates of the primary end point were 0.77 and 1.36 per 100 person-years of follow-up in the rosuvastatin and placebo groups, respectively), and/or questioned the utility of C-reactive protein. It is unclear as of this writing whether CRP will be incorporated in the next NCEP report to select patients for statin therapy who have an LDL less than 130 mg/dL who are not at high risk.

Cross-References

- ▶ Lipid
- ▶ Lipid Metabolism
- ▶ Lipid, Plasma
- ▶ Lipoprotein

References and Readings

American Heart Association website, "Triglycerides." http://www.americanheart.org/presenter.jhtml?identifier=4778

Grundy, S. M., et al. (2004). Implications of recent clinical trials for the national cholesterol education program adult treatment panel III guidelines. *Circulation, 110*, 227–239.

Musunuru, K. (2010). Atherogenic dyslipidemia: Cardiovascular risk and dietary intervention. *Lipids, 45*(10), 907–914.

National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). (2002). Third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III) final report. *Circulation, 106*, 3143–3421.

Ridker, P. M., & Glynn, R. J. (2010, November 1). JUPITER trial: Responding to the critics. *The American Journal of Cardiology, 106*(9), 1351–1356.

Ridker, P. M., et al. (2008 Nov 20). Rosuvastatin to prevent vascular events in men and women with elevated C-reactive protein. *The New England Journal of Medicine, 359*(21), 2195–2207.

Lipid Disorder

- ▶ Hyperlipidemia

Lipid Metabolism

Manjunath Harlapur[1] and Daichi Shimbo[2]
[1]Center of Behavioral Cardiovascular Health, Division of General Medicine, Columbia University, New York, NY, USA
[2]Center for Behavioral Cardiovascular Health, Columbia University, New York, NY, USA

Synonyms

Fat metabolism

Definition

Lipid metabolism is a complex process which involves multiple steps from production within the body or dietary intake to degradation or transformation into several lipid-containing structures in the body.

Description

Lipid metabolism is a complex process that involves multiple steps involving the dietary intake of lipids (exogenous) or the production of lipids within the body (endogenous) to degradation or transformation (catabolism) into several

lipid-containing structures in the body. A brief description of metabolism related to fatty acids and cholesterol is provided below.

The dietary fat in the form of triacylglycerol (TAG), cholesterol, cholesteryl esters, and free fatty acids is absorbed by the intestine after going through various steps during digestion from mouth to intestine.

Fatty acids, once absorbed from intestine, are activated in the intestinal wall and eventually resynthesize the TAG. Some of the shorter-chain fatty acids go to the liver after binding to albumin in the blood. TAG, long-chain fatty acids, and cholesterol, after activation, are packaged in the chylomicron particles and moved to the blood stream via the lymphatic system to reach the entire body. The TAG is mainly broken down in the capillaries of skeletal muscles and adipose tissues. Other organs like heart, lung, liver, and kidneys are also involved. TAG in the chylomicrons is degraded to free fatty acids and glycerol by lipoprotein lipase. This enzyme is primarily produced in the adipose tissues and muscle cells. Thus formed free fatty acids may be either used for energy or may be stored as TAG after reesterification. However, glycerol that is released from TAG is used almost exclusively by the liver to produce glycerol 3-phosphate and this can be used either in the glycolytic pathway to produce energy or in the production of new molecules of glucose(gluconeogenesis).Fatty acids can also be produced by the body and this process occurs mainly in the liver and adipose tissues.

Fatty acids that are stored in the adipose tissue in the form of TAG serves as a major energy storage. The complete oxidation of fatty acids produces 9 kcal/g of fat as compared to proteins and carbohydrates which produce about 4 kcal.

The major degradation (catabolism) of saturated fatty acids occurs in mitochondria and this process is called beta oxidation. During beta oxidation, two-carbon fragments are removed successively generating high-energy-rich substances (like NADH and FADH2 molecules). After the oxidation of fatty acids, acetyl Co-A is produced and eventually it is used in the formation of ketone bodies. Thus formed ketone bodies can be used as energy source or for production of other important biochemical substrates. However, the oxidation of unsaturated fatty acids results in lesser calories and this process requires additional enzymes in the metabolic pathway.

Lipid metabolism also includes the formation and degradation of several other important lipid particles like phospolipids which plays a very important role in the structure of cellular membranes, prostaglandins, and several hormonal activities in the body.

Cholesterol is not only provided to the body by the dietary intake but also synthesized by virtually all the tissues in the body, although liver, intestine, adrenal cortex, reproductive tissues including ovaries, testes, and placenta produce most of the cholesterol in the body. These sites also utilize cholesterol in the process of production of multiple hormones in the body.

Degradation of the cholesterol happens mainly in the liver, where the sterol nucleus is eliminated from the body after conversion into bile acids and bile salts, which are excreted in the feces or it may undergo enterohepatic circulation.

The lipoproteins (chylomicrons, high-density lipoproteins, and low-, intermediate-, very-low-density lipoproteins) are involved with transporting these various lipid particles in the body with the apolipoproteins on their surface.

Cross-References

- ▶ Fat Absorption
- ▶ Lipoprotein
- ▶ Plasma Lipid

References and Readings

Harvey, R. A., & Ferrier, D. R. (2008). Cholesterol and steroid metabolism. In R. A. Harvey (Ed.), *Lippincott's illustrated reviews biochemistry* (pp. 181–200). Philadelphia: Wolters Kluwer/Lippincott Williams and Wilkins.

Lipid, Plasma

Manjunath Harlapur[1] and Daichi Shimbo[2]
[1]Center of Behavioral Cardiovascular Health, Division of General Medicine, Columbia University, New York, NY, USA
[2]Center for Behavioral Cardiovascular Health, Columbia University, New York, NY, USA

Synonyms

Total cholesterol in the blood

Definition

Plasma lipid is the cholesterol content in the blood. Plasma lipid profile includes total cholesterol, triglycerides (TG), high-density lipoproteins (HDL), intermediate-density lipoproteins (IDL), low-density lipoproteins (LDL), and very low-density lipoproteins (VLDL). The fasting lipid profile is generally recommended due to significant variations in the TG levels due to food contents.

Elevated LDL cholesterol levels and lower levels of HDL are associated with increased risk for incident cardiovascular disease. Non-HDL cholesterol is calculated as the difference between total cholesterol and HDL levels. Non-HDL cholesterol includes all the cholesterol present in the lipoproteins attributed to atherogenicity. Non-HDL cholesterol is considered as better tool for risk assessment than the assessing LDL cholesterol alone.

Cross-References

- ▶ Dean Ornish
- ▶ Heart Disease
- ▶ Lipid Metabolism

References and Readings

Cui, Y., Blumenthal, R. S., Flaws, J. A., Whiteman, M. K., Langenberg, P., Bachorik, P. S., et al. (2001). Non-high-density lipoprotein cholesterol level as a predictor of cardiovascular disease mortality. *Archives of Internal Medicine, 161*(11), 1413–1419.

Friedewald, W. T., Levy, R. I., & Fredrickson, D. S. (1972). Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clinical Chemistry, 18*(6), 499–502.

Harvey, R. A., & Ferrier, D. R. (2008). Metabolism of lipids. In R. A. Harvey (Ed.), *Lippincott's illustrated reviews biochemistry* (pp. 173–180). Philadelphia: Wolters Kluwer/Lippincott.

Third Report of the National Cholesterol Education Program (NCEP) Expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III) final report. *Circulation*, 2002, *106*(25), 3143–3421.

Lipoprotein

Manjunath Harlapur[1] and Daichi Shimbo[2]
[1]Center of Behavioral Cardiovascular Health, Division of General Medicine, Columbia University, New York, NY, USA
[2]Center for Behavioral Cardiovascular Health, Columbia University, New York, NY, USA

Synonyms

Cholesterol

Definition

Lipoproteins are complex substances containing lipids and specific proteins called apolipoproteins or apoproteins.

Description

Lipoprotein particles include chylomicrons, high-density lipoproteins (HDL), very low-density lipoproteins (VLDL), intermediate low-density lipoproteins (IDL), and low-density lipoproteins (LDL).

Each of these particles differs in their lipid and protein composition, density and size, and site of origin. Lipoproteins act as transporter of lipids

and render the lipids as water-soluble. Each lipoprotein is described in terms of their origin and metabolism in the following sections.

Chylomicrons are assembled in the intestinal cells, and they carry the dietary triacylglycerol, cholesterol, fat-soluble vitamins, and cholesterol esters to the peripheral tissues of the body. Chylomicrons are highest in their lipid content and lowest in density as compared to other lipoproteins. The characteristic apolipoprotein associated with chylomicron is B-48. The molecule of the chylomicron is modified after the attachment of apolipoprotein E and apolipoprotein C, mainly apolipoprotein C-II. Once the chylomicrons are released into blood circulation, the lipid component of chylomicrons is extracted from the adipose tissue, cardiac and skeletal muscle with the help of enzyme called lipoprotein lipase. This enzyme is activated by apolipoprotein C-II on the surface of chylomicron molecule. Thus, extracted lipid is further used for the energy supply and the storage. After extraction of lipids, chylomicrons are called chylomicron remnants, and these particles are metabolized and degraded in the cells of liver. This process generates amino acids, free cholesterol, and fatty acids.

VLDL are produced by the liver and composed of triacylglycerol. The function of VLDL is to transport the lipid from the liver to peripheral tissue. Apolipoprotein B-100 is located on surface of VLDL. After secreted into blood circulation, VLDL obtains apolipoprotein C-II and apolipoprotein E from HDL. During this exchange, some of the triacylglycerols are transferred from VLDL to HDL and some of the cholesteryl esters from HDL to VLDL. This exchange is mediated by cholesteryl ester transfer protein. With this modification, VLDL is converted in to LDL. During this transition, intermediate dense lipoproteins (IDL) and VLDL remnants are produced. Furthermore, IDL may be further taken up by the cells with the help of apo E. There are three isoforms of apolipoprotein E (E2, E3, and E4). Type III hyperlipoproteinemia (familial dysbetalipoproteinemia or broad beta disease) is manifested when apolipoprotein E2 is deficient and the patients usually present with premature atherosclerosis.

LDL contains lesser amount of triacylglycerol than VLDL and higher concentration of cholesterol and cholesteryl esters. The role of the LDL is to provide the cholesterol to the peripheral tissues. Apolipoprotein B-100 is located on the surface of LDL and binds to the LDL receptors on the cell surface. If there is deficiency of functional LDL receptor, this condition produces significant elevation in the LDL as seen in type II hyperlipidemia (familial hypercholesterolemia) causing premature atherosclerosis.

HDL are formed in the blood, and they have apolipoprotein A-1 on their surface and serve several important functions. HDL selectively transfer the cholesterol from the peripheral tissue and transport it back into liver for the bile acid synthesis and to steroidogenic cells for hormone synthesis. Thus, HDL help in the homeostasis of cholesterol. HDL also pick up the unesterified cholesterol in the circulation, and they also act as circulating reservoirs of apolipoprotein C-II. With all these functions, HDL are sometimes referred to as good cholesterol.

Cross-References

- ▶ Fat Absorption
- ▶ Lipid Metabolism
- ▶ Plasma Lipid

References and Readings

Harvey, R. A., & Ferrier, D. R. (2008). Cholesterol and steroid metabolism. In R. A. Harvey (Ed.), *Lippincott's illustrated reviews biochemistry* (pp. 219–244). Philadelphia: Wolters Kluwer/Lippincott.

Literacy

- ▶ Health Literacy

Load-High

► Hyperglycemia

Lobes

► Brain, Cortex

Locus

► Gene

Locus (Genetics)

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Gene

Definition

A locus is a spot or "address" on a chromosome at which a gene for a particular trait is located in all members of a species. It can also refer to the location of a mutation or other genetic marker. A given locus can be found on any pair of homologous chromosomes (Brown, 2009).

In 1909, the botanist Wilhelm Johannsen coined the term "gene" to describe the heritable characters discussed by Mendel. Between 1907 and 1919, the geneticist Thomas Hunt Morgan (a Nobel Laureate in Medicine) and his colleagues conducted an elegant series of experiments using the fruit fly *Drosophila melanogaster*. Fruit flies have marked advantages for use in transmission genetics research: they are abundant in nature, and they are very easily fed and accommodated. In addition, they are prodigious reproducers. They have a generation time of 10 days, and females produce around 300 eggs (of which half are female). This mathematics means that in the space of a single month, i.e., just three generation times, one fruit fly couple can lead to over three million flies (Watson, 2006).

In 1907, there was no current knowledge of established genetic differences, making it difficult for Morgan to know precisely where to start his research, as reflected by a comment by Watson (2006): "You cannot do genetics until you have isolated some characteristics to track through the generations." A search for mutant genes was started since mutant genes (variations from the normally occurring genes, sometimes called abnormal variants) lead to distinct characteristics. This led to the identification of some flies with white eyes (the normal color is red). The trait for white eye color was found to be located on the X chromosome and was inherited with a factor that determines the fly's sex. Morgan and others subsequently showed that each gene for a particular trait was located at a specific spot, i.e., a locus, on a chromosome in all individuals of a species.

Cross-References

► Chromosomes
► Gene
► Heritability
► Marker (Genetics)

References and Readings

Brown, S. M. (2009). *Essentials of medical genomics* (2nd ed.). Hoboken, NJ: Wiley-Blackwell.
Watson, J. D. (2006). *DNA : The Secret of life*. New York: Alfred A. Knopf.

Locus of Control

Julia R. Van Liew
Department of Psychology, University of Iowa, Iowa City, IA, USA

Synonyms

Attributional style; Hardiness; Self-efficacy

Definition

Locus of control refers to one's general predisposition to perceive control, or lack thereof, across various situations. The extent to which one attributes valued outcomes or reinforcement to either internal or external circumstances reflects their dimension of locus of control. Individuals with an internal locus of control believe in the power of their own decisions and behaviors to impact life events and determine their own future. Those with an external locus of control, on the other hand, view life events as dictated by environmental factors outside of one's control, such as luck, fate, or powerful others.

As originally defined by Rotter (1966), locus of control is a broad concept that is present across dimensions of functioning. The field of behavioral medicine has specifically applied it to the control individuals feel in regard to their physical health, termed health locus of control. The Health Locus of Control scale (HLC; Wallston, Wallston, Kaplan, & Maides, 1976) and Multidimensional Health Locus of Control scale (MHLC; Wallston, Wallston, & DeVellis, 1978) assess this specific construct. Researchers are interested in the relationship between health locus of control and a variety of health behaviors and outcomes, such as engagement in health-promoting and preventive behaviors, adherence to medical regimens, health care utilization, and coping with chronic illness.

References and Readings

Rotter, J. B. (1966). Generalized expectancies for internal versus external control of reinforcement. *Psychological Monographs, 80*(1), 1–28.
Wallston, K. A., Wallston, B. S., & DeVellis, R. (1978). Development of the multidimensional health locus of control (MHLC) scales. *Health Education & Behavior, 6*(1), 160–170.
Wallston, B. S., Wallston, K. A., Kaplan, G. D., & Maides, S. A. (1976). Development and validation of the health locus of control (HLC) scale. *Journal of Consulting and Clinical Psychology, 44*(4), 580–585.

Loneliness

Elizabeth A. Majka and John T. Cacioppo
Department of Psychology, The University of Chicago, Chicago, IL, USA

Synonyms

Social isolation; Social pain

Definition

A subjective sense of social isolation produces feelings of *loneliness* or social pain (Cacioppo & Patrick, 2008). This distressing constellation of feelings and emotions results from a discrepancy between one's actual and desired social relationships. Thus, lonely individuals are not satisfied with the quality of their actual social relationships, leaving them lacking a sense of social inclusion and belonging. Not to be confused with objective social isolation or a low quantity of social relationships, individuals can feel lonely when alone just as much as they can feel lonely when surrounded by a sea of people. Identifying individuals who suffer from loneliness is important, since over time loneliness can seriously impair physical, cognitive, and psychological health.

Loneliness is typically assessed using self-report items assessing the degree to which individuals endorse statements describing thoughts and

feelings commonly associated with loneliness such as "I lack companionship" or "I feel alone" (Russell, Peplau, & Cutrona, 1980). Scores on these sorts of measures fall on a continuum, ranging from very low in loneliness (i.e., feeling highly socially connected) to very high. Therefore, although labeling individuals as lonely or non-lonely may be convenient shorthand, people may fall anywhere on the loneliness continuum. Feelings of loneliness can be experienced transiently in response to an experience of social rejection or to a shift in circumstances (e.g., beginning college, divorce, widowhood, relocating to a new city), or loneliness can be experienced more chronically, functioning as a trait-like characteristic.

Three theoretical perspectives dominate the loneliness literature (Cacioppo & Patrick, 2008). One theory of loneliness posits that dissatisfaction with specific types of relationships (e.g., social network members vs. romantic partner) leads to different types of loneliness (e.g., social vs. emotional). Another perspective suggests that social skill deficits and personality traits (e.g., shyness) can hinder relationship development and maintenance, thereby leading to feelings of loneliness. Finally, a third theory of loneliness takes an evolutionary approach, positing that feelings of loneliness – although aversive – are adaptive in that they motivate humans to pursue and maintain social connections, thereby enhancing survival of oneself and one's offspring. Adoption and twin studies indicate loneliness has a heritability coefficient of approximately 0.50 (Boomsma, Willemsen, Dolan, Hawkley, & Cacioppo, 2005).

Loneliness also predicts various cognitive impairments, including poorer executive functioning, depressive symptomatology, and sleep fragmentation (Cacioppo & Hawkley, 2009). When people feel lonely, they are more likely to cope with stressors by withdrawal rather than by active coping and seeking emotional and instrumental support from others. Loneliness is also associated with an implicit hypervigilance for social threats, which has a cascading effect on social cognition. Briefly, although people who feel lonely want to connect with others, they tend to anticipate rejection and engage in self-protective behavior that paradoxically can be self-defeating. Accordingly, a meta-analysis of loneliness interventions indicated that the most effective interventions addressed these maladaptive social cognitions (Masi, Chen, Hawkley, & Cacioppo, 2011).

Cross-References

- ▶ Depression: Symptoms
- ▶ Executive Function
- ▶ Loneliness and Health
- ▶ Negative Thoughts

References and Readings

Aarsen, M., & Jylha, M. (2011). Onset of loneliness in older adults: Results of a 28 year prospective study. *European Journal of Ageing, 8*, 31–38.

Boomsma, D. I., Willemsen, G., Dolan, C. V., Hawkley, L. C., & Cacioppo, J. T. (2005). Genetic and environmental contributions to loneliness in adults: The Netherlands twin register study. *Behavior Genetics, 35*, 745–752.

Cacioppo, J. T., & Hawkley, L. C. (2009). Perceived social isolation and cognition. *Trends in Cognitive Science, 13*, 447–454.

Cacioppo, J. T., & Patrick, B. (2008). *Loneliness: Human nature and the need for social connection*. New York: W. W. Norton.

Masi, C. M., Chen, H., Hawkley, L. C., & Cacioppo, J. T. (2011). A meta-analysis of interventions to reduce loneliness. *Personality and Social Psychological Review, 15*, 219–266.

Russell, D., Peplau, L. A., & Cutrona, C. E. (1980). The revised UCLA loneliness scale: Concurrent and discriminant validity evidence. *Journal of Personality and Social Psychology, 39*, 472–480.

Loneliness and Health

Louise C. Hawkley and John T. Cacioppo
Department of Psychology, The University of Chicago, Chicago, IL, USA

Definition

Loneliness is the distress that accompanies feelings of social isolation and lack of connectedness

and belonging. Although people who are married and have close friends tend to be less lonely than those who do not, people can live relatively solitary lives and not feel lonely, and conversely, they can live an ostensibly rich social life and feel lonely nevertheless. As many as 80% of those under 18 years of age and 40% of adults over 65 years of age report being lonely at least sometimes. For as many as 15–30% of the general population, loneliness is a chronic state. Left untended, loneliness has serious consequences for physical and cognitive health and well-being.

Description

Loneliness and Physical Health and Mortality

Loneliness has been shown to predict increased morbidity and mortality (reviewed in Hawkley & Cacioppo, 2010). The effects of loneliness seem to accrue over time to accelerate physiological aging and mortality. For instance, loneliness exhibited a dose-response relationship with cardiovascular health risk in young adulthood such that the greater the number of measurement occasions at which participants were lonely (i.e., childhood, adolescence, and at 26 years of age), the greater their number of cardiovascular health risks (i.e., BMI, systolic blood pressure (SBP), total and HDL cholesterol levels, glycated hemoglobin concentration, and maximum oxygen consumption). Loneliness has been associated with increased systolic blood pressure in middle-aged adults, and a persistent trait-like aspect of loneliness accelerated the rate of systolic blood pressure increase over a 4-year follow-up period. Notably, the loneliness effect was unique and independent of the effects of depressive symptoms, perceived stress, social support, and hostility, variables that are correlated and sometimes mistakenly assumed to be synonymous with loneliness. Similarly, in a study of women, chronic high-frequency loneliness was prospectively associated with incident coronary heart disease over a 19-year follow-up in analyses that adjusted for depressive symptoms and a range of demographic and cardiovascular risk factors. Finally, a number of large epidemiologic studies have shown that all-cause or cardiovascular mortality risk is greater in chronically than situationally or rarely lonely adults.

Loneliness and Mental Health and Cognitive Functioning

Loneliness has been associated with personality disorders and psychoses, suicide, impaired cognitive performance and cognitive decline over time, increased risk of Alzheimer's disease, diminished executive control, and increases in depressive symptoms (Cacioppo & Hawkley, 2009). Loneliness and depressive symptoms are correlated, and recent analyses have demonstrated a causal role for loneliness; loneliness predicted increases in depressive symptoms over 1-year intervals, but depressive symptoms did not predict loneliness (Cacioppo, Hawkley, & Thisted, 2010). This is consistent with experimental evidence which has shown that hypnotically induced feelings of loneliness increase depressive symptoms while also increasing perceived stress, fear of negative evaluation, anxiety, and anger, and diminishing optimism and self-esteem (Cacioppo et al., 2006).

Feelings of social isolation have been associated with cognitive decline and dementia. A causal role for loneliness is supported by evidence that cognitive functioning in 75–85-year-olds did not differ as a function of loneliness at baseline but diminished to a greater extent among those high than low in loneliness over a 10-year follow-up, an effect that persisted after adjusting for objective measures of social activity and social network size. In another prospective study, loneliness was inversely associated with performance on a battery of cognitive measures in initially dementia-free older adults and predicted a faster decline in cognitive performance on most of these measures over a 4-year follow-up. In addition, incidence of Alzheimer's disease was predicted by degree of baseline loneliness adjusting for relevant covariates, including depressive symptoms.

Mechanisms for Health Effects of Loneliness

Health behaviors and sleep. Physical activity is a well-known protective factor for physical health,

mental health, and cognitive functioning. Loneliness has been associated with a lower likelihood of engaging in physical activity and a greater likelihood of discontinuing physical activity over time in middle-aged adults. This effect is not attributable to frequency of social contact or social network size, ruling out social control as a mechanism for the effects of felt isolation on physical activity. Loneliness is also a risk factor for obesity and a propensity to abuse alcohol. A compromised ability to exercise self-discipline and regulate emotions has been implicated in the poorer health behaviors exhibited in lonelier individuals.

Sleep affords physiological restoration, and sleep quality is important in accomplishing sleep's restorative effects. Non-restorative sleep (i.e., sleep that is non-refreshing despite normal sleep duration) results in daytime impairments such as physical and intellectual fatigue, role impairments, and cognitive and memory problems. Loneliness heightens feelings of vulnerability and unconscious vigilance for social threat, implicit cognitions that are antithetical to relaxation and sound sleep (Cacioppo & Hawkley, 2009). Cross-sectional and longitudinal studies have shown that loneliness predicts poor self-reported sleep quality, greater daytime dysfunction (i.e., low energy, fatigue), and more nightly micro-awakenings, effects that are largely independent of sleep duration and depressive symptoms (Hawkley, Preacher, & Cacioppo, 2010).

Physiological functioning. In young adults, loneliness has been associated with elevated levels of total peripheral resistance (Cacioppo et al., 2002; Hawkley, Burleson, Berntson, & Cacioppo, 2003), the primary determinant of systolic blood pressure (SBP) until at least 50 years of age. In older adults, loneliness has been associated with elevated SBP and predicted an accelerated increase in SBP over a 4-year follow-up, an effect that was adjusted for and independent of age, gender, race/ethnicity, cardiovascular risk factors, medications, health conditions, and the effects of depressive symptoms, social support, perceived stress, and hostility (Hawkley, Thisted, Masi & Cacioppo, 2010).

Changes in TPR levels are themselves influenced by a variety of physiological processes, including activity of the autonomic nervous system and the hypothalamic-pituitary adrenal (HPA) axis. To date, loneliness has not been shown to correlate with sympathetic nervous system activity at the heart, but was associated with a greater concentration of epinephrine in overnight urine samples in a middle-aged and an older adult sample (Hawkley et al., 2010). At high concentrations, circulating epinephrine binds α-1 receptors on vascular smooth muscle cells to elicit vasoconstriction and could thereby serve as a mechanism for increased SBP in lonely individuals.

Dysregulation of the hypothalamic-pituitary-adrenal axis contributes to inflammatory processes that play a role in hypertension, atherosclerosis, and coronary heart disease. Loneliness has been associated with urinary excretion of significantly higher concentrations of cortisol and with higher levels of salivary or plasma cortisol (reviewed in Hawkley & Cacioppo, 2010). A 3-day diary study clarified the causal direction; prior-day feelings of loneliness were associated with a higher cortisol awakening response the next day, but morning cortisol awakening response did not predict feelings of loneliness later the same day (Adam, Hawkley, Kudielka, & Cacioppo, 2006).

Gene Effects. Cortisol exerts broad anti-inflammatory effects, but although loneliness is associated with elevated cortisol levels, it increases risk for inflammatory disease. This apparent contradiction may be explained by glucocorticoid insensitivity. Evidence consistent with glucocorticoid insensitivity has been demonstrated in chronically lonely versus socially connected older adults and has further been shown to vary across the loneliness continuum (Cole et al., 2007; Cole, Hawkley, Arevalo, & Cacioppo, 2011). Genome-wide microarray analyses showed that markers of immune activation and inflammation (e.g., pro-inflammatory cytokines and inflammatory mediators) were over-expressed and markers of inhibitors of the pro-inflammatory NF-κB (nuclear factor-kappa B) transcript were under-expressed in genes of the lonely relative to the socially connected group. Analyses also indicated a possible

decrease in glucocorticoid receptor–mediated transcription in the lonely group, despite the fact that there were no group differences in circulating glucocorticoid levels. These results are consistent with a functional desensitization of the glucocorticoid receptor, which permits increased NF-κB activity and thereby induces a pro-inflammatory bias in gene expression. Loneliness differences in NF-κB/glucocorticoid receptor–mediated transcription activity were not attributable to objective indices of social isolation, nor were they explained by demographic, psychosocial (i.e., perceived stress, depression, hostility), or medical risk factors.

In an extension of this work, a recent study showed that feelings of social isolation were associated with a proxy measure of functional glucocorticoid insensitivity, namely, an attenuation in lonelier individuals of the typically positive correlation between cortisol levels and the ratio of neutrophil percentages relative to lymphocyte or monocyte percentages. This result signifies a diminished sensitivity of leukoctyes and leukocyte trafficking to the influence of cortisol in lonelier individuals (Cole, 2008).

Immune Functioning. Loneliness has been associated with impaired cellular immunity as reflected in lower natural killer (NK) cell activity and higher antibody titers to the Epstein-Barr virus and human herpes viruses. In addition, loneliness has been associated with poorer antibody response to a component of the flu vaccine, suggesting that the humoral immune response may also be impaired in lonely individuals (reviewed in Hawkley & Cacioppo, 2010).

Summary

Social integration was once thought to have effects on health via health behaviors, but we now know that the effects of perceived social isolation include biological consequences, therefore implicating central nervous system control. Evidence consistent with central influences includes implicit hypervigilance for social threat and diminished self-regulatory capacity. Loneliness is itself a product of central processes, i.e., maladaptive social cognitions that bias lonely individuals relative to socially connected individuals to perceive, expect, and remember more negative social information. Interventions that address maladaptive social cognitions have been shown to be more successful in reducing felt loneliness than interventions that work to increase number of social contacts. A role for central processes in the health effects of loneliness suggests that interventions that address maladaptive social cognitions may also influence biological, behavioral, and health outcomes.

Cross-References

- ▶ Alzheimer's Disease
- ▶ Autonomic Nervous System (ANS)
- ▶ Cognitive Function
- ▶ Cognitive Impairment
- ▶ Cortisol
- ▶ Gene Expression
- ▶ Health Risk (Behavior)
- ▶ Hostility
- ▶ Hypothalamic-Pituitary-Adrenal Axis
- ▶ Immune Function
- ▶ Inflammation
- ▶ Loneliness
- ▶ Negative Thoughts
- ▶ Sleep Quality
- ▶ Sympathetic Nervous System (SNS)
- ▶ Systolic Blood Pressure (SBP)

References and Readings

Adam, E. K., Hawkley, L. C., Kudielka, B. M., & Cacioppo, J. T. (2006). Day-to-day dynamics of experience-cortisol associations in a population-based sample of older adults. *Proceedings of the National Academy of Sciences United States of America, 103*, 17058–17063.

Cacioppo, J. T., & Hawkley, L. C. (2009). Perceived social isolation and cognition. *Trends in Cognitive Science, 13*, 447–454.

Cacioppo, J. T., Hawkley, L. C., Crawford, L. E., Ernst, J. M., Burleson, M. H., Kowalewski, R. B., Malarkey, W. B., Van Cauter, E., & Berntson, G. G. (2002). Loneliness and health: Potential mechanisms. *Psychosomatic Medicine, 64*, 407–417.

Cacioppo, J. T., Hawkley, L. C., Ernst, J. M., Burleson, M. H., Berntson, G. G., Nouriani, B., & Spiegel, D. (2006). Loneliness within a nomological net: An

evolutionary perspective. *Journal of Research in Personality, 40*, 1054–1085.
Cacioppo, J. T., Hawkley, L. C., & Thisted, R. A. (2010). Perceived social isolation makes me sad: Five year cross-lagged analyses of loneliness and depressive symptomatology in the Chicago Health, Aging, and Social Relations Study. *Psychology and Aging, 25*, 453–463.
Cole, S. W. (2008). Social regulation of leukocyte homeostasis: The role of glucocorticoid sensitivity. *Brain, Behavior, and Immunity, 22*, 1049–1055.
Cole, S. W., Hawkley, L. C., Arevalo, J. M. G., & Cacioppo, J. T. (2011). Transcript origin analysis identifies antigen presenting cells as primary targets of socially regulated leukocyte gene expression. *Proceedings of the National Academy of Sciences, 15*, 3080–3085.
Cole, S. W., Hawkley, L. C., Arevalo, J. M., Sung, C. Y., Rose, R. M., & Cacioppo, J. T. (2007). Social regulation of gene expression in humans: Glucocorticoid resistance in the leukocyte transcriptome. *Genome Biology, 8*, R189.1–R189.13.
Hawkley, L. C., Burleson, M. H., Berntson, G. G., & Cacioppo, J. T. (2003). Loneliness in everyday life: Cardiovascular activity, psychosocial context, and health behaviors. *Journal of Personality and Social Psychology, 85*, 105–120.
Hawkley, L. C., & Cacioppo, J. T. (2010). Loneliness matters: A theoretical and empirical review of consequences and mechanisms. *Annals of Behavioral Medicine, 40*, 218–227.
Hawkley, L. C., Preacher, K. J., & Cacioppo, J. T. (2010). Loneliness impairs daytime functioning but not sleep duration. *Health Psychology, 29*, 124–129.
Hawkley, L. C., Thisted, R. A., Masi, C. M., & Cacioppo, J. T. (2010). Loneliness predicts increased blood pressure: Five-year cross-lagged analyses in middle-aged and older adults. *Psychology and Aging, 25*, 132–141.
Masi, C. M., Chen, H.-Y., Hawkley, L. C., & Cacioppo, J. T. (2010). A meta-analysis of interventions to reduce loneliness. *Personality and Social Psychology Review, 15*, 219–266.

Longevity

Denis Gerstorf[1] and Christiane A. Hoppmann[2]
[1]Institute of Psychology, Humboldt University, Berlin, Germany
[2]Department of Psychology, University of British Columbia, Vancouver, BC, Canada

Synonyms

Life expectancy

Definition

The population-level concept of life expectancy refers to the average number of years of life remaining at a given age under the assumption that everyone will experience (for the rest of their lives) the same risk of death as evinced by current life tables. At the individual level, there is tremendous heterogeneity in the length of individual life spans within any society.

Description

The last century has witnessed unprecedented increases in longevity. This chapter provides an overview of population-level trends of longevity and describes important individual-level factors that contribute to between-person differences in longevity. In a first step, the terminology is clarified and population trends regarding past, present, and future developments in life expectancy are considered. In a second step, the chapter illustrates the importance of taking into account psychosocial predictors of between-person differences in longevity and discusses how and why sociodemographic characteristics, levels of functioning, as well as trajectories of change in behavioral and experiential characteristics are uniquely associated with mortality risks. The chapter concludes by pointing to key societal challenges for the future.

Population Trends in Longevity

The oldest reliably documented human being was a French woman by the name of Jeanne Calment who died in 1997 at the age of 122 years. This historically unparalleled life span demonstrates that, under optimal circumstances, humans can live up to 125 years. Such an age, however, is only reached by very few exceptional cases. A more realistic estimate of how long people can expect to live is provided by the population concept of life expectancy. Life expectancy at birth, for example, is the average number of years of life remaining for newborns under the assumption that everyone will experience (for the rest of their lives) the same risk of death as

evinced by current life tables (Olshansky, Hayflick, & Carnes, 2002). Over the past century, the world has witnessed a remarkable rise in life expectancy. For example, the average US American could expect to live for 49 years in 1900. In 2000, this number was 77 years, an increase of 64%. Another impressive illustration is that over the past 170 years, increases in life expectancy, in nations with the highest life expectancies, progressed at a rate of about 2.5 years per decade or 6 h per day (Vaupel, 2010). This remarkable increase in life expectancy was primarily due to a reduction in infant mortality (in the US, approximately 10% of infants born alive died before 1 year of age in 1900 vs. less than 1% in 2000). This trend originated in substantial advances in public health, hygiene, and nutrition (e.g., sewage, refuse disposal, safe drinking water) as well as medical care and technology. For example, key medical milestones in the early twentieth century were the discovery of antibiotics in 1928 and penicillin entering mass production in the 1940s, which substantially reduced mortality due to bacterial infections, diarrhea, and pneumonia.

Currently, life expectancy at birth is over 80 years in many industrialized nations such as Japan, Australia, or Canada (Organisation for Economic Co-operation and Development, OECD Factbook, 2010). Whereas rising life expectancy was historically driven by reductions in infant mortality, the fastest growing segment of the population today are the oldest old. For example, the number of individuals aged 85+ in the US has increased by 300% between 1960 and 2000, and the number of individuals aged 100+ is expected to grow from 37,000 in 1990 to 850,000 in 2050 (U.S. Bureau of the Census, 2004). Notwithstanding such general increases, there are tremendous geographical disparities in life expectancies. For example, the US only ranks 49 worldwide with a current life expectancy of 78 years (Central Intelligence Agency, CIA World Factbook, 2011), probably due to limitations in health-care coverage, unhealthy lifestyles (e.g., obesity), and public health problems (e.g., sanitation, pollution). In addition, some nations are currently experiencing dreadfully low or even declining life expectancies. According to the World Health Organization, for example, life expectancy in several African regions such as Angola is below the age 45 because of the AIDS pandemic. Similarly dramatic, Russia experienced a drop in average life expectancy by 6% in the 1990s that is only slowly coming back up. Alcohol abuse (as proxy for self-destructive behaviors and external causes of death due to homicide, suicide, and accidents) and stress (related to economic uncertainty and a poor outlook for the future) each accounted for about 25% of the documented increase in mortality rates (Brainerd & Cutler, 2005).

For the future of life expectancy, some demographers expect that every other girl born today in the developed world can expect to live up to the age 100 years (Vaupel, 2010). Such an optimistic prospect, however, rests heavily upon the assumption that progress in reducing mortality rates continues at the same pace as over the past two centuries. This assumption is not shared by other more conservative demographers (Olshansky et al., 2005) who instead predict that increases in life expectancy may slow down (e.g., because of little room for further reducing infant mortality) or even come to an end (e.g., due to unhealthy lifestyles resulting in sharp increases in obesity-related illnesses). The verdict for which of these estimates is going to be correct is out, but the aging population will put considerable strain on the health-care, welfare, and pensions systems.

Individual Differences in Survival

Besides such demographic trends, there is tremendous heterogeneity in the length of individual life spans within any society. Some individuals die relatively young (e.g., in their 40s or 50s), whereas others reach very advanced ages (e.g., age 90). This observation raises the pivotal question of key individual-level factors that are associated with heterogeneity in length of life. One such factor is sociodemographic characteristics. More specifically, individuals with low education and low income who belong to an ethnic minority have a lower life expectancy than highly educated, affluent Caucasians (Kaplan, Pamuk,

Lynch, Cohen, & Balfour, 1996). Another example is gender, with a considerable female survival advantage (e.g., up to six female centenarians per one male centenarian; Vaupel, 2010). This pervasive gender differential in life expectancy is thought to reflect a combination of genetic (e.g., with the second X chromosome, women have genetic information stored in duplicate, thereby reducing the risk for genetic defects), physiological (e.g., sturdier body makeup to sustain childbirth), and behavioral factors (e.g., better health behaviors; Owens, 2002).

In recent years, behavioral and experiential factors have also been identified as key predictors of heterogeneity in mortality hazards. Among the most obvious candidates are health behaviors and lifestyle factors, including lack of regular physical activity, unhealthy diets, stress, smoking, and heavy drinking. It has further been documented that impaired cognitive functioning (Deary, Weiss, & Batty, 2011), personality characteristics such as neuroticism (Wilson et al., 2005), lack of social integration (Seeman, 2001), diminished well-being (Danner, Snowdon, & Friesen, 2001), and perceptions of poor health (Idler & Benyamini, 1997) each uniquely predict mortality over and above established mortality correlates. What are the pathways and underlying mechanisms that link psychosocial factors with differences in mortality? It is an open conceptual question if psychosocial factors constitute mortality risks on their own or if they reflect the effects of pathologic processes. For example, poor well-being may have detrimental physiological effects on cardiovascular and immune functioning (Pressman & Cohen, 2005), resulting in further loss of functioning and ultimately death. Alternatively, well-being may reflect quite accurate perceptions of levels and particularly changes in functioning across a variety of other domains that are more directly linked to mortality such as physical health (Maier & Smith, 1999). Examining such questions will shed light on crucial, potentially modifiable risk factors.

Importantly, evidence is accumulating that the direction and size of changes in psychosocial characteristics evince unique and additional associations to mortality that go beyond those reported for levels of functioning. The probably most prominent example of such processes is the terminal decline concept. The general notion is that at some point shortly before death, individual functioning declines rapidly. Empirical evidence for precipitous mortality-related deteriorations has accumulated in the cognitive (Bäckman & MacDonald, 2006), personality (Mroczek & Spiro, 2007), and well-being domains (Gerstorf et al., 2010). It is possible that such declines represent a pervasive phenomenon and are indicative of general processes of brain atrophy and system breakdown. As more and more individuals live longer and longer lives, further understanding how terminal decline progresses and what factors may mitigate these processes will become one of the top priorities on the research agenda of the social and behavioral sciences.

Synopsis

The chapter concludes by raising three major questions for scientific inquiry and social policy regarding longevity-related insights at the population and individual level. First, the prospect of living a longer life will profoundly shape the way individuals allocate their time and resources during different life phases. More flexible and individualized ways of living are required to integrate different spheres of life, including education, employment, child rearing, retirement, and leisure (Carstensen, 2009). The second question revolves around whether the years gained are also characterized by the quality of life. Here, issues of vitality and time free of chronic and debilitating diseases in the face of increasing life expectancy come into focus (Hoppmann & Gerstorf, this volume, ▶ Vitality). A third issue concerns the modifiability of psychosocial risk factors and proximal steps to translate research findings into prevention and intervention strategies. The overarching aim is to strengthen individual resources, capabilities, and behaviors that are linked to living longer, thereby addressing the needs of a growing population of elderly, worldwide.

Cross-References

▶ Vitality

References and Readings

Bäckman, L., & MacDonald, S. W. S. (2006). Death and cognition: Synthesis and outlook. *European Psychologist, 11*, 224–235.

Brainerd, E., & Cutler, D. M. (2005). Autopsy on an empire: Understanding mortality in Russia and the former Soviet Union. *Journal of Economic Perspectives, 19*, 107–130.

Carstensen, L. L. (2009). *A long bright future*. New York: Crown.

CIA world factbook. https://www.cia.gov/library/publications/the-world-factbook/index.html

Danner, D. D., Snowdon, D. A., & Friesen, W. V. (2001). Positive emotions in early life and longevity: Findings from the Nun Study. *Journal of Personality and Social Psychology, 80*, 804–813.

Deary, I. J., Weiss, A., & Batty, G. D. (2011). Intelligence and personality as predictors of illness and death: How researchers in differential psychology and chronic disease epidemiology are collaborating to understand and address health inequalities. *Psychological Science in the Public Interest, 11*, 53–79.

Gerstorf, D., Ram, N., Mayraz, G., Hidajat, M., Lindenberger, U., Wagner, G. G., et al. (2010). Late-life decline in well-being across adulthood in Germany, the United Kingdom, and the United States: Something is seriously wrong at the end of life. *Psychology and Aging, 25*, 477–485.

Idler, E. L., & Benyamini, Y. (1997). Self-rated health and mortality: A review of twenty-seven community studies. *Journal of Health and Social Behavior, 38*, 21–37.

Kaplan, G. A., Pamuk, E. R., Lynch, J. W., Cohen, R. D., & Balfour, J. L. (1996). Inequality in income and mortality in the United States: Analysis of mortality and potential pathways. *British Medical Journal, 312*, 999–1003.

Maier, H., & Smith, J. (1999). Psychological predictors of mortality in old age. *Journals of Gerontology: Series B Psychological Sciences, 54B*, P44–P54.

Mroczek, D. K., & Spiro, A. (2007). Personality change influences mortality in older men. *Psychological Science, 18*, 371–376.

OECD. 2010. *Factbook 2010: Economic, environmental and social statistics*. ISBN 92-64-08356-1 – © OECD 2010.

Olshansky, J. S., Hayflick, L., & Carnes, B. A. (2002). Position statement on human aging. *Journal of Gerontology: Biological Sciences, 57A*, B292–B297.

Olshansky, J. S., Passaro, D. J., Hershow, R. C., Layden, J., Carnes, B. A., Brady, J., et al. (2005). A potential decline in life expectancy in the United States in the 21st century. *The New England Journal of Medicine, 352*, 1138–1145.

Owens, I. P. F. (2002). Sex differences in mortality rate. *Science, 297*, 2008–2009.

Pressman, S. D., & Cohen, S. (2005). Does positive affect influence health? *Psychological Bulletin, 131*, 925–971.

Seeman, T. (2001). How do others get under our skin? Social relationships and health. In C. D. Ryff & B. H. Singer (Eds.), *Emotion, social relationships, and health* (pp. 189–210). New York: Oxford University Press.

U.S. Bureau of the Census. (2004). *U.S. interim projections by age, sex, race, and Hispanic origin*. Washington, DC: Author.

Vaupel, J. W. (2010). Biodemography of human ageing. *Nature, 464*, 536–542.

Wilson, R. S., Krueger, K. R., Gu, L., Bienias, J. L., Mendes de Leon, C. F., & Evans, D. A. (2005). Neuroticism, extraversion, and mortality in a defined population of older persons. *Psychosomatic Medicine, 67*, 841–845.

World Health Organization. http://www.who.int/countries/en/

Longitudinal Research

▶ Cohort Study

Longitudinal Study

▶ Bogalusa Heart Study
▶ Follow-up Study

Long-Term Care

▶ Institutional Care

Loss

▶ Grieving

Low Back Pain

▶ Back Pain

Low Blood Glucose

▶ Hypoglycemia

Low Glycemic Index

Sheah Rarback
Department of Pediatrics, University of Miami, Miami, FL, USA

Synonyms

Glycemic index

Definition

The glycemic index (GI) measures how much a 50-g portion of carbohydrate raises a person's blood-sugar level when compared to glucose with a GI of 100. Carbohydrates that are rapidly broken down during digestion and release glucose rapidly into the blood stream have a high GI. Carbohydrates that break down and release glucose more slowly have a low GI. A diet of low GI foods can help control diabetes, improve the body's sensitivity to insulin, assist with weight loss by minimizing hunger, and lower blood lipid levels. Whole fruits, vegetables, beans, and foods without added sugar and refined grains usually have a lower glycemic index.

Dr. David Jenkins and Dr. Thomas Wolever of the University of Toronto developed the GI in 1981. They classified foods according to the following guide:

- Low GI ranging 55 and below
- Medium GI ranging between 56 and 69
- High GI ranging 70 and above

Glycemic index is one aspect of looking at a food. Since foods are usually not eaten in isolation, the other foods eaten at a meal will influence the glycemic load of a meal.

Cross-References

► Glycemia
► Hyperglycemia
► Hypoglycemia
► Nutrition

References and Readings

http://www.glycemicindex.com
Moore, C. S., Lindroos, A. K., Kreutzer, M., Larsen, T. M., Astrup, A., van Baak, M. A., et al. (2010). Dietary strategy to manipulate ad libitum macronutrient intake, and glycaemic index, across eight European countries in the Diogenes study. *Obesity Reviews, 11*(1), 67–75.
Solomon, T. P., Haus, J. M., Kelly, K. R., Cook, M. D., Filion, J., Rocco, M., et al. (2010). A low glycemic index diet combined with exercise reduces insulin resistance, postprandial hyperinsulinemia, and glucose dependent insulinotropic responses in obese humans. *American Journal of Clinical Nutrition, 92*(6), 1359–68.

Low Self-Efficacy

► External Locus of Control

Lumbago

► Back Pain

Lung Cancer and Smoking

► Cancer and Smoking

Lung Function

Akihisa Mitani
Department of Respiratory Medicine, Mitsui Memorial Hospital, Chiyoda-ku, Tokyo, Japan

Synonyms

Pulmonary function

Definition

Lung function is measured by various tests, including the pulmonary function test (PFT),

arterial blood gases (ABG) analysis, and oxygen desaturation during exercise. These tests can evaluate symptoms and signs of lung disease, reveal the cause of breath shortness, and may help confirm lung diseases, such as asthma and chronic obstructive lung disease (COPD). They are also useful for monitoring the progression of lung disease or evaluating the risk of surgery in the preoperative patients (ATS Committee on Proficiency Standards for Clinical Pulmonary Function Laboratories, 2002; American Thoracic Society, 1995; Crapo, 1994; Hughes & Pride, 1999; Hyatt, Scanlon, & Nakamura, 1997).

Pulmonary function tests include spirometry and the quantitation of lung volume and diffusing capacity. Spirometry is in widespread use, because it takes 10–15 min without any risk. It measures mainly forced expiratory volume in 1 s (FEV1.0) and forced vital capacity (FVC), which classify pulmonary disorder into a restrictive disorder and an obstructive one (American Thoracic Society, 1995). The patients with a restrictive disorder have reduced FVC, which means that the expansion of the lung is restricted and the effort of breathing is increased. This type of disorder is caused not only by diseases of the lung parenchyma (such as lung fibrosis) but also pleural or extrapulmonary diseases (such as pleural effusion, pneumonectomy, vertebral deformity, and myasthenia gravis). On the other hand, obstructive lung disorder, in which airway obstruction limits airflow, is defined by reduced FEV1.0/FVC. Obstructive disorder is the first attribute of asthma and COPD, and spirometry is essential to diagnose these diseases (especially in the case of COPD). The crucial difference between them is the reversibility of the obstructive disorder. Reduced FEV1.0/FEV of the patient with asthma could recover 10 min after administration of a short-acting bronchodilator, unlike in the case of COPD.

Lung volume measurement is important when FVC is decreased and useful for interpreting the result of diffusing capacity test. The diffusing capacity is a capacity of lung to transfer gas across alveoli. Carbon monoxide is widely used, and the amount of carbon monoxide disappeared from the lung is measured. The diffusing capacity for carbon monoxide (DLCO) is useful both in restrictive and obstructive disorders. In the patients with pulmonary fibrosis, DLCO is reduced because of increased alveolar membrane thickness. These two tests are so useful, but tend to be performed in specialized centers.

Arterial blood gas (ABG) analysis is used to measure the partial pressures of oxygen (PaO_2) and carbon dioxide ($PaCO_2$) and the pH of an arterial sample. The decreased level of PaO_2 (hypoxia) means insufficient oxygen supply to the body, principally indicating the existence of respiratory dysfunction. The high level of $PaCO_2$ (hypercapnia) may result from conditions that impair pulmonary ventilation, not infrequently occurring in the patients with severe COPD.

Oxygen desaturation during exercise is also indicative especially in patients with chronic lung disease, such as COPD and pulmonary fibrosis. It can assess lung function as part of physical function. The 6-min walk test is in widespread use. A decrease in SpO_2 of more than 4% reflects significant desaturation.

Cross-References

- ▶ Chronic Obstructive Pulmonary Disease
- ▶ Pulmonary Function

References and Readings

American Thoracic Society. (1995). Standardization of spirometry – 1994 update. *American Journal of Respiratory and Critical Care Medicine, 152*, 1107.

ATS Committee on Proficiency Standards for Clinical Pulmonary Function Laboratories. (2002). ATS statement: Guidelines for the six-minute walk test. *American Journal of Respiratory and Critical Care Medicine, 166*, 111.

Crapo, R. O. (1994). Pulmonary-function testing. *The New England Journal of Medicine, 331*, 25.

Hughes, J. M. B., & Pride, N. B. (1999). *Lung function tests: Physiological principles and clinical applications*. London: WB Saunders.

Hyatt, R. E., Scanlon, P. D., & Nakamura, M. (1997). *Interpretation of pulmonary function tests. A practical guide*. Philadelphia: Lippincott Williams & Wilkins.

Lupus: Psychosocial Impact

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

Lupus is a brief name of the formal medical term systemic lupus erythematosus (SLE). This is an autoimmune disease, affecting different organs in the body including the heart, lungs, skin, joints, and nervous system. It can be life threatening, though the advancement in medical treatment has made this outcome rare. Sadly, SLE mimics other illnesses in that its symptoms overlap with many other diseases; hence, patients may be misdiagnosed for long periods of time. It is prevalent mainly in women, between 15 and 35 years old. In SLE, the immune system produces antibodies against proteins in cell nuclei. Furthermore, various immune cells show excessive apoptosis, whose level correlates with disease activity. About 30% have skin symptoms (e.g., red rashes); many patients have joint pain, particularly in the hands and wrist, muscle pain, and anemia. Finally, cardiac inflammation and atherosclerosis are common. Some of the symptoms such as weight gain and retinal damage also result from treatment side effects.

Among the risk factors of SLE are environmental exposure to cigarette smoke, Epstein-Barr virus, and silica, which may interact with genetic predisposition (Simard & Costenbader, 2007). Common psychosocial sequels of SLE include worry about one's appearance due to skin problems on the face, uncertainty about the future, anxiety, and depression (Beckerman et al., 2011). Additional psychosocial consequences of SLE also include various phobias including agoraphobia and social phobia, possibly due to facial disfigurement as a result of the disease (Líndal, Thorlacius, Steinsson, & Stefánsson, 1995). One unique prospective study found that daily hassles concerning interpersonal issues and mood changes, assessed over a period of time electronically, predicted future flares in SLE patients (Pawlak et al., 2003). Thus, SLE is a complex poorly understood disease, which involves multiple systems and is affected by and affects patients' psychosocial well-being. The role of behavior medicine in SLE is essential given that psychosocial factors predict its course possibly since they could be related to the immunological changes in SLE due to neuroimmune interactions, and as they are outcomes of this disease as well.

References and Readings

Beckerman NL, Auerbach C., & Blanco I. (2011). Psychosocial dimensions of SLE: implications for the health care team. J Multidiscip Healthc., 4, 63–72.

Líndal, E., Thorlacius, S., Steinsson, K., & Stefánsson, J. G. (1995). Psychiatric disorders among subjects with systemic lupus erythematosus in an unselected population. *Scandinavian Journal of Rheumatology, 24*, 346–351.

Pawlak, C. R., Witte, T., Heiken, H., Hundt, M., Schubert, J., Wiese, B., et al. (2003). Flares in patients with systemic lupus erythematosus are associated with daily psychological stress. *Psychotherapy and Psychosomatics, 72*, 159–165.

Simard, J. F., & Costenbader, K. H. (2007). What can epidemiology tell us about systemic lupus erythematosus? *International Journal of Clinical Practice, 61*, 1170–1180.

Luvox®

▶ Selective Serotonin Reuptake Inhibitors (SSRIs)

Lymphocyte-Activating Factor (LAF)

▶ Interleukins, -1 (IL-1), -6 (IL-6), -18 (IL-18)

Lymphokines

▶ Cytokines

Lymphoma

▶ Cancer, Lymphatic

M

Macrophages

Riyad Khanfer
School of Sport and Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

Definition

Macrophages are white blood cells which form an important part of the innate immunity (non-specific). They are widely distributed in the body's tissues. Macrophages have irregular shape with granules in their cytoplasm. They are phagocytes and play a crucial role in the initiation of the adaptive (specific) immune response. They engulf, remove, and digest necrotic cell debris, bacteria, and other foreign bodies and also play important roles in the early phases of infection and inflammation (Levinson 2006; Waugh and Grant 2001).

Known as antigen-presenting cells, macrophages migrate between tissues, and when they encounter pathogen/antigen, they engulf it, more importantly, they digest the antigen via specific intracellular processes and transport the most antigenic fragment to their own cell membrane and display it on their surface, where they carry it until they come into contact with the adaptive immune cells (T lymphocytes) in the lymphoid tissues. These lymphocytes become activated and sensitized as a result and eventually proliferate and differentiate into three main types of memory T cells which generate a specific memory against this particular original antigen (Janway et al. 2005).

In the inflammatory response, macrophages produce many cytokines (cell messengers); among them are two important proinflammatory cytokines: interferon-γ and tumor necrosis factor (TNF). Macrophages have also been found to play a role in tumor immunology; they can infiltrate tumor mass and destroy tumor cells in tissue culture by producing reactive oxygen intermediates (ROIs) and tumor necrosis factor (TNF) (Janway et al. 2005; Weinberg 2007).

Cross-References

▶ Immune Function

References and Readings

Janeway, C. A., Travers, P., Walport, M., & Shlomchik, M. J. (2005). *Immunobiology* (6th ed.). New York/London: Garland Science.

Levinson, W. (2006). *Review of medical microbiology and immunology* (9th ed.). New York: McGraw-Hill Medical.

Waugh, A., & Grant, A. (2001). *Ross and Wilson anatomy and physiology in health and illness*. Edinburgh: Churchill Livingstone.

Weinberg, R. (2007). *The Biology of Cancer*. New York: Garland Science.

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

Magnetic Resonance Imaging (MRI)

John Ryan[1] and Howard Aizenstein[2]
[1]Department of Psychiatry, Western Psychiatric Institute and Clinic, University of Pittsburgh, Pittsburgh, PA, USA
[2]Geriatric Psychiatry Neuroimaging, Western Psychiatric Institute and Clinic, University of Pittsburgh Medical Center, Pittsburgh, PA, USA

Synonyms

MRI

Definition

Magnetic resonance imaging is a noninvasive imaging method used for visualizing structures in the body. The participant is placed in a strong static magnetic field that aligns all atomic nuclei in the body. A radiofrequency pulse is applied at a resonant frequency to selectively energize one type of atom (typically hydrogen). When the pulse is discontinued, the energy is emitted and detected by a detector coil. Due to the differences in concentrations of atoms in different structures, this energy can be used to reconstruct an image of the tissue in the detector coil.

Description

Magnetic resonance imaging (MRI) uses several separate components to collect an image. First, a superconducting electromagnet (created by passing electrical current through super-cooled wires) provides a static electromagnetic field. The strength of the magnet is denoted by "Tesla" strength. Most research scanners have a strength of 3.0 T – approximately 600 times more powerful than a kitchen magnet. As such, it is extremely important that participants remove any metal prior to entering the scanner room. Anyone with metal implants cannot have an MRI scan. Second, radiofrequency coils send energy to, and receive energy from, the sample being analyzed. Finally, gradient coils create a small alteration in the static magnetic field to create a spatial distribution in the signal to allow for image reconstruction in 3D space.

MRI data is collected in "slices" which are a certain thickness. Within each slice, space is divided into small squares known as "voxels." A voxel is the smallest sampling unit and is typically a few millimeters on each side. The size of a voxel determines the spatial resolution of the image. Smaller voxels will result in a clearer image. Most MRI paradigms take at least two separate scans: a high-resolution anatomical image with voxels approximately 1 mm^3 and a lower-resolution functional image (see below) with voxels approximately 9 mm^3. The lower-resolution functional image is then placed over the higher-resolution image to determine the anatomy that corresponds to the functional activity.

When tissue is placed into a strong magnetic field, the nuclei of atoms align in parallel with the magnetic field. Most nuclei align in a parallel (low energy) state, but some nuclei align with the magnetic field in an antiparallel (high-energy) state. An electromagnetic pulse is then applied to the sample, which flips some of the nuclei from low-energy to high-energy states. When the pulse is discontinued, the nuclei revert to the low-energy state and release the energy as photons, which are then detected with receiver coils. This is the basis of the MR signal. The return to the low-energy state and release of energy at the end of a pulse is termed "relaxation" and produces two parameters. "Transverse relaxation" (or T2 decay) is a measurement of how quickly the spins of the nuclei lose coherence after the pulse is discontinued. "Longitudinal relaxation" is a measurement of how long it takes the spins to return to the low-energy (parallel) state and is quantified using a time constant denoted as T1.

Numerous parameters can be manipulated to record certain properties of the MRI signal that make MRI a very versatile method for collecting images. First, the time between successive excitation pulses is the "repetition time" (TR). If the TR is too short, full recovery of the T1 parameter will not occur before the subsequent pulse occurs. Second, it is important to consider how long of a delay to include between the discontinuation of

the pulse and the recording of the MR signal. This parameter is termed the "echo time" (TE). By selecting optimal TR and TE values, the MR image can be tuned to either T1 or T2 contrast. T1 images are most commonly recorded for anatomical images and include a short TE and a medium TR. A final parameter that can be manipulated is the flip angle, which indicates the degree to which the net magnetization changes following the excitation pulse.

The selection of certain parameters to maximize the image of a particular feature of the image is referred to as a pulse sequence. Common pulse sequences include echo planar imaging (EPI) in BOLD fMRI (discussed below), fluid-attenuated inversion-recovery imaging (FLAIR), diffusion tensor imaging, and magnetization transfer imaging.

Structural MRI

Structural MRI is most commonly utilized to quantify certain types of tissue and identify patterns of change in structure that co-occur with disease or time. It can be used to measure the volume in grey mater, white matter, and cerebrospinal fluid using high-resolution T1 images discussed above. FLAIR images can be used to identify white matter hyperintensities that co-occur with aging, as well as certain psychiatric disorders. Diffusion tensor imaging measures the directions of water diffusion along multiple vector directions to assess the white matter tracts that connect disparate regions of the brain. Finally, magnetization transfer imaging can be used to assess the integrity of myelin in regions of interest.

Functional MRI

The methods described above are primarily used for visualizing structures in the body. In the 1990s, it was discovered that with appropriate tuning of the pulse sequence, brain activity could be detected. The key to this discovery is the magnetic properties of blood. Red blood cells contain hemoglobin that binds to oxygen to be carried to the tissues. Oxygenated hemoglobin has very little sensitivity to magnetic fields, whereas deoxygenated hemoglobin is paramagnetic and will distort a magnetic field. This distortion will alter the decay of the transverse magnetic component following the termination of the excitation pulse – termed T2* ("T2 star"). When neurons in the brain increase activity, they require more oxygen. The body responds by increasing blood flow to that region of the brain, which flushes out deoxygenated blood. This reduction in deoxygenated blood creates a stronger MR signal intensity. This change in signal is known as the blood-oxygenation-level-dependent (BOLD) contrast. This discovery has led to an explosion of research on human brain function as researchers can now noninvasively monitor brain activity. By comparing BOLD activity while performing a task with activity during a control condition, researchers can identify regions of the brain whose activity increases or decreases in response to performing the task.

One limitation of BOLD contrast is that it is a relative measure – signal is compared to a control condition – and therefore an absolute measure of blood flow to an area is unreliable. An alternative method for quantifying blood flow is arterial spin labeling (ASL). As arterial blood flows through the carotid artery toward the brain, a 180° excitation pulse is applied. This has the effect of magnetizing the water in the blood, which then travels to the brain. When this paramagnetic water arrives in the brain, it alters the magnetization of the area and causes an increase in the MR signal intensity. This signal intensity is compared to a control image to quantify the blood that was flowing to the region during the transit time.

Recent Developments

High-Field MRI

Early MRI scanners had strengths of 1.5 T, and research scanners in hospitals now are commonly 3.0 T. As the strength of the magnet increases, there is a nearly linear increase in signal-to-noise ratio. Some research centers now have 7.0 T magnets, and stronger magnets are currently under development. These stronger magnetic fields may allow better information about tissue composition, including iron deposition in the basal ganglia.

Functional and Effective Connectivity

As computing power and software have increased in the past decade, new methods for measuring

networks and interactions of brain regions have been developed. Seed-voxel correlation maps extract the signal from a small group of voxels and then correlate the activity over time with the signal from all other voxels in the brain. The result is a "functional connectivity" map of regions whose activity correlates with the seed region. Such a method cannot infer causal influence between regions, but it has been useful in understanding so-called Resting State Networks – networks of brain activity that are active in the absence of an experimental task. More sophisticated techniques have been developed for inferring causality between regions, including Granger Causality Analysis, Dynamic Causal Modeling, and Structural Equation Modeling.

Cross-References

- ▶ Brain, Imaging
- ▶ Neuroimaging

References and Readings

Duyn, J. H. (2010). Study of brain anatomy with high-field MRI: Recent progress. *Magnetic Resonance Imaging, 28*, 1210–1215.

Huettel, S. A., Song, A. W., & McCarthy, G. (2004). *Functional magnetic resonance imaging* (2nd ed.). Sunderland, MA: Sinauer Associates.

Rogers, B. P., Morgan, V. L., Newton, A. T., & Gore, J. C. (2007). Assessing functional connectivity in the human brain by fMRI. *Magnetic Resonance Imaging, 25*, 1347–1357.

Maintenance Phase of the Transtheoretical Model of Change

Seema Mutti
School of Public Health & Health Systems, University of Waterloo, Waterloo, ON, Canada

Definition

The Transtheoretical Model of Change (TTM) is characterized by six 'stages of change' (precontemplation, contemplation, preparation, action, maintenance, and termination) that individuals move through when attempting to change problematic health behavior, such as smoking (Shumaker, Ockene, & Riekert, 2009).

Individuals reach the 'maintenance' phase of the TTM when the positive health behavior, for example, complete abstinence from smoking, is sustained for a period of 6 months or longer (Glanz, Rimer, & Viswanath, 2008). Self-efficacy (or situation-specific confidence) plays an integral role in this stage. As an individual's ability to abstain from the risky health behavior increases, so too does their ability to avoid relapse. The risk of relapse is eliminated once an individual reaches the final 'termination' stage of the TTM, in which the positive health behavior has become automatic (Shumaker et al., 2009).

Cross-References

- ▶ Behavior Change
- ▶ Self-Efficacy

References and Readings

Glanz, K., Rimer, B. K., & Viswanath, K. (Eds.). (2008). *Health behavior and health education: Theory, research, and practice* (4th ed.). San Francisco: Jossey Bass.

Miller, W. R., & Heather, N. (Eds.). (1998). *Treating addictive behaviors* (2nd ed.). New York: Plenum Press.

Prochaska, J. O., & Norcross, J. C. (2010). *Systems of psychotherapy: A transtheoretical analysis* (7th ed.). Belmont, CA: Brooks/Cole.

Shumaker, S. A., Ockene, J. K., & Riekert, K. A. (Eds.). (2009). *The handbook of health behavior change* (3rd ed.). New York: Springer.

Major Adverse Cardiac and Cerebrovascular Event (MACCE)

- ▶ Cardiac Events

Major Adverse Cardiac Event (MACE)

▶ Cardiac Events

Major Adverse Cardiovascular Event (MACE)

▶ Cardiac Events

Major Depressive Disorder

▶ Postpartum Depression
▶ Unipolar Depression

Major Depressive Disorder, with Seasonal Pattern

▶ Seasonal Affective Disorder

Maladaptation

▶ Maladaptive/Maladjustment

Maladaptation of Symptom Behaviors to Chronic Illness

▶ Symptom Magnification Syndrome

Maladaptive/Maladjustment

David Busse and Ilona S. Yim
Department of Psychology and Social Behaviour, University of California, Irvine, Irvine, CA, USA

Synonyms

Dysfunctional/dysfunction; Maladaptation; Maladjustive

Definition

Maladjustment is the result of insufficient responses to demands that may occur throughout the life span and result in impaired functioning, distress, and/or poor health. The term *maladaptive* refers to processes (e.g., specific behaviors, patterns of thought or emotion that yield negative outcomes) whereas maladjustment is the result or outcome of this process.

Description

The terms maladjustment and maladaptive are used in a wide range of contexts, which may broadly be categorized as social, psychological, and biological. Social maladjustment refers to how a person develops and maintains interpersonal relationships, especially with peers. Maladaptive behaviors in this realm often emerge during childhood when individuals learn how to navigate their social world and solve interpersonal problems. During this period, children face changing school settings and social networks. Children who are not able to successfully adjust to these new environments may exhibit a range of maladaptive behaviors, such as aggression or rough play, which may lead to peer rejection (Ladd & Price, 1987).

The terms maladjustment and maladaptive, when applied to the psychological domain, may also refer to how well somebody is able to regulate their emotions. Emotions typically serve adaptive purposes in how individuals interact with the environment. Thus, psychological maladjustment may be characterized by high levels of "emotional inertia" (Kuppens, Allen, & Sheeber, 2010), or the inability to respond appropriately to the dynamically changing demands of a given situation.

Finally, maladjustment and maladaptie processes can also pertain to how individuals physiologically respond and adapt to environmental demands. The human body attempts to respond optimally to the continually changing environment, a concept that has been termed *allostasis* (McEwen & Stellar, 1993). Key systems involved in this

process are the autonomic nervous system, the hypothalamic pituitary-adrenal axis, and the immune and cardiovascular systems. Repeated activation of these systems exerts wear and tear on the body, termed *allostatic load*. High levels of allostatic load, when quantified using objective health parameters, have been associated with increased risk of cardiovascular disease and lower physical and cognitive functioning (Seeman, Singer, Rowe, Horwitz, & McEwen, 1997).

Maladjustment and maladaptive processes have been studied throughout the life span, ranging from the prenatal period to old age. These studies provide important insight into the immediate and long-term health consequences of maladjustment at different times in life. Researchers study maladjustment in the context of how certain prenatal events during critical periods can have lasting implications on development throughout the life span. For example, the Dutch Famine Studies are a series of studies that examine the mechanisms linking disease outcomes to deprivation in utero. A blockade of food supplies in the Netherlands between November 1944 and April 1945 created a natural experiment from which to study the effects of maternal malnutrition. It was later found that children who were in gestation during the famine had higher rates of adult diseases, including coronary heart disease (Roseboom et al., 2001) and schizophrenia (Hoek, Brown, & Susser, 1998). Similarly, a study of 141 Canadian families who endured a severe and prolonged ice storm during the winter of 1998 demonstrates how severe stressors can result in postnatal maladjustment. Children whose mothers were exposed to more severe stress had poorer cognitive and language development at age two than children whose mothers who experienced less stress during the ice storm (King & Laplante, 2005).

Typically, the effect of maladaptive behavior is studied during later periods of life. A classic example for how maladaptive behavior can lead to adverse health outcomes is the link between certain components of Type A behavior patterns (i.e., hostility) and coronary heart disease (CHD). Type A behavior is characterized by aggressiveness, impatience, and competitiveness. Although these behaviors often lead to high status in the workplace, they may also be maladaptive in the context of maintaining health. By itself, maladaptive behavior does not have an effect on disease pathogenesis. Rather, this process occurs through the repeated and exaggerated activation of biological regulatory systems (e.g., high blood pressure), conflictual and unsupportive social relationships, and health-compromising behaviors (e.g., smoking). A link between maladaptive personality traits and development of disease has been demonstrated in past research. A longitudinal study with over 4,700 college students showed that greater hostility at age 19 predicted more risk factors for CHD, including higher caffeine consumption, larger body mass index (BMI), higher cholesterol, and more tobacco use (Seigler, Peterson, Barefoot, & Williams, 1992).

References and Readings

Hoek, H. W., Brown, A. S., & Susser, E. (1998). The Dutch famine and schizophrenia spectrum disorders. *Social Psychiatry and Psychiatric Epidemiology, 33*, 373–379.

King, S., & Laplante, D. P. (2005). The effects of prenatal maternal stress on children's cognitive development: Project ice storm. *Stress, 8*, 35–45.

Kuppens, P., Allen, N. B., & Sheeber, L. B. (2010). Emotional inertia and psychological maladjustment. *Psychological Science, 21*, 984–991. doi:10.1177/0956797610372634.

Ladd, G. W., & Price, J. M. (1987). Predicting children's social and school adjustment following the transition from preschool to kindergarten. *Child Development, 58*, 1168–1189.

McEwen, B., & Stellar, E. (1993). Stress and the individual: Mechanisms leading to disease. *Archives of Internal Medicine, 153*, 2093–2101.

Roseboom, T. J., van der Meulen, J. H. P., Ravelli, A. C. J., Osmond, C., Barker, D. J. P., & Bleker, O. P. (2001). Effects of prenatal exposure to the Dutch famine on adult disease in later life: An overview. *Molecular and Cellular Endocrinology, 185*, 93–98.

Seeman, T. E., Singer, B. H., Rowe, J. W., Horwitz, R. I., & McEwen, B. S. (1997). Price of adaptation-Allostatic load and its health consequences: MacArthur studies of successful aging. *Archives of Internal Medicine, 157*, 2259–2268.

Siegler, I. C., Peterson, B. L., Barefoot, J. C., & Williams, R. B. (1992). Hostility during late adolescence predicts coronary risk factors at mid-life. *American Journal of Epidemiology, 136*, 145–154.

Maladjustive

▶ Maladaptive/Maladjustment

Malignant Neoplastic Disease

▶ Carcinoma

Malingering

▶ Symptom Magnification Syndrome

Managed Care

▶ Disease Management

Management

▶ Lifestyle Changes

Management of Depression

▶ Depression: Treatment

Marital Dissolution

▶ Divorce and Health

Marital Satisfaction

▶ Marriage and Health

Marital Stress

▶ Family Stress

Marital Therapy

▶ Couple-Focused Therapy

Marker (Genetics)

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

A genetic marker is a DNA sequence with a known physical location on a chromosome. Genetic markers can help link an inherited disease with the responsible gene. DNA segments close to each other on a chromosome tend to be inherited together. Genetic markers are used to track the inheritance of a nearby gene that has not yet been identified but whose approximate location is known. The genetic marker itself may be a part of a gene or may have no known function (Genome.gov, 2011).

Cross-References

▶ Chromosomes
▶ DNA
▶ Gene

References and Readings

Genome.gov (2011). Accessed November 15th, 2011 http://www.genome.gov/glossary/?id=86

Marriage and Health

Wendy Troxel[1] and Julianne Holt-Lunstad[2]
[1]Psychiatry and Psychology, University of Pittsburgh, Pittsburgh, PA, USA
[2]Department of Psychology, Brigham Young University, Provo, UT, USA

Definition

Socially connected people live longer, healthier, and happier lives than their socially isolated counterparts (Holt-Lunstad, Smith, & Layton, 2010). Given the central role that marriage plays for most adults, a great deal of research has focused on this particular social relationship in association with physical and mental health. For instance, prior research suggests that married adults have lower rates of morbidity and mortality compared to unmarried adults (Jaffe, Manor, Eisenbach, & Neumark, 2007; Johnson, Backlund, Sorlie, & Loveless, 2000). Likewise, married individuals have greater life satisfaction, happiness, and are at lower risk for depression (Gove, Hughes, & Style, 1983; Inaba et al., 2005). The conclusion often drawn from this literature is that being married is beneficial for health, but perhaps less so for women than for men. Ironically, several lines of evidence suggest that marriage *should* be associated with better health, perhaps, particularly so in women. Specifically, marriage may protect against social isolation, may promote health-enhancing behaviors and deter health-damaging behaviors, and may affect health indirectly, by increasing socioeconomic resources, particularly for women. In contrast, given the health costs of conflictual social relationships and the fact that marriage is the primary social relationship for most adults, being in an unhappy marriage may serve as a potent psychosocial stressor, thereby elevating disease risk. In aggregate, these divergent perspectives on the advantages of having a spouse on the one hand, and the disadvantages of marital discord, on the other hand, suggest the utility of examining marital status and marital quality simultaneously. However, few studies to date have adopted this approach of simultaneously examining marital status and marital quality (for exceptions see Gallo et al., 2003a; Holt-Lunstad, Birmingham, & Jones, 2008; Troxel, Matthews, Gallo, & Kuller, 2005). Theoretically, individuals in a high-quality marriage should be at a health advantage relative to unmarried individuals and to individuals in low-quality marriages because they reap the material benefits of marriage *and* the social support benefits of being in a harmonious union.

Description

Marital Quality Matters

Indeed, mounting evidence suggests that various indicators of marital quality, including satisfaction or strain, are associated with increased risk of the development of CVD risk factors or prognosis in cardiac patients (as reviewed in Kiecolt-Glaser & Newton, 2001). For instance, Orth-Gomer and colleagues (2000) found that marital strain was associated with a 2.9-fold increased risk of recurrent cardiac events in female cardiac patients. Similarly, Coyne and colleagues (2001) reported that among male and female coronary heart disease (CHD) patients, poor marital quality was a more robust predictor of mortality in women than in men. Our laboratory has shown in an initially healthy sample of women that those who are satisfied in their marriage show less atherogenic risk trajectories over time (Gallo, Troxel, Matthews & Kuller, 2003b), have lower levels of subclinical measures of carotid atherosclerosis (Gallo et al., 2003a), and are at lesser risk of developing the metabolic syndrome over an 11-year follow-up, as compared to their dissatisfied or unmarried counterparts (Gallo et al., 2003a; Troxel et al., 2005). In aggregate, findings suggest that the quality of the marital relationship is implicated in the development and progression of CVD; however, the effects may be stronger in women. Moreover, how marriages "get under the skin" to influence cardiovascular risk and outcomes remains a question of critical importance.

Pathways Linking Marriage with Health

Both the presence of a spouse as well as the quality of the marital relationship may influence health directly and indirectly through behavioral, psychological, and physiological mechanisms. For instance, social relationships may influence the extent to which individuals engage in health-relevant behaviors (e.g., smoking, sedentary lifestyle, fatty diet intake, and excessive alcohol consumption). More recent data suggests that being stably married or being in a happy marriage is associated with better sleep (Troxel, Buysse, Hall, & Matthews, 2009; Troxel et al., 2010), another critical health behavior. Marriage has also been linked to psychological factors such as lowered stress and depression, which have demonstrated influences on cardiovascular health themselves (Rozanski, Blumenthal, & Kaplan, 1999; Smith & Ruiz, 2002). Notably, social relationships may also exert direct physiological effects on autonomic, inflammatory, or neuroendocrine pathways (Robles & Kiecolt-Glaser, 2003).

Sex Differences in the Effects of Marriage on Health

Based on their review of the marriage and health literature, Kiecolt-Glaser and Newton (2001) concluded that women may be more vulnerable to the health consequences associated with marital distress than men. For example, Coyne and colleagues (2001) reported that among male and female coronary heart disease (CHD) patients, poor marital quality was a more robust predictor of mortality in women than in men. Research further suggests that this gender disparity may be attributable to gender differences in physiological responding to marital interactions. Indeed, compared with men, women display more pronounced physiological responses (i.e., cardiovascular, endocrine, and immunologic) to marital interactions, particularly when negative interactions are assessed (e.g., Kiecolt-Glaser, Glaser, Cacioppo, & Malarkey, 1998; Smith, Gallo, Goble, Ngu, & Stark, 1998). However, more recent data suggests that these sex differences may be less pronounced than formerly demonstrated (Manzoli, Villari, Pirone, & Boccia, 2007), perhaps suggestive of a cohort effect.

Summary

Social isolation is a significant risk factor for morbidity and mortality. The marital relationship is arguably the single-most important adult social relationship and can protect against social isolation. Consistent evidence shows that married individuals have greater life satisfaction, happiness, mental health, and lower rates of morbidity and mortality compared to unmarried adults (Johnson et al., 2000; Manzoli et al., 2007). However, not all marriages are created equal. More recent data points to the importance of assessing qualitative aspects of marriage as well marital status. Indeed, evidence suggests that marriage is health-protective, particularly if it is a high-quality marriage. Understanding the mechanisms underlying the health effects of marital status and quality remains a critical next step in order to understand how relationships "get under the skin" to impact health and well-being.

Cross-References

- ▶ Alcohol Abuse and Dependence
- ▶ Alcohol Consumption
- ▶ Smoking Behavior
- ▶ Social Stress
- ▶ Social Support

References and Readings

Coyne, J. C., Rohrbaugh, M. J., Shoham, V., Sonnega, J. S., Nicklas, J. M., & Cranford, J. A. (2001). Prognostic importance of marital quality for survival of congestive heart failure. *The American Journal of Cardiology, 88*, 526–529.

Gallo, L. C., Troxel, W. M., Kuller, L. H., Sutton-Tyrrell, K., Edmundowicz, D., & Matthews, K. A. (2003a). Marital status, marital quality, and atherosclerotic burden in postmenopausal women. *Psychosomatic Medicine, 65*, 952–962.

Gallo, L. C., Troxel, W. M., Matthews, K. A., & Kuller, L. H. (2003b). Marital status and quality in middle-aged women: Associations with levels and trajectories of cardiovascular risk factors. *Health Psychology, 22*, 453–463.

Gove, W. R., Hughes, M., & Style, C. B. (1983). Does marriage have positive effects on the psychological well-being of the individual? *Journal of Health and Social Behavior, 24*, 122–131.

Holt-Lunstad, J., Birmingham, W., & Jones, B. Q. (2008). Is there something unique about marriage? The relative impact of marital status, relationship quality, and network social support on ambulatory blood pressure and mental health. *Annals of Behavioral Medicine, 35*, 239–244.

Holt-Lunstad, J., Smith, T. B., & Layton, J. B. (2010). Social relationships and mortality risk: A meta-analytic review. *PLoS Medicine, 7*, e1000316.

Inaba, A., Thoits, P. A., Ueno, K., Gove, W. R., Evenson, R. J., & Sloan, M. (2005). Depression in the United States and Japan: Gender, marital status, and SES patterns. *Social Science & Medicine, 61*, 2280–2292.

Jaffe, D. H., Manor, O., Eisenbach, Z., & Neumark, Y. D. (2007). The protective effect of marriage on mortality in a dynamic society. *Annals of Epidemiology, 17*, 540–547.

Johnson, N. J., Backlund, E., Sorlie, P. D., & Loveless, C. A. (2000). Marital status and mortality: The national longitudinal mortality study. *Annals of Epidemiology, 10*, 224–238.

Kiecolt-Glaser, J. K., Glaser, R., Cacioppo, J. T., & Malarkey, W. B. (1998). Marital stress: Immunologic, neuroendocrine, and autonomic correlates. *Annals of the New York Academy of Sciences, 840*, 656–663.

Kiecolt-Glaser, J. K., & Newton, T. L. (2001). Marriage and health: His and hers. *Psychological Bulletin, 127*, 472–503.

Manzoli, L., Villari, P., Pirone, M., & Boccia, A. (2007). Marital status and mortality in the elderly: A systematic review and meta-analysis. *Social Science & Medicine, 64*, 77–94.

Orth-Gomer, K., Wamala, S. P., Horsten, M., Schenck-Gustafsson, K., Schneiderman, N., & Mittleman, M. A. (2000). Marital stress worsens prognosis in women with coronary heart disease: The Stockholm Female Coronary Risk Study. *Journal of the American Medical Association, 284*, 3008–3014.

Robles, T. F., & Kiecolt-Glaser, J. K. (2003). The physiology of marriage: Pathways to health. *Physiology and Behavior, 79*, 409–416.

Rozanski, A., Blumenthal, J. A., & Kaplan, J. (1999). Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation, 99*, 2192–2217.

Smith, T. W., Gallo, L. C., Goble, L., Ngu, L. Q., & Stark, K. A. (1998). Agency, communion, and cardiovascular reactivity during marital interaction. *Health Psychology, 17*, 537–545.

Smith, T. W., & Ruiz, J. M. (2002). Psychosocial influences on the development and course of coronary heart disease: Current status and implications for research and practice. *Journal of Consulting and Clinical Psychology, 70*, 548–568.

Troxel, W. M., Buysse, D. J., Hall, M., & Matthews, K. A. (2009). Marital happiness and sleep disturbances in a multi-ethnic sample of middle-aged women. *Behavioral Sleep Medicine, 7*, 2–19.

Troxel, W. M., Buysse, D. J., Matthews, K. A., Kravitz, H. M., Bromberger, J. T., Sowers, M., et al. (2010). Marital/cohabitation status and history in relation of sleep in midlife women. *Sleep, 33*, 862–863.

Troxel, W. M., Matthews, K. A., Gallo, L. C., & Kuller, L. H. (2005). Marital quality and occurrence of the metabolic syndrome in women. *Archives of Internal Medicine, 165*, 1022–1027.

Marriage Counseling

▶ Couple-Focused Therapy

Martyr Behavior

▶ Symptom Magnification Syndrome

Masculine Role

▶ Gender Role

Massage

▶ Massage Therapy

Massage Therapy

Beate Ditzen[1] and Tiffany Field[2]
[1]Division of Clinical Psychology and Psychotherapy, Department of Psychology, University of Zurich, Binzmuhlestrasse, Zurich, Switzerland
[2]Touch Research Institute, University of Miami, School of Medicine, Mailman Center for Child Development, Miami, FL, USA

Synonyms

Massage; Touch

Definition

Massage therapy (MT) has been defined as the manual manipulation of soft tissue intended to promote health and well-being (Moyer, Rounds, & Hannum, 2004). This definition includes therapeutic treatments of different duration and types of touch administered. Additionally, the sites of the body where the treatment is applied can vary depending on the treatment method and the desired effects.

Types of Massage Therapy

Today, several different massage methods are in use. The most popular forms include the following.

Swedish massage (or "classic massage"), the most common form of massage in Western countries, is based on five techniques termed "effleurage" (sliding or gliding), "petrissage" (kneading), "tapotement" (rhythmic tapping), friction, and vibration/shaking, which are applied to different parts of the body (e.g., back, shoulders, neck, legs, arms).

Reflexology massage is based on the theory that specific zones in the hands and the feet represent an image of the body. Manipulation techniques, such as pressure, applied to these areas are thought to improve functioning in related parts of the body (stomach, back, head, etc.).

Shiatsu, by origin a Japanese technique, uses pressure of fingers and palms but also rolling, brushing, vibrating, and grasping of the hands centered on the energy meridians of the whole body.

The Trager method, named after its founder, involves a gentle, rhythmic movement and touch of different body parts with the aim to relax the body, reduce pain, and increase mobility.

Effects of Massage Therapy

Efficacy varies considerably between different forms of MT and with regard to specific desired outcomes. Moreover, short-term effects of MT (e.g., immediate reduction in anxiety and stress) may differ from long-term benefits (e.g., reduced trait anxiety or depression). However, overall beneficial effects of MT have been documented for several psychiatric and medical conditions, including depression, posttraumatic stress disorder, eating disorders, attention-deficit disorder, chronic fatigue, fibromyalgia, back pain, migraine headache, postoperative pain, labor, rheumatoid arthritis, multiple sclerosis, spinal cord injury, autism, diabetes, asthma, HIV, and breast cancer, as well as in preterm neonates (Field, 2002; Field, Diego, & Hernandez-Reif, 2007; Moyer et al., 2004).

Underlying Mechanisms

Despite the beneficial overall effects of MT, the underlying mechanisms are only partly understood to date. General and specific effects of MT have been reported, and accordingly, the underlying biological mechanisms might vary. The stimulation of pressure receptors was suggested to promote vagal activity, and thereby to improve parasympathetic activity (Diego, Field, Sanders, & Hernandez-Reif, 2004). Beyond this, increases in serotonin, endorphins, and oxytocin might generally link MT to reduced anxiety, arousal, and depressiveness. Specific analgesic effects of MT have been interpreted in line with the gate control theory (Melzack & Wall, 1965) and with increased circulation and waste elimination in the affected/treated muscles.

Overall Evaluation

MT has proven effectiveness in a variety of psychiatric and medical conditions. The underlying mechanisms and long-lasting effects of MT remain to be identified.

Cross-References

▶ Gate Control Theory of Pain
▶ Oxytocin

- ▶ Pain
- ▶ Parasympathetic Nervous System (PNS)
- ▶ Serotonin

References and Readings

Diego, M. A., Field, T., Sanders, C., & Hernandez-Reif, M. (2004). Massage therapy of moderate and light pressure and vibrator effects on EEG and heart rate. *International Journal of Neuroscience, 114*(1), 31–44.

Field, T. M. (2002). Massage therapy. *Medical Clinics of North America, 86*(1), 163–171.

Field, T. M., Diego, M., & Hernandez-Reif, M. (2007). Massage Therapy Research. *Developmental Review, 27*, 75–89.

Melzack, R., & Wall, P. D. (1965). Pain mechanisms: A new theory. *Science, 150*(699), 971–979.

Moyer, C. A., Rounds, J., & Hannum, J. W. (2004). A meta-analysis of massage therapy research. *Psychological Bulletin, 130*(1), 3–18.

Masters of Public Health

- ▶ MPH (Masters of Public Health)

Maternal Stress

Kristin A. Long[1] and Melissa A. Alderfer[2]
[1]Department of Psychology, University of Pittsburgh, Pittsburgh, PA, USA
[2]Division of Oncology, The Children's Hospital of Philadelphia, Philadelphia, PA, USA

Definition

For the purpose of this entry, maternal stress is defined as the objective stressors and subjective feelings of distress or strain experienced by mothers related to having a child with a chronic illness.

Description

One out of every ten children in the USA experiences a chronic illness such as asthma, diabetes, or cancer. These illnesses may impair the child's functioning, decrease health-related quality of life, and/or contribute to emotional distress. However, because children are typically embedded within families, the impact of chronic illnesses extends beyond the individual child and includes parents and siblings. Despite changing gender roles over the past 50 years, mothers remain at the forefront of child rearing in many American families. Therefore, the reciprocal relationships between a child's illness or treatment and mothers' emotional health are particularly important.

Many aspects of childhood chronic illnesses may be stressful for mothers. Initially, the mother may be the first person to notice the child's symptoms and suspect a medical problem. She also may be responsible for seeking medical care for her child. Although a medical problem may have been suspected, the confirmation of a diagnosis may be shocking and devastating. Chronic illnesses may be life threatening or life limiting for the child and are typically unpredictable and incurable, requiring long-term management of symptoms and side effects of treatment. Complex treatment regimens may involve frequent and/or extended hospitalizations, invasive procedures, and daily medication management and adjunct treatments, such as physical therapy. Mothers often assume the burdens of managing the child's medical treatment at the hospital and at home. These responsibilities can alter the typical mother role – nurturing her child(ren)'s development may take a backseat to ensuring adherence to a treatment regimen. Furthermore, daily demands of treatment may constrain mothers' employment and career options, potentially limiting professional goals and altering self-perceptions. These employment restrictions, coupled with medical costs stemming from the child's diagnosis and treatment, may impact the family's financial stability. In sum, mothers of children with chronic illnesses experience a range of emotional, personal,

professional, and practical stressors related to their child's illness and well-being.

Evidence of Maternal Stress

Maternal stress in the face of a child's medical illness is ubiquitous and well documented. Feelings of distress and symptoms of depression and anxiety have been reported in mothers of children with cancer (Vrijmoet-Wiersma et al., 2008), cystic fibrosis (Berge & Patterson, 2004), irritable bowel disease (Mackner, Sisson, & Crandall, 2004), epilepsy (Rodenberg, Meijer, Dekovic, & Aldenkamp, 2005), congenital heart disease (Lawoko, 2007), and spina bifida (Vermaes, Janssens, Bosman, & Gerris, 2005). Across diagnoses, shock, fear, uncertainty, and helplessness are common experiences. For example, parents of children with kidney disease report unremitting feelings of uncertainty about prognosis; fatigue and distress arising from vigilant monitoring of symptoms and management of complex medical regimens; and feelings of anger, depression, stress, and frustration (Tong, Lowe, Sainsbury, & Craig, 2008). Parents of children with cancer report higher levels of anxiety, depression, and traumatic stress symptoms and lower quality of life than normative or comparison samples (Alderfer & Kazak, 2006). In addition to distress, mothers of children with cerebral palsy, for example, report more stressful life events than mothers of typically developing children (Rentinck, Ketelaar, Jongsmans, & Gorter, 2006), and higher levels of parenting stress have been reported by mothers of children with spina bifida (Vermaes, Gerris, & Janssens, 2007) and cancer (Alderfer & Kazak, 2006). The literature suggests that distress is generally greatest at the time of diagnosis, and for most mothers, improves with time.

Risk and Protective Factors

Although stress is common among mothers of children with chronic illnesses, there is considerable variability in the intensity and duration of distress experienced by mothers. Perceived amount of and satisfaction with social support has been found to be a powerful protective factor against distress for mothers of children with chronic illnesses including cerebral palsy (Rentinck et al., 2006), congenital heart disease (Lawoko, 2007), cancer (Vrijmoet-Wiersma et al., 2008), and spina bifida (Vermaes et al., 2005). Similarly, a positive family environment buffers against maternal distress in the context childhood chronic illnesses such as spina bifida (Vermaes et al., 2005) and cancer (Vrijmoet-Wiersma et al., 2008). In addition to social and family factors, mothers' positive beliefs about the illness, the medical team, and one's ability to manage the illness seem to be important for adjustment. For example, in the context of childhood cancer, more positive adjustment is reported when parents conceptualize the illness as a manageable, meaningful experience; report higher self-efficacy; and endorse greater trust in the healthcare team. Personal characteristics, such as trait anxiety and reliance upon disengaged or emotion-focused coping strategies, have been found to be associated with greater levels of distress (Alderfer & Kazak, 2006). Across illnesses, sociodemographic factors predicting greater maternal stress include minority status, unemployment, financial strain, lower levels of education, and higher levels of concurrent, non-illness-related stressors. Surprisingly, objective indices of illness severity generally do not influence maternal stress. In general, parents who endorse greater emotional strain at the time of their child's diagnosis continue to experience elevated distress even after the treatment ends.

Effects of Maternal Stress on the Family

Systems theories suggest that both the child's illness and maternal adjustment to illness-related stressors impact the family. For example, the literature shows that children with cancer report more anxiety, hopelessness, depression, internalizing problems, and externalizing problems when their mothers report more depression and anxiety, or poorer global mental health (Alderfer & Kazak, 2006). Healthy siblings of children with various medical diagnoses are also at elevated risk for psychological distress (Sharpe & Rossiter, 2002), and a small literature suggests that siblings of children with medical illnesses exhibit more behavioral problems and poorer overall adaptation when their mothers report higher levels of distress (Alderfer & Kazak, 2006).

The literature remains inconclusive about how maternal and paternal stress are related, with some work suggesting that congruence in coping style may be important for understanding the relationship between levels of parents' distress. Higher levels of anxiety and depression and discrepancies in perceptions of appropriate levels of emotional expression in mothers and fathers have been linked to greater marital distress in the context of childhood cancer (Alderfer & Kazak, 2006). At the family level, there are likely to be bidirectional relationships between maternal stress and aspects of family functioning (e.g., cohesion and communication), but more research is needed.

Effects of Maternal Stress on the Medical Condition

Pediatric illnesses are increasingly conceptualized using transactional models considering biological, psychological, and social influences. Accordingly, maternal stress is one psychosocial influence on the child's disease course. For example, the pediatric asthma literature shows that family characteristics, including caregiver mental health and perceived stress, impact outcomes such as asthma symptoms, healthcare utilization, and quality of life (Kaugars, Klinnert, & Bender, 2004). One possible mechanism is that maternal stress interferes with asthma management by reducing adherence to treatment protocols, increasing exposure to allergens or irritants (e.g., tobacco smoke) that exacerbate asthma symptoms, or limiting the degree to which treatment protocols are incorporated into daily routines.

In illnesses known to be exacerbated by stress (e.g., asthma, cardiovascular disease, diabetes), the relationship between maternal stress and child health outcomes may be mediated by stress-induced physiological changes to the child's immune and neuroendocrine systems. In pediatric asthma, the patient's reactivity to maternal stress can lead to alterations in the immune response resulting in increased inflammation or susceptibility to upper respiratory infections, leading to asthma exacerbations (Wright, Rodriguez, & Cohen, 1998).

Interventions Addressing Maternal Stress

Given increased stress in mothers of children with chronic illnesses, along with the impact of this stress on the family's psychosocial functioning and the child's medical status, interventions have been designed to alleviate mothers' distress and/or improve their adaptive functioning. Also, family interventions have been designed to provide education, promote adherence, improve family functioning, or reduce family members' distress.

A relatively small number of psychosocial interventions have been empirically evaluated. Results consistently show that educational interventions are not effective in reducing maternal distress across illnesses, while social support and cognitive-behavioral therapy interventions show more promise (Drotar, 2006). For example, mothers of children with cystic fibrosis, asthma, and diabetes enrolled in a social support intervention that paired mothers of children with the same diagnosis reported reduced distress. In childhood cancer, a meta-analysis evaluating behavioral, cognitive-behavioral, family systems, or psychoeducational interventions reported small but significant mean effect sizes for decreased parent distress and improved parent adjustment (Pai, Drotar, Zebracki, Moore, & Youngstrom, 2006).

Summary

As primary caretakers, mothers of children with chronic illnesses experience a range of practical and emotional stressors related to their child's medical diagnosis and treatment, on top of normative childrearing, employment, and/or household management tasks. These sustained responsibilities can take an emotional toll. Mothers of children with chronic illnesses endorse strong negative emotions including shock, fear, and helplessness, along with elevated levels of depression, anxiety, and traumatic stress symptoms. Positive family environments and greater perceived social support may buffer against maternal distress, and at the same time, maternal stress may influence the psychosocial functioning of other family members. Maternal stress also can impact the child's illness course via less effective illness management behaviors

and/or changes to the immune or endocrine systems. Interventions targeting maternal distress have modest empirical support, but more research is needed.

Cross-References

- ▶ Caregiver/Caregiving and Stress
- ▶ Family Social Support
- ▶ Family Systems Theory
- ▶ Family, Caregiver
- ▶ Stress, Caregiver

References and Readings

Alderfer, M. A., & Kazak, A. E. (2006). Family issues when a child is on treatment for cancer. In R. T. Brown (Ed.), *Comprehensive handbook of childhood cancer and sickle cell disease: A biopsychosocial approach*. New York: Oxford University Press.

Berge, J. M., & Patterson, J. M. (2004). Cystic fibrosis and the family: A review and critique of the literature. *Families, Systems & Health, 22*, 74–100.

Drotar, D. (2006). *Psychological interventions in childhood chronic illness*. Washington, DC: Americal Psychological Association.

Kaugars, A. S., Klinnert, M. D., & Bender, B. G. (2004). Family influences on pediatric asthma. *Journal of Pediatric Psychology, 29*, 475–491.

Lawoko, S. (2007). Factors influencing satisfaction and well-being among parents of congenital heart disease children: Development of a conceptual model based on a literature review. *Scandinavian Journal of Caring Sciences, 21*, 106–117.

Mackner, L. M., Sisson, D. P., & Crandall, W. V. (2004). Review: Psychosocial issues in pediatric inflammatory bowel disease. *Journal of Pediatric Psychology, 29*, 243–257.

Pai, A. L. H., Drotar, D., Zebracki, K., Moore, M., & Youngstrom, E. (2006). A meta-analysis of the effects of psychological interventions in pediatric oncology on outcomes of psychological distress and adjustment. *Journal of Pediatric Psychology, 31*, 978–988.

Ratliffe, C. E., Harrigan, R. C., Haley, J., Tse, A., & Olson, T. (2002). Stress in families with medically fragile children. *Issues in Comprehensive Pediatric Nursing, 25*, 167–188.

Rentinck, I. C. M., Ketelaar, M., Jongsmans, M. J., & Gorter, J. W. (2006). Parents of children with cerebral palsy: A review of factors related to the process of adaptation. *Child: Care, Health and Development, 33*, 161–169.

Rodenberg, R., Meijer, A. M., Dekovic, M., & Aldenkamp, A. P. (2005). Family factors and psychopathology in children with epilepsy: A literature review. *Epilepsy & Behavior, 6*, 488–503.

Sharpe, D., & Rossiter, L. (2002). Siblings of children with a chronic illness: A meta-analysis. *Journal of Pediatric Psychology, 27*, 699–710.

Tong, A., Lowe, A., Sainsbury, P., & Craig, J. C. (2008). Experiences of parents who have children with chronic kidney disease: A systematic review of qualitative studies. *Pediatrics, 121*, 349–360.

Vermaes, I. P. R., Gerris, J. R. M., & Janssens, J. M. A. M. (2007). Parents' social adjustment in families of children with spina bifida: A theory-driven review. *Journal of Pediatric Psychology, 32*, 1214–1226.

Vermaes, I. P. R., Janssens, J. M. A. M., Bosman, A. M. T., & Gerris, J. R. M. (2005). Parents' psychological adjustment in families of children with Spina Bifida: A meta-analysis. *BMC Pediatrics, 5*, 32.

Vrijmoet-Wiersma, C. M. J., et al. (2008). Assessment of parental psychological stress in cancer: A review. *Journal of Pediatric Psychology, 33*, 694–706.

Wright, R. J., Rodriguez, M., & Cohen, S. (1998). Review of psychosocial stress and asthma: An integrated biopsychosocial approach. *Thorax, 53*, 1066–1074.

Matthews, Karen

Rebecca C. Thurston
Department of Psychiatry, School of Medicine, University of Pittsburgh, Pittsburgh, PA, USA

Biographical Information

Karen Matthews was born in Los Angeles, CA. She received her undergraduate degree in psychology from the University of California, Berkeley, and her Ph.D. in psychology from the

University of Texas at Austin, where she worked with David C. Glass, studying the Type A behavior pattern. Her first faculty position was at Kansas State University, where she joined in 1976 as an Assistant Professor. She later joined the faculty of the University of Pittsburgh's Psychiatry Department in 1979, jointly appointed to the Departments of Psychology and Epidemiology. She was promoted to Professor in 1989 and named as Distinguished Professor in 2009.

Matthews investigates the psychosocial characteristics of individuals and their early life experiences that ultimately lead to coronary atherosclerosis and hypertension later in life. Her work has focused on two stages of the life span when change in cardiovascular risk reliably occurs: adolescence and midlife. Change can provide an optimal setting for observing how hormonal and other biologic processes, social roles, and psychological characteristics interact to accelerate an individual's cardiovascular risk. Her approach has integrated knowledge and methods derived from psychology, psychiatry, epidemiology, and cardiology.

Major Accomplishments

Matthews has made influential contributions to several areas of health psychology. Her early work was in the Type A behavior pattern, and her detailed analysis of psychological perspectives of Type A later became a *Citation Classic*. She has since been a major contributor to the understanding of how stress impacts the cardiovascular system. She has made key contributions to understanding how psychological stress serves as a potential link between personality characteristics, such as hostility and Type A; social and environmental factors, such as low socioeconomic status; and the development of cardiovascular disease. In work that has been ongoing for over three decades, she has proven herself to be one of the earliest health psychologists to incorporate a lifespan perspective in health psychology, tracing the early origins of stress reactivity and stressful environments to the development of cardiovascular risk later in life.

Matthews has also conducted seminal work in understanding cardiovascular risk in women. Her work, supported by a MERIT award by the National Heart, Lung, and Blood Institute (NHLBI), has included elucidating gender differences in cardiovascular and neuroendocrine stress reactivity, including the role of the hypothalamic-pituitary-gonadal axis in stress reactivity in women. She has also made major contributions to the understanding of the epidemiology of cardiovascular risk in women. With epidemiologist Lewis Kuller, she has played a central role in elucidating the symptomatic and cardiovascular changes occurring with the menopausal transition. This work has helped form the basis of one of the largest and currently ongoing multiethnic studies of women transitioning through menopause, for which she is a principal investigator. Reflective of the multidisciplinary nature of this work, Matthews' approach represents a unique blend of concepts and methods from psychophysiology, developmental psychobiology, and epidemiology.

Matthews has had a major role in establishing health psychology as a formal subdiscipline of psychology. With Steve Manuck and Al Shapiro (deceased), she designed one of the first cardiovascular behavioral medicine research training programs, which began in 1983 (HL07560 through 2013). This program was one of the earliest formal high-quality training programs in health psychology. Matthews also served as Editor-in-Chief of *Health Psychology* from 1990 to 1995, with the goal of making the journal the most important health psychology journal in the field and negotiating with the American Psychological Association (APA) to make it the primary APA health psychology journal. Further, she provided the perspectives of a health psychologist on several NIH advisory panels, including the National Heart, Lung, Blood Advisory Council and the Center for Scientific Review Advisory Committee (Chair). She also was President of the American Psychosomatic Society (1990–1991), the second female President of the Society and one of few Presidents with a Ph.D. by that time. As the field matured, she continued to be instrumental in providing an infrastructure for advanced training and

resources for health psychology research as Director of the Pittsburgh Mind-Body Center. In this role, she directed scientists at Carnegie Mellon University and the University of Pittsburgh to identify the important common pathways of risk across multiple diseases.

Reflective of her distinguished work, Matthews has received awards from diverse organizations, including American Heart Association Established Investigator; Cardiovascular Research Award from the North American Menopause Society and Pfizer; American Psychosomatic Society President's Award; Award for Distinguished Contributions to Health Psychology, Division 38 of APA; Award for Significant Research Contribution from the Society of Pediatric Psychology; Distinguished Scientist Award from the Society of Behavioral Medicine; and APA Award for Distinguished Scientific Applications of Psychology. She became a member of the Institute of Medicine in 2002 and was awarded the Philosophiae Doctor Honoris Causa from the University of Helsinki in 2007.

Cross-References

- ▶ Cardiovascular Disease
- ▶ Hypertension
- ▶ Psychosocial Characteristics
- ▶ World Health Organization (WHO)

References and Readings

Chen, E., Matthews, K. A., & Boyce, W. T. (2002). Socioeconomic differences in children's health: How and why do these relationships change with age? *Psychological Bulletin, 128*, 295–329.

Gallo, L. C., & Matthews, K. A. (2003). Understanding the association between socioeconomic status and physical health: Do negative emotions play a role? *Psychological Bulletin, 129*, 10–51.

Low, C. A., Thurston, R. C., & Matthews, K. A. (2010). Psychosocial factors in the development of heart disease in women: Current research and future directions. *Psychosomatic Medicine, 72*, 842–854.

Matthews, K. A. (1982). Psychological perspectives on the type A behavior pattern. *Psychological Bulletin, 91*, 293–323.

Matthews, K. A. (1989). Are sociodemographic variables markers for psychological determinants of health? *Health Psychology, 8*, 641–648.

Matthews, K. A. (2005). Psychological perspectives on the development of coronary heart disease. *The American Psychologist, 60*, 783–796.

Matthews, K. A., Glass, D. C., Rosenman, R. H., & Bortner, R. W. (1977). Competitive drive, pattern A, and coronary heart disease: A further analysis of some data from the Western Collaborative Group Study. *Journal of Chronic Diseases, 30*, 489–498.

Matthews, K. A., & Gump, B. B. (2002). Chronic work stress and marital dissolution increase risk of posttrial mortality in men from the Multiple Risk Factor Intervention Trial. *Archives of Internal Medicine, 162*, 309–315.

Matthews, K. A., Gump, B. B., Harris, K. F., Haney, T. L., & Barefoot, J. C. (2004). Hostile behaviors predict cardiovascular mortality among men enrolled in the Multiple Risk Factor Intervention Trial. *Circulation, 109*, 66–70.

Matthews, K. A., Kamarck, T. W., Hall, H., Strollo, P. J., Owens, J. F., Buysse, D. J., et al. (2008). Blood pressure dipping and sleep disturbance in African-American and Caucasian men and women. *American Journal of Hypertension, 21*, 826–831.

Matthews, K. A., Katholi, C. R., McCreath, H., Whooley, M. A., Williams, D. R., Zhu, S., et al. (2004). Blood pressure reactivity to psychological stress predicts hypertension in the CARDIA study. *Circulation, 110*, 74–78.

Matthews, K. A., Kuller, L. H., Chang, Y., & Edmundowicz, D. (2007). Premenopausal risk factors for coronary and aortic calcification: A 20-year follow-up in the healthy women study. *Preventive Medicine, 45*, 302–308.

Matthews, K. A., Kuller, L. H., Sutton-Tyrrell, K., & Chang, Y. F. (2001). Changes in cardiovascular risk factors during the perimenopause and postmenopause and carotid artery atherosclerosis in healthy women. *Stroke, 32*, 1104–1111.

Matthews, K. A., Kuller, L. H., Wing, R. R., Meilahn, E. N., & Plantinga, P. (1996). Prior to use of estrogen replacement therapy, are users healthier than nonusers? *American Journal of Epidemiology, 143*, 971–978.

Matthews, K. A., Meilahn, E., Kuller, L. H., Kelsey, S. F., Caggiula, A. W., & Wing, R. R. (1989). Menopause and risk factors for coronary heart disease. *The New England Journal of Medicine, 321*, 641–646.

Matthews, K. A., Owens, J. F., Salomon, K., Harris, K. F., & Berga, S. L. (2005). Influence of hormone therapy on the cardiovascular responses to stress of postmenopausal women. *Biological Psychology, 69*, 39–56.

Matthews, K. A., Wing, R. R., Kuller, L. H., Meilahn, E. N., Kelsey, S. F., Costello, E. J., et al. (1990). Influences of natural menopause on psychological characteristics and symptoms of middle-aged healthy women. *Journal of Consulting and Clinical Psychology, 58*, 345–351.

Matthews, K. A., & Woodall, K. L. (1988). Childhood origins of overt Type A behaviors and cardiovascular

reactivity to behavioral stressors. *Annals of Behavioral Medicine, 10*, 47–59.
Matthews, K. A., Woodall, K. L., Kenyon, K., & Jacob, T. (1996). Negative family environment as a predictor of boys' future status on measures of hostile attitudes, interview behavior, and anger expression. *Health Psychology, 15*, 30–37.
Midei, A. J., & Matthews, K. A. (2011). Interpersonal violence in childhood as a risk factor for obesity: A systematic review of the literature and proposed pathways. *Obesity Reviews, 12*, e159–e172.
Raikkonen, K., & Matthews, K. A. (2008). Do dispositional pessimism and optimism predict ambulatory blood pressure during school days and nights in adolescents? *Journal of Personality, 76*, 605–630.
Thurston, R. C., Kuller, L. H., Edmundowicz, D., & Matthews, K. A. (2010). History of hot flashes and aortic calcification among postmenopausal women. *Menopause, 17*, 256–261.
Thurston, R. C., & Matthews, K. A. (2009). Racial and socioeconomic disparities in arterial stiffness and intima media thickness among adolescents. *Social Science & Medicine, 68*, 807–813.

Maximal Aerobic Capacity Test

► Maximal Exercise Stress Test

Maximal Aerobic Power Test

► Maximal Exercise Stress Test

Maximal Exercise Heart Rate

James Turner
School of Cancer Sciences, The University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

HR_{max}; Tachycardia

Definition

Maximum exercise heart rate refers to the highest or fastest rate at which the heart can beat during exercise and is expressed as the number of heartbeats per unit of time, usually beats per minute (bpm).

Heart rate increases linearly with oxygen consumption and therefore exercise intensity. Maximal exercise heart rate is most accurately recorded upon volitional exhaustion and termination of a maximal exercise stress test. Maximal exercise heart rate declines with aging; therefore, several formulae are also available to estimate maximal heart rate on the basis of age. The most widely used formula, $HR_{max} = 220 - \text{age (in years)}$, provides a rough approximation. However, due to lower maximal heart rates among well-trained individuals, several population-specific formulas also exist. In behavioral research, estimated or measured maximal exercise heart rate can be used to prescribe exercise (e.g., walking speed) based upon the relationship between oxygen consumption, heart rate, and exercise intensity.

References and Readings

McArdle, W. D., Katch, F. I., & Katch, V. L. (2001). *Exercise physiology. Energy, nutrition and human performance* (5th ed.). Baltimore, MD: Lippincott Williams & Wilkins.

Maximal Exercise Stress Test

James Turner
School of Cancer Sciences, The University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Cardiac stress test; Exercise tolerance test; Fitness test; Graded exercise test; Maximal aerobic capacity test; Maximal aerobic power test; Maximal oxygen uptake test; Symptom-limited exercise test; VO_2max test

Definition

An exercise stress test is a general term used to describe the assessment of cardiorespiratory

functional capacity for either health and fitness reasons, or for clinical and diagnostic purposes. Such tests, primarily conducted on a motorized treadmill or stationary cycle ergometer, involve exercising at several workloads or exercise intensities/difficulties, during which cardiovascular and respiratory measurements are recorded. Depending on the rationale for testing, the type of test employed, and the health and fitness status of the individual taking part, exercise stress tests might terminate at volitional exhaustion, upon observation of a particular clinical symptom, or at some other predetermined outcome measure.

Description

Maximal oxygen uptake (VO_2max) is the accepted measure of cardiorespiratory fitness and is the product of cardiac output and arterial-venous oxygen difference (i.e., the ability to extract and utilize oxygen from the blood). During exercise, cardiac output is markedly increased above resting levels and shows further elevations when the intensity or difficulty of exercise is increased. Using a treadmill-based exercise test, for example, increases in exercise intensity are achieved by increasing the speed and gradient of the treadmill belt. VO_2max is measured using closed-circuit spirometry: analyzing the fractions of expired oxygen and carbon dioxide. VO_2max is reached when, despite subsequent increases in exercise intensity, oxygen consumption plateaus. VO_2max is expressed as milliliters or liters of oxygen consumed per minute ($ml/l \cdot min^{-1}$) and is often adjusted for body mass or fat-free mass (i.e., dividing oxygen consumption by kilograms). This value is dependent on the health and fitness status of individuals, age, gender, body size and composition, as well as genetic factors.

Exercise stress tests can be maximal or submaximal. Maximal tests, in which individuals exercise with verbal encouragement until they are unable to continue (i.e., perceived volitional exhaustion), offer the most accurate assessments of VO_2max. Submaximal tests are terminated at an endpoint, for example, when heart rate reaches a predetermined percentage of an estimated age-predicted maximum (see ▶ Maximal Exercise Heart Rate). The oxygen consumption upon termination of submaximal tests is extrapolated to predict the value at the predicted maximum heart rate (i.e., VO_2max). The decision to employ submaximal exercise testing is usually based upon the availability of equipment and possibly medical supervision. In clinical and diagnostic exercise testing, maximal or submaximal exercise tests can be used to assess the severity of disease and prognosis of patients with known or suspected coronary artery disease. Tests in this scenario often include more detailed physiological measurements in addition to expired gas fractions and heart rate measurement, such as blood pressure assessments, electrocardiogram recordings, and use of pharmacological agents and radioactive tracers. In the health and fitness industry, or in behavioral research, exercise stress tests can be used to characterize individuals in terms of cardiorespiratory fitness, or used to accurately prescribe exercise training interventions.

Cross-References

- ▶ Cardiac Output
- ▶ Cardiovascular Psychophysiology: Measures
- ▶ Pulmonary Function

References and Readings

McArdle, W. D., Katch, F. I., & Katch, V. L. (2001). *Exercise physiology. Energy, nutrition and human performance* (5th ed.). Baltimore: Lippincott Williams & Wilkins.

Whaley, M. H., Brubaker, P. H., & Otto, R. M. (Eds.). (2006). *ACSM's guidelines for exercise testing and prescription* (7th ed.). Baltimore: Lippincott Williams & Wilkins.

Maximal Exercise Test

- ▶ Graded Exercise

Maximal Oxygen Uptake Test

▶ Maximal Exercise Stress Test

MBMD

▶ Millon Behavioral Medicine Diagnostic (MBMD)

McGill Pain Index

▶ McGill Pain Questionnaire

McGill Pain Questionnaire

Tavis S. Campbell, Jillian A. Johnson and Kristin A. Zernicke
Department of Psychology, University of Calgary, Calgary, AB, Canada

Synonyms

McGill pain index

Definition

The McGill Pain Questionnaire (MPQ) was developed in 1971 by pain researcher Ronald Melzack. It was devised as a system for subjectively assessing pain experiences along three dimensions. Scoring of this inventory provides a quantitative index of pain (Melzack, 1975).

- The first dimension, *sensory quality*, focuses on the variations that occur in the sensation of pain, such as temporal, spatial, pressure, and thermal properties.
- The second dimension, *affective quality*, highlights the fear, tension, and autonomic properties that are part of the pain experience.
- The third dimension, *evaluative quality*, refers to the words that describe the subjective overall intensity of the pain experience.

In addition to these three dimensions of pain, the MPQ is subdivided into four parts (Melzack, 1975).

- Part 1 consists of front and back drawings of the human body. Individuals indicate the location of pain by marking the drawing with an "I" or "E" to indicate if the pain experienced is internal or external.
- Part 2 consists of 20 sets of descriptive words to describe the pain experience. The individual circles the word in each set that best describes their experience of pain. The words are ordered from least to most painful.
- Part 3 asks how the patient's pain changes with time (continuous, intermittent, etc.).
- Part 4 measures the strength or intensity of the pain on a five-point scale from "mild" to "excruciating". The Present Pain Index (PPI) score is derived from this section of the scale.

The McGill Pain Questionnaire is among the most widely used brief assessment tools for pain.

Cross-References

▶ Pain

References and Readings

Melzack, R. (1975). The McGill pain questionnaire: Major properties and scoring methods. *Pain, 1*, 277–299.
Melzack, R. (1987). The short-form McGill pain questionnaire. *Pain, 30*, 191–197.
Melzack, R., & Torgerson, W. S. (1971). On the language of pain. *Anesthesiology, 34*, 50–59.

Mean (Average)

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Arithmetic mean; Average

Definition

The mean of a set of data points is defined as their sum divided by the total number of data points. The actual name for this parameter is the "arithmetic mean," a term that distinguishes it unambiguously from the geometric mean, which is defined quite differently. Having said this, the geometric mean is seldom used in behavioral medicine, and it is a reasonable assumption that the "default" value of the term "mean" in the literature is the arithmetic mean.

The mean is a measure of central tendency that is most appropriately used for continuous data, i.e., variables that are measured on a continuous, uninterrupted scale and can take any value on that scale. Relevant examples are height, weight, blood pressure, and heart rate. Other common measures of central tendency are the median and the mode. Each of these can provide a more meaningful summary of a data set depending on the characteristics of the data, and care should be taken when deciding which measure to present when reporting the data. If there is any doubt in this choice, a good practice is to present more than one of them.

Cross-References

- ▶ Central Tendency
- ▶ Median
- ▶ Mode

Meaning (Purpose)

Login S. George and Crystal L. Park
Department of Psychology, University of Connecticut, Storrs, CT, USA

Synonyms

Beliefs; Goals; Purpose; Worldview

Definition

Most definitions of meaning encompass a sense of understanding, significance, and purpose in life. Meaning refers to how an individual perceives and conceptualizes life, one's self, and relationships between the two. Meaning consists of global and situational aspects. Global meaning refers to individuals' general orienting systems and consists of beliefs, goals, and subjective feelings of purpose or meaning in life. Situational meaning refers to the meaning associated with specific events or contexts. Situational meaning is the result of the interaction between specific contexts and individuals' global meaning systems (e.g., the degree to which a specific event violates or fits an individual's general beliefs regarding the world).

Description

Research has established links between each of the components of global meaning (beliefs, goals, and a subjective sense of meaning) with general levels of physical and psychological health (see Park, 2011, for a review). Global beliefs regarding the amount of control individuals have over the circumstances in their lives have been shown to be positively related to health and well-being (see Lachman & Firth, 2004, for a review). Other global beliefs, such as beliefs in a just world, appear to buffer stressors in both lab-based and workplace settings. The effects of global beliefs on health appear to be the result of both physiological and behavioral pathways. For example, a high sense of control appears to moderate physical and psychological reactivity to daily stressors and has also been found to be inversely related to aortic calcification. Many studies have found that performance of health behaviors mediated the relationship between control beliefs and health benefits, suggesting that behavioral pathways are involved in the relationship between global beliefs and health.

Research on the relationship between global goals and health suggests that various dimensions of goals have different relations with health.

Goals have multiple dimensions such as structure, process, content, pursuit, attainment, maintenance, and disengagement, and relationships with health may vary across them (Maes & Karoly, 2005). Preliminary research suggests that being committed to goals that are perceived as attainable, feeling that one is making progress toward his/her goals, and experiencing less goal ambivalence and goal conflict are related to better health.

The subjective experience of meaning in life seems to be related to better physical and psychological health as well. Studies have found an inverse relationship between meaning and mortality rates, and a positive relationship with meaning and self-rated health and psychological well-being. A sense of meaning in life appears to affect well-being in both direct and indirect pathways. A higher level of meaning in life has been related to better autonomous functioning, lower heart rate, and decreased heart rate reactivity. It has also been related to the performance of health-promoting activities in various samples such as Anglo women, Japanese adults, and cardiac outpatients.

The impact of meaning on health seems to be particularly salient during crises or highly stressful experiences perceived to entail a high probability of damage or loss (e.g., receiving a diagnosis of serious illness). Crises may disrupt global meaning, which can diminish one's sense of meaning or purpose in life. Global beliefs affect how individuals deal with and recover from stressful situations. For example, beliefs regarding control have been found to moderate the impact of economic hardship on health and to predict lower levels of pain, stress, fatigue, and blood pressure in individuals suffering from rheumatoid arthritis. A high sense of control may help individuals make more adaptive appraisals of stressful situations (e.g., "this is a situation I can handle") and enable persistence in coping efforts.

Crises may also disrupt individuals' global goals. Studies suggest that the extent to which individuals perceive an event to disrupt or hinder their goals affect physical and psychological well-being. The higher the perceived disruption of goals, the more negative the impact. The subjective sense of meaning in life seems to play a role in the impact of crises as well. A high sense of meaning can serve as a resource that allows individuals to maintain and achieve higher health-related quality of life amidst stressful situations.

Current literature on meaning and its impact on emotional and physical health are largely correlational, which renders it impossible to draw conclusions about causal links between meaning and health. It is possible that there is a bidirectional relationship between the two or that the direction of causality is the reverse of what has been commonly presumed (i.e., that the better one's physical and psychological health, the more likely one is to perceive meaning in life). It is also possible that unidentified third variables (confounds) are influencing the relationship between the two.

Cross-References

- ▶ Beliefs
- ▶ Coping

References and Readings

Lachman, M. E., & Firth, K. M. P. (2004). The adaptive value of feeling in control during midlife. In O. G. Brim, C. D. Ryff, & R. C. Kessler (Eds.), *How healthy are we? A national study on well-being at midlife* (pp. 320–349). Chicago: The University of Chicago Press.

Lachman, M. E. (2006). Control: Perceived control over aging-related declines: Adaptive beliefs and behaviors. *Current Directions in Psychological Science, 15*, 282–286.

Maes, S., & Karoly, P. (2005). Self-regulation assessment and intervention in physical health and illness: A review. *Applied Psychology: An International Review, 54*, 267–299.

Park, C. L. (2010). Making sense of the meaning literature: An integrative review of meaning making and its effects on adjustment to stressful life events. *Psychological Bulletin, 136*, 257–301.

Park, C. L. (2011). Meaning, spirituality, and growth: Protective and resilience factors in health and illness. In A. Baum, T. Revenson, & J. Singer (Eds.), *Handbook of health psychology* (2nd ed.). London: Psychology Press.

Wong, P., & Fry, P. (1998). *The human quest for meaning: A handbook of psychological research and clinical applications*. Mahwah, NJ: Lawrence Erlbaum Associates.

Measures of Perceived Control of Health

▶ Multidimensional Health Locus of Control Scales

Measures of Quality of Life

Natalie E. Bustillo
Department of Psychology, University of Miami, Coral Gables, FL, USA

Synonyms

Measures of well-being; Quality of life assessments; Quality of life instruments

Definition

General quality of life (QOL) refers to how individuals function in the various life domains (The WHOQOL Group, 1998). General QOL encompasses how well individuals are able to carry their daily responsibilities (e.g., work and family). Factors that contribute to general QOL include physical well-being, emotional well-being, social well-being, and functional well-being. Health-related QOL refers to physical and mental well-being as it relates to specific diseases (Centers for Disease Control and Prevention [CDC], 2000). Health-related QOL includes how well individuals are able to cope with the physical limitations that result from specific diseases as treatments may be accompanied by decrements in QOL. Health-related QOL is also referred to as disease-specific QOL.

Description

General QOL

The following are examples of commonly used measures that have been developed to assess general QOL.

The Medical Outcomes Study 36-Item Short-Form Health Survey (SF-36; Ware & Sherbourne, 1992) consists of eight QOL domains. The eight domains are: (1) limitations in physical activities because of health problems, (2) limitations in social activities because of physical or emotional problems, (3) limitations in usual role activities because of physical health problems, (4) bodily pain, (5) general mental health (psychological stress and well-being), (6) limitations in usual role activities because of emotional problems, (7) vitality (energy and fatigue), and (8) general health perceptions. The measure is a subjective, self-report measure that is easy to administer. The questions have Likert scale response options.

The Medical Outcomes Study SF-36 was used to develop the Medical Outcomes Study 12-Item Short-Form Health Survey SF-12 (SF-12; Ware, Kosinski, & Keller, 1996). The SF-12 consists of two subscales that each contains 12 items. The first subscale is Physical Component Summary and the second subscale is the Mental Component Summary. The advantage of using the SF-12 instead of the SF-36 is that it takes less time to administer due to having fewer items and may be more beneficial for research studies characterized by long assessment batteries. The reliability of the Physical Component Summary ranged between 0.864 and 0.890, while the reliability of the Mental Component Summary ranged between 0.760 and 0.774 among populations in the United States and United Kingdom.

The World Health Organization Quality of Life (WHOQOL) instrument was designed to assess cross-cultural QOL (World Health Organization [WHO], 1993). The WHOQOL-BREF was later developed as a brief version of the original measure in order to make its use more convenient (The WHOQOL Group, 1998). The WHOQOL-BREF consists of 26 items that tap into the domains of physical health (e.g., sleep, pain, and energy), psychological health (e.g., happiness, concentration, and feeling positive about self), social relationships (e.g., social support and sexual life), and environment (e.g., safety, financial resources, and home

environment). The measure also contains one overall health question and one overall QOL question. The WHOQOL-BREF has been developed in several countries across the world, including United States, China, Czech Republic, Indonesia, Poland, Russia, and Thailand. The development of the WHOQOL-BREF allows for the comparability of general QOL across various cultures.

Health-Related QOL

Disease-specific QOL can also be assessed for various illnesses. The following are examples of measures that have been used to evaluate the health-related QOL of cancer, heart disease, and diabetes.

The Memorial Symptom Assessment Scale Short Form (MSAS-SF) is a shortened version of the Memorial Symptom Assessment Scale (Chang, Hwang, Feuerman, Kasimis, & Thaler, 2000). The MSAS-SF consists of 32 items that tap into physiological and psychological symptoms. Each item contains two parts. The first part of the item assesses whether or not the symptom was present in the past 7 days. The second part of the item assesses the distress caused by the symptom (if the symptom was indeed present). The MSAS-SF yields a physical symptom subscale score, psychological symptom subscale score, and global distress index. Although the measure does not assess QOL specific to any one specific illness, psychological and physical symptom severity is an important component of health-related QOL. The MSAS-SF was validated among a sample of cancer survivors and demonstrated adequate reliability. The Cronbach's alpha coefficients for the composite scores were 0.82, 0.76, and 0.80 for the physical symptom subscale score, psychological symptom subscale score, and global distress index, respectively.

The Functional Assessment of Cancer Therapy (FACT) scale was developed to assess general cancer-related QOL for survivors receiving cancer treatment (FACT-G; Cella et al., 1993). The FACT-G was validated using a sample of breast, lung, and colorectal cancer and consisted of 29 items. The FACT-G yields five composite scores: physical well-being, social/family well-being, relationship with doctor, emotional well-being, and functional well-being. A total score may derived by calculating the sum of the five subscales. Although the FACT-G was originally designed to assess general cancer QOL, new scales have been developed in order to assess cancer-specific QOL. Specifically, the FACT instrument is available for survivors of breast cancer, bladder cancer, bone marrow transplant, primary brain tumors, colorectal cancer, cervical cancer, head and neck cancer, lung cancer, ovarian cancer, prostate cancer, and HIV infection. The FACT is commonly used to assess cancer-specific QOL and has been translated to seven languages (Cella & Bonomi, 1996).

The MacNew Heart Disease Health-related Quality of Life (MacNew) Questionnaire was developed to evaluate health-related QOL in patients with coronary heart disease (Höfer, Lim, Guyatt, & Oldridge, 2004). The MacNew is comprised of 27 items and yields three domains: physical limitations, emotional function, and social function. The internal consistency of the MacNew reflects adequate reliability (Cronbach's alpha ranges from 0.93 to 0.95 for the three domains). The MacNew is available in various languages, including English, Dutch, Farsi, German, and Spanish.

The Diabetes-39 is a measure designed to assess the disease-specific QOL of individuals diagnosed with diabetes (Boyer & Earp, 1997). The scale is comprised of 39 items and 5 subscales: (1) energy and mobility, (2) diabetes control, (3) anxiety and worry, (4) social burden, and (5) sexual functioning. The scale has been found to correlate with the well-established Sf-36 instrument, suggesting the measure is a valid tool for assessing diabetes-specific QOL.

Overall, several measures have been developed in order to assess general and health-related QOL. Assessing QOL may aid in further understanding how individuals are impacted by diseases, both physically and psychologically. Advances in the field of QOL measurement may play a role in the development of effective interventions to treat individuals who have experienced decrements in QOL due to illness.

Cross-References

- ▶ Health-Related Quality of Life
- ▶ Physical Well-being
- ▶ Quality of Life: Measurement

References and Readings

Aaronson, N. K., Ahmedzai, S., Bergman, B., Bullinger, M., Cull, A., Duez, N. J., et al. (1993). The European organization for research and treatment of cancer QLQ-C30: A quality-of-life instrument for use in international clinical trials in oncology. *Journal of the National Cancer Institute, 85*, 365–376.

Boyer, J. G., & Earp, J. A. L. (1997). The development of an instrument for assessing the quality of life of people with diabetes: Diabetes-39. *Medical Care, 35*, 440–453.

Cella, D. F., & Bonomi, A. E. (1996). The functional assessment of cancer therapy (FACT) and functional assessment of HIV infection (FAHI) quality of life measurement system. In B. Spilker (Ed.), *Quality of life and pharmacoeconomics in clinical trials* (2nd ed., pp. 203–214). New York: Raven Press.

Cella, D. F., Tulsky, D. S., Gray, G., Sarafian, B., Linn, E., Bonomi, A., et al. (1993). The functional assessment of cancer therapy scale: Development and validation of the general measure. *Journal of Clinical Oncology, 11*(3), 570–579.

Centers for Disease Control and Prevention. (2000). *Measuring healthy days*. Atlanta: CDC.

Chang, V. T., Hwang, S. S., Feuerman, M., Kasimis, B. S., & Thaler, H. T. (2000). The memorial symptom assessment scale short form (MSAS-SF). *Cancer, 89*(5), 1162–1171.

El Achhab, Y., Nejjari, C., Chikri, M., & Lyoussi, B. (2008). Disease-specific health-related quality of life instruments among adults diabetic: A systematic review. *Diabetes Research and Clinical Practice, 80*(2), 171–184.

EuroQol GRoup. (1990). EuroQol: A new facility for the emasurement of health-realted quality of life. *Health Policy, 16*, 199–208.

Groenvold, M., Petersen, M. A., Aaronson, N. K., Arraras, J. I., Blazeby, J. M., Bottomley, A., et al. (2006). The development of the EORTC QLQ-C15-PAL: A shortened questionnaire for cancer patients in palliative care. *European Journal of Cancer, 42*(1), 55–64.

Höfer, S., Lim, L., Guyatt, G., & Oldridge, N. (2004). The MacNew heart disease health-related quality of life instrument: A summary. *Health and Quality of Life Outcomes, 2*, 3.

Jolly, M., Pickard, A. S., Wilke, C., Mikolaitis, R. A., Teh, L., McElhone, K., et al. (2010). Lupus-specific health outcome measure for US patients: The LupusQoL-US version. *Annals of the Rheumatic Diseases, 69*, 29–33.

Litwin, M. S., Hays, R. D., Fink, A., Ganz, P. A., Leake, B., & Brook, R. H. (1998). The UCLA prostate cancer index: Development, reliability, and validity of a health-related quality of life measure. *Medical care, 36*(7), 1002–1012.

The WHOQOL Group. (1998). Development of the world health organization WHOQOL-BREF quality of life assessment. *Psychological Medicine, 28*(3), 551–558.

Ware, J. E., Kosinski, M., & Keller, S. D. (1996). A 12-item short form health survey: Construction of scales and preliminary tests of reliability and validity. *Medical Care, 34*, 220–233.

Ware, J. E., & Sherbourne, C. D. (1992). The MOS 36-item short form health survey (SF-36). *Medical Care, 30*, 473–483.

Wei, J. T., Dunn, R. L., Litwin, M. S., Sandler, H. M., & Sanda, M. G. (2000). Development and validation of the expanded prostate cancer index composite (EPIC) for comprehensive assessment of health-related quality of life in men with prostate cancer. *Urology, 56*(6), 899–905.

World Health Organization. (1993). WHOQoL study protocol (MNH7PSF/93.9). Geneva, Switzerland: WHO

Measures of Well-Being

- ▶ Measures of Quality of Life

Medial Mammillary Nucleus

- ▶ Hypothalamus

Medial Preoptic Area

- ▶ Hypothalamus

Median

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

The median is a measure of central tendency. It a value such that, when the data are arranged

in order of magnitude, an equal number of data points lie above and below it. For any odd number of data points (e.g., 9), obtaining the median is straightforward (here, it is the fifth value). For an even number of data points (e.g., 10), it is the mean of the middle two observations (here, the mean of the fifth and sixth).

In comparison to the mean of a data set, the median has the advantage that it is less influenced by outliers. Consider the following data set: 3, 3, 4, 4, 5, 5, 100. The median is 4, a value which provides a good indication of the central tendency of the data set. The mean of the data set is 17.71, a value that is intuitively much less indicative of the data set's central tendency.

Cross-References

- ▶ Central Tendency
- ▶ Mode

Mediating Cognitions

- ▶ Cognitive Mediators

Mediators

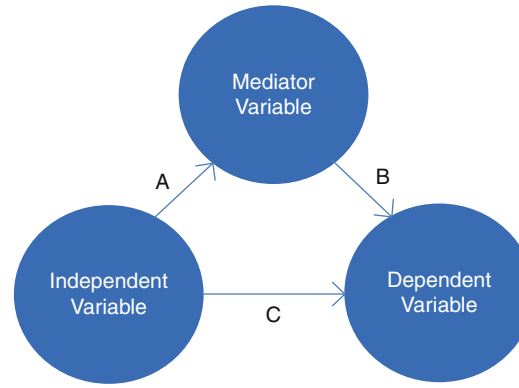
Chris Zehr
Department of Health Studies and Gerontology, University of Waterloo, Waterloo, ON, Canada

Synonyms

Intermediate variable

Definition

A mediator is a variable that serves to explain the nature of the relationship between an independent variable and a dependent variable or between a predictor and criterion variable (Baron and Kenny, 1986; MacKinnon, Krull, & Lockwood, 2000). It provides an indirect means by which the independent variable may affect the dependent variable. Specifically, a mediator influences the dependent variable while being influenced by the independent variable itself (MacKinnon et al., 2000). In experimental research, researchers manipulate a mediator to more clearly understand the means through which a causal effect is operating.



Mediators, Fig. 1 A meditational model as depicted in Baron and Kenny (1986)

A simple meditational model is depicted in Fig. 1 as shown in Baron and Kenny (1986). As illustrated, path (C) represents a direct effect of the independent variable on the dependent variable when no mediator is accounted for. In contrast, the causal path linking the independent variable to the mediator (A) and the mediator to the dependent variable (B) represents an indirect effect of the independent variable on the dependent variable via the mediator (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001). In this case, the independent variable influences the dependent variable through the mediator. If controlling for the mediator reduces the direct effect that the independent variable has on the dependent variable to zero, it can be said that complete mediation is present. However, if after controlling for the mediator, the effect of the independent variable on the dependent variable is significantly reduced but remains statistically different from zero, it can be said that partial mediation has occurred (Judd & Kenny, 1981). In behavioral medicine, as in any science, researchers use mediation analyses to test causal

pathways. For example, one could examine whether levels of various biomarkers such as C-reactive protein mediate the relation between hostility and acute coronary syndrome, by measuring hostility, the biomarkers and the disease endpoint, and implementing the Baron and Kenny test.

Cross-References

- ▶ Causal Diagrams
- ▶ Intermediate Variable

References and Readings

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 51*(6), 1173–1182.

Judd, C. M., & Kenny, D. A. (1981). Process analysis: Estimating mediation in treatment evaluations. *Evaluation Review, 5*(5), 602–619.

Kraemer, H. C., Stice, E., Kazdin, A., Offord, D., & Kupfer, D. (2001). How do risk factors work together? Mediators, moderators, and independent, overlapping, and proxy risk factors. *The American Journal of Psychiatry, 158*(6), 848–856.

MacKinnon, D. P., Krull, J. L., & Lockwood, C. M. (2000). Equivalence of the mediation, confounding and suppression effect. *Prevention Science, 1*(4), 173–181.

Medical Agreement

- ▶ Clinical Agreement

Medical Decision-Making

William Whang
Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Definition

Medical decision-making is the process by which a diagnosis or treatment plan is formulated from the available test information, often with incorporation of known patient preferences.

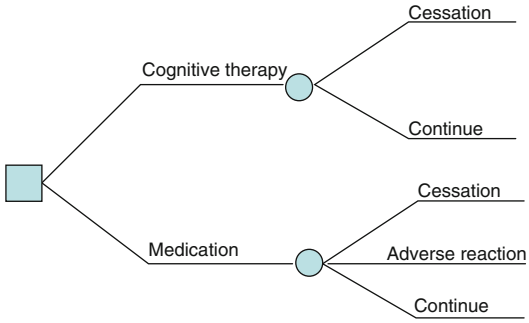
Description

Diagnoses and treatment choices by health-care providers and their patients are made under uncertainty. Medical decision-making can describe the process by which a diagnosis is derived from the retrieved facts at hand (Mark, 2008). It can also define the choices that are available for treatment or prevention of illness and attempt to maximize the expected health benefits given the probable outcomes of each choice.

Most clinicians use cognitive shortcuts, or heuristics, in order to guide them in their decision-making (Mark, 2008). For instance, the representativeness heuristic involves assessing whether a patient's clinical features match the representative features of different diagnostic hypotheses. Medical decision-making often involves the generation of initial hypotheses and testing of these hypotheses against data obtained through diagnostic tests.

The accuracy of diagnostic tests is defined in relation to a reference standard that is meant to reflect the true condition of a patient (Weinstein & Fineberg, 1980). For a test with binary results, it is useful to employ a 2 by 2 table to illustrate the concepts related to test assessment. The *sensitivity* of a test represents the extent to which a test identifies patients with disease and is defined as the proportion of patients with true disease who test positive (in the table, sensitivity equals [true positive]/[true positive + false negative]). *Specificity* represents the extent to which a test identified patients without disease and is defined as the proportion of patients without disease who test negative ([true negative]/[true negative + false positive]). The sensitivity and specificity of a diagnostic test can be incorporated in calculation of the *positive predictive value* of a test or the proportion of patients with a positive test result who actually have disease ([true positive]/[true positive + false positive]).

		Disease	
		Present	Absent
Test result	Positive	True positive	False positive
	Negative	False negative	True negative



Medical Decision-Making, Fig. 1 Hypothetical decision tree of treatment for smoking cessation

Decision analysis is a formal method that illustrates and estimates the likely outcomes associated with different medical choices and has been used frequently in cost-effectiveness research (Weinstein & Fineberg, 1980). In Fig. 1, a hypothetical choice between medication and behavior therapy for smoking cessation is outlined in a decision tree. The probability of each outcome, and the health "units" associated with each (e.g., quality-adjusted life years), can be estimated in order to assess the choice that is more likely to result in improved health.

Cross-References

► Clinical Decision-Making

References and Readings

Mark, D. B. (2008). Chapter 3. Decision-making in clinical medicine. In A. S. Fauci, E. Braunwald, D. L. Kasper, S. L. Hauser, D. L. Longo, J. L. Jameson, & J. Loscalzo (Eds.), *Harrison's principles of internal medicine* (17th ed.). New York: McGraw-Hill.

Weinstein, M. C., & Fineberg, H. V. (1980). *Clinical decision analysis*. Philadelphia: Saunders.

Medical Dialogue

► Doctor-Patient Communication: Why and How Communication Contributes to the Quality of Medical Care

Medical Interaction

► Doctor-Patient Communication: Why and How Communication Contributes to the Quality of Medical Care

Medical Outcomes Study

William Whang
Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Definition

The Medical Outcomes Study (MOS) was a 2-year observational study of patients with chronic conditions, conducted in a cross-sectional phase among 22,462 patients and in a longitudinal phase among 2,349 patients (Tarlov et al., 1989). One of its two main purposes was to analyze how outcomes vary depending on factors such as the type of delivery system in which a patient receives care, clinician specialty training, and intensity of resource use. Another main purpose was to develop practical tools for monitoring patient outcomes and their determinants. For instance, the MOS core survey includes 116 items that measure quality of life in terms of physical, mental, and general health. In addition, the MOS Short-Form General Health Survey has been used in 12-item, 20-item, and 36-item versions for quality of life assessment in numerous conditions (Stewart et al., 1989). One of the initial publications from the MOS defined the impact of depression on patient functioning and well-being (Wells et al., 1989).

References and Readings

Stewart, A. L., Greenfield, S., Hays, R. D., et al. (1989). Functional status and well-being of patients with chronic conditions. Results from the Medical Outcomes Study. *Journal of the American Medical Association, 262*, 907–913.

Tarlov, A. R., Ware, J. E., Jr., Greenfield, S., Nelson, E. C., Perrin, E., & Zubkoff, M. (1989). The Medical Outcomes Study. An application of methods for monitoring the results of medical care. *Journal of the American Medical Association, 262*, 925–930.

Wells, K. B., Stewart, A., Hays, R. D., et al. (1989). The functioning and well-being of depressed patients. Results from the Medical Outcomes Study. *Journal of the American Medical Association, 262*, 914–919.

Medical Psychology

Pamela S. King
Pediatric Prevention Research Center, Department of Pediatrics, Wayne State University School of Medicine, Detroit, MI, USA

Synonyms

Clinical health psychology

Definition

Medical psychology has historically been defined as the branch of psychology concerned with the application of psychological principles to the practice of medicine. Medical psychology shares with the fields of health psychology and behavioral medicine an interest in the ways in which biological, psychological, and social factors interact to influence health. Medical psychologists use psychological theories and principles in order to improve the health and well-being of patients with physical illness. They are clinical psychologists who work in hospitals, medical centers, and health care facilities. Medical psychologists use techniques of psychotherapy, behavior modification, and cognitive, interpersonal, and family therapy to help patients manage chronic illness, reduce physical symptoms of disease or treatment, and manage emotional aspects of their illness.

Recently, the term "medical psychologist" has also been used to describe psychologists who prescribe medication. In the United States, psychologists in New Mexico, Louisiana, and all branches of the armed forces can prescribe medication after passing an examination and receiving board certification (e.g., American Board of Medical Psychology, Louisiana State Board of Medical Examiners). In order to receive board certification, these professional psychologists must have a doctoral degree in clinical psychology from an accredited institution, and must pursue postdoctoral training in medical psychology (including specialized training in psychopharmacology). The use of the term "medical psychologist" to describe psychologists with prescription privileges has been controversial. Division 38 of the American Psychological Association has stated that "the term 'medical psychologist' should not be equated with having prescription authority," citing the historical usage of the term "medical psychologist" (which had not previously been associated with medication prescription by psychologists) and the potential for confusion among psychologists, healthcare professionals, and the general public.

Cross-References

- ▶ American Psychological Association Division 38 (Health Psychology)
- ▶ Behavioral Medicine
- ▶ Health Psychology
- ▶ Psychologist

References and Readings

Academy of Medical Psychology. Retrieved from http://www.amphome.org.

Statement on the use of the term Medical Psychology from Division 38 of the American Psychological Association (Health Psychology). Retrieved from http://www.health-psych.org/MedPsych.cfm.

Medical Sociology

Richard Peter
Institute of Epidemiology and Medical Biometry, University of Ulm, Ulm, Germany

Definition

Medical sociology is a theory-based discipline which applies theory and methods from social sciences and epidemiology to analyze the impact of micro (individual)- and macro (societal)-social environments on disease onset and on the course of disease. Core aspects of interest concerning the micro-social level are social relationships in different areas of life, i.e., the workplace, the family, and their associations with health. One special aspect of a micro-social environment is the doctor-patient relationship. The focus on the macro level is on social inequalities in health, i.e., the distribution of morbidity and mortality along social classes, and on social inequalities in health care and health-care systems. More recent developments in medical sociology attempt to explain the macro-social associations with the help of micro-social factors, e.g., social inequality in health by higher levels of work-related stress in lower social class subjects.

Cross-References

- ▶ Occupational Health
- ▶ Social Class
- ▶ Social Stress
- ▶ Work-Related Stress

References and Readings

Cockerham, W. C. (2011). *Medical sociology* (12th ed.). Boston: Pearson International Edition.
Marmot, M. G., & Wilkinson, R. (Eds.). (2006). *Social determinants of health*. Oxford: Oxford University Press.

Medical Specialty

- ▶ Gerontology

Medical Utilization

Karl-Heinz Ladwig
Institut für Epidemiologie, Neuherberg, Germany

Synonyms

Adherence; Compliance

Definition

Medical utilization describes the degree to which a population or a given group uses medical and dental services, procedures, facilities, and practitioners in a specified time period. The utilization rates of health services are often based on exposure variables like the number of outpatient visits, number of physicians consulted, use of emergency services, hospitalization, and total health care costs that are quantitatively assessed as the number of services used per year or per 1,000 patients eligible for a medical service.

Description

Medical utilization research assesses not only the extent of medical service use, but also analyzes the necessity, appropriateness, and efficiency of medical services. In a hospital setting, this analysis may include the appropriateness or possible delay of decisions regarding admissions, duration of stay, and discharge practices. The concept is tied to the concepts of ▶ adherence and ▶ compliance which describe the degree to which a patient correctly follows medical advice.

Medical utilization is largely dependent on societal and personal determinants (Andersen & Newman, 1973). The societal determinants ("supply-side") encompass mainly resources and the particular organization that a national health care system offers to its consumers. In highly developed western countries, the total volume of resources relative to the population may be similar. Nevertheless, there may also be particular substantial differences which may not be addressed sufficiently enough in utilization research so far.

The progress in health research may alter patterns of medical utilization by itself. For example, a progress in diagnostic tools may decrease the number of patients with manifest acute myocardial infarction when referred to a chest pain unit but may increase the admission of sub-acute cases (McManus et al., 2011). Progress in antipsychotic medication may result in a shift from custodial care to treatment on an outpatient basis thus decreasing hospital admission of mental health conditions (Andersen & Newman, 1973).

Medical utilization can also be viewed as a type of individual behavior ("demand-side"). Psycho-pathological health conditions strongly impact medical over- and underutilization:

Medical *overutilization* is often seen in patients who suffer from medically unexplained symptoms, and the concept has received attention as part of the diagnosis of somatization disorder. These patients consult multiple physicians for the same problem ("doctor shopping") but remain dissatisfied and distressed with their medical care. Characteristically, they deny any psychological influence on the symptoms, resist psychiatric referral, and remain unreassured after receiving somatic evaluation results (Barsky, Orav, & Bates, 2005).

Medical *underutilization* is highly prevalent in mental health patients. Only 50% of bipolar patients may be adherent to their prescribed medication. Side effects and dissatisfaction with treatment are strong predictors of underutilization in these patients (DiMatteo, Lepper, & Croghan, 2000). Underutilization of basic treatment requirements are also often seen in patients with severe somatic disease conditions (e.g., coronary heart disease) and co-morbid depressed mood. Here, successful anti-depressive treatment may improve adherence to all therapeutic options (e.g., cardiac medications) required for the patient (Rieckmann et al., 2006).

Individual characteristics of patients beyond clinical features of psychopathology may also impact medical utilization patterns. It has been sufficiently demonstrated in population-based studies that females (less-convincing education level and older age) are independent unspecific contributors to medical utilization (Ladwig, Marten-Mittag, Formanek, & Dammann, 2000). Furthermore, inadequate health information, attitudes, health beliefs, and the quality of the physician-patient relationship are strong determinants of medical utilization (O'Donohue & Cucciare, 2005).

Cross-References

- ▶ Adherence
- ▶ Compliance

References and Readings

Andersen, R., & Newman, J. F. (1973). Societal and individual determinants of medical care utilization in the United States. *Milbank Memorial Fund Quarterly Health and Society, 51*(1), 95–124.

Barsky, A. J., Orav, E. J., & Bates, D. W. (2005). Somatization increases medical utilization and costs independent of psychiatric and medical comorbidity. *Archives of General Psychiatry, 62*(8), 903–910.

DiMatteo, M. R., Lepper, H. S., & Croghan, T. W. (2000). Depression is a risk factor for noncompliance with medical treatment: Meta-analysis of the effects of anxiety and depression on patient adherence. *Archives of Internal Medicine, 160*(14), 2101–2107.

Ladwig, K. H., Marten-Mittag, B., Formanek, B., & Dammann, G. (2000). Gender differences of symptom reporting and medical health care utilization in the German population. *European Journal of Epidemiology, 16*(6), 511–518.

McManus, D. D., Gore, J., Yarzebski, J., Spencer, F., Lessard, D., & Goldberg, R. J. (2011). Recent trends in the incidence, treatment, and outcomes of patients with STEMI and NSTEMI. *American Journal of Medicine, 124*(1), 40–47.

O'Donohue, W., & Cucciare, M. (2005). Pathways to medical utilization. *Journal of Clinical Psychology in Medical Settings, 12*(2), 185–197.

Rieckmann, N., Gerin, W., Kronish, I. M., Burg, M. M., Chaplin, W. F., Kong, G., et al. (2006). Course of depressive symptoms and medication adherence after acute coronary syndromes: An electronic medication monitoring study. *Journal of the American College of Cardiology, 48*(11), 2218–2222.

Medically Underserved Populations

▶ Health Disparities

Medically Unexplained Physical Symptoms

▶ Somatoform Disorders

Medically Unexplained Symptoms

Benjamin H. Natelson
Department of Pain Medicine & Palliative Care, Beth Israel Medical Center and Albert Einstein College of Medicine, Bronx, NY, USA

Synonyms

Unexplained symptoms; Unexplained patient complaints

Definition

A set of patient complaints that with careful history, physical examination, and lab testing has no apparent medical cause is thus unexplained. When this happens, some physicians jump to a psychiatric explanation in lieu of no other – often leaving the patient stigmatized. Instead, a group of experts has arrived at operational definitions of symptom-based syndromes to allow for diagnosis and research. When the symptom is predominantly fatigue, severe enough to reduce activity substantially, and lasting at least 6 months, the diagnosis is idiopathic chronic fatigue. When the symptom is predominantly body-wide pain, also lasting many months, and the patient is tender on palpation, the diagnosis is fibromyalgia. When the pain is limited to face and jaw, the diagnosis is temporomandibular joint syndrome, and when the pain is in the abdomen and associated with constipation and/or diarrhea, the diagnosis is irritable bowel syndrome. The fact that patients with one of these syndromes often have at least one other has supported the psychiatric interpretation of these being variants of somatization disorder (Barsky & Borus, 1999). However, data are accumulating that differences do exist among these syndromes (Evengård et al., 1998; Weaver, Janal, Aktan, Ottenweller, & Natelson, 2010), and focus on pathogenesis has gradually shifted from the focal areas in the body affected to the brain with the notion that nociceptive systems are dysregulated (Schweinhardt, Sauro, & Bushnell, 2008). Functional brain imaging studies show patients with widespread pain and chronic fatigue, respectively, to have much larger responses to painful or fatigue-producing stimuli than control subjects (Cook et al., 2004; Lange et al., 2005) – suggesting a sensitized response to these stimuli. Another major factor influencing thinking about these syndromes in the physician community is the emergence of nationally approved drugs that effectively treat the symptoms of some of these disorders. All of this work suggests that, at least for some patients, the pathophysiological process underlying these syndromes is not the same. In addition to these newly available drugs, patients report substantial symptomatic improvement with a program of gentle physical conditioning (Hauser et al., 2010); similarly, providing patients behavioral ways to cope with chronic symptoms via cognitive behavioral therapy (Price, Mitchell, Tidy, & Hunot, 2008) also can help with symptom reduction and improve health-related quality of life.

Cross-References

- ▶ Brain, Imaging
- ▶ Fatigue
- ▶ Fibromyalgia
- ▶ Functional Somatic Syndromes
- ▶ Health-related Quality of Life
- ▶ Pain

References and Readings

Barsky, A. J., & Borus, J. F. (1999). Functional somatic syndromes. *Annals of Internal Medicine, 130*(11), 910–921.

Cook, D. B., Lange, G., Ciccone, D. S., Liu, W. C., Steffener, J., & Natelson, B. H. (2004). Functional imaging of pain in patients with primary fibromyalgia. *Journal of Rheumatology, 31*(2), 364–378.

Evengård, B., Nilsson, C. G., Lindh, G., Lindquist, L., Eneroth, P., Fredrikson, S., et al. (1998). Chronic fatigue syndrome differs from fibromyalgia. No evidence for elevated substance P levels in cerebrospinal fluid of patients with chronic fatigue syndrome. *Pain, 78*(2), 153–155.

Hauser, W., Klose, P., Langhorst, J., Moradi, B., Steinbach, M., Schiltenwolf, M., et al. (2010). Efficacy of different types of aerobic exercise in fibromyalgia syndrome: A systematic review and meta-analysis of randomised controlled trials. *Arthritis Research & Therapy, 12*(3), R79.

Lange, G., Steffener, J., Bly, B. M., Cook, D. B., Christodoulou, C., Liu, W. C., et al. (2005). Chronic fatigue syndrome affects verbal working memory: A BOLD fMRI study. *NeuroImage, 26*(2), 513–524.

Price, J. R., Mitchell, E., Tidy, E., & Hunot, V. (2008). Cognitive behaviour therapy for chronic fatigue syndrome in adults. *Cochrane Database of Systematic Reviews, 3*, CD001027.

Schweinhardt, P., Sauro, K. M., & Bushnell, M. C. (2008). Fibromyalgia: A disorder of the brain? *The Neuroscientist, 14*(5), 415–421.

Weaver, S. A., Janal, M. N., Aktan, N., Ottenweller, J. E., & Natelson, B. H. (2010). Sex differences in plasma prolactin response to tryptophan in chronic fatigue syndrome patients with and without comorbid fibromyalgia. *Journal of Women's Health, 19*(5), 951–958.

Medication Compliance

- ▶ Adherence

Medication Event Monitoring Systems

Jessica Haberer
Medicine and Center for Global Health, Massachusetts General Hospital, Harvard University, Boston, MA, USA

Definition

Medication event monitoring systems are devices and reporting mechanisms for keeping track of pill-taking behavior.

Description

Introduction

Estimates from the World Health Organization indicate that patients in developed countries only take about 50% of prescribed medicine for chronic diseases like hypertension and diabetes. Many factors contribute to incomplete adherence, including side effects, stigma, forgetfulness, depression, and poverty. Behavioral science plays a critical role in understanding and improving adherence.

A variety of approaches to measuring adherence are discussed in the topic Adherence. This topic focuses on medication event monitoring systems, which are devices and reporting mechanisms for keeping track of pill-taking behavior.

The First-Generation Monitor

The first well-known device is the Medication Event Monitoring System (MEMS^{TM}), which was introduced in 1986. With MEMS, a specialized bottle cap contains a microelectronic switch, a clock, and a memory chip; the cap fits on multiple standard and large sized medication bottles. Every opening and closing of the MEMS cap records a time and date stamp that is stored and later downloaded to a computer via a USB cable. The data can be displayed graphically or in spreadsheet format for analysis. MEMS data has been used primarily in research projects, as well as some clinical scenarios.

There are several advantages to this type of electronic medication adherence monitoring. First, the time and date stamp provides a dose-by-dose estimate of adherence, thus enabling an understanding of patterns of pill-taking behavior. Most other adherence measures, such as self-report and pill counts, provide average estimates that do not distinguish between contiguous and intermittent lapses in adherence, which may have significantly different effects on the effectiveness of therapy.

MEMS caps, however, also have significant limitations. First, a pill bottle opening does not necessarily equate to pill ingestion. Second, the cap can only be used with a single bottle, which makes the use of pill boxes and other adherence aids impossible and prevents monitoring of multiple medications with a single device. Third, patients may take out multiple pills at a time for later dosing (i.e., a "pocket dose"). Similarly, patients may open the bottle without taking a pill (i.e., a "curiosity event"). Subjective reports may be used to correct for these shortcomings; however, the accuracy of these self-reports is unknown and dilutes the objectivity of MEMS.

Another significant limitation of MEMS is the fact that the data must be periodically downloaded onto a computer for analysis. Researchers and clinicians are typically unaware of adherence problems until regularly scheduled visits, which may occur weeks to months after the lapse(s) occurred. For some conditions, like HIV/AIDS, lapses as short as a few doses may have irreversible clinical consequences, and real-time data transmission could allow for interventions prior to these clinical consequences.

Second-Generation Monitors

In the past few years, many new devices have been introduced for medication event monitoring, which add a variety of features to build on the first-generation monitor. See Table 1 for a summary description of which features are associated with each of these devices.

Several second generation monitors offer multiple compartments, each with a microelectronic sensor, to allow for monitoring of multiple medications. Another approach to multiple medications is to store all medications in the device and dispense one or more pills at predefined time periods. These devices serve as monitors by recording when the dispensing compartment is opened to retrieve the pill(s). An alternate system for dose monitoring is to equip blister packs with microelectronics, such that a wire is broken when the pill is removed from its compartment. Dosing can then be recorded through a radio-frequency identification (RFID) signal. A similar yet less sophisticated RFID system monitors the removal and insertion of entire blister packs into a device.

Many second-generation monitors offer reminder features and have liquid crystal displays indicating which pills to take, as well as other clinically relevant information, such as potential side effects or health education messages. A newer version of the MEMS cap displays the number of openings to help track if a dose was taken. Other devices have auditory alarms and/or visual cues to remind patients when to take their medications.

Another common feature of second-generation monitors is the capacity for data transmission via the phone or internet. While some devices require cable connections, others are wireless, using Bluetooth to transmit to cell phone networks. These devices, thus, allow for real-time monitoring and the potential for real-time adherence interventions. Some systems have options to send phone calls, emails, and/or text messages to the patient, as well as friends, family members, health-care providers, and/or researchers to facilitate adherence support.

Despite these new features, this second generation of medication event monitors still has limitations. First, some devices have more pill capacity than others, and those with larger capacity have less portability. Second, few devices have options for liquid medications or medication applicators. Additionally, pocket doses and curiosity events remain a limitation for most devices, although some have compartment covers that limit each opening to one dose of medicine.

Third Generation

A third generation of monitors seeks to document actual ingestion of a medication. The ability to detect actual drug ingestion is highly

Medication Event Monitoring Systems, Table 1 Summary of second-generation device features[a]

Device	Web site	Time/ date stamp	Multiple compartments per device	Cover for unit dosing	Educational features (e.g., LCD)	Auditory and/ or visual reminders	Transmission of data for reminders and monitoring		Capacity for liquids/applicators
							Phone/web	Wireless	
MEMS™	www.aardexgroup.com	X			X				
Med-eMonitor™	www.informedix.com	X	X		X	X	X		
MedSignals™	www.medsignals.com	X	X		X	X	X		
MedMinder™	www.medminder.com	X	X			X	X		
Epill MedSmart™	www.epill.com	X	X	X	X	X	X		
Ubox	www.innovatorsinhealth.org	X	X	X		X			
Med-ic®	www.med-ic.com	X	X	X					
Cypak	www.cypak.com	X	X	X					
Wisepill	www.wisepill.com	X				X	X	X	X
SimPill®	www.simpill.com	X				X	X	X	
GlowCaps™	www.vitality.net	X				X	X	X	

[a]Due to the rapidly evolving nature of technology development, this table may not be exhaustive. It is meant to give examples of devices currently in development or commercially available. All information on device features was obtained through the device web sites.

Medication Event Monitoring Systems, Table 2 Summary of third-generation device features[a]

Device	Web site	Time/date stamp	Wireless transmission of monitoring data	Metabolite detection	Ingestible marker
XoutTB	www.xoutb.info	X	X	X	
MagneTrace	users.ece.gatech.edu/~mghovan	X	X		Magnet
IDCap	www.etectbio.com	X	X		Antennae
ChipSkin™	www.proteusbiomed.com	X	X		Microchip
SMART™	www.xhale.com	X		X	Drug taggant

[a]Due to the rapidly evolving nature of technology development, this table may not be exhaustive. It is meant to give examples of devices currently in development or commercially available. All information on device features was obtained through the device web sites

appealing; however, such an approach may seem invasive and inappropriate. Ultimately, acceptability remains to be seen. These third-generation monitors use two approaches: metabolite detection and documentation of taggant ingestion (a taggant is chemical or physical marker added to materials to allow various forms of testing). See Table 2 for a summary description of which features are associated with each of several third-generation devices.

In a monitoring system using metabolic detection, the patient urinates on a filter paper after medication ingestion. If a specific metabolite is present, a code is revealed on the filter paper. The patient then sends a text message with that code to document medication ingestion and in return automatically receives mobile phone airtime credit as an incentive. The Taggant ingestion systems involve a variety of materials and technologies, including a biocompatible antenna, magnet, and digestible microchip. A taggant is attached to each pill and detected by a small, wearable patch, wristwatch, or handheld reader as it travels through the digestive tract. Data is then transmitted in real time via cell phone networks. Another approach combines both metabolite detection and taggant ingestion. In this system, a taggant is released as the medication reaches the stomach and intestines. Following absorption, the taggant is metabolized to a volatile breath marker, and a breath sample is analyzed and data stored in the monitoring device for later assessment.

While these technologies are intriguing, the sensitivity and specificity of the ingestion detection mechanisms needs to be established. Results, for instance, may vary with pharmacokinetics. Additional complications include the requirement of collaboration with drug manufacturers and the need to adhere to the adherence monitoring strategy (e.g., patients must wear the taggant detection device or urinate on a filter paper) over time.

Summary and Conclusions

All of the above described medication event monitoring devices provide objective information for understanding pill-taking behavior, which is critical for both interpretation of clinical trials and evaluation of treatment effectiveness. Emerging methods and strategies offer improvements in objective discrimination of adherence, especially those with the capacity for real-time data transmission. This approach allows for proactive prevention of the consequences of nonadherence, which has previously not be possible. Any detection strategy, however, is only as good at the accompanying intervention.

A final, important consideration for all medication event monitors, regardless of their generation, is cost. In the United States alone, medication nonadherence is estimated to cost hundreds of billions of dollars annually. Moreover, studies in both developed and developing settings indicate the cost-effective nature of adherence interventions (e.g., for HIV/AIDS

treatment). Further research is needed into the effectiveness of medication event monitors as adherence interventions, and policies must be established for public and private insurance coverage.

Cross-References

► Adherence

References and Readings

Ailinger, R. L., Black, P. L., & Lima-Garcia, N. (2008). Use of electronic monitoring in clinical nursing research. *Clinical Nursing Research, 17*(2), 89–97.

Goldie, S. J., Paltiel, A. D., Weinstein, M. C., Losina, E., Seage, G. R., 3rd, Kimmel, A. D., et al. (2003). Projecting the cost-effectiveness of adherence interventions in persons with human immunodeficiency virus infection. *American Journal of Medicine, 115*(8), 632–641.

Haberer, J. E., Kahane, J., Kigozi, I., Emenyonu, N., Hunt, P., Martin, J., et al. (2010). Real-time adherence monitoring for HIV antiretroviral therapy. *AIDS and Behavior, 14*(6), 1340–1346.

Nachega, J. B., Leisegang, R., Bishai, D., Nguyen, H., Hislop, M., Cleary, S., et al. (2010). Association of antiretroviral therapy adherence and health care costs. *Annals of Internal Medicine, 152*(1), 18–25.

New England Healthcare Institute (NEHI). (2009). *Thinking outside the pillbox: A system-wide approach to improving patient medication adherence for chronic disease*. Cambridge: Author.

World Health Organization. (2003). *Adherence to long-term therapies: Evidence for action*. Geneva: Author.

Meditation

Alan M. Delamater
Department of Pediatrics, University of Miami Miller School of Medicine, Miami, FL, USA

Synonyms

Attention training; Concentration; Contemplation; Mental training

Definition

Meditation refers to a variety of mental techniques designed to focus the mind, improve well-being, and facilitate spiritual development. Most of the world's religions include some type of meditation technique. In recent years, mediation techniques have also become increasingly recognized as helpful in the treatment of many physiological and emotional disorders, as well as promoting wellness in healthy individuals.

There are two major types of meditation techniques: concentration and insight. Concentrative meditations involve the development of single-pointed attention, i.e., concentration on a single object. In insight meditation, the object is to focus the attention on changing objects as they occur in the mind on a moment to moment basis. A good example of concentrative meditation is transcendental meditation in which an individual focuses on a mantra, paying attention to the silent repetition of the mantra in the mind while screening out other stimuli. As concentration deepens, feelings of calm or tranquillity are experienced. Research has shown that when individuals practice this type of meditation, they experience a restful hypometabolic state in which their respiration, heart rate, blood pressure, muscle tension, and other indicators of sympathetic nervous system activation all decrease. This state of hypometabolic alertness has been termed the relaxation response by Herbert Benson. The relaxation response can be reliably elicited by the repetition of a mental stimulus (e.g., a mantra) while the individual adopts a relaxed mental attitude in a quiet environment.

Concentration is also an important component of insight meditation, but is used in insight meditation to focus on changing objects rather than a single object of attention. A key factor in insight meditation is the development of bare attention and mindfulness. Bare attention is observing phenomenon as they are, without comment, evaluation, or judgment. Mindfulness is the application of bare attention to changing objects as they occur in each moment, with an attitude of acceptance so that there is no clinging to objects that

are desirable and there is no aversion to objects that are not desirable. In mindfulness practice, one does not identify with objects of attention. The goal is to increase the frequency of noticing objects as they arise and pass away in consciousness. As mindfulness becomes stronger and more sustained, insights about the nature of the mind and reality develop, including three fundamental characteristics of existence: impermanence, suffering, and the concept that the self is a creation. Insight or vipassana meditation has its origins in Buddhism and was developed over 2,500 years ago; vipassana is the Pali word meaning to clearly see the nature of. In recent years, mindfulness has been incorporated into psychotherapeutic techniques. In addition to promoting spiritual development, research has shown that mindfulness can be helpful for stress reduction in healthy people as well as people with chronic health conditions and be an important aid in the treatment of a variety of psychological disorders.

Cross-References

- ▶ Mindfulness
- ▶ Prayer
- ▶ Relaxation
- ▶ Transcendental Meditation

References and Readings

Benson, H. (2000). *The relaxation response–updated and expanded (25th anniversary edition)*. New York: Avon.

Bohlmeijer, E., Prenger, R., Taal, E., & Cuijpers, P. (2010). The effects of mindfulness-based stress reduction therapy on mental health of adults with a chronic medical disease: A meta-analysis. *Journal of Psychosomatic Research, 68*(6), 539–544.

Goldstein, J. (1976). *The experience of insight: A simple and direct guide to Buddhist meditation*. Boulder, CO: Shambhala.

Goleman, D. (1996). *The meditative mind: Varieties of meditative experience*. New York: Tarcher.

Hofmann, S. G., Sawyer, A. T., Witt, A. A., & Oh, D. (2010). The effect of mindfulness-based therapy on anxiety and depression: A meta-analytic review. *Journal of Consulting and Clinical Psychology, 78*(2), 169–183.

Menarche

▶ Puberty

Menopausal Hormone Therapy

▶ Hormone Treatment

Menopause

Suzana Drobnjak
Department of Psychology, University of Zurich, Binzmuehlestrasse, Switzerland

Definition

Menopause is a natural event in a woman's life. It describes the end of menstruation or, more precisely, a permanent cessation of the primary functions of the human ovaries. Menopause can only be determined retrospectively, once 12 months have lapsed with no menstrual cycle. At this point a woman is considered to be infertile. Women usually enter menopause at the age of 50–52 years.

Menopause is distinguished from other related terms. Premenopause describes the years before the Perimenopause and it begins around the age of 40. At this time, the levels of reproductive hormones are becoming lower. Perimenopause, on the other hand, describes the beginning of irregular menstrual cycles until the final menstruation cycle, which then leads to Menopause. And finally, the Postmenopause is the time after 12 months of Menopause.

Menopause is associated with many biological and psychological changes. It begins when follicular maturation stops due to lack of follicles in the ovaries. At this time, the concentration of estrogen falls to low levels. Ovulation and the production of progesterone stop. The absence of estrogen disturbs the homeostasis of the

hypothalamus-pituitary-gonadal axis. The low estrogen levels lead to a lack of negative feedback to the hypothalamus and pituitary. Consequently, the hypothalamus raises production of gonadotropin-releasing hormones, which leads to an increased release of follicle-stimulating hormone and luteinizing hormone. The body adjusts to the new hormonal balance and this leads to the appearance of several menopause symptoms, such as vasomotoric symptoms (hot flashes, headache, sweating, etc.), neuro-vegetative symptoms (insomnia, anxiety, depressive mood, etc.), organic symptoms (vaginal dryness, itchiness, etc.), metabolic symptoms (reduction of bone density, fat redistribution, etc.), and sexual symptoms (loss of libido function, etc.).

Aside from biological factors, Menopause transition can also lead to psychological changes. On the one hand, the risk of depression, anxiety, problems with concentration, and fatigue increases. On the other hand, the aforementioned symptoms of Menopause could affect well-being and life quality. It seems that psychological distress can also be influenced by social changes in this phase of life, such as the moving out of adult children. Confrontation with the process of aging can also negatively affect general well-being. These symptoms, however, generally depend on individual predispositions and manifest differently in every women.

References and Readings

Alexander, J. L., Dennerstein, L., Woods, N. F., McEwen, B. S., Halbreich, U., Kotz, K., & Richardson, G. (2007). Role of stressful life events and menopausal stage in wellbeing and health. *Expert Review Neurotherapeutics, 7*(Suppl. 11), 93–113.

Buckler, H. (2005). The menopause transition: Endocrine changes and clinical symptoms. *The Journal of the British Menopause Society, 11*(2), 61–65.

Sherman, S. (2005). Defining the menopausal transition. *The American Journal of Medicine, 118*(Suppl. 12B), 3–7.

Menstrual Headache

▶ Migraine Headache

Mental Ability

▶ Cognitive Factors
▶ Cognitive Function

Mental Disengagement

▶ Distraction (Coping Strategy)

Mental Disorder

▶ Psychological Disorder

Mental Function

▶ Cognitive Factors
▶ Cognitive Function

Mental Health Professional

▶ Psychologist

Mental Health Surveillance

William Reeves
Office of Surveillance,
Epidemiology and Laboratory Services Centers for Disease Control and Prevention,
Atlanta, GA, USA

Synonyms

Mental illness monitoring or tracking; Mental illness surveillance; Mental illness (mental health surveys); Population health monitoring or tracking

Definition

According to the World Health Organization, "mental health represents a state of well-being in which the individual realizes his/her own abilities, can cope with the normal stresses of life, can work productively and fruitfully, and is able to make a contribution to the community." Mental health is not merely the absence of mental illness; mental health and mental illness represent distinct constructs. However, reflecting perceived stigmatization, *mental health* is commonly used rather than *mental illness*.

Mental illness comprises conditions affecting cognition, mood, or behavior associated with distress or impaired functioning. The American Psychiatric Association (APA) classification system separates mental illnesses into categories based on symptoms observed by a health professional or reported by the patient; these are currently defined in the Diagnostic and Statistical Manual of Mental Disorders 4th edition (DSM-IV-TR). The World Health Organization's International Classification of Disease, 10th Revision, Clinical Modification (ICD-10-CM) defines mental illness categories similar, but not identical, to those in DSM-IV-TR. The APA is developing the DSM-V and working to achieve greater coordination with future editions of the ICD.

According to the US Centers for Disease Control and Prevention, "public health surveillance is the ongoing systematic collection, analysis, interpretation, and dissemination of data about a health-related event for use in public health action to reduce morbidity and mortality and to improve health." In practice, mental health surveillance systems collect information concerning occurrence of mental illness.

Description

Mental illness surveillance serves several public health functions as follows: (1) informing public health interventions; (2) estimating the impact of mental illness; (3) portraying the natural history of various conditions and impact of treatments on outcomes; (4) determining the distribution and occurrence of mental illness; (5) generating hypotheses and stimulating research; (6) evaluating prevention and control measures; and (7) facilitating program planning. Individual surveillance systems cannot serve all these functions, and multiple approaches must be used in combination to provide a complete mosaic.

As with infectious and chronic medical diseases, public health strategies for mental illness aim to reduce its incidence, prevalence, clinical course, impairment, and economic impact. Surveillance provides the evidence base to develop prevention and control strategies. Fundamentally, surveillance must quantify the burden imposed by mental illness and identify the high risk, often underserved, populations in need of intervention services. In addition to representing an important public health problem in its own right, mental illness is significantly associated with chronic medical diseases (e.g., obesity, diabetes, and cardiovascular disease), which adds additional morbidity and mortality. Mental illness surveillance must consider these associations.

Surveillance also serves to monitor trends, and detect and characterize changes. The greatest public health successes related to infectious disease (e.g., small pox, polio, influenza) have utilized this aspect of surveillance. More recently, chronic disease (e.g., cancer, cardiovascular disease) prevention and control strategies have successfully used surveillance trend-data. Recognition is increasing as to the importance of tracking trends in mental illnesses associated with exposure to military combat or large-scale disasters (e.g., 9/11, Katrina, Deep Water Horizon).

Three categories of surveillance systems collect mental illness data. Optimally, their measures should be combined to plan, implement, and evaluate mental illness control strategies.

Population surveys measure occurrence of mental illness at national, state, and local levels. The US Department of Health and Human Services conducts national mental illness surveillance as part of ongoing surveys (e.g., National Survey on Drug Use and Health and National Health Interview Survey). More detailed national mental illness surveys occur sporadically (e.g., 1990/1992 and 2001/2003

National Comorbidity Surveys and 2001/2002 National Epidemiologic Survey on Alcohol and Related Conditions). National-level mental illness surveillance provides the evidence base for formulating policy and tracking progress on goals such as *Healthy People 2010 and 2020*. However, national-level surveillance has limited utility for state and local authorities responsible for designing, managing, and evaluating mental illness control programs. Currently, the Behavioral Risk Factor Surveillance System (BRFSS) represents the only ongoing US state/county-specific surveillance system. BRFSS measures mental illness, in selected states, through optional modules. Some population surveys define mental illness by stringent APA criteria from clinical evaluation using the Structured Clinical Interview for DSM (SCID) and Composite International Diagnostic Interview (CIDI). More often, large-scale mental illness surveys use standardized and validated screening instruments (e.g., Patient Health Questionnaire-9), which have varying degrees of diagnostic sensitivity and specificity. Currently, US population surveys of mental illness measure only depression and nonspecific psychological distress and so data is not available concerning anxiety disorders which are at least as common, equally severe and also associated with chronic medical conditions.

Health care surveys gather information from providers and insurers concerning patients' diagnoses and health care use. They provide data on occurrence of mental illness in people who have access to and utilize medical care. Evaluation of health care surveys in conjunction with population surveys provides information on health disparities. Examples of US health care surveys include the National Ambulatory Medical Care Survey (NAMCS), National Hospital Discharge Survey (NHDS), and the National Nursing Home Survey (NNHS). The manner by which health care surveys identify mental illness varies. In the USA, coding systems used by hospitals and medical providers typically involve the ICD-9 coding system, which is not fully congruent with the DSM. Mental health professionals generally use the DSM nomenclature while primary care providers may use other terminology. In practice, regardless of which diagnostic system is used, diagnosis varies based on training, local practice, availability of treatment resources, and available payment codes.

Vital statistics records represent the fundamental source of demographic, geographic, and cause-of-death information. Over 99% of deaths in developed countries, such as the USA, are registered. Suicide is the most serious potential outcome of mental illness. In 2006, suicide was the 11th leading cause of death in the USA and most people who commit suicide have a psychiatric diagnosis at the time of their death. Suicide rates provide one of the few indicators of mental illness impact that can be compared cross-nationally.

As discussed above, most current public health efforts aim to provide mental illness services with the goal of reducing the impact of mental illness on the population. Public health research efforts have also been initiated to explore protection and promotion of mental, emotional, and behavioral health, and primary prevention of mental illness.

Cross-References

- ▶ Absolute Risk
- ▶ Benefit Evaluation in Health Economic Studies
- ▶ Benefit-Risk Ratio
- ▶ Bias
- ▶ Bogalusa Heart Study
- ▶ Centers for Disease Control and Prevention
- ▶ Chronic Disease or Illness
- ▶ Community Sample
- ▶ Cost-Effectiveness Analysis (CEA)
- ▶ Cultural Factors
- ▶ Data
- ▶ Decision Making
- ▶ Disease Burden
- ▶ Dissemination
- ▶ Framingham Heart Study
- ▶ General Population
- ▶ Generalizability
- ▶ Geographic Information System (GIS) Technology

- Health Departments
- Health Disparities
- Healthy Cities
- Income Inequality and Health
- Longitudinal Research
- Medical Outcomes Study
- Mental Illness
- Methodology
- Migration and Health Services
- Mini-Finland Health Survey
- Minority Health
- Missing Data
- Mortality
- Mortality Rates
- Multiethnic Cohort Study
- National Health and Nutrition Examination Survey (NHANES)
- National Health Interview Survey
- National Institute of Mental Health
- Neighborhood-Level Studies
- Population Health
- Population Stratification
- Population-Based Study
- Psychosocial Variables
- Public Health
- Reliability and Validity
- Screening
- Selection Bias
- Statistics
- Women's Health Initiative (WHI)
- World Health Organization (WHO)

References and Readings

Behavioral Risk Factor Surveillance System. http://www.cdc.gov/brfss/index.htm

Galea, S., & Norris, F. H. (2006). Public mental health surveillance and monitoring. In F. H. Norris, S. Galea, M. J. Friedman, & P. J. Watson (Eds.), *Methods for disaster mental health research*. New York: Guilford Press. Chapter 11.

Guidelines Working Group. (2001). *Updated guidelines for evaluating public health surveillance systems. Recommendations from the Guidelines Working Group*. (MMWR 50:/RR-13)

Li, C., Ford, E. S., Zhao, G., Strine, T., Dhingra, S., Barker, L., et al., (2009). Association between diagnosed diabetes and psychological distress among U.S. adults: Findings from 2007 BRFSS. *International Journal of Public Health, 54*(Suppl. 1), S1–S119.

National Ambulatory Medical Care Survey. http://www.cdc.gov/nchs/ahcd.htm

National Comorbidity Survey-Replication. http://www.nimh.nih.gov/health/topics/statistics/ncsr-study/index.shtml

National Health Interview Survey. http://www.cdc.gov/nchs/nhis.htm

National Hospital Discharge Survey. http://www.cdc.gov/nchs/nhds.htm

National Nursing Home Survey. http://www.cdc.gov/nchs/nnhs.htm

Substance Abuse and Mental Health Services Administration. (2010). Results from the 2009 National Survey on Drug Use and Health. http://www.oas.samhsa.gov/NSDUH/2k9NSDUH/2k9Results.htm

Mental Illness

Andrew Fox
Recovery and Wellbeing Inpatient Services, Birmingham and Solihull Mental Health NHS Trust, Birmingham, West Midlands, UK

Synonyms

Psychiatric illness; Psychological disorder

Definition

Mental illnesses are particular ways of thinking, feeling, or behaving that are typically associated with distress or impairment and that are not accepted as normal by the dominant local culture. The concept remains controversial, and there is ongoing debate about how best to describe and understand the experiences often subsumed under this term.

Description

Classification of Mental Illnesses

Mental illnesses are made up of clusters of symptoms (abnormal or statistically infrequent

behavior, emotion, or thoughts) and are typically classified according to different categories, or diagnoses, so that a particular mental illness is considered as either present or not. There are many different forms of mental illnesses, and people may be classed as displaying several at any one time (known as comorbidity). The key manuals used to diagnose mental illnesses are the International Statistical Classification of Diseases and Related Health Problems (currently on its tenth revision; ICD-10) published by the World Health Organization and the Diagnostic and Statistical Manual of Mental Disorders (currently on its fourth edition), published by the American Psychiatric Association. According to these established manuals, individuals must demonstrate a certain number of symptoms in order to be considered as displaying evidence of the presence of a mental illness. Some example diagnoses from the ICD-10 include moderate depressive episode, bipolar affective disorder, panic disorder, posttraumatic stress disorder, generalized anxiety disorder, paranoid schizophrenia, and anorexia nervosa. More recent "dimensional" attempts to classify mental illnesses (e.g., "salience syndrome"; Van Os, 2009) have incorporated the notion that symptoms of mental illness lie at one end of a continuum of human experiences, with less debilitating but similar experiences within the range of what may be considered present within the general population.

An alternative to diagnosis of mental illness is psychological formulation (Johnstone & Dallos, 2006). This approach attempts to integrate hypothesized etiological factors into an explanatory framework for the individuals' current difficulties (e.g., low mood, anxiety). There are many different possible frameworks used for formulation, often – but not always – structured around the perspective of a school of psychotherapy.

Causes of Mental Illnesses

The precise cause of different mental illnesses remains unclear, with many hypothesized mechanisms for each of the many different categories of mental illness. The popular diathesis-stress model proposes that individuals are vulnerable to experiencing symptoms of mental illness through their genetic predisposition, and environmental stress triggers the onset of symptoms. Another popular and somewhat related general approach is the "biopsychosocial" perspective that suggests that multiple biological, psychological, and social factors are all implicated in the onset of mental illness. Psychological formulations often attempt to integrate hypothesized etiological factors with those that maintain the difficulties.

Treatment of Mental Illness

Given the multiple etiological and maintenance factors that can be considered in mental illness, there are many possible treatment options; however, the most popular remain medication and psychotherapy ("talking therapy"). Individuals may be offered a combination of treatment options including medication and psychotherapy.

Specialist practitioners, often psychiatrists, but sometimes nurses and psychologists, prescribe medication that is licensed for particular mental illnesses when there is evidence that it may reduce some or all of the unwanted symptoms that a person may be experiencing. Individuals may be encouraged to try several different forms of medication until they find one that they and their mental health practitioner feel helps them.

There are also numerous psychotherapies that are offered for different mental illnesses, often by psychiatrists, psychologists, nurses, social workers, and other individuals trained in specific forms of psychological therapy. Popular forms of psychotherapy include psychoanalysis, cognitive behavioral therapy, systemic (or family) therapy, and humanistic therapy. All forms of psychotherapy involve meeting for discussion with a trained mental health practitioner; however, the focus and content of these discussions will vary according to the type of psychotherapy.

Controversies Regarding the Concept of Mental Illness

The concept of "mental illness" is highly controversial; the psychiatrist Thomas Szasz (1961) has been an influential opponent of the term, and the concept continues to attract criticism.

The varieties of human experience and behavior that comprise what is viewed as a mental illness are often fluid, and what is termed mental illness in one time or context may not be termed mental illness in another (e.g., homosexuality was classified as a mental illness by the original Diagnostic and Statistical Manual of Mental Disorders). As there are often legal powers available to control the freedom of individuals labeled as mentally ill, there has been criticism of how diagnostic labels of mental illness have been used to control and oppress minority groups. Some of the symptoms linked to particular categories of mental illness – schizophrenia in particular – are so heterogeneous that they have been widely criticized as lacking validity and reliability (Bentall, 2003). Furthermore, the term itself ("illness") may lend itself to the false assumption that experiences subsumed under the category of a mental illness are primarily the result of a physiological disease process and should be treated in a medical way. Indeed, there is often a social stigma associated with the label of a mental illness, especially if understood as resulting from some underlying physiological cause, and this can have negative consequences for those labeled as mentally ill (e.g., reduced employment opportunities).

Given the criticisms of the concept of "mental illness," alternative terms have been suggested, such as mental, psychological, or emotional distress.

Cross-References

- ▶ Psychiatric Diagnosis
- ▶ Psychiatric Illness
- ▶ Psychological Disorder

References and Readings

Bentall, R. P. (2003). *Madness explained: Psychosis and human nature*. London: Allen Lane.

Johnstone, L., & Dallos, R. (2006). *Formulation in psychology and psychotherapy: Making sense of people's problems*. Hove, East Sussex: Routledge.

Szasz, T. S. (1961). *The myth of mental illness: Foundations of a theory of personal conduct*. New York: Hoeber-Harper.

Van Os, J. (2009). A salience dysregulation syndrome. *British Journal of Psychiatry, 194*, 101–103.

World Health Organisation. (2010). *International statistical classification of diseases and related health problems, 10th revision, version for 2010*. Retrieved April 11, 2012 from http://apps.who.int/classifications/apps/icd/icd10online/

Mental Illness Monitoring or Tracking

▶ Mental Health Surveillance

Mental Illness Surveillance

▶ Mental Health Surveillance

Mental Illness (Mental Health Surveys)

▶ Mental Health Surveillance

Mental Imagery

▶ Guided Imagery

Mental Models of Illness

▶ Common-Sense Model of Self-regulation

Mental Representations of Illness

▶ Common-Sense Model of Self-regulation

Mental Status Examination

▶ Screening, Cognitive

Mental Strain

▶ Psychosocial Work Environment

Mental Strategies

▶ Cognitive Strategies

Mental Stress

Kristen Salomon
Department of Psychology, University of South Florida College of Arts & Sciences, Tampa, FL, USA

Synonyms

Anxiety; Distress; Psychological stress

Definition

A form of stress that occurs because of how events in one's external or internal environment are perceived, resulting in the psychological experience of distress and anxiety (Lazarus & Folkman, 1984). Mental stress is often accompanied by physiological responses (Cacioppo, 1994). Mental stress is most often induced in the laboratory by demanding and/or noxious stimuli, involving motivation to meet a performance criterion (Blascovich, Mendes, Tomaka, Salomon, & Seery, 2003) and/or social-evaluative threat (Dickerson & Kemeny, 2004), or interpersonal interactions, particularly those involving conflict (Glass & Singer, 1972). Common mental stress tasks include preparing and giving a speech, performing arithmetic, tracing around star with only a mirror image as a guide, performing a reaction time task (Steptoe & Vögele, 1991), and discussing a disagreed upon topic with another person (Gottman & Levenson, 1992).

Cross-References

▶ Anxiety
▶ Perceptions of Stress
▶ Physiological Reactivity
▶ Stress
▶ Stress Responses
▶ Stress, Emotional

References and Readings

Blascovich, J., Mendes, W. B., Tomaka, J., Salomon, K., & Seery, M. (2003). The robust natures of the biopsychosocial model: A reply to Wright and Kirby. *Personality and Social Psychology Review, 7*, 234–243.
Cacioppo, J. T. (1994). Social neuroscience: Autonomic, neuroendocrine, and immune responses to stress. *Psychophysiology, 31*, 113–128.
Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130*, 355–391.
Glass, D. C., & Singer, J. E. (1972). *Urban stress. Experiments on noise and social stressors*. New York: Academic Press.
Gottman, J. M., & Levenson, R. W. (1992). Marital processes predictive of later dissolution: Behavior, physiology, and health. *Journal of Personality and Social Psychology, 63*, 221–233.
Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal and coping*. New York: Springer.
Steptoe, A., & Vögele, C. (1991). Methodology of mental stress testing in cardiovascular research. *Circulation, 83*, II-14–II-24.

Mental Stress Task

▶ Stress Test

Mental Stressor

▶ Stroop Color-Word Test

Mental Training

▶ Meditation
▶ Transcendental Meditation

Mental Work Load

► Job Demand/Control/Strain

Messenger RNA

► RNA

Meta-Analysis

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

A meta-analysis is a quantitative evaluation of the evidence provided by two or more individual research studies that have addressed the same question. It commonly involves the statistical combination of summary statistics from various trials, but it also refers to analyses performed on the combination of raw data.

Description

Purposes of meta-analyses include:

- Providing a more precise estimate of the overall treatment effect of interest.
- Evaluating whether overall positive results are also seen in prespecified subgroups of participants.
- Evaluating apparently conflicting study results.

The conceptual basis of meta-analysis is straightforward: More data will provide a better opportunity to get a meaningful answer to a research question. However, appropriate implementation of the required statistical techniques and the appropriate interpretation of the results obtained are not so straightforward. Synthesizing the results from many individual studies can be difficult and confusing. Inconsistency between published studies addressing the same research question is not uncommon, particularly when different study designs have been employed.

Even though a meta-analysis does not require a new study to be conducted, it is still a research method in its own right. Therefore, "to ensure that a meta-analysis is scientifically valid, it is necessary to plan and conduct the analysis in an appropriate way. It is not sufficient to retrospectively go to a bunch of studies that you like the look of and stick them together!" (Kay, 2007).

One way to combat inconsistencies is to employ the pooled data approach, i.e., to combine the data from all of the studies and to conduct a new analysis on these data. The size of the new data set will be (much) larger than the size of any of the individual data sets, and the analysis of a larger data set lends itself more readily to the identification of a relatively small but still clinically important treatment effect. Such treatment effects can have considerable public health benefits if they are seen in a very large number of patients with a common disease.

At the outset, the purpose for a meta-analysis must be stated, and the treatment effect of interest determined. Subsequently, the basic steps required for the conduct of a meta-analysis are as follows:

- Establishing rules for which of the identified studies will be allowed into the meta-analysis. This includes determining and detailing approaches to ensure consistent quality of the studies and how to handle poor quality studies.
- Identification of all relevant studies.
- Data abstraction and acquisition.
- Data analysis.
- Evaluating robustness.
- Dissemination of results and conclusions.

One straightforward approach to meta-analysis is to include every study identified and obtained. A counterargument is that, almost certainly, some studies will be "better" than others, and that "less good" studies perhaps should not be included if the "best" information is to be provided to the clinicians who will read

the meta-analysis and therefore may base treatment decisions on its results. In the latter case, a priori inclusion and exclusion criteria must be created in advance of searching for studies. These effectively define "better" and "less good," and hence determine which of the studies about to be identified will be included in the meta-analysis.

The results of a completed meta-analysis would typically include the following:

- The estimated treatment effect (a point estimate) for each individual study included in the analysis, and a 95% confidence interval about each study's estimate.
- The overall estimated treatment effect and its 95% confidence interval.

This information can be displayed in tabular form or in a graphical form called a confidence interval plot.

Having calculated the result of a meta-analysis, it can be informative to assess the robustness of the analysis. In any meta-analysis some of the studies included will be larger than others, and sometimes a small percentage of included studies can be considerably larger than the majority of others. The nature of the calculations performed in meta-analysis mean that the larger trials tend to influence the result more, since they tend to have greater precision. It can therefore be helpful to assess the robustness of the overall conclusion by performing the analysis without the data from the largest study or studies to see if the result remains qualitatively the same. If it does, then the result is deemed robust. If it does not, confidence in the overall result can be undermined. Additionally, if the results are considerably different, and the studies are considerably different from each other (heterogeneous), it may not be appropriate to conduct any form of meta-analysis using them.

While an analysis is certainly conducted when performing a meta-analysis, the term *meta-methodology* may better reflect the methodology used to collect the data before conducting the actual analysis (Turner, 2011). This term emphasizes the methodological rigor that is needed in getting ready to conduct the analysis.

Cross-References

▶ Systematic Review

References and Readings

Kay, R. (2007). *Statistical thinking for non-statisticians in drug regulation*. Chichester, UK: Wiley.

Turner, J. R. (2011). Editor's commentary: Additional associate editors, new submission category, and meta-methodology. *Drug Information Journal, 45*, 405–411.

Metabolic Processes

▶ Metabolism

Metabolic Syndrome

Anna Maria Patino-Fernandez
Department of Pediatrics, University of Miami, Miami, FL, USA

Synonyms

Insulin resistance (IR) syndrome; Syndrome X

Definition

The metabolic syndrome (MS) in adults has been defined in the literature by various organizations, including the National Cholesterol Education Program Expert Panel (2001), although a standard definition does not exist in either the adult or child literature. In adults, the clustering of four health risk factors, obesity, hyperinsulinemia, high blood pressure, and dyslipidemia, has been referred to as syndrome X, insulin resistance syndrome, and/or metabolic syndrome (Reaven, 1988). The dominant underlying risk factors for this syndrome appear to be abdominal obesity and

insulin resistance. Insulin resistance is a generalized metabolic disorder in which the body can no longer use insulin efficiently. This is why the metabolic syndrome has also been called the insulin resistance syndrome. The MS risk factors manifest in childhood and have a major impact on the development of medical problems throughout the life course. Although the clustering of these risk factors is not well understood in children, children who are overweight are more likely to have hyperlipidemia, elevated blood pressures, and elevated insulin levels (Cook, Weitzman, Auinger, Nguyen, & Dietz, 2003). A decrease in insulin sensitivity is one of the first manifestations of MS in youth (Arslanian & Suprasongsin, 1996), and insulin levels in children are positively associated with levels of triglycerides, blood pressure, and body mass index, and negatively associated with high-density lipoprotein cholesterol (Batey et al., 1997). The MS definition has been modified for the examination of MS in children and includes the presence of at least three of the following abnormalities: overweight, hypertension, hypertriglyceridemia, low HDL cholesterol, and impaired glucose tolerance (Cruz Weigensberg, Huang, Ball, Shaibi, & Goran, 2004).

The American Heart Association and the National Heart, Lung, and Blood Institute recommend that MS in adults be identified as the presence of three or more of the following:

- Elevated waist circumference:
  - Men – equal to or greater than 40 in. (102 cm)
  - Women – equal to or greater than 35 in. (88 cm)
- Elevated triglycerides:
  - Equal to or greater than 150 mg/dL
- Reduced HDL ("good") cholesterol:
  - Men – less than 40 mg/dL
  - Women – less than 50 mg/dL
- Elevated blood pressure:
  - Equal to or greater than 130/85 mmHg
- Elevated fasting glucose:
  - Equal to or greater than 100 mg/dL

Individuals with MS are at increased risk for coronary heart disease and other diseases related to plaque buildups in artery walls (e.g., stroke and peripheral vascular disease) and type 2 diabetes. The metabolic syndrome has become increasingly common in the United States, with over 50 million Americans having it. Some people are genetically predisposed to insulin resistance. Acquired factors, such as excess body fat and physical inactivity, can elicit insulin resistance and the metabolic syndrome in these people.

For managing both long- and short-term risk, lifestyle interventions are the first-line treatments to reduce the MS risk factors. These lifestyle interventions include:

- Weight loss to achieve a desirable weight (in adults, a BMI less than 25 kg/m^2)
- Increased physical activity, with a goal of at least 30 min of moderate-intensity activity on most days of the week
- Healthy eating habits that include fruits and vegetables; reduced intake of saturated fat, trans fat, and cholesterol; and appropriate portions

Cross-References

- ▶ Coronary Heart Disease
- ▶ Insulin Resistance
- ▶ Insulin Resistance (IR) Syndrome
- ▶ Obesity
- ▶ Type 2 Diabetes

References and Readings

Arslanian, S. A., & Suprasongsin, C. (1996). Insulin sensitivity, lipids, and body composition in childhood: Is syndrome X present? *Journal of Clinical Endocrinology Medicine, 81*, 1058–1062.

Batey, L. S., Goff, D. C., Tortolero, S. R., Nichaman, M. Z., Chan, W., Chan, F. A., et al. (1997). Summary measures of the insulin resistance syndrome are adverse among Mexican American versus Non-Hispanic white children. *Circulation, 96*, 4319–4325.

Cook, S., Weitzman, M., Auinger, P., Nguyen, M., & Dietz, W. H. (2003). Prevalence of a metabolic syndrome phenotype in adolescents. *Archives of Pediatric and Adolescent Medicine, 157*, 821–827.

Cruz, M. L., Weigensberg, M. J., Huang, T. T., Ball, G., Shaibi, G. Q., & Goran, M. I. (2004). The metabolic syndrome in overweight Hispanic youth and the role of insulin sensitivity. *Journal of Clinical Endocrinology and Metabolism, 89*, 108–113.

http://www.mayoclinic.com/health/metabolic%20syndrome/DS00522

National Institutes of Health. (2001). *The third report of the national cholesterol education program expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III)*. NIH Publication 01-3670. Bethesda, MD: National Institutes of Health.

Reaven, G. M. (1988). Pathophysiology of insulin resistance in human disease. *Physiology Review, 75*, 473–486.

Metabolic Syndrome X

► Hyperinsulinemia

Metabolism

Wen B. Leong[1], Shahrad Taheri[2] and G. Neil Thomas[3]
[1]Diabetes and Endocrinology, University of Birmingham, Heart of England NHS Foundation Trust, Birmingham, West Midlands, UK
[2]University of Birmingham, Heart of England NHS Foundation Trust, Birmingham, UK
[3]Department of Public Health, University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Metabolic processes

Definition

The word metabolism is derived from Greek and means "change." It involves all chemical reactions or changes in a living organism. In simple terms, "metabolism is the chemistry of life (Bing, 1971)." Due to the importance of metabolic processes to life, the processes have been highly conserved throughout evolution.

Description

Metabolism consists of multiple processes resulting in the breakdown and formation of molecules that sustain life. Metabolic processes are complex and can be influenced by internal and external factors. These processes are highly regulated and aim to maintain physiological balance or homeostasis in the face of a constantly changing environment. There are two types of metabolic reactions: anabolic and catabolic. These processes work hand in hand, although one may predominate depending on physiological or pathological state. Anabolic reactions predominate at times of high substrate availability and involve transformations of smaller molecules into larger complex storage compounds. Catabolic reactions predominate at times of low substrate availability and are involved in the breakdown of molecules for energy utilization (Berg, Tymoczko, & Stryer, 2012).

Catabolism involves energy production by breaking down complex molecules to simpler ones for energy usage or cell repair. For example, break down of glycogen, a carbohydrate, to glucose, a monosaccharide, produces cellular energy. This energy in turn can be used to create other complex products through anabolic reactions. Anabolic reactions are important as they produce important compounds essential for basic function of life including the synthesis of deoxyribonucleic acid (DNA) for genetic coding or peptide and steroid hormones from amino acids and cholesterol, respectively (Dominiczak, 2007).

In metabolic processes, one molecule is transformed to another or to energy via cellular respiration. The steps of metabolic pathways are regulated by various enzymes that act as catalysts to speed up the chemical reactions. These enzymes are regulated at several levels from production to function by several factors including the cell's environmental milieu. Abnormalities in several metabolic pathways are associated with various disease conditions, both genetic and acquired. Manipulation of metabolic pathways is also important in treatment of several medical conditions.

Important Biochemical Compounds in Metabolism

Living organisms need food to grow and survive. Food can be divided into essential biochemical

compounds called macro- and micronutrients: proteins, carbohydrates, lipids, vitamins, and minerals. Other important biochemical compounds include nucleotides, which are important in the transfer of genetic information for growth, maintenance, and repair.

Protein

Protein is made from amino acids joined together via peptide bonds. Degraded amino acids are converted to nitrogen which in humans is transformed to urea (Brody & ScienceDirect, 1999). Urea is excreted in urine by the kidneys. Protein is vital in life as it is involved in many metabolic pathways either as catalytic enzymes or as peptide hormones (e.g., insulin) (Brownie & Kernohan, 2005) regulating metabolic enzymes and processes. Proteins also function as the cellular cytoskeleton, part of immune system as antibodies and help in transport of substrates into cells as transporters of part of cell membrane channels.

Carbohydrate

Carbohydrate is formed by simple sugars or monosaccharides. These monosaccharides are fructose, galactose, and glucose. Monosaccharides are stored as complex carbohydrates (polysaccharides) such as glycogen in animal or starch in plants for energy utilization and help in structural formation of cells alongside with proteins (Dominiczak, 2007).

Lipids

Lipids consist of a broad group of hydrophobic molecules (Brownie & Kernohan, 2005), which are soluble in organic solvents. Lipids function as structural components in building of cell membranes (Berg et al., 2012). Although they are less accessible as substrates, lipids are also important source of energy storage (Dominiczak, 2007). Cholesterol is an example of a lipid, which can subsequently be converted into bile acids or steroids to function as hormones in the endocrine system (Molina, 2004).

Vitamins

Vitamins are organic compounds derived from the diet. They can be divided into water-soluble (vitamin B and C) or fat-soluble vitamins (vitamin A, D, E, and K). For most metabolic pathways, coenzymes or metabolic intermediates are essential to carry functional groups to be used in specific enzyme activity. Without these coenzymes, the chemical reaction will not be successful. Many vitamins act as coenzymes, for example, vitamin B1 (or thiamine) is a coenzyme in carbohydrate metabolism. Thiamine deficiency is common in chronic excessive alcohol consumption, and this can lead to disease of the nervous system known as Wernicke's encephalopathy or beriberi causing problems to the cardiovascular, muscular, and nervous systems. Other functions of vitamins include maintenance in life and support for growth (King, 2011).

Minerals

Minerals or inorganic compounds are needed for many different metabolic pathways. Some common examples of minerals are sodium, potassium, chloride, magnesium, iron, calcium, and phosphate. They all have different functions. Some examples are as follows: the electrolytes sodium, potassium, chloride, and water are important for blood pressure regulation; iodine is essential for the production of thyroid hormone; and iron is a key component of hemoglobin which carries oxygen. Calcium and phosphorus are important minerals for bone metabolism. Calcium is also the key to several cellular functions including muscle contraction, neurotransmission, hormone release, and metabolic enzyme regulation (Brody & ScienceDirect, 1999).

Nucleotides

Within cells, there are heterocylic compounds called purines and pyrimidines. These serve as bases for nucleotides and are formed from amino acids. Purine bases include adenine and guanine, while pyrimidines are thymine, cytosine, and uracil. When purine or pyrimidine bases are linked to carbohydrate, they are called nucleosides. Nucleotides are phosphorylated nucleosides (King, 2011). Functions of nucleotides include acting as coenzymes, (e.g., nicotinamide adenine dinucleotide, NAD+), cell mediators (e.g., cyclic adenosine monophosphate, cyclic-AMP), and as

energy storage in the form of adenosine triphosphate (ATP) for future energy transfer. Importantly, nucleotides are involved in gene coding in the form of deoxyribonucleic acid (DNA) and ribonucleic acid (RNA) (Berg et al., 2012; King, 2011).

Energy Metabolism

Unit of Measurement

Energy is measured in joules or calories. Joule, named after English physicist James Prescott Joules, is the unit of measurement for energy in the international system of unit. One joule is equal to 0.2390 calorie, and one calorie is equal to 4.184 J (Brody & ScienceDirect, 1999). Calories printed on food labels provide the measured energy gained within the body when the food is eaten and absorbed.

Krebs Cycle

After food is absorbed from the digestive system, they can be converted into energy. Fatty acids are used as a major energy source in most organs except the brain. The brain gets energy through breaking down glucose into carbon dioxide. It also uses ketone bodies during starvation or fasting states (Brody & ScienceDirect, 1999). The liver on the other hand, uses mainly fatty acid for energy so it can function to produce, store, and release glucose. The glucose stored within the liver can be used by other organs when in need. Both the breakdown of glucose and fatty acids are metabolized through a pathway called the Krebs cycle (Brownie & Kernohan, 2005).

The Krebs cycle is also known as the tricarboxylic acid cycle. It is a pathway which uses oxygen and nutrients to produce adenotriphosphates (ATP), the energy currency of cells (Krebs & Roberts, 1973). Proteins, carbohydrates, and fatty acids can be metabolized to acetyl-CoA which then produces energy. These processes involved energy transfer or release for maintenance of life (Brownie & Kernohan, 2005).

Energy Transfer

A main aim of metabolism is to transfer energy or release energy for usage. There are two basic laws of thermodynamics for energy, they are:

1. Energy can neither be created nor destroyed. It can only change forms. Therefore, the total amount of energy in an organism is the same.
2. Entropy always increases. This means energy can be produced by converting high-energy compounds to low-energy end product.

In life, energy changes can be expressed using the term free energy, which is the difference in the energy content from the initial compound and the end product. For example, the energy content from glucose (initial compound) will be different when it is converted to carbon dioxide and water (end product). If there is a negative change in free energy, meaning there is release of energy from the initial high-energy compound when converted to a low-energy end product. One common example is the conversion of adenosine triphosphate (ATP) to adenosine diphosphate (ADP). The process which releases energy is called exergonic reaction (Brownie & Kernohan, 2005).

A positive change in free energy means extra energy is needed for the process to convert the initial compound to the end product(s). This process is called endergonic reaction. The energy released is used to maintain the metabolic rate (Brownie & Kernohan, 2005).

Metabolic Rate

The minimum body energy requirement within a body is called basal metabolic rate (BMR). It is the body's energy usage at rest in the postabsorptive state (no processes of digestion). The metabolic rate varies during different activities. During sleeping state, for example, metabolic rate may fall by 10% from the BMR, while BMR may increase by 10% postmeals. It also changes with different physical activities, aging, temperature, environment, and stress (Brody & ScienceDirect, 1999).

A useful way to measure metabolic rate is by measuring respiratory quotient (RQ). As mentioned above, one of the end products of Krebs cycle is carbon dioxide and, during metabolic pathways, oxygen is an essential compound. So by measuring the ratio between carbon dioxide released from respiration and the oxygen consumed, the result will be the total energy usage. The consumption of different substrates has

different respiratory quotients and is regulated closely by different mechanisms (Brody & ScienceDirect, 1999).

Regulatory Mechanisms

Metabolic processes are very complex and can be influenced by many factors to ensure responsiveness to alterations in the environment. This regulation occurs through several mechanisms: the nervous system, the endocrine system, and at cellular level, the activation of enzymes and feedback inhibition.

Nervous System

The nervous system controls metabolism through the sympathetic and parasympathetic nervous systems. These systems can stimulate a metabolic pathway directly or indirectly through the endocrine system via release of hormones. For example, release of catecholamines stimulates energy production by breaking down fat and glycogen stores within the body. Epinephrine released can also act indirectly by activation of different receptors causing release of hormones, which in turn trigger a cascade of reactions. An example of this will be epinephrine causing the release of insulin or glucagon via α and β receptors within the endocrine organ, pancreas (Molina, 2004).

Endocrine System

The endocrine system regulates metabolism through release of hormones into the circulation. Hormones act via their receptors to regulate different metabolic pathways. For example, growth hormone released from the pituitary gland will act on receptors in the liver to stimulate released of peptides called insulin-like growth factors (IGF), and this will lead to different metabolism thus stimulating growth (Molina, 2004).

Activation of Enzymes

Many proteins are inactive and need addition of specific molecule to be converted to the active form. These activated proteins will then be able to function in metabolic pathways as active enzymes. The availability of the specific activating enzymes will thus determine balance of metabolic pathways.

Adenosine triphosphate or ATP is one of the commonest compounds which will donate a phosphate to attach to another protein, such as tyrosine resulting in active form of tyrosine ready for metabolism. The process to break and free phosphate from ATP needs specific enzymes, namely, protein kinases, to be available. Therefore, the availability of protein kinases acts as a regulatory factor. This process of attaching phosphate to proteins is called phosphorylation. There are many different protein kinases available (Berg et al., 2012).

On the other hand, dephosphorylation can stop or slow down some metabolic pathways. It will remove the phosphate from the active protein by hydrolyzing phosphate to become orthophosphate. Without the additional phosphate molecule, the active protein will become dormant. The key enzymes on dephosphorylation are called protein phosphatases (Berg et al., 2012).

Feedback Inhibition

The availability of the final products of the metabolic pathway can act as part of a feedback regulation causing preceding steps in the pathway to slow or stop. This prevents overproduction of certain compounds, for example, the end product of pyrimidine base nucleotide cytidine triphosphate will inhibit aspartate transcarbamoylase, the enzyme required for the first step of metabolism of cytidine triphosphate (Dominiczak, 2007).

In summary, metabolism is the chemistry of life. There are many essential biochemical compounds needed to maintain life, and these compounds are used for storage, maintenance, or conversion into energy. Metabolic pathways are regulated by different mechanisms to maintain homeostasis. Any problems in any steps of metabolic pathway can lead to different diseases. Through manipulation of metabolic pathways, it is also possible to treat several medical conditions.

Cross-References

- ▶ Basal Metabolic Rate
- ▶ Metabolic Syndrome

References and Readings

Berg, J. M., Tymoczko, J. L., & Stryer, L. (2012). *Biochemistry*. Basingstoke/New York: WH Freeman and Company.
Bing, F. C. (1971). The history of the word "metabolism". *Journal of the History of Medicine and Allied Sciences, 26*(2), 158–180.
Brody, T. & ScienceDirect. (1999). *Nutritional biochemistry (electronic resource)*. San Diego: Academic Press.
Brownie, A. C., & Kernohan, J. C. (2005). *Medical biochemistry: A core text with self-assessment*. Edinburgh: Churchill Livingstone.
Dominiczak, M. H. (2007). *Flesh and bones of metabolism*. Edinburgh/New York: Elsevier Mosby.
King, M. (2011). *The medical biochemistry page*. Retrieved March 3, 2011, from http://www.themedicalbiochemistrypage.org/
Krebs, H. A. S., & Roberts, M. B. V. (1973). *The citric acid cycle. A further analysis of metabolism and its implications slide*. London: Audio-Learning.
Molina, P. E. (2004). *Endocrine physiology*. New York: McGraw-Hill.

Methodology

► Research Methodology

Methylation

Jana Strahler
Clinical Biopsychology, Department of Psychology, University of Marburg, Marburg, Germany

Synonyms

DNA-methylation; Gene methylation; Methylation of bases; Protein methylation; Transmethylation

Definition

Addition of a methyl group ($-CH_3$), simple four-atom molecule, to a chemical compound.

Description

In biological systems, methylation is involved in multiple biochemical processes such as the regulation of gene expression, protein function, and RNA metabolism. Enzymes are the main catalyzers constantly working to methylate or demethylate in a dynamic process.

DNA methylation refers to the modification of DNA caused by enzyme-induced addition of methyl groups to nucleobases, which alters the amount of messenger RNA. In general, methylated genes are ineffective, while unmethylated genes are active. The pattern of methylation differs between gene regions with the coding region being highly methylated while the promotor region is mostly free of methylation. DNA methylation typically occurs at sites where cytosine is directly followed by guanine. While there is no change in DNA sequence, DNA methylation prevents translation and therefore, alters gene expression. Thus, the addition of methyl groups to DNA can affect whether the gene's signal to produce a protein is triggered. The absence of the protein product alters the development and function of the cell.

Protein methylation refers to the posttranslational addition of methyl groups to basic amino acid residues in DNA- and RNA-binding proteins which regulates protein function.

Transmethylation denotes the transfer of a methyl group of methyl group donators to C-, O-, or N-atoms of a biomolecule under the catalytic influence of transmethylase. Example: Synthesis of creatine, a nitrogenous organic acid that increases the formation of adenosine triphosphate.

Failures in DNA methylation cause reduced or heightened gene activity. Gene methylation is likely to be a major mechanism in different disease states such as atherosclerosis or lung cancer development and progression. In this context, methylation state is discussed as a potential marker for the early detection of cancer, that is, a biomarker for tumorigenesis. Furthermore, methylation plays a role in gene-environment interaction. Previous studies have shown that early life experiences may alter

gene expression mediated via differential methylation patterns. Subjects suffering from Posttraumatic Stress Disorder show more unmethylated, active genes than unaffected subjects, and most of those genes are involved in immune system regulation. Thus, there is evidence for an association between stress-related disorders and epigenetic changes in immune system genes. Furthermore, there is also evidence for an association between diet and dietary supplements and gene methylation status implicating a possible mechanism for the effects of nutrients on human health.

Importantly, the *pattern of gene methylation is heritable*. Modifications made to DNA are maintained every time the cell divides. As already noted, lifetime experiences, dietary habits, or environmental factors can change gene activity, that is, the amount of DNA methylation. There is evidence for genetic transmission of methylation patterns to the offspring (called transgenerational epigenetic inheritance). This suggests that epigenetic mechanisms may impact health for several subsequent generations but are also attractive targets for interventions in complex diseases. Future research will provide new advances in treatments and new tools for the identification of the function of genes. The identification, cataloging, and interpretation of DNA methylation is subject of the Human Epigenome Project.

Cross-References

- ▶ DNA
- ▶ Epigenetics
- ▶ Gene
- ▶ Gene-Environment Interaction
- ▶ RNA

References and Readings

Alberts, B., Johnson, A., Walter, P., Lewis, J., Raf, M., & Roberts, K. (2007). *Molecular biology of the cell* (5th ed.). New York/London: Garland Science.

Haslberger, A. G. (Ed.). (2010). *Epigenetics and human health: Linking hereditary, environmental and nutritional aspects*. Weinheim: Wiley-VCH Verlag GmbH & Co. KGaA.

Hedgecoe, A. (2004). *The politics of personalized medicine: Pharmacogenetics in the clinic*. Cambridge, UK: University Press.

Miller, G. E., & Cole, S. W. (2010). Functional genomic approaches in behavioral medicine research. In A. Steptoe (Ed.), *Handbook of behavioral medicine. Methods and application* (pp. 443–454). New York/Dordrecht/Heidelberg/London: Springer.

Szyf, M. (2005). *DNA methylation and cancer therapy*. Georgetown, TX: Landes Bioscience.

Methylation of Bases

▶ Methylation

MI

▶ Acute Myocardial Infarction

Micro Data Collection and Analysis System

▶ Surveys

Migraine Headache

Jeanetta Rains[1] and Donald Penzien[2]
[1]Center for Sleep Evaluation, Elliot Hospital, Manchester, NH, USA
[2]Head Pain Center, University of Mississippi Medical Center, Jackson, MS, USA

Synonyms

Classic migraine; Common migraine; Headache with aura; Menstrual headache; Migraine with aura; Premenstrual headache; Sick headache; Vascular headache

Definition

Migraine is the most prevalent neurological disorder, and is ranked by the World Health

Organization as 19th worldwide of all diseases causing disability. Migraine is recurrent headache characterized by moderate to severe pain, pulsating pain, unilateral location, and pain that is aggravated by routine physical activity (e.g., walking, climbing stairs). Associated features include nausea and/or vomiting, and photophobia(heightened sensitivity to light and sound). Headaches generally last 4–72 h (untreated or unsuccessfully treated) although migraine in children are often less than 4 h in duration and chronic migraine – considered a complication or migraine – may exceed 72 h and in some cases may present as unremitting. The majority of migraineurs receive treatment in the primary care setting with a wide variety of acute and preventative medications. But even the best pharmacologic options have their limits. Headache is recognized as not merely a physiological problem but rather a *psycho*physiological disorder (i.e., a physical disorder subject to psychosocial influences and environmental stressors). Thus, identification of modifiable headache risk factors and management of headache triggers are common goals of headache care. Specific behavioral headache treatments may be utilized to augment, facilitate, or in certain cases offered in lieu of pharmacologic treatment. Behavioral treatments target the patient's headache-related physiological responses (relaxation training, biofeedback) or behaviors, emotions, and cognitions (cognitive-behavioral therapy, stress management). A very substantial quantity of empirical research evaluating these treatments has been produced within the past three decades.

Description

Migraine Diagnoses

The widely used nosology for headache is *The International* diagnosis *Classification of Headache Disorders-II* (IHS, 2004) which provides detailed DSM-style hierarchical diagnostic criteria for primary and secondary headache disorders; a revised edition, the ICHD-II-R, is forthcoming. The majority of migraines can be subclassified according to IDHD-II as: 1.1 Migraine without aura (previously termed common migraine) or 1.2 Migraine with aura (classic migraine). About one in five migraine sufferers experiences an aura prior to onset of a migraine headache. The migraine aura is a phenomenon in which one or more visual or sensory disturbances develop over a few minutes and disappear within 60 min. Migraine headache usually follows within 1 h, but may occur before or during the aura or may be entirely absent. There are many different types of visual and sensory auras which may differ between attacks. Visual disturbance may be positive features (e.g., flickering, blurred, curved, or luminescent area in the visual field) and/or negative features (e.g., loss of vision). Aura may involve one such symptom or a succession of visual disturbances. Unilateral sensory disturbances are also common, such as pins-and-needles, arm or leg numbness or tingling, or one-sided weakness. Disturbances of speech may also occur, though they are a much less common aura symptom. Such neurological symptoms are fully reversible.

Chronic migraine is the most common headache diagnosis seen in neurology practice and multidisciplinary clinic. Most cases of chronic migraine evolve over time from less frequent migraine without aura. As chronicity develops, the headaches tend to lose their episodic character. Chronic migraine is characterized by having daily or almost daily head pain more than 15 days per month and the average headache duration greater than 72 h (aka, transformed migraine, chronic daily headache). The pattern of progression of migraine from episodic to chronic over time has been well documented in headache literature, sometimes referred to as *chronification* or *transformation* of migraine. Medication overuse is the variable that has received the greatest attention as a potential cause for progression. Medication-overuse headache (MOH) has been associated with over-the-counter and prescriptions medications commonly used in the treatment of migraine. Though these medications are effective for intermittent use, frequent use over an extended period of time may complicate headache itself. ICHD-II defines minimum patterns of medication use considered to be associated with 8.2

Medication-overuse headache: ergotamines or triptans $\geq$10 days/month on a regular basis $\geq$3 months; opioids or combination analgesics $\geq$10 days/month on a regular basis for $>$3 months; and simple analgesics $\geq$15 days/month $>$3 months. MOH is generally recognized to be relatively resistant to traditional pharmacological as well as behavioral treatments. However, following withdrawal of the overused medications MOH generally reverts to an episodic pattern which is responsive to behavioral and prophylactic treatments.

Epidemiology

Migraine afflicts an estimated 28 million people in the United States. Surveys from the United States and elsewhere find 8% of children, 6% of men, and up to 18% of all women (about 12% of the population as a whole) experience migraine. Roughly, three out of four migraine sufferers are female. Migraine prevalence peaks in the third and fourth decades of life. At this time period, for example, migraine is 3.3 times more common in women than men. The female preponderance and variance with age is presumed to be related to hormonal milestones in women. Females with migraine often notice a change in their migraine pattern with hormonal fluctuation. Migraine increases at menarche and with the menstrual period. Improvement or worsening occurs during pregnancy and menopause. The headaches improve in the 1950s and 1960s in some patients, and are replaced with isolated aura symptoms.

Genetic Predisposition

There is a genetic predisposition to migraine. Approximately, 80% of migraine sufferers have a family history of migraine affecting a first-degree relative. Twin studies have demonstrated a familial influence. The concordance for migraine in monozygotic twins is greater than it is for dizygotic twins. However, it is also clear that the genetic background of the disorder is complex.

Disability and Impact

Migraine is associated with significant disability, lost work, and productivity. Headache is among the most prevalent medical problems and migraine is ranked by the World Health Organization as 19th worldwide of all diseases causing disability. Although only about one fourth of headache sufferers seek professional attention, over 40 million Americans consult a physician each year because of headache. Estimated aggregate indirect costs to employers in the United States for reduced productivity due to migraine range from 6.5 to 17 billion dollars annually. The problem can range from sporadic, infrequent headache with only minimal impact, to chronic, persistent headache that can severely disrupt the patient's life.

Risk Factors in the Transformation from Episodic to Chronic Migraine

Transformed migraine refers to the condition in which episodic migraine headache progresses to or "transforms" to chronic migraine – occurring at least 180 days per year. That is, high frequency chronic headache emerges, or escalates in frequency over time, from an earlier pattern of lower frequency episodic headache. Epidemiological studies suggest that there are specific variables or risk factors that might lead to progression from episodic to chronic migraine in selected patients: medication overuse, psychiatric comorbidity, stress, obesity, snoring, sleep disturbance, and others. Such modifiable risk factors may become potential targets for headache prevention.

Medication overuse: One of the most important and common causes of transformation is headache medication itself. When taken often, the very medications used to treat tension-type and migraine headache attacks can transform episodic headache into chronic headache. The medications known to play a role in this process include many over-the-counter and prescribed analgesics, analgesics combined with caffeine, opiates, ergotamines, and triptans. All these medications can be effective in treating episodic headache when used on an occasional basis. However, when used frequently, they may transform and aggravate headache. This process is called medication-overuse headache. MOH usually improves when the overuse ceases. Within 2 months (and frequently sooner), the

chronic headache pattern will revert back to the earlier episodic headache pattern or will remit. When episodic headache persists, it is often much more responsive to conventional treatment after the medication overuse has been eliminated.

Stress: Stress is the most commonly identified trigger for a headache in the average headache sufferer. Therefore, it is not surprising that frequent life changes and chronic daily stressors or "hassles" are also implicated in the development of chronic headaches.

Sleep Disturbance: Headache may be aggravated by frequent sleep disturbance. The most common sleep problem for headache sufferers is insomnia, including difficulty falling asleep, difficulty staying asleep, or poor quality "non-restful" sleep. Voluntarily limiting sleep or "burning the candle at both ends" can also increase risk of headache. Snoring during sleep is a specific risk factor for chronic headache in some patients. Though the cause is not known, it could be because snoring disturbs sleep quality or compromises breathing at night.

Obesity: Obesity is an independent risk factor for chronic migraine after adjusting for comorbidities and demographic variables. Interestingly, obesity is not a risk factor for migraine in general. The prevalence of episodic migraine in general does not vary significantly with the body mass index (BMI). However, studies have shown among migraineurs, high BMI was associated with more frequent headache attacks.

Caffeine: Finally, although caffeine can be beneficial in moderate amounts, and is used in some medications, it can also be a risk factor. Caffeine is the most widely used mood-altering substance in the United States. It is present in many beverages, dietary supplements, and in some foods such as chocolate. Many Americans consume caffeine daily with very little awareness that they are ingesting a drug with potent effects. For some headache sufferers, caffeine can aggravate headache in much the same way that medication overuse can.

Others: Female gender, presence of comorbid pain conditions, and psychiatric comorbidity have also been linked to transformation.

Psychiatric Comorbidity

Epidemiological studies have determined migraine and several psychiatric disorders to be comorbid. Psychiatric comorbidity portends a poorer prognosis for both pharmacologic and behavioral treatments. Migraine is comorbid with depression (OR 2.2–4.0), generalized anxiety (OR 3.5–5.3), panic disorder (OR 3.7), and bipolar disorder (OR 2.9–7.3). Patients with chronic migraine are at increased risk for major depression (OR 6.4 vs. 2.7) as well as for anxiety disorders (ORs 6.9 vs. 3.2 for all migraineurs).

Pharmacologic Treatment

A wide range of acute and prophylactic medications are available for treatment of migraine. Acute medications fall into general classes of medicines including analgesics, ergotamines, and triptans. Analgesics are considered nonspecific migraine medications as they work on pain systems in general, not just activated pain pathways involved in migraine. In contrast, triptans and ergotamines are migraine-specific medications. Analgesic and abortive medications are recommended for intermittent use. The American Migraine Prevalence and Prevention workgroup recommends a preventive medication for 4–5 non-disabling attacks or two or more disabling attacks per month. In addition, the use of behavioral headache medicine techniques combined with a daily medication will reduce the frequency of headaches further. A headache preventive therapy goal is to reduce attacks by 50% or more. Prophylactic or preventive medications are taken daily to prevent migraine attacks from occurring and commonly include a variety of antiepileptic, antidepressant, and antihypertensive medications. OnabotulinumtoxinA (BOTOX) is an injectable neurotoxin that has recently been FDA approved for migraine treatment. The mechanisms of action for migraine prevention remains unknown, but inhibition of nociceptive signaling has been suggested. Other medications (e.g., antiemetics, steroids) may also be utilized.

Behavioral Treatments for Migraine

Over the past three decades, several behavioral treatments for migraine prevention have been

used widely as independent therapies or in combination with medications. Most behavioral treatments are classified into three broad categories: cognitive-behavioral therapy (i.e., stress-management training), relaxation training, and biofeedback (often administered in conjunction with relaxation). In most instances, behavior therapies are used for preventing migraine episodes rather than for alleviating symptoms, once an attack has begun. Although these modalities may be effective as monotherapy, they are more often used in conjunction with medications. Goals for behavioral treatments include: reducing the frequency and severity of headache episodes; reducing headache-related disability; reducing reliance on medications; enhancing personal control over headache; and reducing distress and psychological symptoms.

Over 300 studies evaluating behavioral treatments for migraine have been published since the earliest empirical report evaluating a behavioral intervention for recurrent headache was published in 1969. The overwhelming majority of published clinical trials yielded positive outcomes, leading many professional practice organizations to recommend use of behavioral headache treatments alongside the preferred pharmacologic treatments for primary headache. An exhaustive meta-analysis of behavioral treatments for migraine funded by the Agency for Healthcare Research and Quality found these behavioral interventions yielded 35–55% reduction in migraine versus 5% reduction for no-treatment controls. The meta-analysis was the basis for an evidence-based guideline for migraine management produced by the *U. S. Headache Consortium* whose membership included the American Academy of Family Physicians, American Academy of Neurology, American Headache Society, American College of Emergency Physicians, American College of Physicians, American Osteopathic Association, and the National Headache Foundation. The Consortium's recommendations pertaining to behavioral interventions for migraine are as follows:

(a) Relaxation training, thermal biofeedback combined with relaxation training, electromyographic biofeedback, and cognitive-behavioral therapy may be considered as treatment options for prevention of migraine (Grade A Evidence).
(b) Behavioral therapy may be combined with preventive drug therapy to achieve added clinical improvement for migraine (Grade B Evidence).

Behavioral treatment typically entails 6–12 individual clinic sessions with a professional. Cost and availability of behavioral medicine specialists that provide services severely limit access of the average patient to behavioral treatments. In recent years, alternative delivery formats have been developed to increase availability and cost-effectiveness, such as group administration and minimal therapist-contact treatments. Minimal-contact or "home-based" formats have been the most extensively developed in this arena and provide similar treatment components to their clinic-based counterparts. Skills are introduced in the clinic but training occurs primarily at home guided by written materials and audio recordings. Consequently, only three or four clinic sessions may be necessary when behavioral techniques are delivered via this format. Meta-analyses have demonstrated the utility of the minimal-contact treatment approach, indicating that for many patients such treatments can be as effective as those delivered in a clinic setting.

Self-management

Headache Diary: Daily quantitative recordings of headache yield measures of headache frequency, duration, and intensity and may be employed to identify medication overuse, chronobiological patterns, menstrual and other headache triggers (see below), and to assess treatment outcomes. Diaries should be recorded daily, since memory for pain is prone to distortion. Diaries encourage patients to be systematic in their assessment of headache patterns and enable health-care providers to observe efficacy of treatments and dosages over time.

Headache Triggers: Identification and management of individual headache triggers is a common component of behavioral headache therapies. A trigger is an event that provokes

headache within 24 h at least 50% of the time. The level of vulnerability to any specific headache trigger factor varies from person to person. An individual's vulnerability to any given trigger factor also can rise and fall over time. Just the same, a person's overall tendency to experience a headache in response to trigger factors is likely to be an inherited trait. Prospective headache diaries are used to correlate headache onset or exacerbation with potential stimuli. The more common headache triggers reported by patients across studies is stress, menstruation, sleep disturbance, fasting or skipping meals, and weather. Additional triggers include alcohol, smoking, exercise, sexual activity, bright lights, odors, specific dietary additives, and others. There may be a threshold effect, in which multiple triggers or a combination of triggers tend to provoke headache. Headache trigger management includes: education about common headache precipitants, training patients to identify their unique triggers through prospective self-monitoring in a headache diary, applying behavior change principles to limit exposure to avoidable triggers (e.g., sleep deprivation) or apply self-management skills in responses to unavoidable triggers (e.g., stress).

Cross-References

- ▶ Biofeedback
- ▶ Cognitive Behavioral Therapy (CBT)
- ▶ Headaches: Psychological Management
- ▶ Relaxation: Techniques/Therapy
- ▶ Stress Management

References and Readings

Headache Classification Subcommittee of the International Headache Society (IHS). (2004). The international classification of headache disorders: 2nd edition. *Cephalalgia, 24*(Suppl. 1), 1–160.

Holroyd, K. A., Penzien, D. B., Rains, J. C., Lipchik, G. L., & Buse, D. A. (2007). Behavioral management of headache (562–598). In S. D. Silberstein, R. B. Lipton, & D. W. Dodick (Eds.), *Wolff's headache and other head pain* (8th ed.). New York: Oxford University Press.

Rains, J. C., Penzien, D. B., McCrory, D. C., & Gray, R. N. (2005). Behavioral headache treatment: History, review of the empirical literature and methodological critique. *Headache, 45*(Suppl.), S91–S108.

Migraine with Aura

- ▶ Migraine Headache

Migration and Health Services

- ▶ Acculturation
- ▶ Health Disparities

Milieu Interieur

- ▶ Homeostasis

Miller, Neal

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Biographical Information

Neal Elgar Miller was born on August 3, 1909, in Milwaukee, Wisconsin. He earned a Bachelor's degree from the University of Washington in

1931, a Master's from Stanford the following year, and his doctorate from Yale in 1935. He was then a social science research fellow at the Institute of Psychoanalysis, Vienna, for a year before returning to Yale as a faculty member in 1936.

During the Second World War, he served as an officer in charge of research in the Army Air Corps' Psychological Research Unit #1 in Nashville, Tennessee, and later directed the Psychological Research Project at the headquarters of the Flying Training Command in Randolph Field, Texas. Miller returned to Yale as Professor of Psychology and in 1952, he was appointed the first James Rowland Angell Professor of Psychology.

In 1966, Miller transferred to Rockefeller University, where he spent an additional 15 years of service. He became professor emeritus at Rockefeller in 1981 and research affiliate at Yale in 1985.

Major Accomplishments

Miller is often credited as being the founder of behavioral medicine. He made significant contributions to our understanding of the relationship between reinforcement mechanisms and the control of autonomic behavior, and pioneered the field of biofeedback, which is used successfully today to treat a variety of medical conditions. He was also active in many other fields of scientific investigation.

His early research focused on the investigation of Freudian theory and clinical phenomena using experimental analysis of behavior techniques. He concluded that fear is a learnable drive, and began to investigate other autonomic behaviors to determine if they could be modified through instrumental conditioning. He used behavioral methodologies and neurophysiological techniques to investigate hunger and thirst. This integration of ideas and practices laid the foundation for modern neuroscience, and also fundamentally changed understanding of behavior and motivation. Interdisciplinary collaboration was a hallmark of his work.

Following animal model work on anxiety, Miller began to investigate other autonomic behaviors, trying to find out if they could also be modified through instrumental conditioning. He investigated hunger and thirst, using behavioral methodologies and neurophysiological techniques. He concluded that the autonomic nervous system could be as susceptible to classical conditioning as the voluntary nervous system. This led to his work on biofeedback. Initial reaction in the scientific community to his claim that people could learn to control autonomic functions was not positive. However, his sustained investigation revealed that this is indeed the case, and biofeedback became gradually accepted in scientific circles as a method to help treat various medical conditions, including high blood pressure, epilepsy, attention deficit and hyperactivity disorder (ADHD), and migraines.

During his career, Miller was president of many learned societies, including the American Psychological Association (APA), the Society for Neurosciences, the Biofeedback Society of America, and the Academy of Behavioral Medicine Research.

Miller received the APA Distinguished Scientific Contribution Award in 1959 and the APA Citation for Outstanding Lifetime Contribution to Psychology in 1991. In 1993, APA's Board of Scientific Affairs voted to honor him by establishing the Annual Neal Miller Distinguished Lecture, to be dedicated to neuroscience and animal research and presented at each APA convention. Miller himself presented the first lecture in 1994. The Academy of Behavioral Medicine Research established the Neal E. Miller New Investigator Award in his honor, and he received the National Medal of Science in 1964.

Editor's note: Dr Miller passed away in 2002.

Cross-References

- ▶ Autonomic Nervous System (ANS)
- ▶ Behavioral Medicine
- ▶ Biofeedback
- ▶ Hypertension
- ▶ Migraine Headache

References and Readings

Coons, E. E. (2002). Neal Elgar Miller (1909–2002). *American Psychologist, 57*, 784–786.
Miller, N. E. (1975). Behavioral medicine as a new frontier: Opportunities and dangers. In S. M. Weiss (Ed.), *Proceedings of the national heart and lung institute working conference on health behavior, 1975* (pp. 1–11). Washington, DC: DHEW Publ. #NIH, 76-868.
Miller, N. E. (1979). Behavioral medicine: New opportunities but serious dangers. *Behavioral Medicine Update, 1*(2), 5–7.
Miller, N. E. (1981). An overview of behavioral medicine: Opportunities and dangers. In S. M. Weiss, J. A. Herd, & B. H. Fox (Eds.), *Perspectives on behavioral medicine* (pp. 3–22). New York: Academic Press (Note: Based on his Presidential address given at the meeting of the Academy of Behavioral Medicine Research, Snowbird, Utah, June 1979).
Miller, N. E. (1983). Behavioral medicine: Symbiosis between laboratory and clinic. *Annual Review of Psychology, 34*, 1–31.
Miller, N. E. (1984). Behavioral medicine. In R. J. Corsini (Ed.), *Encyclopedia of psychology* (Vol. 1, pp. 126–130). New York: Wiley Interscience.
Miller, N. E. (1987). Behavioral medicine. In G. Adelman (Ed.), *Encyclopedia of neuroscience* (pp. 122–124). Boston, MA: Birkhauser.
Miller, N. E. (1992). Some trends from the history to the future of behavioral medicine. *Annals of Behavioral Medicine, 14*(4), 307–309.
Miller, N. E. (1995). Perspective on behavioral medicine and the brain's hierarchy of homeostatic controls. In T. Kikuchi, H. Sakuma, I. Saito, & K. Tsuboi (Eds.), *Biobehavioral self-regulation: Eastern and Western perspectives* (pp. 229–245). Tokyo: Springer.
Spielberger, C. D. (1992). American Psychological Association citation for outstanding lifetime contribution to psychology. *American Psychologist, 47*, 847.
Zimbardo, P. G., & Miller, N. E. (1958). The facilitation of exploration by hunger in rats. *Journal of Comparative and Physiological Psychology, 51*, 43–46.

Millon Behavioral Medicine Diagnostic (MBMD)

Mike Antoni
Department of Psychology, University of Miami, Sylvester Cancer Center, Miller School of Medicine, Miami, FL, USA

Synonyms

MBMD

Definition

Short Definition

The Millon Behavioral Medicine Diagnostic (MBMD) is a broadband self-report psychosocial instrument designed to assess physically ill and behavioral medicine patients to help develop effective treatment and management plans.

Description

The Millon Behavioral Medicine Diagnostic (MBMD) is a broadband self-report psychosocial instrument designed to assess physically ill and behavioral medicine patients aged 18–85 years to develop effective treatment and management plans. It provides information on test response patterns, health behaviors, psychiatric features, personality-based interpersonal coping styles, stress-moderating variables, and predicted responses to medical treatments.

The test was developed using a 3-step validation process wherein theory-generated items were used to form the 29 clinical scales representing the domains tapped by the test (Millon, Antoni, Millon, Minor, & Grossman, 2006). The initial validation sample comprised 720 patients (cancer, cardiac, diabetes, HIV, neurological, pain) drawn from medical settings across 22 states in the USA and Canada. Item content was refined to optimize scale internal consistency and test-retest reliability. Concurrent validity was determined through correlations with scales widely used in behavioral medicine research and with medical staff ratings. The MBMD is a 165-item true-false test in English and Spanish versions appropriate for a sixth grade education reading level. The test requires 20–25 min to complete and can be scored through a computer program, a fax-in service to the test publisher, or using hand-scoring templates.

The MBMD reports result in a profile depicting the raw scores and norm- and gender-adjusted prevalence scores for each scale representing the major test domains (Psychiatric Indicators [5 scales], Coping Styles [11 scales], Stress Moderators [6 scales], and Treatment Prognostics [5 scales] and Management Guides [2 scales]). Scores on other test domains,

Negative Heath Behaviors and Response Patterns, are expressed as categories. The test also yields a multipage report that highlights patient assets and liabilities in each domain, and uses "cross-domain" synthesis to modulate the interpretation of individual scores based on other domains in the profile (e.g., response pattern, psychiatric indicators, and coping style). At the end of the report there is a one-page Health Provider Summary that includes the most salient aspects of the patient's test results expressed in bulleted short phrases, and a list of noteworthy item endorsements that may require immediate attention and follow-up.

Subsequent validation studies based on 711 patients undergoing bariatric surgery procedures and 1,200 patients undergoing treatment for chronic pain in a variety of treatment settings across the USA have produced specialized test reports designed for use in these settings. Designed to accompany the general MBMD report, each of these "specialized" reports contains additional report sections germane to pretreatment considerations and longer-term patient management. The bariatric report contains information across 24 variables describing hypothesized benefits of additional presurgical assessments and interventions, and the benefits of postsurgical adjunctive behavioral interventions. The pain report comes in two versions – a presurgical report and a nonsurgical report. The pain reports include general medical prevalence scores, and chronic pain-specific percentile scores. The pain reports contain probabilistic statements relevant to pretreatment and posttreatment patient management. The MBMD is associated with health behaviors and medical outcomes in diverse clinical populations (Cruess, Meagher, Antoni, & Millon, 2007, Cruess, Localio, Platt, & Kimmel, 2010; Farrell, Shen, Mallon, Penedo, & Antoni, 2011; Harper, Wager, & Chacko, 2010; Lavoie et al., 2010; Pereira et al., 2010).

References and Readings

Cruess, D., Localio, R., Platt, A., & Kimmel, S. (2010). Patient attitudinal and behavioral factors associated with warfarin non-adherence at outpatient anticoagulation clinics. *International Journal of Behavioral Medicine, 17*, 33–42.

Cruess, D., Meagher, S., Antoni, M. H., & Millon, T. (2007). Utility of the millon behavioral medicine diagnostic (MBMD) to predict adherence to highly active antiretroviral therapy (HAART) medication regimens among HIV-positive men and women. *Journal of Personality Assessment, 89*, 277–290.

Farrell, K., Shen, B.-J., Mallon, S., Penedo, F., & Antoni, M. H. (2011). Utility of the millon behavioral medicine diagnostic (MBMD) to predict medication adherence in patients diagnosed with heart failure. *Journal of Clinical Psychology in Medical Settings, 18*(1), 1–12.

Harper, R., Wager, J., & Chacko, R. (2010). Psychosocial factors in noncompliance during liver transplant selection. *Journal of Clinical Psychology in Medical Settings, 17*, 71–76.

Lavoie, K., Bouthiller, D., Bacon, S., Lemiere, C., Martin, J., Hamid, Q., et al. (2010). Psychologic distress and maladaptive coping styles patients with severe vs moderate asthma. *Chest, 137*, 1324–1331.

Millon, T., Antoni, M., Millon, C., Minor, S., & Grossman, S. (2006). *Millon behavioral medicine diagnostic (MBMD) manual* (2nd ed.). Minneapolis, MN: NCS Pearson.

Pereira, D., Christian, L., Patidar, S., Bishop, M., Dodd, S., Athanason, R., et al. (2010). Spiritual absence and 1-year mortality after hematopoeitic stem cell transplant. *Biology of Blood and Marrow Transplantation, 16*, 1171–1179.

Mindfulness

Alan M. Delamater
Department of Pediatrics, University of Miami Miller School of Medicine, Miami, FL, USA

Synonyms

Insight meditation; Meditation

Definition

A key factor in insight meditation is the development of bare attention and mindfulness. Bare attention is observing phenomenon as they are, without comment, evaluation, or judgment.

Mindfulness is the application of bare attention to changing objects as they occur in each moment, with an attitude of acceptance so that there is no clinging to objects that are desirable and there is no aversion to objects that are not desirable. In mindfulness practice, one does not identify with objects of attention. The goal is to increase the frequency of noticing objects as they arise and pass away in consciousness. Concentration is also an important component of insight meditation, but is used in insight meditation to focus on changing objects rather than a single object of attention (Goldstein, 1976; Goleman, 1988).

As mindfulness becomes stronger and more sustained, insights about the nature of the mind and reality are experienced, including three fundamental characteristics of existence: impermanence, suffering, and not self, i.e., the concept that the self is a creation. Insight or vipassana meditation has its origins in Buddhism and was developed over 2,500 years ago; vipassana is the Pali word meaning to clearly see the nature of phenomenon.

In recent years, mindfulness has been incorporated into psychotherapeutic techniques and health-care interventions. There is a growing evidence base for its efficacy in treating pain, anxiety, stress, depression, and chronic health conditions, as well as for stress reduction and improved well-being in healthy people (Baer, 2006; Bohlmeijer, Prenger, Taal, & Cuijpers, 2010; Hofmann, Sawyer, Witt, & Oh, 2010; Shapiro & Carlson, 2009). Mindfulness has been adapted to several treatment programs, including mindfulness-based stress reduction (Kabat-Zinn, 2003), mindfulness-based cognitive therapy (Segal, Wiliams, & Teasdale, 2002), dialectical behavior therapy (Linehan, 1993), and acceptance and commitment therapy (Hayes, Strosahl, & Wilson, 1999).

Cross-References

- ▶ Meditation
- ▶ Stress Management
- ▶ Transcendental Meditation

References and Readings

Baer, R. (2006). *Mindfulness-based treatment approaches: Clinician's guide to evidence base and applications*. Boston: Elsevier Academic Press.

Bohlmeijer, E., Prenger, R., Taal, E., & Cuijpers, P. (2010). The effects of mindfulness-based stress reduction therapy on mental health of adults with a chronic medical disease: A meta-analysis. *Journal of Psychosomatic Research, 68*(6), 539–544.

Goldstein, J. (1976). *The experience of insight: A simple and direct guide to Buddhist meditation*. Boulder, CO: Shambhala.

Goleman, D. (1988). *The meditative mind: Varieties of meditative experience*. New York: Tarcher.

Hayes, S., Strosahl, K., & Wilson, K. (1999). *Acceptance and commitment therapy*. New York: Guilford.

Hofmann, S. G., Sawyer, A. T., Witt, A. A., & Oh, D. (2010). The effect of mindfulness-based therapy on anxiety and depression: A meta-analytic review. *Journal of Consulting and Clinical Psychology, 78*(2), 169–183.

Kabat-Zinn, J. (2003). Mindfulness-based interventions in context: Past, present, and future. *Clinical Psychology: Science and Practice, 10*, 144–154.

Linehan, M. (1993). *Cognitive-behavioral treatment of borderline personality disorder*. New York: Guilford.

Segal, Z. V., Wiliams, M. G., & Teasdale, J. D. (2002). *Mindulness-based cognitive therapy for depression: A new approach to preventing relapse*. New York: Guilford Press.

Shapiro, S. L., & Carlson, L. E. (2009). *The art and science of mindfulness: Integrating mindfulness into psychology and the helping professions*. Washington, DC: American Psychological Association.

Mini-Finland Health Survey

Arpo Aromaa
Health and Functional Capacity,
National Institute for Health and Welfare,
Helsinki, Finland

Definition

The Mini-Finland health survey (MFhes) is the first comprehensive nationwide study combining interview, questionnaire, and examination methods. Its baseline field work was carried out by the Social Insurance Institution's (SII) Mobile Clinic Unit (*Autoklinikka*) during 1978–1980 in a representative sample of adults aged 30 years,

and 96% of the sample was interviewed and 90% examined. The register-based follow-up of the sample of 8,000 is complete until 2010.

Description

In Europe in the twentieth century, the SII Mini-Finland health examination survey (MFhes; *in Finnish: Mini-Suomi terveystutkimus*) carried out in 1978–1980 was the first comprehensive nationally representative study of health, chronic diseases, functional limitations, their determinants (social environmental, behavioral and biological risk, and protective factors), use of health services comprising rehabilitation, and need and unmet need for care and help. It combined the sociomedical, epidemiological, and social science research traditions of the SII, i.e., the paradigms and methodological skills of the Mobile Clinic Unit's health examinations and the Social Security Health Interview surveys, both initiated in 1964, soon after the introduction of the sickness insurance scheme covering the whole population of Finland. In addition to the general goals of a health survey, a special aim was to compare data on chronic diseases and need for care independently obtained by a health interview and a health examination.

The Sample

A two-stage cluster sample of 8,000 people aged 30 years and over in 40 study areas (clusters) was designed, and a list of eligible individuals with contact information was drawn from the national population register. The sampling frame comprised the whole population, i.e., also institutionalized people.

The Concept of Health

Health was conceptualized as a perceived, biological, and social phenomenon. Special emphasis was placed to the measurement of functional capacity, its limitations, handicaps, and disabilities. Work ability and functioning were concepts used to measure the state of "health" independently of the presence or absence of diseases.

The Phases of the Field Work

The main phases of the field survey were the health interview; the health examination, comprising a two phase field work; and a third phase, the in-depth examination in the SII rehabilitation research center.

The Health Interview and the Examination

First, nurses (626 nurses over the whole country) carried out a structured health interview in the persons' home (or institution), and 7,703 persons (96% of the sample) participated. Within 1–6 weeks of the interview, all individuals in the sample were invited by letter to the Mobile Clinic Unit's health examination. The unit was situated centrally in the study area. In the initial screening phase, 7,217 individuals (90% of the sample) were interviewed and examined. A few months later, on the basis of findings in the screening phase and health-related data drawn from the SII's registers, those that might suffer from one of the core chronic diseases were asked to come to the clinical phase, 5,819 individuals (73%) attended the clinical phase and 4,840 (61%) were subjected to a doctor's structured history and examination, which as indicated by the screening phase findings concentrated on the cardiovascular and respiratory system, on the musculoskeletal system, or on all of them.

Non-attendants were approached by a mail questionnaire after the field study proper. Therefore, some health data are available on 98% of the whole sample.

Health-Related Content of the Health Interview

Perceived health, chronic disease or defect, care received, illness-induced change of occupation or duties, handicap caused by illness or defect in the present work, functional limitations (ADL and IADL), sickness days, psychological symptoms, perceived need of care or help for psychological symptoms and visits to a psychiatrist or a psychologist, use of medical care, prescription medicines (name and classification according to the main indication) currently used, dental care, preventive health examinations, smoking, and dietary habits.

Methods of the Health Examination

The health data were obtained by questionnaires, symptom interviews, tests of functioning, and clinical measurements. In the screening phase, the order of the measurements was the following: (1) height, weight, and BMI; (2) interviews on symptoms; (3) a urine sample; (4) blood pressure (mercury sphygmomanometer) and heart rate; and (5) ECG and respiratory tests by a Vitalograph spirometer. Other tests and measurements were carried out in varying order. They comprised supplementary interviews, a joint function test, a dental examination, a chest X-ray, and a blood sample. Finally, psychometric group tests were carried out and reaction time was measured. The screening phase comprised, i.e., questions on psychological symptoms and the general health questionnaire (GHQ-36). The clinical examination for mental health problems consisted of a present state examination interview (short version) carried out by one trained psychiatric nurse. Half of these interviews were carried out during the screening phase and half during the clinical phase. In the clinical phase proper, to assess reliability/repeatability, all screening measurements were repeated on a 20% randomly drawn subsample of the subjects, and a clinical doctor's examination directed according to the earlier findings was carried out. The examining doctor recorded also any other concurrent diagnoses and assessed each examinee's work and functional ability, disabling diseases and need for care. Current good diagnostic and therapeutic practice was used as a yardstick when assessing the need for care and unmet need for care.

Finally, 991 persons were examined in depth for workability and rehabilitation needs in the SII Rehabilitation Research Centre.

Information Obtained by Questionnaires and Interviews During the Health Examination

Perceived health, chronic disease, diseases diagnosed by a doctor (a disease list of 32 diseases) and follow-up questions for each disease mentioned (hospital care ever, medicine use ever, current care by a medical doctor, current use of medicines), hospital care, surgical operations, care by a medical doctor, prescription medicines (name and classification according to indication), nonprescription medicines, work ability and work disability, ability to participate and perform tasks and duties outside gainful employment, as well as in leisure time activities. There were also a set of questions on the ability to perform IADL and ADL activities. The final part comprised questions on physical activity such as physical strenuousness of work, leisure time physical activity comprising also details of regular exercise, and physical activity on the way to and from work. The last part of the main questionnaire of the health examination inquired about work and working conditions. A set of questions were specially constructed to describe leisure time activities and hobbies, and the final questions inquired about alcohol use in a way to allow quantification of the amount of absolute alcohol consumed on average during a week over the past month.

Record Linkage at the Time of the Field Work and Follow-up of the Cohort

By record linkage, after the field survey, further data were obtained from national registers on use of services, diseases, and disabilities. Final diagnostic assessments were made by combining all available information. As a result, diagnostic and other clinical data are available on 5,292 people invited to come to the doctor's examination. These are all of the individuals meeting the criteria screening positivity for the clinical phase and equal 66% of the sample.

Record linkage was used also for a follow-up of the sample, which today is complete until year 2009.

Biobank and Data Set

Frozen serum and plasma are stored by the National Institute for Health and Welfare (THL), and samples are being used for prospective studies to assess the impact of many biochemically determined risk and protective factors on mortality and risk of chronic diseases.

The large data set has been fully characterized and described, and upon proper agreement, the data can be and have been used extensively for collaborative research by THL and outside scientists.

Social Circumstances, Living Conditions, and Behavior

The readership may be especially interested in some of the available data on social circumstances and living conditions as well as methods used for measuring health-related behavior.

The usual information on family/household is available such as data on the number of family members, their age distribution, the subject's marital status, the family head's occupation (current or latest previous) and his/her receipt of any pension benefit, the subject's educational attainment, the community (municipality) in which the subject has lived longest, the subject's receipt of a pension, and total family income.

The subject's main occupation and employer, working hours, possible unemployment or layoff, the salary basis (e.g., hourly or monthly payment, paid by the piece), and comparable information related to job and employment.

Furthermore, information such as the distance to the nearest public health center doctor, to the nearest private doctor, and to the nearest dentist was gathered.

Current Context of the Mini-Finland Health Survey

The Mini-Finland health survey was originally designed as a single-handed cross-sectional and epidemiological follow-up study. However, it is now part of a unique set of nationally representative cross-sectional and individual level follow-up studies based on the same concept and with the same or similar health interview and health examination data. I refer to the Health 2000 national survey (Aromaa & Koskinen, 2004) and the ongoing Health 2011 national survey. The data gathered on these studies are even richer, and in addition to serum and plasma samples, the biobank now also contains DNA and RNA samples. After complete sequencing of the genome has become possible, the genetic information on the Health 2000 subjects has been extensively used to identify genes increasing the risk of the major public health problems, and corresponding effects on and interactions with social determinants, living conditions, and biological risk and protective factors of chronic diseases.

References and Readings

Aromaa, A., & Koskinen, S. (Eds.). (2004). *Health and functional capacity in Finland: Baseline results of the health 2000 Health Examination Survey*. Helsinki: Publications of the National Public Health Institute.

Mini-Mental State Examination

Jane Upton
School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Definition

This is a practical method for grading the cognitive state of patients for the clinician (Folstein & McHugh, 1975).

Description

The mini-mental state examination was developed in 1975 (Folstein & McHugh, 1975) and has since been widely used in clinical and research settings. It is most commonly used by clinicians in a medical setting to assess for dementia, where it is administered serially to assess the effect of time on the progression of dementia. It is a 30-item screening tool which takes approximately 10 min to complete by a trained administrator. Severe cognitive impairment is indicated by a score of ($\leq$9 points) moderate (10–20 points) and mild (21–24 points). A score of 25 and over indicates normal cognition.

It includes the following tasks of cognition:

- Orientation to time and place
- Registration: The ability to learn the names of three objects

- Attention and calculation: The performance of serial 7's, or spelling "world" backward
- Recall: Remembering the three objects that they had learned in the registration task
- Language: Naming a pencil and watch, repeating a sentence, following a three-stage commend, reading and obeying a command, and writing a sentence
- Basic motor skills: Copying a design of two intersecting pentagons

Psychometric Properties

Reliability was investigated by Molley and colleagues, who reported an intraclass correlation for the mini-mental state examination of 0.69 (Molloy, Alemayehu, & Roberts, 1991). The inter-rater agreement, particularly for the attention and calculation task, has been reported as being poor (Davey & Jamieson, 2004). This examination has been found to be responsive to change (Uhlmann, Larson, & Buchner, 1987).

The MMSE can be obtained from the current copyright owner Psychological Assessment Resources http://www4.parinc.com/

Cross-References

► Cognitive Appraisal

References and Readings

Davey, R., & Jamieson, S. (2004). The validity of using the mini mental state examination in NICE dementia guidelines. *J Neurology Neurosurgery and Psychiatry*, *75*, 341–345. Retrieved from http://jnnp.bmj.com/content/75/2/343.2.full.pdf

Folstein, M., & McHugh, P. (1975). Mini mental state a practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research, 12*, 189–198.

Molloy, D., Alemayehu, E., & Roberts, R. (1991). Reliability of a standardised mini mental state examination compared with the traditional mini mental state examination. *The American Journal of Psychiatry, 148*, 102–105.

Uhlmann, R., Larson, E., & Buchner, D. (1987). Correlations of mini-mental state and modified dementia rating scale to measures of transitional health status in dementia. *Journal of Gerontology, 42*, 33–36. doi:10.1093/geronj/42.1.33.

Minor Tranquilizer

► Anxiolytic

Minority Health

Luz M. Garcini[1], Eleshia J. P. Morrison[2] and Georita Marie Frierson[3]
[1]Ethnic Minority & Multicultural Health SBM SIG Co-Chair, SDSU/UCSD Joint Doctoral Program in Clinical Psychology, San Diego, CA, USA
[2]Department of Psychology, Ethnic Minority & Multicultural Health SBM SIG Chair, The Ohio State University, Columbus, OH, USA
[3]Department of Psychology, Southern Methodist University, Dallas, TX, USA

Synonyms

Health disparities; Multicultural health

Definition

Minority health refers to the special medical, psychosocial, and/or health needs of minority groups, including addressing the amelioration and prevention of health disparities. Health disparities refer to inequalities in the overall rate of disease prevalence, incidence, morbidity, mortality, or survival rates among certain groups of people when compared to the general population, as well as differences in the quality, outcomes, cost, or use of and access to health-care services (Minority Health and Health Disparities Research and Education

Act, 2000). Although primarily associated with health issues among ethnic and racial minorities, minority health also addresses the medical, psychosocial, and/or health needs of people with disabilities and minorities on the basis of age, socioeconomic inequality, and sexual orientation. Thus, any sociodemographic variable could lead to a minority health concern.

Description

Historical Background of Minority Health in the United States

As early as the beginning of the 1900s, intellectual leaders and activists such as W.E.B. Du Bois and Booker T. Washington documented the effect of contextual factors (i.e., economic poverty, inferior education, segregation and racism) on the health and quality of life of minority groups, particularly African Americans (Thomas, Benjamin, Almario, & Lathan, 2006). In 1915, Washington launched a comprehensive public health education campaign, the Negro Health Improvement Week, to promote health education among segregated Blacks and to increase awareness of existing health disparities. Later, the campaign evolved into the National Negro Health Movement, one of the most effective movements to promote direct and indirect cooperation between government organizations and members of a minority group for the betterment of minority health. An important legacy of the National Negro Health Movement was that the Black community became among the most active American groups in the national and worldwide movement for minority health improvement.

During the 1960s and inspired by the civil rights movement, other ethnic minority groups also became increasingly involved in advocacy efforts aimed at community health improvement. A notable example was that of Latinos and the development of organizations such as the National Organization for Mexican American Services (NOMAS) and the Southwest Council of La Raza (SWCLR), which later became the National Council of La Raza (National Council of La Raza [NCLR], 2011). These organizations aimed to increase representation of the Latino perspective at the local and national level on issues of health, education, immigration, housing, employment, training, and civil rights enforcement. Nevertheless, despite these early efforts, it was not until two decades later that minority health issues made it onto the national agenda.

In 1985, Margaret Heckler, Secretary of the US Department of Health and Human Services, released the Federal Report of the Secretary's Task Force on Black and Minority Health (Department of Health & Human Services [DHHS], 1985) to introduce minority health onto the national agenda. This report provided the first comprehensive national minority health study published by a government department to document widespread and persistent disparities in health status between various ethnic/racial minorities (i.e., African Americans, Hispanics, Native Americans, Asians and/or Alaskan Natives) and Whites. Additionally, Heckler's report provided several primary recommendations to address for the improvement of minority health. These recommendations were to (1) implement the dissemination of health education through community outreach; (2) develop culture- and context-sensitive health education programs and interventions; (3) improve access, delivery, and financing of health services to minority populations; (4) promote communication, coordination, and collaboration among federal and local agencies in administering programs addressing minority health issues; (5) improve the quality, availability, and use of health data pertaining to minority populations; and (6) increase and support research to inform the improvement of minority health. Given their relevance, the aforementioned recommendations continue to be at the forefront of the research and practice of minority health issues even to this date.

To manage the implementation of recommendations set forth by Heckler's report, the DHHS created the federal Office of Minority Health (OMH) in 1986 (OMH, 2011). Two years later, and also in response to the same report, the Centers for Disease Control and Prevention (CDC) established its own Office of the Associate Director for Minority Health (ADMH), which later became the CDC's Office of Minority Health

(OMH) in 2002 and most recently the Office of Minority Health and Health Disparities (OMHD) (CDC, 2011). Unique to the OMHD is its expanded focus on addressing minority health issues for populations defined not only by race/ethnicity but also by socioeconomic, age, disability, and risk status related to sex and gender. Also, acknowledging the importance of research as essential to the advancement of minority health, the National Institutes of Health (NIH) established the Office of Research on Minority Health in 1990. In 2000, the Minority Health and Health Disparities Research and Education Act signed by President Bill Clinton elevated the NIH Office of Research on Minority Health to become the National Institute on Minority Health and Health Disparities (NIMHD, 2011). Since its establishment, the NIMHD has focused on emphasizing the need for and improvement of the visibility of basic, clinical, social, and behavioral research focusing on minority health issues and health disparities, as well as promoting research infrastructure and training of highly qualified minority scientists.

Important national and state initiatives exist that aim at health promotion and disease prevention and management among minority groups. For three decades, Healthy People, the nation's health agenda, has set a target for reaching health objectives for minority populations. In Healthy People 2000, the focus was on reducing health disparities, while Healthy People 2010 aimed to eliminate health inequality. Expanding on the goals of these two previous agendas, Healthy People 2020 aims at improving the health of all groups, thus extending its focus to address minority health beyond racial/ethnic groups to the inclusion of people with disabilities and minorities on the basis of age, socio-economic inequality, and sexual orientation. Expanding minority health to focus on vulnerable populations at risk beyond ethnic/racial groups is a more appropriate way to represent the needs and changing demographic profile of the US population.

Behavioral Medicine and Minority Health

The United States is undergoing fundamental changes in its minority populations that will challenge the current health-care system and the field of behavioral medicine (Institute for the Future, 2003; Yali & Revenson, 2004). Continuing with current trends, there will be an increase in older populations, as well as growth in the number of people varying in sexual orientation. Likewise, the prevalence of certain ethnic/racial minorities will also increase. It is estimated that by 2050, non-Hispanic Whites will make up less than half of the US population (46.3%), with minority and immigrant groups becoming a majority. Of all ethnic/racial minorities, Hispanics or Latinos are expected to experience the largest population growth, from 16% today to 30% by 2050 (US Census Bureau, 2010). As these population changes take place, the need for culture-sensitive and contextual approaches to the research and practice of minority health will be increasingly necessary. Given its interdisciplinary approach and biopsychosocial perspective, behavioral medicine provides an appropriate platform for the conceptualization, assessment, treatment, research, and policy development of minority health issues as defined by changing demographics in the USA. In developing an understanding of the effect of cultural and contextual determinants of health and illness to the prevention, promotion, etiology, diagnosis, treatment, and rehabilitation of minority health, behavioral medicine can efficiently and effectively address even the most complex issues pertaining to minority health.

Cross-References

- ▶ Behavioral Medicine
- ▶ Ethnic Identities and Health Care
- ▶ Health Disparities

References and Readings

Centers for Disease Control and Prevention (CDC)/Office of Minority Health and Health Disparities (OMHD). (2011). Retrieved from http://www.cdc.gov/omhd

Institute for the Future. (2003). *Health and health care 2010: The forecast, the challenge*. Princeton, NJ: Jossey-Bass.

Minority Health and Health Disparities Research and Education Act of 2000. (2000). 106th congress.
National Council of La Raza (NCLR). (2011). Retrieved from http://www.nclr.org
National Institute on Minority Health and Health Disparities (NIMHD). (2011). Retrieved from http://www.nimhd.nih.gov
Thomas, S. B., Benjamin, G. C., Almario, D., & Lathan, M. J. (2006). Historical and current policy efforts to eliminate racial and ethnic health disparities in the United States: Future opportunities for public health education. *Health Promotion Practice, 7*(3), 324–330.
U.S. Census Bureau. (2010). Projections of the population by sex, race and Hispanic origin for the Unite States: 2010 to 2050. http://www.census.gov/population/www/projections/files/nation/summary/np2008-t4.xls
U.S. Department of Health and Human Services Office of Minority Health. Office of Minority Health (OMH). (2011). Retrieved from http://minorityhealth.hhs.gov
Yali, A. M., & Revenson, T. A. (2004). How changes in population demographics will impact health psychology: Incorporating a broader notion of cultural competence into the field. *Health Psychology, 23*(2), 147–155.

Minority Subgroups

Orit Birnbaum-Weitzman
Department of Psychology, University of Miami, Miami, FL, USA

Synonyms

Ethnicity subgroups; Subethnic groups; Subgroup heterogeneity

Definition

Minority subgroups refer to the within-group variation that is notably manifested among individuals from the same ethnic and racial group according to one or more sociodemographic factors including but not limited to nationality, language, religion, gender, social/immigration histories, educational level, and socioeconomic status.

Description

All racial/ethnic minority populations, including persons of Hispanic/Latino, African/Black, and Asian descent in the United States, are characterized by considerable diversity and can be further classified into minority subgroups. However, data on health aspects among minority groups have usually been combined in behavioral health analysis (For example see Whitfield, Weidner, Clark, and Anderson, 2002). To a large extent, the health-care system still fails to understand the complexity and diversity of immigrant and ethnic populations (Srinivasan and Guillermo, 2000). There is a need for greater specificity of racial and ethnic subgroups in behavioral medicine research (Feinleib, 1993). Studies that fail to disaggregate minority subgroups may be inaccurately reporting health research and overlooking health risks experienced by some immigrant groups (Srinivasan and Guillermo, 2000). Methodological difficulties typically arise in assessing the variables that are important in further classifying ethnic minorities into subgroups. However, by subsuming them under larger racial categories aggregating their data into one undifferentiated group, the health and health-care needs of specific minority subgroups are overlooked. Although scant, recent epidemiological studies are making an effort to collect additional data to characterize the heterogeneity of the standard racial/ethnic categories and provide data on subgroup differences. This trend will improve development of effective treatment interventions to address public health needs of ethnic minorities.

American ethnic subgroups vary substantially with respect to demographic and socioeconomic characteristics (see McCracken et al., 2007 for example in Asian Americans). There are extreme variations in income and rates of poverty among ethnic subgroups (Stone and MCQuillan, 2007; Srinivasan and Guillermo, 2000), and these variations can affect health. Minority subgroups also vary markedly in terms of religion, culture, and language. The social and political histories of minority subgroups and the variability within these subgroups have implications for the general health and mental health of this population (see

for example Williams, Nazroo, Kooner, and Steptoe, 2010). Time since immigration is directly related to language spoken at home and English-language fluency and is also a source of variability within minorities (McCracken et al., 2007). Ethnic subgroups that have lived longer in the United States are more likely to speak English better or speak only English at home. This suggests that certain minority subgroups face different barriers to medical care as a result of linguistic and cultural differences (McCracken, et al., 2007). Generational factors and the process of adjustment to living in the United States also have an impact on health and mental health.

Acculturation and adoption of westernized diets and behaviors can also contribute to explanations of health differences in minority subgroups. Within ethnic populations, minority subgroups show mixed profiles of health behaviors such as smoking, alcohol consumption, and levels of physical activity (Alegria et al., 2008; Williams et al., 2010). Additionally, samples from minorities living in the same geographical catchment areas suggest that despite little variation in environmental context, subgroup variations arise as a consequence of individual differences in religious and psychosocial experiences (Williams et al., 2010). Interreligious variation observed in some studies highlights the relevance of studying subgroup heterogeneity and indicates that a clear understanding and assessment of these concepts will increase the understanding of health differences between ethnic/racial groups (Williams et al., 2010). Furthermore, differential exposure to stressful experiences in particular discrimination stress can further explain inequalities in physical and mental health among minority subgroups and has been an important topic in minority research (Perez, Fortuna, and Alegria, 2008; Gee, Ro, Shariff-Marco, and Chae, 2009). Variations in perceived discrimination are related to sociodemographic and cultural differences across ethnic subgroups.

Hispanics/Latinos are the largest minority group in the United States, but they are not a homogeneous group. Hispanics are a diverse group in terms of nativity status, country of origin, racesocioeconomic status, and exposure to U.S. culture (Borrell and Crawford, 2009; Amaro and Zambrana, 2000). Recent findings suggests that nativity status, racial identification, and country of origin may have implications for Hispanics' mortality and morbidity outcomes (Borrell and Crawford, 2009). In contrast to the paradox of lower mortality rates for Hispanics as compared with non-Hispanics, disaggregated data suggest that the mortality advantage does not apply to all Hispanics (Borrell and Crawfors, 2009). Younger Hispanics, Puerto Rican men and women, Mexican American women, and those Hipanics who identify themselves as White exhibit higher death rates than non-Hispanic Whites (Borrell and Crawford, 2009). Health status varies within Hispanics according to nationality and can differ between Puerto Ricans, Mexicans, and Cubans living in the USA (Borrell and Crawford, 2008). The prevalence of hypertension among Hispanic Americans falls between that of Blacks and non-Hispanic Whites but appears to increase with the process of acculturation within Hispanics (Borrell and Crawford, 2008). In addition, the prevalence of hypertension and other cardiovascular risk factors increases with decreasing socioeconomic status within Hispanic/Latino subgroups. (Borrell and Crawford, 2008). Similarly, in contrast to the more commonly established belief that Latino populations are at lower risk of psychiatric disorders, when Latinos are disaggregated into ethnic subgroups and by nativity, a different picture of Latino mental health emerges, exhibiting a more limited application of the immigrant paradox (Alegria et al., 2008). Research shows that some Latino subgroups suffer from psychiatric disorders at rates comparable to non-Latino White individuals (Alegria et al., 2008).

Although there is a great quantity of detailed data available about the health of minorities, the need for greater specificity of minority subgroups is also evident in studies of African Americans and other Black populations in the United States. While an African American born and raised in the South, a Jamaican, a Haitian, a Kenyan, and an African American born and raised in the Northeast can all "Black", they are likely to differ in

terms of beliefs, behavior, and mental and physical functioning (Williams and Jackson, 2000). Such diversity within the Black population may predict important differences in terms of health status (Williams and Jackson, 2000). There have been few empirical studies of ethnic differences in health within the American Black population. One recent study used data from a national survey of the US Black population to examine the relationships among ethnicity, nativity, depressive symptoms, and physical health in the two largest ethnic groups of American Blacks, African Americans, and Caribbean Blacks (Griffith, Johnson, Zhang, Neighbors, and Jackson, 2011). Results showed that African Americans, US-born Caribbean Blacks, and Caribbean-born Blacks had significantly different self-ratings of their health and self-reports of being diagnosed with a chronic physical health condition: Caribbean-born Blacks had the best health outcomes, and US-born Caribbean Blacks had the worst (Griffith, et al., 2011). This data highlights the importance of considering the ethnic diversity, within the American Black population.

Asian Americans and Pacific Islanders are another major US racial/ethnic group, defined by the US Census Bureau as individuals with origins in the Far East, Southeast Asia, or the Indian subcontinent (see McCracken et al., 2007). Asian groups are not limited to nationalities and include ethnic terms as well, such as the "Hmong". Most incidence data and national level mortality data are available only for all Asian/Pacific Islander groups combined (McCracken et al., 2007). However, these populations are extraordinarily different with respect to country of origin, time since immigration, socioeconomic status, and other characteristics that affect health (McCracken et al., 2007). Chinese Americans are the largest Asian ethnic group in the United States and have one of the oldest immigration histories. The differences in cancer incidence and mortality among the Asian American ethnic subgroups illustrate the heterogeneity of this population (McCracken et al., 2007). In a recent review McCracken and colleagues (2007) showed that subgroups of Asian Americans have different type of cancers according to their immigration histories. For example, subgroups of Asian Americans with more recent immigration histories, such as Vietnamese and Koreans, have a higher burden of cancers not typically observed at high rates in westernized countries, such as stomach and liver cancer. In contrast, other groups with older immigration histories, such as Japanese and Filipinos, have a higher rate of cancers commonly observed in the United States such as colorectal and breast cancers (McCracken et al., 2007).

Cross-References

- ▶ Aggregate Data
- ▶ Cultural and Ethnic Differences
- ▶ Diversity
- ▶ Ethnic Differences
- ▶ Hispanic/Latino Health

References and Readings

Alegria, M., Canino, G., Shrout, P., Woo, M., Duan, N., Vila, D., et al. (2008). Prevalence of mental health illness in immigrant and non-immigrant US Latino groups. *American Journal of Psychiatry, 165*, 359–369.

Amaro, H., & Zambrana, R.E. (2000). Criollo, Mestizo, Mulato, LatiNegro, Indigena, White or Black? The US Hispanic/Latino Population and multiple responses to the 2000 Census. *American Journal of Public Health*, *90*, 1724–1727.

Borrell, L.N., & Crawford, N.D. (2008). Disparities in self-reported hypertension in Hispanic subgroups, non-Hispanic black and non-Hispanic white adults: the national health interview survey. *Annals of Epidemiology*, *18*, 803–812.

Borrell, L. N., & Crawford, N. D. (2009). All-cause mortality among Hispanics in the United States; Exploring heterogeneity by nativity status, country of origin, and race in the National Health Interview Survey-linked mortality file. *Annals of Epidemiology, 19*, 336–343.

Feinleb, M., (1993). Data needed for improving the health of minorities. *Annals of Epidemiology*, *3*, 199–202.

Griffith, D. M., Johnson, J. L., Zhang, R., Neighbors, H. W., & Jackson, J. S. (2011). Ethnicity, nativity, and the health of American blacks. *Journal of Health Care for the Poor and Underserved, 22*, 142–156.

Gee, G. C., Ro, A., Shariff-Marco, S., & Chae, D. (2009). Racial discrimination and health among Asian Americans: Evidence, assessment, and directions for future research. *Epidemiological Reviews, 31*, 130–151.

McCracken, M., Olsen, M., Chen, M. S., Jemal, A., Thun, M., Cokkinides, V., et al. (2007). Cancer incidence, mortality, and associated risk factors among Asian Americans of Chinese, Filipino, Vietnamese, Korean, and Japanese ethnicities. *CA: A Cancer Journal for Clinicians, 57*, 190–205.

Perez, D. J., Fortuna, L., & Alegria, M. (2008). Prevalence and correlates of everyday discrimination among U.S. Latinos. *Journal of Community Psychology, 36*, 421–433.

Srinivasan, S., & Guillermo, T. (2000). Toward improved health: Disaggregating Asian American and native Hawaiian/Pacific islander data. *American Journal of Public Health, 90*, 1731–1734.

Stone, R.T., & MCQuillan, J. (2007). Beyond Hispanic/Latino: The importance of gender/ethnicity-specific earnings analysis. *Social Science research*, *36*, 175-200.

Whitfield, K.E., Weidner, G., Clark, R., & Anderson, N.B. (2002). Sociodemographic diversity in behavioral medicine. *Journal of Consulting and Clinical Psychology*, *70*, 463–481.

Williams, D. R., & Jackson, J. S. (2000). Race/ethnicity and the 2000 census: Recommendations for African American and other black populations in the United States. *American Journal of Public Health, 90*, 1728–1730.

Williams, E.D., Nazroo, J.Y., Kooner, J.S., & Steptoe, A. (2010). Subgroup differences in psychosocial factors relating to coronary heart disease in the UK south Asian population. *Journal of Psychosomatic Research*, *69*, 379–387.

Missing Data

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Missing values

Definition

The term "missing data" refers to data that are not present in a final data set. While the term can apply to one or more subjects' entire set of data that should be present, the more usual situation is where only part of one or more subjects' entire set of data is missing.

Various methodologies have been developed to address this issue. One common and straightforward approach is to delete such records from any analyses that involve variables for which the records have missing values. This is called the complete-subject analysis approach (Greenland & Rothman, 2008). It is a viable option whenever subjects with complete data have effectively been randomly sampled from all the subjects in the study, in which case the missing data are regarded as "missing completely at random."

However, this approach does have drawbacks. It can be very inefficient if many subjects have missing values, and particularly if many subjects have just a few missing data values each, since so many data points are effectively discarded (it discards all the data present in a subject's record, even if only one study variable in the record has a missing value). Therefore, many alternative approaches have been developed (Greenland & Rothman, 2008).

As Donders et al. (2006) observed, "Missing data are a common problem in all types of medical research." Simple methods of handling missing data include complete or available case analysis, the missing-indicator method, and overall mean imputation. Unfortunately, however, these approached lead to inefficient analyses and, more seriously, they often produce severely biased estimates of the association(s) being investigated (Donders et al. 2006). More sophisticated imputation techniques, such as multiple imputation, yield better results.

Missing data are also a problem for researchers employing structural equation modeling (SEM) techniques. Different approaches include full information maximum likelihood (FIML), listwise deletion, pairwise deletion, and similar response pattern imputation (Enders & Bandalos, 2001). These authors performed a Monte Carlo simulation examining the performance of these missing data methods and found that FIML estimation was superior across all conditions of the design.

Cross-References

- ▶ Data
- ▶ Randomization
- ▶ Structural Equation Modeling (SEM)

References and Readings

Donders, A. R. T., van der Heijden, G. J. M. G., Stijnen, T., & Moons, K. G. M. (2006). A gentle introduction to imputation of missing values. *Journal of Clinical Epidemiology*, *59*:1087–1091.

Enders, C. K., & Bandalos, D. L. (2001). The relative performance of full information maximum likelihood estimation for missing data in structural equation models. *Structural Equation Modeling*, *8*(3):430–457.

Greenland, S., & Rothman, K. J. (2008). Fundamentals of epidemiologic data analysis. In K. J. Rothman, S. Greenland, & T. L. Lash (Eds.), *Modern epidemiology* (3rd ed., pp. 213–237). Philadelphia: Wolters Kluwer/ Lippincott Williams & Wilkins.

Missing Values

- ▶ Missing Data

Mistrust

- ▶ Hostility

Mixed-Effects Modeling

- ▶ Multilevel Modeling

Mode

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

The mode is a measure of central tendency. It is the most frequently occurring value in a data set.

Unlike the mean, which is extremely well suited as a measure of central tendency for continuous data, the mode is better suited to numeric data for which there are only a few unique values.

There may be more than one value of the mode for a data set, which happens when two or more values occur equally often. It is also possible for there to be no mode for a data set, which happens when each value is unique. These properties of the mode, i.e., that it may have multiple values in some instances and be undefined in others, are considerable drawbacks to its use.

Cross-References

- ▶ Central Tendency
- ▶ Median

Model

- ▶ Theory

Model of Self-Regulation

- ▶ Self-Regulation Model

Moderate-Vigorous Physical Activity

- ▶ Aerobic Exercise

Moderators/Moderating Factors

Pamela S. King
Pediatric Prevention Research Center, Department of Pediatrics, Wayne State University School of Medicine, Detroit, MI, USA

Definition

A moderator variable is a qualitative (e.g., gender, SES) or quantitative (e.g., amount of social

support) variable that affects the direction and/or strength of the relationship between an independent or predictor variable and a dependent or criterion variable. In research, in order to infer that a variable is a moderating variable, there must be a significant statistical interaction between the predictor and the moderator (i.e. $p < .05$).

Description

Conceptual Meaning

A moderator variable affects the relationship between a predictor variable (X) and an outcome variable (Y). Moderator variables commonly affect the strength of the relationship between X and Y. For example, social support is thought to function as a moderator of the relationship between stress and negative health outcomes: in the presence of low social support, there is a strong relationship between stress and negative health outcomes; in the presence of high social support, however, the association between stress and health is weak to nonexistent. In some cases, a moderator can affect the direction of association between X and Y (e.g., a positive association between X and Y at high levels of the moderator, and a negative association between X and Y at low levels of the moderator).

Statistical Approach

Researchers typically use regression analyses to detect a moderator effect. In this type of analysis, the moderator variable and the predictor variable (X) are both entered as predictors in the regression model (i.e., "main effects"). The interaction between the predictor and moderator (i.e., the product of the moderator and predictor variables) is also entered as a predictor in the regression model. Because of the way the interaction term is computed (moderator × predictor), the main effects for the predictor and moderator will be highly correlated with the interaction term, which could result in problems with the regression model due to multicollinearity. To prevent problems due to multicollinearity, it is recommended that the predictor and moderator variables be "centered" before calculating their interaction. Centering simply means that these variables are transformed by subtracting each variable by its sample mean (e.g., X−mean of X). If a moderator effect is present, the interaction term in the regression model will be significant ($p < .05$). In the presence of a significant interaction, main effects for the predictor and moderator are usually not interpreted.

In order to interpret statistically significant interactions, researchers usually plot regression lines (representing the association between X and Y) for high and low values of the moderator. This allows researchers to see how the association between X and Y is different for different levels of the moderator. After plotting the simple regression lines for high and low values of the moderator, researchers can test the significance of the slopes of the regression lines. In some cases, one slope might be significant, but the other might not, indicating a significant association between X and Y for one level of the moderator, but not for the other level of the moderator.

Distinction Between Moderator and Mediator Variables

It is important to note the distinction between the term moderator and the term mediator, as these two terms are often confused in the literature. Both moderators and mediators are "third variables" discussed in the context of the relationship between a predictor and an outcome. As discussed above, moderator variables are those that can affect the strength and/or direction of the relationship between X and Y (i.e., X × Moderator → Y). A mediator variable, on the other hand, is a mechanism; it explains how a predictor and an outcome are related. The relationship between the predictor, the mediator, and the outcome can be written as X → Mediator → Y. In other words, the predictor causes the mediator, which then causes Y (i.e., the mediator is in the middle of a causal chain linking the predictor to the outcome).

Cross-References

▶ Mediators
▶ Social Support

References and Readings

Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions*. Thousand Oaks, CA: Sage.

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic and statistical considerations. *Journal of Personality and Social Psychology, 51*, 1173–1182.

Bauer, D. J., & Curran, P. J. (2005). Probing interactions in fixed and multilevel regression: Inferential and graphical techniques. *Multivariate Behavioral Research, 40*, 373–400.

Cohen, J., Cohen, P., West, S. G., & Aiken, L. S. (2003). *Applied multiple regression/correlation analysis for the behavioral sciences* (3rd ed.). Hillsdale, NJ: Erlbaum.

Holmbeck, G. N. (1997). Toward terminological, conceptual, and statistical clarity in the study of mediators and moderators: Examples from the child-clinicial and pediatric psychology literatures. *Journal of Consulting and Clinical Psychology, 65*(4), 599–610.

Holmbeck, G. N. (2002). Post hoc probing of significant moderational and mediational effects in studies of pediatric populations. *Journal of Pedaitric Psychology, 27*(1), 87–96.

McClelland, G. H., & Judd, C. M. (1993). Statistical difficulties of detecting interactions and moderator effects. *Psychological Bulletin, 114*(2), 376–390.

Online utilities to probe interaction effects (e.g., testing the significance of simple slopes), *Probing interactions in multiple linear regression, latent curve analysis, and hierarchical linear modeling*. Retrieved from http://www.quantpsy.org/interact/index.html

"Mono" or Mononucleosis

► Epstein-Barr Virus

Monokines

► Cytokines

Monounsaturated Fats

► Fat, Dietary Intake

Monounsaturated Fatty Acids

► Fat: Saturated, Unsaturated

Monozygotic Twins

Jennifer Wessel
Public Health, School of Medicine, Indiana University, Indianapolis, IN, USA

Synonyms

Identical twins

Definition

Monozygotic (MZ) twins are pairs of individuals resulting from the fertilization of one egg (monozygote literally means one egg, or zygote). The zygote goes through several cell divisions, and the cells then separate into two groups, resulting in two separate embryos.

MZ twins have identical copies of all of their genes. Therefore, unlike dizygotic (DZ) twins who can be same-sex or opposite-sex pairs, all MZ twins are same-sex pairs. At young ages their epigenetic material is nearly identical, but as they age the different environmental influences they experience can change their epigenetic material. Phenotypes, e.g., physical and mental traits and disease states, can therefore differ between pairs of twins.

Cross-References

► Dizygotic Twins

References and Readings

Elston, R. C., Olson, J. M., & Palmer, L. (2002). *Biostatistical genetics and genetic epidemiology* (1st ed.). Chichester: Wiley.

Nussbaum, R. L., Mc Innes, R. R., & Willard, H. F. (2001). *Genetics in medicine* (6th ed.). Philadelphia: W.B. Saunders.
Spector, T. D., Snieder, H., & MacGregor, A. J. (2000). *Advances in Twin and Sib-pair analysis* (1st ed.). London: Greenwich Medical Media.

Mood

Maria Kleinstäuber
Department of Clinical Psychology and Psychotherapy, Johannes Gutenberg-University of Mainz, Mainz, Germany

Synonyms

Affective state; Feeling; Feeling state

Definition

In contrast to *emotion* mood is defined as a transient, low-intensity, nonspecific, and subtle affective state that often has no definite cause. Along with all the transient affective states of everyday life, mood also includes low-activation, low-energy states such as fatigue or serenity. Although the terms *mood* and *affect* are often used synonymously, affect is more of an umbrella term that includes both emotions and moods. Short-term and transient feelings are called mood states or affect states, whereas stable, long-term individual differences in the tendency to experience a certain mood state are defined as affective traits. Affective traits can be differentiated from *temperaments* in that they are broader, more general, dispositional constructs, which combine several affective traits and cognitive or behavioral characteristics. Furthermore, the concept of affective traits, in contrast to the concept of temperaments, does not include any implications regarding the origin of the trait.

Description

Affective experiences of everyday life are characterized by mixed mood states, rather than the experience of pure emotion. Mood states inform us continuously about our general state of being and, therefore, play an important role in human self-regulatory processes. Potentially important factors that are involved in intraindividual fluctuations in mood, or differences across individuals can be divided into four broad categories: affective traits and temperaments, exogenous factors (e.g., events, activities, ingestion of substances, physical aspects of environment), endogenous rhythmic patterns and sociocultural rhythms (e.g., circadian rhythms, mood changes across menstrual cycle or the seasons), as well as individual differences in characteristic variability of moods.

Moods and Biobehavioral System

Mood states should not be assumed to be isolated subjective experiences, but instead should be seen as an important component of a complex biobehavioral system that includes affective, cognitive, biological, and behavioral components. Theories focusing on the issue of causal primacy touch on the question of whether cognitions arise first and produce affective states, or whether affective states appear independently and can induce cognitive changes. These theories have so far turned out to be unproductive. Instead it has been proposed that the various components of the biobehavioral system are connected by complex feedback loops and work synchronously. Changes in one component, therefore, produce changes in all of the others. This assumption supports the behavioral medical conceptualization of health and illness, which integrates biological, behavioral as well as psychological issues in an interactive way. It also emphasizes the role of the affective component (including states of mood) on health status.

Measuring Mood

In affective research, mood is mostly assessed using self-ratings. Some rather contradictory assumptions exist about the dimensional structure of self-reported mood. Current evidence strongly supports the existence of two orthogonal dimensions: Positive Affect and Negative Affect.

This implies that knowledge of an individual's current level of negative mood says very little about their level of positive mood. Therefore, in appraising mood it is very important to assess both dimensions separately. Although most research on mood is based on self-ratings, a variety of concerns regarding this method exist, such as biases due to social desirability, expectancy effects, or the idiosyncratic interpretation of anchoring points of rating scales. Behavioral measures of mood are an alternative approach to self-ratings. A typical behavioral technique employs a consensually accepted manipulation of mood (e.g., autosuggestion techniques such as reading positive, negative or neutral self-referent statements) followed by a battery of behavioral measures (e.g., measures of psychomotor speed or activation). This method also has serious limitations, such as biases due to the motivation of participants to comply with the perceived expectations of the experimenter, or the problem of time-limited effects.

Impact of Mood

Moods can have an impact on a variety of cognitive and behavioral processes. Most of the past research has been on associations between mood and memory, judgmental and evaluative processes, and overt behavior. These associations also play an important role in conceptualizations of illness and health in behavioral medicine. Research on associations between mood and memory has identified various phenomena. *Mood-congruent encoding* implies that material is learned more thoroughly if the affective tone of that material is consistent with an individual's mood state. This is considered a robust phenomenon occurring with most mood states. *Mood-congruent retrieval* is defined as an increased recall of material that is of the same affective tone as the mood experienced during retrieval. This effect has been shown less frequently. *Mood-dependent memory* implies that an individual remembers material learned in a particular mood state better on occasions when the individual is in that same mood state. One of the most popular theories attempting to explain these phenomena is the *Associative Network Theory*. It postulates that memory is an associative network of nodes representing units such as concepts, schemata, events, and also affective states. Due to associative links that can form between these units, activation of a memory unit spreads to neighboring associated nodes. The effects mentioned are also relevant to behavioral medical approaches. A typical example is that of mood-congruent effects of depressive mood being associated with poorer memory for positive experiences and/or better memory for negative experiences, which in turn can maintain the vicious cycle of depressive syndrome. An enduring depressive mood can in turn be associated with complications in the healing processes of various diseases.

For explaining the impact of mood on judgments, the *Affect Infusion Model* (AIM; Forgas, 1995) can be applied. *Affect infusion* describes the process whereby affectively loaded information becomes involved in the judgmental process and biases the judgmental outcome. According to the AIM, the occurrence of affect infusion on judgment appears in dependence upon the use of different judgmental strategies, which in turn depend upon features of the target, the judge, and the situation. The model implies that more extensive processing strategies increase the impact of mood on judgment, as such strategies offer a greater scope for the incorporation of affectively loaded information. This association between mood and judgments has extremely important consequences for behavioral medical approaches, for the reason that mood has a major impact on people's perceptions of symptoms of illness, their health efficacy judgments, and their expectations of potential diseases they might have in the future. Individuals in a positive mood seem to judge their health more favorably than individuals in a negative mood. Furthermore, it has been demonstrated that negative mood is associated with negative judgments about symptoms, symptom severity, or health status across

different illnesses (e.g., asthma, Parkinson's disease, rheumatoid arthritis). In addition, negative mood seems to be associated with a lower detection threshold and a higher selective attention for physical symptoms.

Finally, research on the association between mood and behavior has demonstrated relatively consistent results for the mood-congruent effects of positive mood, with the exception that positive mood promotes positive behaviors – only if the behavior is not a threat to continuation of the positive mood. For negative moods the findings are much more contradictory. In behavioral medicine in particular, mood-effects on behavior play an important role concerning disease-preventive as well as disease-management behaviors. Typical examples are that individuals in a negative mood show insufficient asthma control behavior or that negatively tempered individuals with chronic heart failure fail to seek advice for relevant cardiac symptoms.

Mood and Health Status

In general, mood seems to be associated not only with the subjective perception of health status but also with objective aspects of health. For example, individuals with a chronic tendency to experience negative mood report higher levels of health problems, especially more psychophysiological disorders. Similar findings have been demonstrated for highly reactive persons (e.g., persons who quickly change the intensity of their moods). For cardiovascular disease in particular, enduring tendencies toward negative mood seem to be linked to the duration, development, and course of the disease. Enduring negative mood seems to contribute to pathophysiological processes in the complex network of bidirectional signals linking the nervous, endocrine, and immune systems. Thus, negative mood can have an impact on immune function and can indirectly produce a higher risk for infectious diseases. Affective research shows that not only negative mood, but also positive mood has an impact on health status and is associated with favorable health outcomes. It has been demonstrated that positive affect activates the neuroendocrine, autonomic, and immune systems. For example, cortisol level has been shown to be consistently lower in individuals with higher levels of positive mood, and beneficial effects of positive mood on cardiovascular function including blood pressure and heart rate have also been found. The impact of positive mood seems to be independent of the impact of negative mood; therefore, it is speculated that positive mood has its own specific biological correlates.

In conclusion, it is not surprising that mood can impact the healing processes of different diseases. Behavioral interventions focusing on improving mood in patients with chronic diseases, therefore, seem to play an important role in the treatment of medical problems. In patients with different chronic medical conditions (e.g., heart disease, diabetes mellitus, asthma, cancer, arthritis), behavioral therapeutic interventions (e.g., relaxation, biofeedback, graded exercise) have been found to have a positive effect on anxiety and depressive mood.

Cross-References

- ▶ Affect
- ▶ Affect Arousal
- ▶ Coffee Drinking, Effects of Caffeine
- ▶ Emotional Responses
- ▶ Emotions: Positive and Negative

References and Readings

Dalgeish, T., & Power, M. J. (Eds.). (1999). *Handbook of cognition and emotion*. New York: Wiley.

Forgas, J. P. (1995). Mood and judgment: The affect infusion model (AIM). *Psychological Bulletin, 117*(1), 39–66.

Morris, W. N. (1989). *Mood: The frame of mind*. New York: Springer.

Watson, D. (2000). *Mood and temperament*. New York: Guilford.

Mood Variability

► Diurnal Mood Variation

Morbus Crohn (MC)

► Crohn's Disease (CD)

Mortality

G. David Batty
Department of Epidemiology and Public Health, University College London, London, UK

Synonyms

Case fatality; Death; Fatality; Vital status

Definition

Mortality is a measure of human longevity and, by proxy, health that is widely utilized by demographers, epidemiologists, actuaries, and economists. Mortality data typically describe the number of deaths by place and time. Many nations, particularly high-income countries, have a civil registration system of deaths, often accompanied by the underlying cause of death as coded on a death certificate by an attending physician. In accordance with the rules of the International Classification of Diseases (WHO), the underlying cause of death is defined as "the disease or injury which initiated the train of morbid events leading directly to death, or the circumstances of the accident or violence which produced the fatal injury."

Cross-References

► Mortality Rates

References and Readings

World Health Organization. (2011). *International classification of diseases*. Accessed February, 2011, from http://www.who.int/classifications/icd/en/

Mortality Rates

G. David Batty
Department of Epidemiology and Public Health, University College London, London, UK

Synonyms

Death rate

Definition

A mortality rate is an estimate of the proportion of a population group dying during a specific period of time. Mortality rates can be based on how many people die of any cause ("total mortality"), or can be used to describe the death rate of a certain illness or condition, such as dementia or avian influenza.

Mortality rate is calculated as the number of people dying (numerator) derived by the number of people at risk of dying (denominator). The latter estimate is typically based on midyear population data.

In order to produce readily understandable results, the mortality rate – which is typically calculated as a small fraction – is often multiplied up by expressing it as deaths per 100 or 1,000 individuals. For instance, in a town of 10,000 residents, if 10 people die of a heart attack over a given period of time, the mortality rate due to this condition would be said to be 1 in 1,000 persons.

Cross-References

► Mortality

Motivational Interviewing

Demetria Cain
Center for Health Intervention and Prevention, University of Connecticut, Storrs, CT, USA

Definition

Motivational interviewing is a guided, client-centered style of counseling used to help clients explore and resolve ambivalence toward health behavior change. Based on the principles of motivational psychology, it is designed to produce rapid, internally motivated change by mobilizing the client's own change resources. Motivational interviewing is not defined by a technique, but by its spirit as an interpersonal style for facilitating change (Miller & Rollnick, 1991; Rollnick & Miller, 1995).

Description

Motivational interviewing developed from William R. Miller's research on studying behavioral self-control training as a treatment for alcohol addiction. In his early research, Miller noted that a non-confrontational treatment approach lowered drinking levels among alcoholics compared to a therapist outpatient treatment approach (Miller, 1978). Motivational interviewing formed from the notion that counseling can have a huge effect on behavior change when it emphasizes personal responsibility and supports cognitive dissonance in clients who experience ongoing health behavior problems but exhibit awareness for the behavior's negative consequences.

Stemming from these initial concepts, Miller and Rollnick (1991) further elaborated on the fundamental concepts and approaches for this style of counseling in a clinical procedure description. The five basic principles of motivational interviewing are *expressing empathy*, *developing discrepancy*, *avoiding argumentation*, *rolling with resistance*, and *supporting self-efficacy*. In expressing empathy, the therapist seeks to communicate great respect for the client. The therapist's role is a blend of supportive confidant and knowledgeable consultant. The client's freedom of choice and self-direction is supported and respected. Persuasion is given in gentle, subtle ways with the assumption that behavior change is up to the client. In developing discrepancies, motivational interviewing seeks to focus the client's awareness toward discrepancies in their behavior. When people perceive a discrepancy between where they are and where they want to be, they become motivated to change. When the therapist avoids argumentation, they employ strategies to assist the client in accurately seeing the consequences of their behavior. Direct confrontation of behavior can produce resistance. Instead, the therapist passively guides the client to voice their own arguments for change. In rolling with resistance, the therapist views ambivalence toward change as normal, and resistance is explored openly. Instead of challenging a client's resistance, the therapist moves with it, but with the goal of shifting the client's perceptions in the process. Self-efficacy is the belief that one can perform a particular behavior or accomplish a particular task and is a critical determinant of behavior change (Bandura, 1982). Through the therapist's support, the client must be confident that behavior change is possible.

Motivation interviewing was used as the style of counseling in Motivational Enhancement Therapy and tested in the National Institute on Alcohol Abuse and Alcoholism's (NIAAA) multisite cooperative agreement called Project Match. In this clinical trial, patients with alcohol abuse and dependence were matched to different treatment options based on their personal characteristics. Motivational Enhancement Therapy was shown as effective as a 12 Step Facilitation therapy and a Cognitive Behavioral Coping Skills Therapy for alcohol dependency treatment (Miller, Zweben, DiClemente, & Rychtarik, 1992).

Motivational interviewing's efficacy comes from its ability to help guide clients through a natural process of recovery. The Transtheoretical Model of Behavior Change (Prochaska & DiClemente, 1984) describes the stages of change clients move through as they progress in

modifying problem behaviors. The stages of change are precontemplation, contemplation, determination, action, maintenance, and relapse. In motivational interviewing, the therapist assists the client in moving from one stage to the next by addressing where they are and where they want to be.

Motivational interviewing has been synonymous with brief interventions because of terms like "brief motivational counseling," but they are distinctly different concepts (Holder, Longabaugh, Miller, & Rubonis, 1991). The six elements believed to be active ingredients to induce change in brief interventions are summarized in the acronym FRAMES and consist of giving FEEDBACK of personal risk or impairment, placing emphasis on personal RESPONSIBILITY for change, giving clear ADVICE to change, providing a MENU of alternative change options, expression of therapist EMPATHY, and facilitating client SELF-EFFICACY(Miller & Sanchez, 1994). Although the elements of FRAMES are analogous with motivational interviewing, it is important to distinguish the differences between the mechanisms that make brief interventions effective and the methods designed to encourage behavior change in motivational interviewing (Rollnick & Miller, 1995).

Techniques used by therapist in motivational interviewing are structured into the two phases: building motivation for change and strengthening commitment to change (Miller et al., 1993). While building motivation for change, the therapist helps the client make the decision to change by using open-ended questions to elicit self-motivational statements of problem perception, using reflective listening to demonstrate empathy, summarizing themes that need further emphasis, and affirming the client's challenges. While strengthening commitment to change, the therapist helps the client move from contemplation to action by recognizing the client's readiness for change, discussing a potential plan of action, communicating free choice, and discussing consequences of action or inaction.

Although motivational interviewing was formed out of alcohol treatment research, it has moved into other realms of health behavior change research. Motivational interviewing has shown promise in behavior change related to HIV risk behavior, substance abuse, smoking cessation, physical activity, diabetes self-care, and many others.

Those interested in learning more about motivational interviewing can seek information on this style of counseling from resources developed for clinicians, researchers, and trainers. Through the Motivational Interviewing Network of Trainers (MINT), there are opportunities to learn motivational interviewing through one of many training courses.

Cross-References

- ▶ Health Behavior Change
- ▶ Self-efficacy
- ▶ Stages-of-Change Model

References and Readings

Bandura, A. (1982). Self-efficacy mechanism in human agency. *American Psychologist, 37*, 122–147.

Holder, H., Longabaugh, R., Miller, W. R., & Rubonis, A. V. (1991). The cost of effectiveness for treatment for alcoholism: A first approximation. *Journal of Studies on Alcohol, 52*, 517–540.

Miller, W. R. (1978). Behavioral treatment of problem drinkers: A comparative outcome study of three controlled drinking therapies. *Journal of Consulting and Clinical Psychology, 46*(1), 74–86.

Miller, W. R., & Rollnick, S. (1991). *Motivational interviewing: Preparing people for change*. New York: Guilford Press.

Miller, W. R., & Sanchez, V. C. (1994). Motivating young adults for treatment and lifestyle change. In G. Howard (Ed.), *Alcohol use and misuses by young adults* (pp. 55–82). Notre Dame, IN: University of Notre Dame Press.

Miller, W. R., Zweben, A., DiClemente, C. C., & Rychtarik, R. G. (1992). *Motivational enhancement therapy manual: A clinical research guide for therapists treating individuals with alcohol abuse and dependence*. Rockville, MD: National Institute on Alcohol Abuse and Alcoholism.

Prochaska, J. O., & DiClemente, C. C. (1984). *The transtheoretical approach: Crossing traditional boundaries of therapy*. Homewood, IL: Dow Jones/Irwin.

Rollnick, S., & Miller, W. R. (1995). What is motivational interviewing? *Behavioural and Cognitive Psychotherapy, 23*, 325–334. http://motivationalinterview.org/.

Motor Behavior

▶ Benefits of Exercise

Mourning

▶ Bereavement

MPH

▶ MPH (Masters of Public Health)

MPH (Masters of Public Health)

Pedro C. Castellon
Epidemiology and Public Health, Miller School of Medicine, University of Miami, Miami, FL, USA

Synonyms

Masters of public health; MPH; Public health education

Definition

The need for dedicated health professionals to focus on the health of the population led to the creation of a new career and formal training and education in public health. By the end of the nineteenth century, there were several schools of medicine and nursing to train future medical professionals, but there were no formal training programs for the field of public health. In an effort to establish education for a separate public health career, the Rockefeller Foundation provided funding to create the first endowed school of public health at the Johns Hopkins University in 1918. At that time the career and formal education for public health was created for individuals to dedicate their career to controlling infectious diseases, improving sanitation, assuring the safety of food and water, and providing immunizations (Gebbie, Rosenstock, & Hernandez, 2003).

Since the creation of formal education for public health, both schools and programs of public health have awarded the Masters of Public Health (M.P.H.) degree and other public health-related degrees and certificates (Gebbie et al., 2003). Presently (4/21/2011) there are 44 accredited schools and 79 accredited programs of public health in the United States. There are also two accredited schools and four accredited programs of public health outside the United States (Council on Education for Public Health, 2011). In general, the curriculum for the M.P.H. degree and other similar degrees consists of courses in epidemiology, environmental health, health services and administration, biostatistics, and social and behavioral sciences.

This coursework provides the appropriate training and education for public health professionals to establish careers in both the private and public sectors. Those working in private sectors typically work as researchers within an academic institution or a pharmaceutical company, while those working in the public sector work as nutritionists, epidemiologists, food safety inspectors, health educators, and policy analysts in local, state, or federal health departments. Public health professionals continue to control infectious diseases, provide immunizations, and assure the safety of food and water, but today they also confrontcomplex health issues such as obesity and cancer, improving access to health care, and reducing environmental hazards (WhatisPublicHealth.?, n.d.).

Cross-References

▶ Epidemiology
▶ Nutrition
▶ Obesity
▶ Public Health

References and Readings

Council on Education for Public Health. (2011, April 21). Schools of Public Health and Public Health Programs accredited by the Council on Education for Public Health, List by Accreditation Category. Retrieved from http://www.ceph.org

Gebbie, K., Rosenstock, L., & Hernandez, L. M. (Eds.). (2003). *Who will keep the public healthy? Educating public health professionals for the 21st century*. Washington, DC: The National Academies Press.

WhatIsPublicHealth.org (n.d.). Retrieved May 30, 2011, from http://www.whatispublichealth.org

MRI

▶ Magnetic Resonance Imaging (MRI)

mRNA

▶ RNA

Multicultural Health

▶ Minority Health

Multiculturalism

▶ Diversity

Multidimensional Health Locus of Control Scales

Kenneth Wallston
School of Nursing, Vanderbilt University, Nashville, TN, USA

Synonyms

Measures of perceived control of health

Definition

Health locus of control beliefs indicates whether individuals believe their health status is under their own control (termed an internal health locus of control orientation) or is under the control of forces external to themselves, such as other people, fate, luck, chance, or "a higher power."

Description

Multidimensional Health Locus of Control Scales

Internal-external locus of control (LOC) of reinforcement is a generalized expectancy construct consistent with Julian Rotter's social learning theory (Rotter, 1954). Rotter (1966) developed the I-E Scale as a means of assessing individuals' dispositional beliefs as to whether their reinforcements (or valued outcomes) were due to their own behavior or enduring characteristics (an internal LOC orientation) or to external factors such as other people, fate, luck, or chance. Ten years later, Barbara Strudler Wallston, Ken Wallston, and two of their students published a health-related version of the I-E Scale termed the Health Locus of Control (HLC) scale (Wallston, Wallston, Kaplan, & Maides, 1976). Like the I-E Scale, the HLC scale was unidimensional; high scores were termed "health externals," and low scorers were designated "health internals." The HLC scale proved to be very popular; even before publication, health researchers started using it in hopes that HLC scores would be related to measures of health behavior and health status. Soon thereafter, however, the Wallstons recognized that the HLC scale was not unidimensional, with internality on one end of the dimension and externality on the other end, but consisted of two orthogonal dimensions; some people could (and did) simultaneously endorse internal *and* external causes of their health status, while others disagreed with either type of HLC orientation.

With the help of a grant from the National Center for Health Services Research, they set

about developing the Multidimensional Health Locus of Control (MHLC) Scales modeled after Levenson's (1973) I, P, and C scales, a three-dimensional alternative to the unidimensional I-E Scale. They developed two equivalent 18-item forms (A and B) of the MHLC scale (Wallston, Wallston, & DeVellis, 1978). Each form (A or B) contains three, more-or-less orthogonal, 6-item subscales. The IHLC subscale assesses the extent to which people believe their health status is due to their own actions. The PHLC subscale assesses the belief that one's health status is due to the actions of "powerful other" individuals, e.g., health-care professionals, family members, or friends. The belief that nobody or nothing controls a person's health status, and that it is all a matter of fate, luck or chance, is assessed by the CHLC subscale. The early research with Forms A and B was reviewed by B. S. Wallston and K. A. Wallston (1981), and recognizing that designating people as internal or external was too simplistic, an 8-cell MHLC typology was proposed by K. A. Wallston and B. S. Wallston (1982). Although some research has been done using the multidimensional, 8-cell typology, it is much more typical to treat the MHLC scores as continuous rather than categorical.

Forms A and B of the MHLC scales assess LOC orientation toward one's health status in general. Many investigators, however, administered those initial forms of the MHLC to patients with specific medical diagnoses. In those instances, when trying to interpret responses to the MHLC items, investigators had no way of knowing whether the patients were responding with their beliefs about their health in general or their beliefs regarding control of one or more of their medical conditions. This ambiguity led many researchers to modify the MHLC scales in an attempt to make the instrument fit their own purposes. This was all well and good, but each of these modifications used different item stems, and there was little comparability among these modified instruments. K. Wallston, therefore, developed and published Form C of the MHLC scales as a generic instrument that could be easily modified into a condition-specific measure of locus of control beliefs by substituting for the word "condition" in each of the item stems (Wallston, Stein, & Smith, 1994). For example, if studying patients with diabetes, Form C can be used as a multidimensional diabetes locus of control scale. Form C has 6-item subscales for the Internal and Chance dimensions, but the Powerful Other dimension is split into two, 3-item subscales: Doctors and Other People. In the ensuing years, Form C is the instrument of choice for researchers wishing to ascertain control beliefs of patient samples. Those who wish to study healthy samples continue to administer either Form A or B. Also, it is not necessary to administer all of the subscales of the MHLC. If an investigator is only interested in knowing whether a group of respondents believe their health (or health condition) is determined by their own actions, it is perfectly OK just to administer the IHLC subscale.

When the Wallstons developed the initial MHLC scales, they attempted to make the two external dimensions, PHLC and CHLC, as uncorrelated as possible. While Levenson's P and C scales correlated >0.6, PHLC and CHLC usually correlated ≤ 0.3. In accomplishing this, all of the items in the original item pool that mentioned God (or a higher power) as the cause of a person's health status were eliminated, because those items correlated too highly with the other external dimensions. Many years later, K. Wallston received feedback from researchers in "bible belt" states that the MHLC items did not "speak to them," because, in their opinion, God was the entity most responsible for changes in their own health or medical condition. Responding to that feedback, K. Wallston and colleagues developed and published the 6-item God Locus of Health Control (GLHC) scale that could be used as a stand-alone instrument or as another external subscale of the MHLC (Wallston et al., 1999). The GLHC can be administered in either a general health version (similar to Forms A and B) or a condition-specific version (like Form C). As with the other external subscales, the GLHC is uncorrelated with internal HLC, but, depending on the respondents' view of God, GLHC scores correlate positively with one or another of the "traditional" external subscales.

All of the MHLC scales are "in the public domain," which means that they can be used freely by health researchers as long as there is no charge for their administration. They can be downloaded from the MHLC website http://www.vanderbilt.edu/nursing/kwallston/mhlcscales.htm where there are scoring instructions and answers to frequently asked questions (FAQs). The MHLC scales were developed to be used in research, but that has not stopped clinicians from administering the MHLC to individual patients in an attempt to gain insight into patients' health beliefs and to use that information to individualize patient care. The Veterans Administration administers both Form A and Form B as part of a battery of tests to prospective candidates for bariatric surgery. To date, there have been no published reports evaluating the use of the MHLC scales to identify good surgical candidates or their use in routine clinical practice, but there are hundreds of publications in refereed journals that report on the use of the measures, and they have been used in innumerable theses and dissertations. The MHLC scales have also been translated into a number of foreign languages, as well as American Sign Language (Athale et al., 2010).

About 7 years ago, K. Wallston guest edited a special issue of the *Journal of Health Psychology* devoted to research with the MHLC scales. This issue included a critical look at the construct of health locus of control (Luszczynska & Schwarzer, 2005), an overview of the validity of the various MHLC scales (Wallston, 2005), and numerous empirically based articles demonstrating the breadth and creativity of the research that has been done with these measures. Many of the articles in the special issue examined interactions among MHLC subscale scores or interactions between MHLC subscale scores and other constructs as predictors of health behavior or health outcomes. Such analyses were congruent with modified social learning theory (Wallston, 1992), although none directly tested the three-way interaction between internal health locus of control, health value, and self-efficacy beliefs.

High scores on the IHLC, PHLC, and GLHC subscales are all indicative of a belief that health (or health conditions) can be controlled, regardless of whether that control is exercised by one's self (IHLC); powerful other people, such as one's doctors (PHLC); or by God (GLHC). High scores on the chance health locus of control subscale (CHLC) indicate an expectancy that only random occurrences, such as fate luck or chance, control health outcomes. Most research has shown that, in the absence of high scores on the IHLC subscale, scoring high on CHLC is a moderately strong predictor of psychological distress and depressive affect. Recently, K. Wallston reported on a set of analyses from a 10-year longitudinal study of patients with rheumatoid arthritis where it turned out that, controlling for baseline IHLC and PHLC beliefs, higher baseline scores on CHLC not only predicted depressive symptoms at T1 but also predicted an increase in depressive symptoms over the subsequent 10-year period (Wallston, 2011). Baseline scores on neither IHLC nor PHLC predicted change in depression or any other variable in the dataset over that relatively long time.

The MHLC scales have been used by health researchers to assess control beliefs related to health for over 30 years. While research with the MHLC scales has slowed down somewhat in the twenty-first century, interest in the construct and its measurement remains high.

References and Readings

Athale, N., Aldridge, A., Malcarne, V. L., Nakaji, M., Samady, W., & Robins, S. G. (2010). Validity of the multidimensional health locus of control scales in American language. *Journal of Health Psychology, 15*, 1064–1074.

Levenson, H. (1973). Multidimensional locus of control in psychiatric patients. *Journal of Consulting and Clinical Psychology, 41*, 397–404.

Luszczynska, A., & Schwarzer, R. (2005). Multidimensional health locus of control: comments on the construct and its measurement. *Journal of Health Psychology, 10*, 633–642.

Rotter, J. B. (1954). *Social learning and clinical psychology*. Englewood Cliffs, NJ: Prentice-Hall.

Rotter, J. B. (1966). Generalized expectancies for internal versus external control of reinforcement. *Psychological Monographs, 80*(1 whole no. 609), 1–28.

Wallston, K. A. (1992). Hocus-pocus, the focus isn't strictly on locus: Rotter's social learning theory modified for health. *Cognitive Therapy and Research, 16*, 183–199.

Wallston, K. A. (2005). The validity of the multidimensional health locus of control scales. *Journal of Health Psychology, 10*, 623–631.
Wallston, K. A. (April, 2011). *Health locus of control predicts change in depressive symptoms over a 10-year period in patients with rheumatoid arthritis*. Poster presented at the annual meeting of the Society of Behavioral Medicine, Washington, DC.
Wallston, K. A., Malcarne, V. L., Flores, L., Hansdottir, I., Smith, C. A., Stein, M. J., et al. (1999). Does god determine your health? The god locus of health control scale. *Cognitive Therapy and Research, 23*, 131–142.
Wallston, K. A., Stein, M. J., & Smith, C. A. (1994). Form C of the MHLC scales: A condition-specific measure of locus of control. *Journal of Personality Assessment, 63*, 534–553.
Wallston, K. A., & Wallston, B. S. (1981). Health locus of control scales. In H. Lefcourt (Ed.), *Research with the locus of control construct* (Vol. 1, pp. 189–243). New York: Academic Press.
Wallston, K. A., & Wallston, B. S. (1982). Who is responsible for your health: The construct of health locus of control. In G. Sanders & J. Suls (Eds.), *Social psychology of health and illness* (pp. 65–95). Hillsdale, NJ: Erlbaum.
Wallston, K. A., Wallston, B. S., & DeVellis, R. (1978). Development of the multidimensional health locus of control (MHLC) scales. *Health Education Monographs, 6*, 160–170.
Wallston, B. S., Wallston, K. A., Kaplan, G. D., & Maides, S. A. (1976). The development and validation of the health related locus of control (HLC) scale. *Journal of Consulting and Clinical Psychology, 44*, 580–585.

Multidimensional Measure of Religiousness/Spirituality

► Brief Multidimensional Measure of Religiousness/Spirituality (BMMRS)

Multiethnic Cohort Study

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

The Multiethnic Cohort (MEC) Study was a prospective study which included people from Hawaii and California. It recruited people from the main cultural groups residing there – Native Hawaiians, African Americans, Latinos, Japanese Americans, and whites. In such MEC studies, researchers sample subgroups of people belonging to one of several preselected cultural groups. These groups were expected to have a different prevalence of a given disease, and the study aimed to investigate which risk factors accounted for such differences. The MEC study included over 215,000 men and women. Generally speaking, by running the study prospectively and simultaneously for all ethnic subgroups, effects of "historical events" (e.g., development of a new treatment for a disease, a known public figure develops an illness) occur for all participants at the same time. Furthermore, by conducting the study prospectively, researchers used similar assessment tools and controlled for the identical confounders, across ethnic groups. This resulted with more methodological rigor and thus greater inferential validity in the results.

Numerous studies emerged from the MEC study, in relation to several health outcomes. Initially planned to examine ethnic differences in cancer, this research found that Native Hawaiians have 65% higher risk of breast cancer than white Americans. One explanation for this excess, which was partly confirmed, was that Native Hawaiians have more frequent polymorphisms in sex steroid pathways (Pike et al., 2002). Another study compared the rates of mortality from myocardial infarction and other cardiac reasons, between those ethnic groups. Native Hawaiians and African Americans had highest death rates, which were mostly explained by their higher prevalence of diabetes and hypertension (Henderson et al., 2007). Such studies enable health policymakers and public health professionals to target specific risk factors in specific ethnic groups, to prevent such outcomes. Furthermore, MEC studies can shed light on "nature-nurture" interactions, where environmental factors (e.g., smoking, diet, psychosocial stressors) and ethnicity (proxy measure of genetic risk) or actual genetic polymorphism may interact in relation to onset of diseases.

Cross-References

- ▶ Cultural and Ethnic Differences
- ▶ Epidemiology

References and Readings

Henderson, S. O., Haiman, C. A., Wilkens, L. R., Kolonel, L. N., Wan, P., & Pike, M. C. (2007). Established risk factors account for most of the racial differences in cardiovascular disease mortality. *PLoS One, 2*, e377.

Pike, M. C., Kolonel, L. N., Henderson, B. E., Wilkens, L. R., Hankin, J. H., Feigelson, H. S., et al. (2002). Breast cancer in a multiethnic cohort in Hawaii and Los Angeles: risk factor-adjusted incidence in Japanese equals and in Hawaiians exceeds that in whites. *Cancer Epidemiology, Biomarkers & Prevention, 11*, 795–800.

Multi-level Analysis

- ▶ Hierarchical Linear Modeling (HLM)

Multilevel Intervention

Yori Gidron
Faculty of Medicine and Pharmacy,
Free University of Brussels (VUB), Jette,
Belgium

Definition

This term can refer to two concepts. First, a multilevel intervention (MLI) refers to addressing various risk factors in one intervention, either at the prevention stage or at the treatment stage of an illness. Additionally, an MLI refers to an intervention which involves several levels of people or entities such as individual patients, groups, and communities. Both definitions reflect that illnesses and health are a multifactorial problem, requiring either to modify several risk factors or to involve several levels in one's social and health context. In coronary heart disease (CHD) prevention, the Minnesota Heart Health Project (MHHP) combined both meanings of the MLI concept. First, they aimed to conduct a lifestyle behavior change program, targeting diet, physical activity, smoking, and alcohol. Second, this was achieved by involving individuals, community leaders, teachers, physicians, the media, classes, and schools. Thus, they addressed the multiple risk factor aspect of MLI as well as various levels of intervention units, the social-organizational aspect the MLI. Mittelmark et al. (1986) revealed the implementation of the MLI, in which they conducted the MLI on three towns/cities, and compared that to matched towns/cities, over time. However, follow-up studies found modest effects of the MHHP, mainly due to control communities possibly also receiving health education and due to overall increases in certain risk factors such as obesity overtime. Another MLI is the Ornish lifestyle change program, where CHD patients learned to alter their diet, physical activity, smoking, and stress responses. Compared to controls, the MLI-treated group showed regression in atherosclerosis (Ornish et al., 1990). Other types of public health hazards such as traffic accidents could indeed benefit from an MLI approach, where students, teachers, universities, community leaders, safety authorities, schools, and communities can be involved in risk-factor modification. Thus, an MLI approach is suitable for problems involving multiple risk factors or multiple social contexts.

Cross-References

- ▶ Prevention: Primary, Secondary, Tertiary

References and Readings

Mittelmark, M. B., Luepker, R. V., Jacobs, D. R., Bracht, N. F., Carlaw, R. W., Crow, R. S., et al. (1986). Community-wide prevention of cardiovascular disease: Education strategies of the Minnesota Heart Health Program. *Preventive Medicine, 15*, 1–17.

Ornish, D., Brown, S. E., Scherwitz, L. W., Billings, J. H., Armstrong, W. T., Ports, T. A., et al. (1990). Can lifestyle changes reverse coronary heart disease? The lifestyle heart trial. *Lancet, 336*, 129–133.

Multilevel Modeling

Simon Sherry[1] and Anna MacKinnon[2]
[1]Department of Psychology, Dalhousie University, Halifax, NS, Canada
[2]Department of Psychology, McGill University, Montreal, QC, Canada

Synonyms

Hierarchical linear modeling (HLM); Mixed-effects modeling; Random-coefficient regression modeling; Random-effects modeling

Definition

Multilevel modeling is a data analysis technique used to analyze nested data. Nested data refers to data wherein units of analysis at one level are nested within units of analysis at higher levels. Multilevel data are observed in cross-sectional designs which sample individuals nested within groups. An example of this type of multilevel data is patients (level 1) nested within hospitals (level 2). Multilevel data are also found in repeated measures designs (e.g., multiwave longitudinal or experience sampling designs) which sample repeated reports nested within individuals. An example of this type of multilevel data is an experience sampling study where repeated reports of pain (level 1) are nested within individuals (level 2). In multilevel modeling, *level* thus refers to the structure of the data. The lower level (level 1) represents the most detailed unit of analysis and has the greatest number of data points. Level 2 represents the higher level within which level 1 observations are nested.

Description

Why Is Multilevel Modeling Necessary?

Multilevel modeling is necessary because nested data structures violate the assumption of independence required by traditional, single-level data analysis techniques such as analysis of variance and ordinary least squares multiple regression. That is, in nested data, observations at the lower level (level 1) are not independent. For example, individuals (level 1) sampled from the same neighborhood (level 2) may be more similar than individuals sampled from a different neighborhood. Single-level data analysis techniques often fail to take into account such a nested data structure and either ignore the nested data structure (violating the assumption of independence) or collapse across the levels of the nested data structure (ignoring potentially meaningful variability in the data). Violating the assumption of independence may result in underestimation of standard errors and inflation of type I error rates.

In contrast, multilevel modeling allows for data to be analyzed at one level while accounting for variance at other levels. Maximum likelihood algorithms are typically used in multilevel analyses, which allow for simultaneous estimation of multiple error terms. As a result, standard errors are more accurate, and type I error rates are not inflated. In addition, multilevel modeling enables unique types of analyses. Multilevel analyses are similar to single-level regression analyses, where intercepts and slopes are calculated. However, unlike single-level regression analyses, multilevel modeling permits cross-level analyses, wherein a level 2 predictor is used to predict a level 1 outcome. For example, an investigator may test if the neighborhood people live in (level 2) predicts their obesity (level 1). Although such computations are complex, there are many software programs which perform multilevel modeling, including HLM, LISREL, MLwiN, MPlus, R, SAS, SPSS, and Stata.

Considerations When Using Multilevel Modeling

Power. Although sample sizes at both levels warrant consideration, in general, sample size at the higher level has a greater influence on power than sample size at the lower level. For example, in experience sampling studies, the number of participants (level 2) has a greater influence on power than the number of reports per participant (level 1).

Intraclass correlation. It is also necessary to test if multilevel modeling is even necessary. Multilevel modeling is not necessary if there is no variation at higher levels. Variation at higher levels may be computed using the intraclass correlation coefficient. The intraclass correlation coefficient measures the degree to which the lower level units (level 1) belonging to the same higher level unit (level 2) are dependent or clustered. Larger intraclass correlation coefficients indicate more dependence or clustering at higher levels. The occurrence of dependence or clustering at higher levels indicates it is important to use multilevel modeling to protect against inflation of type I error rates and to capture variability at higher levels of the nested data structure.

Missing data. Multilevel modeling is often used to analyze repeated measures data, where missing data are common. Because multilevel analyses typically use maximum likelihood algorithms, participants with missing data may be included in analyses. In multilevel modeling, results are weighted by the amount of data contributed by each participant. That is, participants who provide more data contribute more to the results than participants who provide less data.

Fixed and random effects. Another consideration in multilevel modeling is whether effects are fixed or random. With random effects, the outcome-predictor relationship varies across level 2 units. That is, the slope and the intercept of the regression line are assumed to vary across level 2 units. With fixed effects, the variables of interest do not vary across level 2 units; the slope and the intercept are the same for all level 2 units. If random effects are modeled, the results are assumed to generalize to the population from which cases were sampled, whereas, if fixed effects are modeled, the results are confined to the cases studied. However, random effects models typically require greater sample sizes and may be more complicated to interpret.

Centering. Variables in multilevel modeling may be centered around the group mean (i.e., the mean of each level 1 unit) or centered around the grand mean (i.e., the mean of all the level 2 units). For example, in an experience sampling study involving repeated reports (level 1) nested within individuals (level 2), group mean centering is conceptually equivalent to creating variables that are relative to the individual's own mean based on his/her repeated reports, whereas grand mean centering is conceptually equivalent to creating variables that are relative to the overall mean of all reports provided by all the individuals in the study. Centering aids in interpretation of the results and the choice of centering affects the estimates computed. Decisions regarding centering should be made on a theoretical basis.

Autocorrelation. In repeated measures designs, autocorrelation is an issue. Because of the repeated nature of the data, residual errors in repeated measures data may be correlated (i.e., autocorrelation). The simplest and the most common autocorrelation structure is the first-order autoregressive error structure in which reports closer together in time are more strongly correlated than reports further apart in time. Some software programs (e.g., MPlus or SAS) also model more complex error structures to better account for autocorrelation.

Advantages of Multilevel Modeling

Nested data commonly arises in the field of behavioral medicine. By taking into account the nested structure of the data, multilevel modeling provides more contextualized analyses. For example, multilevel modeling may be used to test neighborhood effects on individuals' obesity, partner effects on patients with cardiovascular disease, and peer influences on adolescents' risky health behaviors. Multilevel modeling may also be used in repeated measures designs, including multiwave longitudinal and experience sampling studies of health behaviors (e.g., smoking, diet, exercise, and medication adherence), chronic illnesses (e.g., pain, diabetes, and HIV), and physiological processes (e.g., cardiovascular reactivity and neuroendocrine levels).

There are several advantages to using multilevel modeling to analyze repeated measures data. Multilevel modeling is able to analyze unbalanced designs, including unequally spaced data and missing data. Using multilevel modeling, it is possible to simultaneously estimate within person and between persons effects. For

example, a researcher could study if on days when a participant experiences more stress, he or she smokes more compared to days when he or she experiences less stress (a within person effect). This effect may then be tested to see if it generalizes across all participants in the study (a between persons effect) or to see whether between person differences, such as personality traits, moderate the relationship between stress and smoking (a cross level interaction).

Limitations of Multilevel Modeling

Multilevel modeling is an advanced statistical technique, which requires a solid grounding in statistics. Increasingly, resources are available to support researchers using multilevel modeling (e.g., Bickel, 2007; Field, 2009; Hox, 2010; Raudenbush & Bryk, 2002). Specialized software is also usually needed to conduct multilevel modeling. However, increasingly, mainstream software also performs multilevel analyses.

New Applications and Developments

Multilevel modeling is not limited to regression analyses. In recent years, researchers are combining multilevel modeling with other data analysis techniques. For example, multilevel modeling may be used in testing moderation, mediation, path models, structural models, growth curves, and meta-analyses. By integrating these data analysis techniques, multilevel modeling is able to analyze a wider variety of research questions.

Conclusion

Multilevel modeling is needed to appropriately analyze the nested data structures that often occur in research on behavioral medicine. Careful consideration of the above issues is critical to appropriately using this data analysis technique.

Cross-References

- ▶ Missing Data
- ▶ Multivariate Analysis
- ▶ Repeated Measures Design

References and Readings

Bickel, R. (2007). *Multilevel analysis for applied research: It's just regression!* New York: Guilford.
Field, A. (2009). *Discovering statistics using SPSS*. Thousand Oaks, CA: Sage.
Hox, J. (2010). *Multilevel analysis techniques and applications*. Mahwah, NJ: Lawrence Erlbaum.
Raudenbush, S., & Bryk, A. (2002). *Hierarchical linear models*. London: Sage.

Multiple Regression

- ▶ Regression Analysis

Multiple Risk Factor Intervention Trial (MRFIT)

Jonathan Newman
Columbia University, New York, NY, USA

Definition

The Multiple Risk Factor Interventional Trial (MRFIT) was a large, randomized primary prevention trial to test the effect of multiple interventions to reduce the risk of premature coronary heart disease (CHD) in 12,866 men, age 35–57, with one or more of three risk factors (hypertension, hyperlipidemia, or cigarette smoking) without a prior history of CHD. The trial was conducted in 22 clinical centers in the United States. MRFIT was conducted by the National Institutes of Health (NIH) and National Heart, Lung, and Blood Institute and was massive in scope, screening 356,222 men for the desired study population. These risk factors were chosen because they are modifiable, and there was an expectation (largely unproven at the time) that reduction of these factors should have beneficial results on the development of premature CHD. A subsample of 3,110 men was recruited to participate in the Behavior Pattern Study, which

investigated the relationship between certain behavior patterns and CHD risk.

Six thousand four hundred and twenty eight participants were assigned to the intervention group, and 6,438 were assigned to the usual care group. Active follow-up was obtained for 6 years, and more than 25 years of surveillance has occurred. The intervention group was instructed to follow a diet designed to result in a nutrient intake of 30–35% of calories from fat, with prespecified intakes of cholesterol and un/saturated fats. This group also received counseling and audiovisual aids to encourage smoking cessation. High blood pressure (BP) was treated with a stepped-care approach using weight loss and medications as validated in prior studies. The usual care group was referred to their individual physician or care provider and received risk-factor management as considered appropriate by these providers. The primary endpoint was CHD death. Participants in both groups had annual assessments of changes in their risk factor levels and were monitored annually for CHD morbidity and mortality.

One of the main findings of the MRFIT study – in both the active follow-up and the 25+ year surveillance – is that the relationship between cholesterol and CHD mortality is continuous, graded, and strong, i.e., that CHD risk is progressively higher at every cholesterol level from normal to higher levels, without a threshold effect. The MRFIT study also clearly demonstrated the importance of dietary cholesterol in CHD risk. This result remained robust after controlling for study factors such as age, systolic BP, diabetes, smoking status, race, and socioeconomic status. MRFIT also demonstrated the near linear relationship between systolic BP and CHD death and the relationship between both systolic and diastolic BP on risk of kidney failure. Importantly, MRFIT participants who stopped smoking had reductions in CHD morbidity and mortality when smoking cessation was implemented along with other risk modification strategies. While failing to demonstrate clear increases in CHD risk associated with type A behavior patterns, the Behavior Pattern Substudy of MRFIT was an important contribution to the field of behavioral cardiology. Lastly, the original 1986 MRFIT paper also introduced the concept of low or optimal CHD risk, proposing a first set of criteria, and emphasized both the rarity and benefit of this phenotype.

References and Readings

Neaton, J. D., Wentworth, D., & The Multiple Risk Factor Intervention Trial Research Group. (1992). Serum cholesterol, blood pressure, cigarette smoking, and death from coronary heart disease: Overall findings and differences by age for 316,099 white men. *Archives of Internal Medicine, 152*, 56–64.

Stamler, J., Wentworth, D., & Neaton, J. D. (1986). Is relationship between serum cholesterol and risk of premature death from coronary heart disease continuous and graded? *Journal of the American Medical Association, 256*(20), 2823–2828.

The Multiple Risk Factor Intervention Trial Group. (1979). The MRFIT behavior pattern study. I. Study design, procedures, and reproducibility of behavior pattern judgments. *Journal of Chronic Disorder, 32*, 293–305.

U.S. Department of Health and Human Services. (1990). Smoking cessation and cardiovascular disease. In: *The health benefits of smoking cessation*. Washington, DC: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 200-5. DHHS publication no. [CDC] 90-8416.

Multiple Risk Factors

Yori Gidron
Faculty of Medicine and Pharmacy,
Free University of Brussels (VUB), Jette,
Belgium

Definition

The etiology and prognosis of many diseases is complex, and assuming single causal agents alone could result in scientific and clinical

"ignorance." Most major causes of morbidity and mortality, such as of cancer, coronary heart disease (CHD), dementia, or traffic accidents (TA), are caused by and are predicted by multiple risk factors. In the case of CHD, for example, researchers often distinguish in behavioral medicine between background demographic risk factors such as age, gender, and education level; known biomedical risk factors such as family history of CHD, smoking, hypertension, physical inactivity, cholesterol level, and inflammatory markers; and behavioral risk factors such as hostility, effort-reward imbalance at work, and little social support. Some of the biomedical risk factors can indeed also be conceptualized as behavioral such as physical inactivity or smoking. Simultaneously testing multiple risk factors is then statistically done by multivariate analyses, where statistically significant risk factors from univariate tests are then simultaneously considered, to identify the unique risk factors of a disease, independent of other ones considered.

For example, after finding that hostility is the main "toxic" element from the type A behavior pattern, studies examined extensively the etiological and prognostic role of hostility in CHD, in multivariate analyses, where multiple risk factors named above where considered. Many studies supported the etiological independent role of hostility in CHD in multivariate analyses (e.g., Dembroski, MacDougall, Costa, & Grandits, 1989). However, some studies did find that certain "known" CHD risk factors, such as physical activity, smoking, and alcohol consumption, accounted for the hostility-CHD relationship, since after statistically controlling for their effects, hostility no longer predicted CHD (Everson et al., 1997). Yet, in a recent meta-analysis of all studies on anger, hostility, and CHD, Chida and Steptoe (2009) concluded that anger and hostility have an independent role in the risk of CHD and in its prognosis. The multivariate analysis serves to examine whether a factor has an etiological or prognostic role, independent of established risk factors, and if not, then to identify which factor mediates its role in the disease. At times, consideration of multiple risk factors also enables to identify interactive effects, which can help to point at a particularly high-risk group, who can benefit from scarce intervention efforts. For example, Gidron, Berger, Lugasi, and Ilia (2002) found that hostility synergistically interacted with CHD family history in relation to severity of coronary artery disease. Thus, for prevention at the population level, it may be better to focus on high-hostile people with a first-degree relative with CHD. Thus, the identification of multiple risk factors enables to better estimate one's risk of a disease and to treat people by targeting all significant independent risk factors (e.g., diet, physical activity, hostility reduction). By identifying and modifying multiple risk factors, we can aim to prevent or treat illnesses more effectively.

Cross-References

- ▶ Epidemiology
- ▶ Etiology/Pathogenesis
- ▶ Risk Ratio

References and Readings

Chida, Y., & Steptoe, A. (2009). The association of anger and hostility with future coronary heart disease: A meta-analytic review of prospective evidence. *Journal of the American College of Cardiology, 53*, 936–946.

Dembroski, T. M., MacDougall, J. M., Costa, P. T., Jr., & Grandits, G. A. (1989). Components of hostility as predictors of sudden death and myocardial infarction in the Multiple Risk Factor Intervention Trial. *Psychosomatic Medicine, 51*, 514–522.

Everson, S. A., Kauhanen, J., Kaplan, G. A., Goldberg, D. E., Julkunen, J., Tuomilehto, J., et al. (1997). Hostility and increased risk of mortality and acute myocardial infarction: The mediating role of behavioral risk factors. *American Journal of Epidemiology, 146*, 142–152.

Gidron, Y., Berger, R., Lugasi, B., & Ilia, R. (2002). Interactions of psychological factors and family history in relation to coronary artery disease. *Coronary Artery Disease, 13*, 205–208.

Multiple Sclerosis: Psychosocial Factors

Kyle R. Noll[1] and Laura H. Lacritz[2]
[1]Department of Physical Medicine & Rehabilitation, Baylor College of Medicine, Houston, TX, USA
[2]Department of Psychology, The University of Texas Southwestern Medical Center at Dallas, Dallas, TX, USA

Synonyms

Demyelinating disease; Quality of life

Definition

Early research into the psychological aspects of multiple sclerosis (MS) focused on an undifferentiated category referred to as "mental symptoms," which included fatigue, sleep, emotional, and cognitive problems. These relatively imperceptible symptoms were initially considered secondary to the more overt physical symptoms believed to be most impairing. However, with improved methodologies for measuring the domains of psychological functioning, impairments in psychosocial functioning are now recognized as a primary and often disabling consequence of the disease.

Description

Disease Characteristics

Pathology

MS is an autoimmune disorder that involves a chronic inflammatory process characterized by neuronal demyelination and inflammation in white matter regions, as well as more subtle tissue damage in diffuse areas of gray matter. The disease causes axonal demyelination in which the fatty myelin sheaths covering the axons of nerve cells are attacked by the body's immune system (Richardson, Robinson, & Robinson, 1997). More specifically, MS attacks oligodendrocytes, glial cells responsible for the production and maintenance of the myelin sheath, causing a thinning of the axonal sheath and transection of axons in the later stages of its more progressive forms (Compston & Coles, 2008). The neuronal damage results in gliosis (i.e., a proliferation of astrocytes) that leads to the formation of glial scars, also referred to as sclerotic plaques or lesions.

Diagnosis

Initial presentation of MS involves one or more of a variety of symptoms that include optic nerve dysfunction (e.g., visual deficits), sensory disturbance (e.g., pain, numbness, and tingling sensations), pyramidal tract dysregulation (e.g., increased muscle tone and hyperreflexia), ataxia, bladder and bowel dysfunction, and sexual problems, as well as cognitive impairment and emotional symptoms (Olek & Dawson, 2002). The varied presentation of MS can make diagnosis difficult, which often requires a combination of clinical symptoms and positive findings from neuroimaging and laboratory tests (see Table 1). Furthermore, the occurrence of one MS attack may be insufficient to make a diagnosis and time between exacerbations can be years. Thus, a definitive diagnosis can sometimes take months to years. However, the existence of disease-modifying agents that can slow progression of the disease makes early diagnosis and treatment particularly important. Multiple sclerosis may have a variable or progressive course. The US National Multiple Sclerosis Society classifies MS phenotypes as relapsing-remitting (RRMS), primary progressive (PPMS), secondary progressive (SPMS), and progressive-relapsing (PRMS).

Course

MS has been estimated to affect between 2 and 150 of every 100,000 people (Rosati, 2001). The majority of patients are diagnosed between 20 and 50 years of age, women are affected 2–3 times as often as men, and its prevalence is greatest in individuals of northern European descent (Prakash, Snook, Lewis, Motl, & Kramer, 2008). RRMS is the most common

Multiple Sclerosis: Psychosocial Factors, Table 1 McDonald criteria for MS diagnosis[a]

Clinical presentation	Additional data needed for MS diagnosis
> = 2 attacks; objective clinical evidence of > = 2 lesions or objective clinical evidence 1 lesion with reasonable historical evidence of a prior attack	None
2 or more attacks; objective clinical evidence of 1 lesion	Lesion(s) dissemination in *space* (DIS) on MRI as demonstrated by: > = 1 lesion in at least 2 of 4 MS typical regions (periventricular, juxtacortical, infratentorial, or spinal cord); or await further clinical attack implicating a different CNS site
1 attack; objective clinical evidence of 2 or more lesions	Lesions dissemination in *time* (DIT) on MRI as demonstrated by simultaneous presence of asymptomatic gadolinium-enhancing and nonenhancing lesions at any time; or a new enhancing lesion(s) on follow-up MRI, irrespective of its timing with reference to a baseline scan; or await a second attack
1 attack; objective clinical evidence of 1 lesion (clinically isolated syndrome)	Lesion dissemination in *space* and *time* (as defined above; or await a second attack
Insidious neurological progression suggestive of MS (PPMS)	1 year of disease progression plus 2 of 3 of the following criteria: 1. Evidence of DIS in the brain based on > = 1 lesion in the MS-characteristic brain regions 2. Evidence of DIS in the spinal cord based on > = 2 lesions in the cord 3. Positive CSF (isoelectric focusing evidence of oligoclonal bands and/or elevated IgG index)

[a]Adapted from Polman et al. (2011)
MRI magnetic resonance imaging, *PPMS* primary progressive multiple sclerosis, *CSF* cerebrospinal fluid, *IgG* immunoglobulin G

subtype and is the initial course of 80% of individuals with MS (Compston & Coles, 2008). Approximately 65% of those with an initial RRMS presentation begin to exhibit progressive neurologic decline classified as SPMS (Lublin & Reingold, 1996). Regardless of subtype, most cases of MS eventually cease to remit and become slowly progressive over time.

Patients with MS have an average life expectancy that is 7 years shorter than the general population, though patients usually die of MS-related disability rather than the disease process itself. Though the disease has no known cure, use of disease-modifying drugs (e.g., interferons, immunosuppressant agents, and cytotoxic agents) has significantly improved outcomes for individuals with MS. Such agents decrease clinical relapses, disability progression, and lesion load, and may also have cognitive benefits, though more research is needed (Amato, Portaccio, & Zipoli, 2006).

Psychosocial Factors

Cognitive Functioning

Cognitive processes rest upon dynamic brain networks that are highly dependent on the integrity of long white matter tracts to facilitate information flow between distant cortical areas. MS lesions can disrupt connections between cortical regions, often resulting in impairments of cognitive functioning. Cognitive deficits have been estimated to be present in up to 65% of patients (Rao, 1997). Impairment can occur even in the early stages of the disease, and complete remission of cognitive symptoms is uncommon across all disease subtypes (Amato et al., 2006).

Although there is no uniform pattern of cognitive impairments in MS, greatest deficits are often seen in learning and memory, with working memory and short-term recall the most significantly impacted (Calabrese, 2006). Other commonly affected domains include information processing speed, complex visuospatial abilities,

conceptual reasoning, and sustained attention, while primary language functions and verbal intellectual skills are usually unaffected. Additional factors that may impact cognition include depression, anxiety, and fatigue, as well as lesion location, size, and medication use.

Psychological Functioning

Individuals with MS are susceptible to emotional problems both as a direct result of disease pathology and as a consequence of coping with its numerous symptoms. Up to 60% of patients seek professional help for emotional problems during the course of their illness. Many MS symptoms are not noticeable to others (e.g., weakness, dizziness, headache, impaired coordination, muscle spasticity, bladder and bowel dysfunction, fatigue, sleep problems, pain, and sexual difficulties), but can result in daily problems and impact emotional functioning (National Multiple Sclerosis Society, 2010).

Given unpredictable exacerbations, symptomatology, and prognosis, it is not surprising that depression and anxiety are commonly associated with MS. The most common mood disorder co-occurring with MS is major depressive disorder, which is present in up to 54% of patients (Patten, Beck, Williams, Barbui, & Metz, 2003). This rate far surpasses those of many other common neurological or medical conditions. Anxiety disorders have also been identified in approximately 25% of MS patients, which is slightly less than their prevalence in the general population (Kessler & Wang, 2008). Comorbid anxiety and major depressive disorders in MS are associated with increased somatic symptoms, suicidal ideation, interpersonal difficulty, and decreased treatment adherence.

Quality of Life

Quality of life (QoL) refers to an individual's subjective well-being as determined by psychosocial, health, economic, and environmental factors. Unsurprisingly, such factors can be significantly impacted by the constellation of cognitive, emotional, and physical symptoms characteristic of MS. MS leads to unemployment in 50–80% of cases within a 10-year disease course, making it the most common cause of neurological disability in young and middle-aged adults in the United States and Europe (Johnson, 2007).

Although physical problems are the most recognizable symptoms of MS, impairments in mental functions appear to be the most important predictor of QoL (Wynia et al., 2008). Mental impairments (including cognitive and emotional problems) are present in more than 80% of patients and are major predictors of disability, unemployment, and caregiver distress. Patients with less impairment in mental functions report better QoL in the domains of mental health, emotional functioning, social functioning, bodily pain, and vitality. All MS patients are at risk for decreased QoL, though QoL is lowest in the more progressive forms of the disease. While fatigue is more prevalent than cognitive and emotional problems and can significantly impact daily functioning, its impact on QOL is limited.

Summary

The demyelinating lesions of MS cause a disconnection of neuronal pathways resulting in a variety of clinical symptoms that tend to be episodic and fluctuate in severity and course. The constellation of symptoms characteristic of the disease often impact physical, cognitive, and psychological functioning, that in turn tend to negatively impact overall quality of life. Further, cognitive impairments (e.g., memory, processing speed, and attention), depression, anxiety, fatigue, and sleep problems are often more functionally impairing than the physical symptoms of the disease, leading to unemployment, disability, and decreased subjective well-being.

Cross-References

- ▶ Fatigue
- ▶ MRI
- ▶ Psychosocial Factors
- ▶ Quality of Life

References and Readings

Amato, M. P., Portaccio, E., & Zipoli, V. (2006). Are there protective treatment for cognitive decline in MS. *Journal of the Neurological Sciences, 245*, 183–186.

Calabrese, P. (2006). Neuropsychology of multiple sclerosis: An overview. *Journal of Neurology, 253* (Suppl. 1), 10–15.

Compston, A., & Coles, A. (2008). Multiple sclerosis. *Lancet, 372*, 1502–1517.

Johnson, S. K. (2007). The neuropsychology of multiple sclerosis. *Disease-a-month, 53*, 172–176.

Kessler, R. C., & Wang, P. S. (2008). The descriptive epidemiology of commonly occurring mental disorders in the United States. *Annual Review of Public Health, 29*, 115–129.

Lublin, F. D., & Reingold, S. C. (1996). Defining the clinical course of multiple sclerosis: Results of an international survey. National multiple sclerosis society (USA) Advisory committee on clinical trials of new agents in multiple sclerosis. *Neurology, 46*, 907–911.

National Multiple Sclerosis Society. (2010). *Symptoms of multiple sclerosis*. Retrieved March 24, 2010, from http://www.nationalmssociety.org/about-multiple-sclerosis/what-we-know-about-ms/symptoms/index.aspx

Olek, M. J., & Dawson, D. M. (2002). *Multiple sclerosis and other inflammatory demyelinating diseases of the central nervous system: Neurology in clinical practice* (3rd ed., Vol. 2). New York: Butterworth Heineman.

Patten, S. B., Beck, C. A., Williams, J. V., Barbui, C., & Metz, L. M. (2003). Major depression in multiple sclerosis: A population-based perspective. *Neurology, 61*, 1524–1527.

Polman, C. H., Reingold, S. C., Banwell, B., Clanet, M., Cohen, J. A., Filippi, M., et al. (2011). Diagnostic criteria for multiple sclerosis: 2010 revisions to the McDonald criteria. *Annals of Neurology, 69*, 292–302.

Prakash, R. S., Snook, E. M., Lewis, J. M., Motl, R. W., & Kramer, A. F. (2008). Cognitive impairments in relapsing-remitting multiple sclerosis: A meta-analysis. *Multiple Sclerosis, 14*, 1250–1261.

Rao, S. M. (1997). Neuropsychological aspects of multiple sclerosis. In C. S. Raine, H. F. McFarland, & W. W. Tourtellotte (Eds.), *Multiple sclerosis: Clinical and pathogenetic basis* (pp. 357–362). London: Chapman and Hall.

Richardson, J. T. E., Robinson, A., & Robinson, I. (1997). Cognition and multiple sclerosis: A historical analysis of medical perceptions. *Journal of the History of the Neurosciences, 6*, 302–319.

Rosati, G. (2001). The prevalence of multiple sclerosis in the world: An update. *Neurological Sciences, 22*, 117–139.

Wynia, K., Middel, B., van Dijk, J. P., De Keyser, J. H., & Reijneveld, S. A. (2008). The impact of disabilities on quality of life in people with multiple sclerosis. *Multiple Sclerosis, 14*, 972–980.

Multistage Submaximal Exercise Test

▶ Graded Exercise

Multivariate Analysis

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

Multivariate analyses analyze several outcome variables at the same time, all as a function of one or more predictor variables (Piantadosi, 2005).

Cross-References

▶ Univariate Analysis

References and Readings

Piantadosi, S. (2005). *Clinical trials: A methodologic perspective* (2nd ed.). Hoboken, NJ: Wiley-Interscience.

Muscle Wasting

▶ Atrophy

Musculoskeletal Pain

▶ Arthritis: Psychosocial Aspects
▶ Pain

Mutagen

► Carcinogens

MyPlate

Sheah Rarback
Department of Pediatrics, University of Miami, Miami, FL, USA

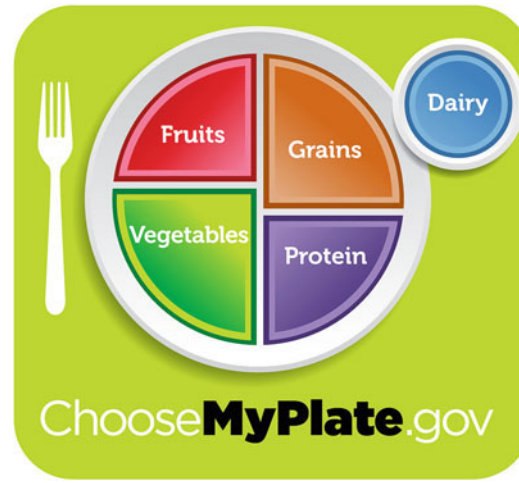
Synonyms

Healthy eating guide

Definition

The Food Guide Pyramid was a visual representation of the recommendations for daily food intake. The Food Guide Pyramid followed the recommendations of the USDA Dietary Guidelines. The Food Guide Pyramid was first published by the United States Department of Agriculture (USDA) in 1992 and was updated approximately every 5 years. In 2005, the Food Guide Pyramid changed to a vertical representation of food groups and emphasized exercise by including a person walking up "stairs" on the side of the pyramid. As the Dietary Guidelines were updated June 2010, the Food Guide Pyramid was eliminated in 2011. The USDA launched a new site in 2011 called Choose My Plate and can be accessed at www.choosemyplate.gov.

Although the Food Guide Pyramid changes every 5 years, the basic message remains the same. Recommendations are to include a variety of foods each day including whole grains, dark-green and orange vegetables, dry beans and peas, fresh or frozen fruit, limited fruit juice, calcium-rich foods such as low-fat and fat-free milk and milk products, and low-fat or lean meats and poultry. There are unique Food Guide Recommendations for preschoolers, children, and pregnant women, as shown in Choose My Plate (Fig. 1).



MyPlate, Fig. 1 The plate is one half fruit and vegetables, a bit over a quarter grains, a bit under a quarter protein and a glass of dairy

Cross-References

► Healthy Eating

References and Readings

Frieden, T. (2010). A framework for public health action: The health impact pyramid. *American Journal of Public Health, 100*, 590–595.

Gao, X., Wilde, P., Lichtenstein, A., & Tucker, K. (2006). The 2005 USDA food guide pyramid is associated with more adequate nutrient intakes within energy constraints than the 1992 pyramid. *Journal of Nutrition, 136*(5), 1341–1346.

Myths

► Cultural Factors

N

N1, N2, N3

► Non-REM Sleep

N3

► Slow-Wave Sleep

n-3 Fatty Acids

► Omega-3 Fatty Acids

National Cancer Institute

Jasmin Tiro and Simon J. Craddock Lee
Department of Clinical Sciences, The University of Texas Southwestern Medical Center, Dallas, TX, USA

Basic Information

The US National Cancer Institute (NCI)

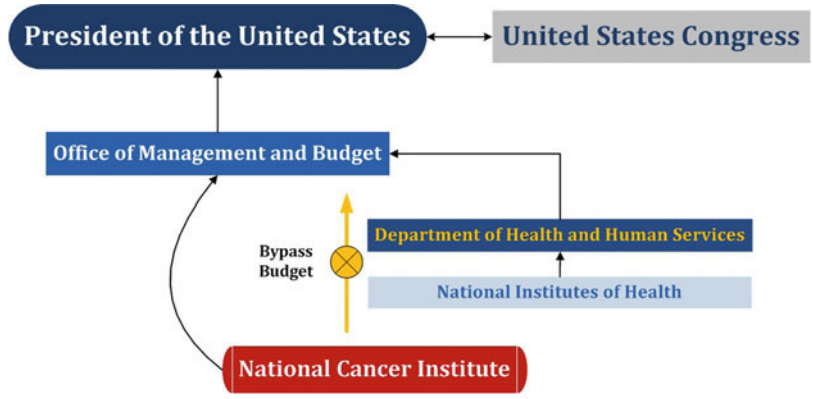
The National Cancer Institute is the oldest and largest of the 27 institutes and 6 centers that comprise the US National Institutes of Health (NIH), which are part of the US Department of Health and Human Services (DHHS) Public Health Service (PHS). First established in 1937 by Congress, NCI's mission and responsibilities were expanded in the *National Cancer Act of 1971*. Currently, the NCI's main responsibility is to coordinate the National Cancer Program, which fosters research, training, and health information dissemination programs with respect to the cause, diagnosis, prevention, and treatment of cancer, rehabilitation from cancer, and survivorship concerns of cancer patients and their families. Through this program, the NCI:

- Supports a national network of regional and community cancer centers
- Conducts and fosters cancer research through intramural (its own laboratories and clinics) and extramural programs (grants and cooperative agreements with universities, hospitals, research foundations, and businesses)
- Reviews, approves, and monitors grants supporting novel research projects on the causes, diagnosis, treatment, and prevention of cancer
- Collects, analyzes, and disseminates cancer research findings
- Trains health professionals in cancer diagnosis and treatment and researchers in basic, clinical, cancer control, behavioral, and population sciences
- Supports collaborative research between US and foreign researchers

Unique Budget Process of the NCI

Unique among the institutes in the NIH, NCI has the authority to submit an annual budget

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,



National Cancer Institute, Fig. 1 Bypass budget process of the National Cancer Institute (The National Cancer Institute, NIH (Bethesda, MD), http://www.cancer.gov/aboutnci/servingpeople/nci-budget-information/budget-process. Accessed date 7/2011)

proposal directly to the president (called the "Bypass Budget" because it circumvents the NIH/DHHS budget process; see Fig. 1). The White House Office of Management and Budget (OMB) reviews and integrates the NCI proposal into the president's executive branch budget which reflects the administration's fiscal and management priorities for the next year. Then, Congress reviews the president's proposal and makes a recommendation, and final appropriations are enacted into law after approval by both houses of Congress and signature by the president. Every year, NCI publishes online the Bypass Budget Proposal for the coming fiscal year and the Annual Fact Book, which summarizes the distribution of budget among the research programs and funding mechanisms for the past fiscal year. In 2009, NCI's budget was $4.97 billion not including the additional $1.26 billion from the American Recovery and Reinvestment Act (ARRA) funds intended to be distributed in fiscal years 2009 and 2010. Approximately 43% of the total NCI 2009 budget was allocated for 5,461 research project grants, including 543 grants made possible by ARRA funds.

The US government has made cancer a national priority and has allocated significant resources to understand the complexity of cancer as a disease process as well as processes of cancer care. Thus, the NCI has become a leading supporter of behavioral, social, and population research in basic, intervention, and applied science related to cancer.

NCI Organizational Structure and Governance

The NCI's organizational structure consists of the Office of the NCI Director, two intramural divisions/centers, and five extramural divisions. The current NCI director is Dr. Harold Varmus, Nobel laureate. The NCI director receives programmatic and scientific advice and counsel from three national bodies of appointed experts. The *National Cancer Advisory Board* (NCAB) advises the secretary of DHHS and the NCI director with respect to the activities of the Institute. Appointed by the president, members are leading representatives of health and scientific disciplines, including at least two experts in public health and the behavioral or social sciences. NCAB approves research grant award decisions made by NCI program staff who prioritize applications ranked according to scientific merit through independent peer review. The *Board of Scientific Advisors* (BSA) provides scientific advice and guidance on a wide variety of matters concerning scientific program policy, progress, and future direction of the NCI's *extramural* research programs and provides concept review of extramural program initiatives. The BSA charter specifically requires that one or more members have expertise with cancer epidemiology, cancer prevention and control, cancer education, cancer information services, and community outreach. The *Board of Scientific Counselors* (BSC) provides analogous scientific advice and guidance regarding *intramural* research programs.

Major Impact on the Field

Division of Cancer Control and Population Sciences (DCCPS)

The main division charged with fostering behavioral medicine is the extramural Division of Cancer Control and Population Sciences (DCCPS), created in 1997 and currently led by social psychologist, Dr. Robert Croyle. Cancer control science is defined as "basic and applied research in the behavioral, social, and population sciences to create or enhance interventions that, independently or in combination with biomedical approaches, reduce cancer risk, incidence, morbidity and mortality, and improve quality of life" (Cancer Control Program Review Group, 1998-modified). For a history of cancer control and commentary on how to advance the science of cancer control, see Hiatt and Rimer (1999).

DCCPS conducts and supports integrated research in a wide range of disciplines and fields including anthropology, behavioral sciences, demography, epidemiology, genetics, health communication, health policy, health services research, psychology, public health, sociology, and surveillance. DCCPS funds research on the full continuum of cancer control that spans prevention, detection, diagnosis, treatment, survivorship, and end-of-life care to "understand the causes and distribution of cancer in populations; support the development and delivery of effective interventions; monitor and explain cancer trends in all segments of the population." As a result of its mission, DCCPS represents one of the largest concentrations of social, behavioral, and population scientists at the NIH.

DCCPS is organized around five research program areas: Epidemiology and Genetics, Surveillance, Applied, Behavioral, and Survivorship. The latter three programs are of central interest to researchers in behavioral medicine. The Applied Research Program fosters research to understand how and why cancer care and control activities in the USA influence patterns and trends in cancer outcomes (incidence, morbidity, mortality, and survival). The Behavioral Research Program fosters a wide range of research from basic behavioral research to evaluation and dissemination of interventions to promote cancer prevention and control behaviors and informed decision-making. The Office of Cancer Survivorship fosters research on the short- and long-term biopsychosocial effects of cancer and its treatment. An overview of the history and scientific accomplishments of the first 10 years of DCCPS (1997–2007) can be found online at the NCI website (www.cancercontrol.gov).

Current Major Programs and Initiatives of DCCPS

DCCPS is a leader in supporting transdisciplinary research in tobacco control, population health disparities, cancer-related energetics (physical activity), cancer screening, and health communication research (Croyle, 2008; Rebbeck, Paskett, & Sellers, 2010; Stokols et al., 2003). DCCPS has led or partnered with other institutes and offices (e.g., National Heart, Lung, and Blood Institute; National Institute on Minority Health and Health Disparities) on large NIH funding mechanisms to establish research centers to leverage transdisciplinary and team science research in these cancer domains.

DCCPS also collaborates with the NIH Office of Behavioral and Social Science Research (OBSSR) to jointly fund innovative grants and projects. For example, the Grid-Enabled Measures Database enables researchers to share standardized measures of theory-based constructs through OppNet, a trans-NIH initiative to fund and advance research elucidating mechanisms and processes within and among individuals and groups that affect health-related behaviors.

Other key theoretic and methodologic initiatives that engage behavioral medicine include cognitive, affective, and social processes in health, health behavior theory development, measurement of health constructs (e.g., quality of cancer care), questionnaire design and testing, and the integration of behavioral and biomedical scientific approaches (Stefanek et al., 2009). These scientific areas are reflected in networks of individual investigators collaborating with NCI scientific staff, as well as in funding opportunities calling for grant applications to advance these efforts.

Cancer Centers Program

The National Cancer Act of 1971 also established NCI-designated Cancer Centers for clinical research, training, and demonstration of advanced diagnostic and treatment methods for cancer. To be awarded designation, these centers must periodically undergo peer review and meet a national series of competitive standards and demonstrate excellence in cancer research through the operation of formal programs. As of 2010, there are 66 Cancer Centers across the USA that have been awarded NCI designation. Forty of these centers carry *Comprehensive* Cancer Center status, which reflects integration and collaborative research in clinical trials and patient care, as well as programmatic emphases in epidemiology and cancer control. As such, NCI-designated Comprehensive Cancer Centers are often leading proponents of cancer-related behavioral medicine.

Conclusion

The US National Cancer Institute is the largest organization dedicated to supporting cancer research in the world. Through its efforts, it provides vision and leadership to the global cancer community. This is especially true in the area of behavioral oncology and cancer control. The NCI recognizes the importance of transdisciplinary research efforts in addressing the cancer continuum.

References and Readings

Croyle, R. T. (2008). The National Cancer Institute's transdisciplinary centers initiatives and the need for building a science of team science. *American Journal of Preventive Medicine, 35*(Suppl. 2), s90–s93.

Hiatt, R. A., & Rimer, B. K. (1999). A new strategy for cancer control research. *Cancer Epidemiology, Biomarkers & Prevention, 8*(11), 957–964.

NCI Division of Cancer Control & Population Sciences. Available at: http://cancercontrol.cancer.gov/index.html. Accessed April 2011.

Niederhuber, J. E. (2007). A look inside the national cancer institute budget process: Implications for 2007 and beyond. *Cancer Research, 67*, 856–862.

Rebbeck, T. R., Paskett, E., & Sellers, T. A. (2010). Fostering transdisciplinary science. *Cancer Epidemiology, Biomarkers & Prevention, 19*(5), 1149–1150.

Stefanek, M. E., Andrykowski, M. A., Lerman, C., Manne, S., Glanz, K., & AACR behavioral science task force. (2009). Behavioral oncology and the war on cancer: Partnering with biomedicine. *Cancer Research, 69*(18), 7151–7156.

Stokols D, Fuqua J, Gress J, Harvey R, Phillips K, Baezconde-Garbanati L, Unger J, Palmer P, Clark MA, Colby SM, Morgan G, Trochim W. (2003). Evaluating transdisciplinary science. *Nicotine Tobacco Research*, *5*(S1): S21–39.

National Children's Study

Steven E. Lipshultz[1], Tracie L. Miller[2], James D. Wilkinson[2] and Miriam A. Mestre[3]
[1]Department of Pediatrics, Epidemiology and Public Health, and Medicine (Oncology), Leonard M. Miller School of Medicine University of Miami Holtz Children's Hospital of the University of Miami-Jackson Memorial Medical Center Batchelor Children's Research Institute Mailman Center for Child Development University of Miami Sylvester Comprehensive Cancer Center, Miami, FL, USA
[2]Department of Pediatrics and Epidemiology and Public Health Division of Pediatric Clinical Research Department of Pediatrics, Leonard M. Miller School of Medicine University of Miami Holtz Children's Hospital of the University of Miami-Jackson Memorial Medical Center Batchelor Children's Research Institute University of Miami Sylvester Comprehensive Cancer Center, Miami, FL, USA
[3]Division of Pediatric Clinical Research Department of Pediatrics, Leonard M. Miller School of Medicine University of Miami, Miami, FL, USA

Synonyms

NCS

Definition

The National Children's Study (NCS) will be the largest longitudinal study of children's health,

growth, and development ever conducted in the United States. The main study aims to recruit women from randomly selected study locations around the country, and a sample of 100,000 children born to these recruited mothers will be followed from before birth to 21 years of age. "It is the first large birth cohort study in any nation to specifically examine the influence of environmental factors on birth outcomes, child health, and human development and the first designed to systematically examine the influence of gene-environment interactions on children's health" (Landrigan et al., 2006). The NCS defines "environment" broadly, such as air, water, soil, noise, stress, and exposure to natural and manufactured products. By studying children through different phases of growth and development, researchers may be better able to understand the role these factors have on health and disease.

The National Children's Study is led by the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development of the National Institutes of Health (NIH) in collaboration with a consortium of federal government partners. Study partners include the National Institute of Environmental Health Sciences of the NIH, the Centers for Disease Control and Prevention, and the Environmental Protection Agency. The NCS will concentrate on collecting data geared toward the following broad outcomes with key public health significance: pregnancy outcome, neurodevelopment and behavior, lung and airway disease, physical growth and body composition, and injury. Analysis of pilot data from field testing at a subgroup of NCS sites has resulted in modifications to the original study design. The NCS main study is currently scheduled to begin in 2013

Cross-References

- ▶ Body Composition
- ▶ Centers for Disease Control and Prevention
- ▶ Gene-Environment Interaction
- ▶ Longitudinal Research
- ▶ National Institute of Child Health and Human Development
- ▶ National Institutes of Health

References and Readings

Landrigan, P. J., Trasande, L., Thorpe, L. E., Gwynn, C., Lioy, P. J., D'Alton, M. E., et al. (2006). The National Children's Study: A 21-year prospective study of 100 000 American children. *Pediatrics, 118*, 2173–2186. Retrieved from www.nationalchildrensstudy.gov

National Health and Nutrition Examination Survey, The

▶ National Health and Nutrition Examination Survey (NHANES)

National Health and Nutrition Examination Survey (NHANES)

Barbara Resnick
School of Nursing, University of Maryland, Baltimore, MD, USA

Synonyms

National health and nutrition examination survey, The

Definition

The National Health and Nutrition Examination Survey (NHANES) is a program of studies designed to assess the health and nutritional status of adults and children in the United States. The survey is unique in that it combines interviews and physical examinations. NHANES is a major program of the National Center for Health Statistics (NCHS). NCHS is part of the Centers for Disease Control and Prevention (CDC) and has the responsibility for producing vital and health statistics for the nation.

The NHANES program began in the early 1960s and has been conducted as a series of surveys focusing on different population groups

or health topics. In 1999, the survey became a continuous program. The focus of the survey changes based on current health care trends and needs. Each year the survey includes a sample of 5,000 persons representative of the general population.

The NHANES interview includes demographic, socioeconomic, dietary, and health-related questions. The examination component consists of medical, dental, and physiological measurements, as well as laboratory tests administered by medical personnel.

Findings from this survey are used to determine the prevalence of major diseases and risk factors for diseases. Information is available to consider such things as nutritional status and its association with health promotion and disease prevention. NHANES data are also used to set norms for height, weight, and blood pressure and to establish public health policy, to guide health maintenance and prevention program and services and expand what is known about health in our country.

References and Readings

The National Health and Nutrition Examination Survey (NHANES). Retrieved from http://www.cdc.gov/nchs/nhanes/about_nhanes.htm

National Health Interview Survey

Donna Miller[1], Cristina A. Fernandez[2] and David J. Lee[2]
[1]Centers for Disease Control and Prevention, National Center for Health Statistics, Hyattsville, MD, USA
[2]Department of Epidemiology and Public Health, Miller School of Medicine, University of Miami, Miami, FL, USA

Synonyms

National health survey; NHIS

Basic Information

The National Health Interview Survey (NHIS) is a principal source of information on the health status of the civilian noninstitutionalized population of the United States. The NHIS is one of the major data collection programs of the National Center for Health Statistics (NCHS), a component of the Centers for Disease Control and Prevention (CDC). The NHIS has been in the field continuously since 1957 and is the nation's largest household health survey. The survey was authorized by Congress in order to obtain national estimates on disease, injury, impairment, disability, and related issues for the US population. The NHIS has evolved over the years, with significant questionnaire redesigns in 1982 and 1997.

Major Impact on the Field

Purpose

The main objective of the NHIS is to monitor the health of the United States population through the collection and analysis of data on a broad range of health topics. The NHIS provides data for the analysis of health trends, barriers to care, health status, health-care access and utilization, health-related behaviors, and risk factors. A major strength of this survey lies in the ability to display these health characteristics by many demographic and socioeconomic characteristics. Survey results have been instrumental in tracking health status and health-care access, and monitoring progress toward achieving national health objectives. The NHIS is a major source of data used for *Healthy People*, which provides 10-year national health objectives and tracks progress toward achieving them for improving the health of all Americans.

Sample Design

The NHIS is a cross-sectional household interview survey designed to be representative of the civilian noninstitutionalized population of the United States. The sampling plan follows a multistage area probability design that permits

the representative sampling of households and noninstitutional group quarters (e.g., college dormitories). The NHIS sample design is reevaluated and modified after every US decennial census. The NHIS sample design oversamples selected minority subgroups including African Americans, Hispanics, and starting in 2006, Asians. Persons aged 65+ in these selected minority groups are also oversampled. Since the last NHIS sample redesign was implemented in 2006, the expected annual interviewed sample size has been about 35,000 households with approximately 87,500 persons, although the sample sizes vary appreciably over time.

The NHIS data files contain sample weights based on the unit of analysis (e.g., household, person). The NHIS sample is chosen such that each person in the covered population has a known nonzero probability of selection. These probabilities of selection, along with adjustments for nonresponse and post-stratification to sex, age, and race/ethnicity census population control totals, are reflected in the sample weights provided. It is necessary to utilize the weights provided in analyses of the NHIS data, along with the stratification and primary sampling unit information, for valid statistical inferences.

Content of the Questionnaire

The NHIS collects basic health and demographic data, which can be used to develop prevalence estimates of a wide variety of health measures. The NHIS questionnaire contains a set of Core questions and Supplemental questions. The Core includes questions about chronic conditions, disability, health behaviors, risk factors, health insurance coverage, and health-care use. Core questions have remained largely unchanged since 1997, which allows for trend analysis and for pooling data from more than one NHIS year, thus increasing the sample size for analytic purposes. The Supplemental questions change from year to year and collect additional data pertaining to current health issues of national importance (e.g., cancer, immunization, diabetes).

A 1997 questionnaire redesign separated the Core questions into three main components: the Family Core (collects information on all family members), the Sample Adult Core (collects information directly from one randomly selected adult, aged 18 and over, in each family in the household), and the Sample Child Core, which is only administered if a child resides in the family (collects information on one randomly selected child, aged 17 and under, from a knowledgeable adult family member).

The Family Core questionnaire includes sections on health status and limitations, injuries and poisoning, health-care access and use, health insurance coverage, and sociodemographic characteristics. The Sample Adult Core questionnaire includes sections on health conditions and health behaviors in addition to more detailed questions on some topics included in the Family Core. The Sample Child Core questionnaire includes questions on health conditions and more detailed questions on some topics included in the family core.

Data Collection Procedures

The NHIS data are collected by interviewers who are employed and trained by the US Census Bureau in accordance with procedures specified by NCHS, via personal household interviews. Prior to 1997, NHIS interviews were conducted using paper and pencil. After a questionnaire redesign was implemented in 1997, all data were collected by interviewers using computer-assisted personal interviewing, allowing interviewers to enter responses directly into the computer during the interviews.

Prior to 1997, all adult members of the household aged 18 and over who were at home at the time of the interview were invited to participate and to respond for themselves; however, a proxy respondent often provided responses for all household members. After the 1997 questionnaire redesign, a greater emphasis was placed on self-reporting to improve data quality. Data collection procedures for conducting the household, family, and children interviews remained largely unchanged, but sample adults must respond for themselves (except in rare cases where they are unable to do so).

Confidentiality

All data collected in the NHIS are used for statistical purposes only and are guaranteed by law to be held in the strictest of confidence. Survey participation is voluntary, and the confidentiality of responses is assured under Section 308(d) of the Public Health Service Act, which forbids the disclosure of any information that may compromise the confidentiality promised to its survey respondents.

Record Linkages

The NHIS data are routinely linked to administrative records enabling researchers to examine factors that influence disability, chronic disease, health-care utilization, morbidity, and mortality. The NHIS is linked to death certificate records from the National Death Index; Medicare and Medicaid enrollment and claims data from the Centers for Medicare and Medicaid Services; and Retirement, Survivor and Disability Insurance and Supplemental Security Income benefit data from the Social Security Administration. Immunization data for children in the 1997–1999 NHIS are linked to physician medical records in the National Immunization Provider Record Check Study. Starting with the 1996 Medical Expenditure Panel Survey (MEPS), the NHIS data can be linked to the MEPS data which are drawn from a subsample of households that participated in the prior year's NHIS, allowing users to link, for example, the 1995 NHIS to the 1996 MEPS. The NHIS data are also linked to contextual data including air monitoring data obtained from the Environmental Protection Agency.

Data Availability

The NHIS data are available via public-use and restricted-use data files. Public-use NHIS data files are released annually and are available for download from the NCHS web site. Restricted-use linked NHIS data files, which contain indirect identifiers such as geographical locations or specific dates, are available for use through the NCHS Research Data Center (RDC). The RDC is a secure environment designed to protect the confidentiality of survey respondents, while also allowing researchers the ability to access restricted-use data for research purposes only.

Documentation containing detailed information on the public-use and restricted-use NHIS data files is available on the NCHS web site. Reports providing statistics based on data collected from NHIS and detailed documentation on the NHIS design, sampling procedures, and guidance for analyzing the data are also available. Information on the NHIS Early Release Program which provides very timely estimates of key health and health-related indicators is also provided on the NCHS web site. Harmonized NHIS public-use data and documentation going back to the early 1960s are available in the Integrated Health Interview Series, maintained by the University of Minnesota. (See section G for more information on these resources.)

References and Readings

For additional information on NHIS, see: http://www.cdc.gov/nchs/nhis/about_nhis.htm
For information on NHIS questionnaires by year of survey, see: http://www.cdc.gov/nchs/nhis/nhis_questionnaires.htm
For information on NHIS design and estimation, see: http://www.cdc.gov/nchs/nhis/methods.htm
For information on NHIS reports and data linked to NHIS, see: http://www.cdc.gov/nchs/nhis/nhis_products.htm
For information on the 1997 NHIS questionnaire redesign, see: http://www.cdc.gov/nchs/nhis/nhis_redesign.htm and ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Dataset_Documentation/NHIS/1997/srvydesc.pdf
For information on NHIS Supplements, see: http://www.cdc.gov/nchs/nhis/supplements_cosponsors.htm
For information on the NHIS Early Release Program, see: http://www.cdc.gov/nchs/nhis/releases.htm
For information on accessing restricted-use NHIS data in the NCHS Research Data Center, see: http://www.cdc.gov/rdc/
For information on NHIS data linked to air quality data, see: http://www.cdc.gov/nchs/data_access/data_linkage/air_quality.htm
For information on the Integrated Health Interview Series (IHIS), see: http://www.ihis.us/ihis/

National Health Survey

► National Health Interview Survey

National Heart, Lung, and Blood Institute

Martica H. Hall
Department of Psychiatry, University of Pittsburgh, Pittsburgh, PA, USA

Basic Information

The National Heart, Lung, and Blood Institute (NHLBI) is one of the National Institutes of Health (NIH). Its mission is the advancement of research on, and the prevention and treatment of, heart, lung, and blood diseases and sleep disorders.

The National Heart Institute (NHI) was established in 1948 as one of the National Institutes of Health (NIH). The National Heart Institute's mission was to advance research and training related to heart disease and functioning. Its functions were broadened in 1969 and 1976 to include lung (National Heart and Lung Institute; NHLI) and blood diseases (National Heart, Lung, and Blood Institute; NHLBI), respectively. The NHLBI is also responsible for the adequacy and safety of the nation's blood supply.

Major Impact on the Field

Organizationally, the NHLBI includes the Office of the Director, an Intramural Research Program, and an Extramural Research Program. The Office of the Director provides strategic planning, policy guidance, program development and evaluation, and institute coordination. It also liaisons with Federal agencies, professional societies, and the public and is responsible for the dissemination of information regarding the prevention of heart, lung, and blood diseases and sleep disorders. The Office of the Director includes divisions, centers, and branches that support bioinformatics development; epidemiological studies of the etiology of heart, lung, and blood diseases and sleep disorders; the translation of scientific evidence into clinical practice; and the elimination of health disparities through the education and career development of a diverse workforce.

The Intramural Research Program includes a multinational team of NIH investigators and clinicians who conduct basic and clinical research and technology development related to heart, lung, and blood diseases and sleep disorders. In contrast, the Extramural Research Program supports heart, lung, and blood diseases and sleep disorders research, research training, and career development of investigators outside of the NIH. These activities are supported through various extramural award mechanisms including research, program project, and center grants; cooperative agreements and research contracts; research career development awards; and institutional and individual national research service awards. Three divisions (Cardiovascular, Lung, Blood Diseases and Disorders) foster research and training in their related areas.

Cross-References

► National Institutes of Health

References and Readings

NHLBI home page. www.nhlbi.nih.gov

National Institute of Child Health and Human Development

Deborah Lee Young-Hyman
Department of Pediatrics, Georgia Prevention Institute Georgia Health Sciences Universtiy, Augusta, GA, USA

Basic Information

The National Institute of Child Health and Human Development (NICHD) was established by federal law in October 1962. The mission of NICHD encompasses that every person is born healthy

and wanted; no harmful effects are suffered by women in the reproductive process; children have the opportunity to live healthy and productive lives; and to ensure the health, productivity, independence, and well-being of people through optimal rehabilitation (http://www.nih.gov/about/almanac/organization/NICHD.htm).

Major Impact on the Field

This mission statement informs the research programs supported by NICHD as follows:

Projects funded through NICHD adopt a life course perspective, such that conditions are studied prior to and during pregnancy, as well as during childhood, as they impact the health and well-being of children and adults, including person based and environmental factors and their interaction. The process of growth and development is assumed to be continual and evolving, such that research focuses also include cellular, molecular, and developmental biology. Basic, clinical, and epidemiologic research is conducted regarding reproductive science, with emphasis on safe and effective regulation of fertility, solving problems of infertility, and understanding consequences of reproductive behavior and population change. This last focus is behavioral and social science based.

NICHD also supports the research training of clinicians in order to address areas of critical public health concern. A current focus is obesity prevention and treatment throughout the life course, with particular emphasis on the maternal-fetal environment. In 2008–2009, the National Children's Study was launched in concert with the Centers for Disease Control (CDC), a longitudinal study of environmental influences on child health. This study will follow approximately 100,000 subjects across diverse areas the United States, and span over 20 years, mapping multiple environmental influences on children's health status. As the issues addressed by NICHD span biologic systems, NICHD often partners with NIDDK and NHLBI to fund projects which target disease prevention and intervention during childhood, particularly the antecedents of diabetes and cardiovascular disease

The division of intramural research addresses the biological and neurobiological, medical, and behavioral aspects of normal and abnormal human development.

In addition to issues of biology, NICHD supports research that addresses health literacy and numeracy, health maintenance, use of health care and community resources to improve child health, and the interaction between risk behaviors, psychological morbidity, and health outcomes.

NICHD supports a systems approach to discovery, whereby the focus of inquiry can range from the maternal-child dyad, to the family-school or family-health care environment. Therefore, besides a strong commitment to developmental biology discovery, there is emphasis on how health prevention attitudes and behavior translate into public health issues.

Cross-References

- ▶ National Children's Study
- ▶ National Institutes of Health

References and Readings

NICHD home page. www.nichd.nih.gov/

National Institute of Diabetes and Digestive and Kidney Diseases

Deborah Lee Young-Hyman
Department of Pediatrics, Georgia Prevention Institute Georgia Health Sciences Universtiy, Augusta, GA, USA

Basic Information

National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) conducts and

supports research ranging from basic science to translational multidisciplinary studies regarding the *endocrine system***.** This consists of *a system of glands*, each of which *secretes a type of* hormone *into the bloodstream to regulate the body*. In addition, related organs such as the kidney, liver, and gonads are studied as secondary contributors of hormonal input to the endocrine system. An endocrine axis denotes the interrelationship of function between glands and organs caused by reciprocal hormonal and cellular functions, including the brain. Functions regulated by endocrine hormones range from growth and pubertal development to basic metabolism and brain-mediated behavior. The endocrine system has systemic effects on the body, and therefore, NIDDK funds and takes a leadership role in studies encompassing the endocrine system, related organ systems, hormonal dysregulation and chronic disease, lifestyle as it relates to prevention and disease course of metabolic diseases, proteinomics, genomics, and gene environment interactions (http://www2.niddk.nih.gov/AboutNIDDK/).

Major Impact on the Field

In addition to its broader mission, NIDDK is dedicated to the funding of projects which address health disparities. Minorities are disproportionately affected by endocrine disorders, in particular diabetes and obesity, and have poorer disease outcomes. Diabetes is the single largest cause of end-stage renal disease (ESRD). In order to address these multifactorial illnesses, the NIDDK is a proponent of multidisciplinary research, reflecting the bench to bedside application of basic research findings to clinical care. NIDDK has a history of funding large clinical trials and epidemiologic studies which have addressed issues such as disease development and progression (types 1 and 2 diabetes), primary and secondary prevention (type 2 diabetes), and secondary prevention (preserving the health of neonates in mothers with gestational diabetes).

Because treatment of endocrine disorders such as autoimmune and weight-related diabetes is based on individual behavior, the NIDDK has an emphasis on behavioral aspects of disease prevention and management and has funding mechanisms (R18, R34) which emphasize the inclusion of behavior change strategies as primary or secondary treatment of disease prevention and management. Clinical trials, in particular the Diabetes Prevention Program (DPP) and Preventing Type II Diabetes (STOPP-T2D), have had major behavioral components and have helped to establish the clinical superiority of behavior/lifestyle change over pharmacologic intervention in lifestyle-based disease development and treatment (http://www2.niddk.nih.gov/Research/ScientificAreas/Diabetes/Type2Diabetes/CTT2.htm). NIDDK also maintains a network of biobehavioral researchers who conduct studies regarding the prevention and early treatment of type 1 diabetes (http://www.diabetestrialnet.org/) and is dedicated to providing funding to train clinician-scientists across disciplines to carry out this work.

Content areas of current NIDDK-funded behavioral research include hormonal regulation of appetite; associations between inflammation, glycemia, and mood; insulin resistance, exercise, and cognition; psychiatric disorders and risk for diabetes; translation of effective care models to underserved populations; and health-care delivery channels to improve pre-disease risk factors such as obesity in children and adults. Applications for funding behavioral approaches are likely to be assigned to the Division of Diabetes, Endocrinology and Metabolic Diseases (DEM).

Cross-References

▶ National Institutes of Health

References and Readings

NIDDK home page. www.niddk.nih.gov/

National Institute of Mental Health

Jennifer Pellowski
Department of Psychology, University of Connecticut, Storrs, CT, USA

Basic Information

The National Institute of Mental Health (NIMH) is a part of the National Institutes of Health (NIH) which focuses on clinical research associated with mental illness and behavioral disorders through biological and behavioral perspectives. It focuses on the prevention, recovery, and cures of mental illness and is the nation's scientific leader in this area.

NIMH was conceived on July 3, 1946 when President Truman signed the National Mental Health Act. This act acknowledged the plight of World War II soldiers coming back from war with mental illnesses caused or exacerbated by the environmental stress of war. It also recognized that the incidence rate of mental illness in the general population was higher than originally thought. To address this need for psychiatric care, the National Institute of Mental Health was formally established on April 15, 1949.

Since the beginning, NIMH has been at the forefront of mental health research focusing especially on assessing psychiatric needs of the nation, using cutting-edge technologies to determine when, where, and how mental illness occurs and finding treatments to slow or cure these mental illnesses. It commenced this long history of research in the 1950s and 1960s, starting with the Mental Health Study Act of 1955. The purpose of the Mental Health Study Act was to access the mental health issues effecting the American public as well as what resources were available at that time to treat those suffering from illness. This resulted in the report *Action for Mental Illness* which called for a national program to address individual needs on specific mental health problems.

NIMH continued to make strides during the 1970s and 1980s with its focus on the use of cutting-edge biological techniques, technologies, and treatments to aid in attempts to answer the questions of how, when, and why mental illness happens and how to treat those who are suffering. These aims resulted in the creation of the PET scan to measure brain function as well as the use of lithium, a revolutionary treatment for mania.

A resolution later signed by President George H. W. Bush declared the 1990s "The Decade of the Brain," and NIMH continued to focus on the advances in imaging technologies as well as the interaction between brain, behavior, and the environment to gain more knowledge about specific mental health illnesses. Currently, NIMH works to fulfill four objectives aimed at further determining the causes of mental illness, charting the course of mental illness, developing interventions, and the dissemination of knowledge acquired through NIMH-supported research.

To fulfill these objectives, NIMH conducts and funds internal and external research studies. With a budget of upwards of $1.5 billion, NIMH provides many grants to independent researchers, as well as contracts through requests for applications (RFAs). It also provides training grants to pre- and postdoctoral students to encourage entry into biomedical and behavior fields of research.

In conjunction with the goals of obtaining new knowledge through research, NIMH also acts to disseminate this newly found knowledge. It accomplishes this through conferences and lectures open to the general public in addition to providing booklets, brochures, and fact sheets. It also supplies curriculum and educational tools to primary and secondary schools to aid in the teaching process.

National Institute of Mental Health
Parklawn Building, 15C-05
5600 Fishers Lane
Rockville, Maryland 20857

Major Impact on the Field

The National Institute of Mental Health is the nation's scientific leader in research on the causes, treatment, and prevention of mental illness. The NIMH acts to bridge the gap between

research and the larger community by providing leadership in the dissemination of information for clinicians, patients, policy makers, and the general public.

Cross-References

- ▶ Depression
- ▶ Dissemination
- ▶ National Institutes of Health
- ▶ Stress, Posttraumatic

References and Readings

National Institute of Mental Health. *A participant's guide to mental health clinical research*. U.S. Department of Health and Human Services.

National Institutes of Health. (1999, September). *National Institute of Mental Health: Important events in NIMH history*. Retrieved February 16, 2011, from NIH 1999 Almanac http://www.nih.gov/about/almanac/archive/1999/organization/nimh/history.html

National Institutes of Health. (2011, February 11). *About NIMH*. Retrieved February 16, 2011, from National Institute of Mental Health http://www.nimh.nih.gov/about/index.shtml

Walls, T. (2008, July 31). *The National Mental Health Act of 1946 and the establishment of NIMH: Ongoing challenges*. Retrieved February 16, 2011, from Scattergood Program for the Applied Ethics of Behavioral Health http://www.scattergoodethics.org/?q=node/1146

National Institute of Nursing Research

Barbara Resnick
School of Nursing, University of Maryland, Baltimore, MD, USA

Basic Information

The development of NINR has had a major impact on building nursing science as delineated by the many successes and the growing number of doctorally prepared nurses engaged in research. Specifically, NINR has had a great influence on genetics research with a focus on bench to bedside, palliative care and symptom management research, end-of-life research, and research in long-term care.

Major Impact on the Field

The major impact of NINR has been in the development of nurse researchers and nursing science in general.

Cross-References

- ▶ National Institutes of Health

References and Readings

National Institute of Nursing Research, Retrieved from www.ninr.nih.gov. Accessed March 2012.

National Institute of Nursing Research publication: Bringing science to life. Retrieved from http://www.ninr.nih.gov/NR/rdonlyres?BCDD9E7E-C6A5-4578-B5B5-333895F54AA5/0/NINR_History_Book_508.pdf. Accessed March 2012.

National Institute on Aging

Natalie E. Bustillo
Department of Psychology, University of Miami, Coral Gables, FL, USA

Basic Information

The National Institute on Aging (NIA) is one of the institutes of the National Institutes of Health (NIH). The mission of the NIA is to conduct research about the aging process, to promote understanding of aging, and to disseminate advances in aging research to the public. The NIA sponsors aging research through the extramural and intramural research programs (National Institute on Aging [NIA], 2009).

The extramural research program consists of aging studies conducted at public and private organizations, such as hospitals and universities. The four extramural research programs supported by the NIA are: Division of Aging Biology, Division of Behavioral and Social Research, Division of Neuroscience, and Division of Geriatrics and Clinical Gerontology. Research conducted by the Division of Aging Biology examines the physiological mechanisms involved in the aging process using humans and animals. Examination of the biological processes involved in aging aid in the understanding of how biochemical changes associated with aging may be risk factors for diseases later in life. The Division of Behavioral and Social Research seeks to understand the aging process at the individual and group level. Emphasis is placed on how individuals change over time and the impact it has on greater society. The Division of Neuroscience supports research related to increasing the understanding of how the nervous system is impacted by the effects of aging. Alzheimer's disease and other dementias are the main research areas in this division. Research studies supported by the Division of Geriatrics and Clinical Gerontology consist of three main areas, which include Geriatrics, Clinical Gerontology, and Clinical Trials. The focus of this division is to examine age-related health concerns and identify risk factors associated with age-related diseases. The four divisions of the extramural research program offer grants and training opportunities to further enhance the aging research field (NIA, 2011).

The intramural research program conducts studies in multiple NIH sites in Maryland. The focus of this research program is to examine the physiological age-related changes that occur throughout the lifespan in order to better understand the physiology of age-related diseases, such as Alzheimer's disease, atherosclerosis, and cancer. In addition to examining the physiology of age-related diseases, the intramural research program also aims to understand the predictors of positive health (NIA, 2010).

Major Impact on the Field

The NIA has made many initiatives in the field of behavioral medicine through their ongoing research studies, clinical trials, and published findings.

Clinical Trials

The NIA supports multiple clinical trials examining multiple age-related conditions. The various medical conditions funded by the NIA-funded clinical trials include atherosclerosis, diabetes mellitus, hypercholesterolemia, hypertension, inflammation, menopause, obesity, and osteoporosis. In addition, the NIA funds clinical research relevant to promoting health behavior and managing psychological stress.

For example, the NIA is committed to understanding more about Alzheimer's disease. One of the clinical trials funded by the NIA tested whether Pioglitazone, a drug traditionally used to treat type 2 diabetes, was effective at slowing the progression of Alzheimer's disease (Geldmaher, 2009). Similarly, the NIA funded a trial to evaluate the effectiveness of antioxidant treatments for patients diagnosed with Alzheimer's disease (Galasko, 2009). Additional clinical trials have been funded by the NIA to assess decision-making abilities of patients diagnosed with Alzheimer's disease and to identify Alzheimer's disease-related brain changes and predictors of memory loss (de Leon, 2009; Karlawish, 2009).

Publications

The NIA has published various resources available to the public that demonstrate its initiatives in the field of behavioral medicine. Published resources include information about the importance of engaging in positive health behaviors such as eating a healthy diet, quitting smoking, exercising, and sleeping well. The NIA has also published resources for issues related to depression, coping with death, memory loss, and sexuality in adulthood.

Cross-References

► Aging

References and Readings

de Leon, M. J. (2009). *PET changes in Alzheimer's disease (AD)*. Retrieved November 28, 2011, from http://clinicaltrials.gov/show/NCT00094913

Galasko, D. (2009). *Anti-oxidant treatment of Alzheimer's disease*. Retrieved November 28, 2011, from http://clinicaltrials.gov/show/NCT00117403

Geldmaher, D. (2009). *Pioglitazone in Alzheimer disease*. Retrieved November 28, 2011, from http://clinicaltrials.gov/show/NCT00982202

Karlawish, J. (2009). *Memory aid for informed consent in Alzheimer's research*. Retrieved November 28, 2011, from http://clinicaltrials.gov/show/NCT00105612

National Institute on Aging. (2009). About NIA. Retrieved November 28, 2011, from http://www.nia.nih.gov/AboutNIA/

National Institute on Aging. (2010). Welcome to the intramural research program. Retrieved November 28, 2011, from http://www.grc.nia.nih.gov/branches/osd/mission.htm

National Institute on Aging. (2011). Research programs (extramural). Retrieved November 28, 2011, from http://www.nia.nih.gov/ResearchInformation/ExtramuralPrograms/

National Institute on Alcohol Abuse and Alcoholism

Jennifer Pellowski
Department of Psychology, University of Connecticut, Storrs, CT, USA

Basic Information

The National Institute on Alcohol Abuse and Alcoholism (NIAAA) is part of the National Institutes of Health and focuses on research associated with the causes, treatment, and prevention of alcoholism. NIAAA became the leading federal agency to address alcoholism as a public health problem after the passage of the Comprehensive Alcohol Abuse and Alcoholism Prevention, Treatment, and Rehabilitation Act of 1970. Also referred to as the Hughes Act of 1970, this legislation established NIAAA as part of the National Institute of Mental Health (NIMH).

Since its conception, NIAAA has been at the forefront of alcohol-related research starting with its investigation of fetal alcohol spectrum disorder (FASD) at a time when many scientists and physicians doubted its existence. In 1977, it held the first national FASD research conference which highlighted epidemiological and clinical research and led to the issue of the first FASD government health advisory. Since then their research has expanded to include the genetics of alcoholism, benefits of alcohol consumption, neurological examinations of the impact of alcohol, and improving health care and treatment of those that suffer from alcohol abuse and alcoholism as well as other topics.

It conducts and funds internal and external research studies. NIAAA focuses on multidisciplinary efforts including epigenetics, neuroscience, public health, epidemiology, genetics, and public policy. It also acts to relay information to clinicians, patients, policymakers, and the general public in an accessible manner. Notable projects include Project MATCH and Project COMBINE which have developed new ways of approaching treatment and therapy for those suffering from alcoholism. Additional studies have also identified targeted populations, specifically pregnant women and youth, and subsequently focused research efforts to highlight specific needs of these populations to increase prevention. In addition to internal research, NIAAA also provides financial and other forms of support to researchers through requests for applications (RFAs) and program announcements (PAs) as well as through other small grants.

In conjunction with the goal of conducting and supporting research, the NIAAA also works to disseminate this newly found information. It accomplishes this goal through Alcohol Alert, which is published quarterly and aimed at professionals and clinicians. Additionally, the NIAAA also provides information to the general public through pamphlets and brochures as well as educational training programs. NIAAA also sponsors several public health programs that range in topics and targeted populations from pregnant women to youth and young adults.

National Institute on Alcohol Abuse and Alcoholism
5635 Fishers Lane, MSC 9304
Bethesda, MD 20892-9304
URL: http://www.niaaa.nih.gov

Major Impact on the Field

The National Institute on Alcohol Abuse and Alcoholism is the largest funder of research concerning alcoholism and alcohol-related problems in the United States. It is a keystone of alcohol abuse and alcoholism research and provides leadership in the dissemination of information for clinicians, patients, and the general public. The NIAAA acts to bridge the gap between research and the community at large through educational programs and changes in public policy.

Cross-References

- ▶ Alcohol Abuse and Dependence
- ▶ Alcohol Consumption
- ▶ Binge Drinking
- ▶ National Institutes of Health

References and Readings

National Institute on Alcohol Abuse and Alcoholism. (2011). *About NIAAA*. Retrieved February 14, 2011, from National Institute on Alcohol Abuse and Alcoholism: http://www.niaaa.nih.gov/AboutNIAAA/Pages/default.aspx

National Institutes of Health Office of the Director. (1998). *National Institute on Alcohol Abuse and Alcoholism: Important events in NIAAA history*. Retrieved February 14, 2011, from NIH Almanac 1998: http://www.nih.gov/about/almanac/archive/1998/organization/niaaa/history.html

Thomas, J. D., Warren, K. R., & Hewitt, B. G. (2010). Fetal alcohol spectrum disorders: From research to policy. *Alcohol Research and Health, 33*, 118–126.

Warren, K. R., & Hewitt, B. G. (2010). NIAAA: Advancing alcohol research for 40 years. *Alcohol Research and Health, 33*, 5–17.

National Institutes of Health

Vaughn Bryant[1] and Anne Frankel[2]
[1]Behavioral and Social Sciences, Brown University, Providence, RI, USA
[2]Robert Stempel College of Public Health and Social Work, Florida International University, Miami, FL, USA

Basic Information

Overview

The National Institutes of Health (NIH) is an agency of the United States Department of Health and Human Services. It consists of the Office of the Director (OD) and 27 institutes and centers which make up the largest source of medical and scientific funding in the world (United States Department of Health and Human Services & National Institutes of Health, 2010). The main function of the OD is to "plan, manage, and coordinate" the specific policies and procedures laid out for these institutes (United States Department of Health and Human Services, 2010a). The policies and procedures are developed and implemented through extensive collaboration and input from the OD and NIH staff. Other sources of information include the extramural scientific community, patient advocacy and volunteer organizations, Congress, and the Director's Council of Public Representatives, a federal committee made up of members of the general public. The current director of the NIH is Francis S. Collins. Dr. Collins' job is to efficiently and effectively integrate the information from his advisors and provide direction for specific "Areas of Research Emphasis," the future goals and directions that the NIH identifies as beneficial to science and society. Themes that are to be addressed in the coming years include translational medicine, health-care reform, global health, and empowering and energizing the research community (United States Department of Health and Human Services, 2010a).

Application Process

Sustained funding is a necessary component for advancing science. In order to facilitate innovative projects that support scientific progress, the NIH funds grants, cooperative agreements, and contracts (United States Department of Health and Human Services, 2010b). The NIH lists several key factors in being awarded funding: (1) the project must be of high scientific caliber, (2) it must be investigator-initiated, and (3) the research must be unique and innovative. In order to provide guidance for funding opportunities, NIH notifies researchers of the availability of funds through Funding Opportunity Announcements (FOAs), which are posted in the NIH Guide for Grants and Contracts and on Grants.gov. Other sources of funding information include Parent Announcements, Program Announcements (PAs), and Requests for Applications (RFAs). The NIH also supports research centers and intramural research (United States Department of Health and Human Services, 2010b; United States Department of Health and Human Services & National Institutes of Health: Office of Extramural Research, 2009).

Funding opportunities exist for a variety of research structures, including individuals, domestic institutions, and foreign institutions. Further, the NIH seeks to fund investigators at various points in their scientific careers. For example, special programs and announcements have been created targeting young and early stage investigators in order to help this population establish their career and ultimately flourish in the realm of scientific research. More senior investigators may choose to be mentors for early stage investigators and directors of projects or institutes (United States Department of Health and Human Services, 2010b).

Historically, it has been considered difficult to be awarded an NIH grant. However, competition is critical for improving the level of applications and ultimately the caliber of scholarly research. In order to determine which grants are awarded funding, applications typically go through a rigorous process known as peer review. The peer review process involves multiple readings of each application by credentialed peers in the relevant field and helps to establish the quality of the grants funded. The NIH lists five major review criteria looked at by reviewers when examining and scoring applications: the significance of the project; investigator background; level of innovation, approach, and methodology; proposed environment for the study; and probability of success (United States Department of Health and Human Services & National Institutes of Health: Office of Extramural Research, 2011). Before reviewers meet, they give each application a preliminary score on a scale from 1 (exceptional) to 9 (poor). Applications which receive a score above a predetermined threshold score progress to a full committee meeting. At this meeting, reviewers discuss the strengths and weaknesses of each application and create an impact/priority score, which determines the percentile ranking that will organize applications for possible funding. Program officers then determine how review scores are translated into funding for a certain number of applications. Often, applications are submitted twice: if an initial application is not granted review, comments are provided to the applicant, and they are encouraged to submit an amendment. Grants are reviewed both by the center for the scientific review and the institutes themselves for special initiatives. Special review processes only allow for one submission (United States Department of Health and Human Services & National Institutes of Health: Office of Extramural Research, 2011).

Funding Success

Funding success is an important measure of the level of competition. Success rates are reported as the percentage of reviewed grant applications that receive funding, which is determined by dividing the number of competing applications funded by the sum of the total number of competing applications reviewed and the number of funded carry-overs (United States Department of Health and Human Service & National Institutes of Health, 2009). Success rates are computed on a fiscal year basis and include applications that are peer-reviewed and either scored or unscored

by an Initial Review Group. Applications that have one or more amendments in the same fiscal year are only counted once. Funding success rates are typically between 10% and 20% (United States Department of Health and Human Services & National Institutes of Health: Office of Extramural Research, 2011; United States Department of Health and Human Service & National Institutes of Health, 2009; United States Department of Health and Human Services, 2010c). In 2010, there was an overall success rate of approximately one in five grants, which accounts for both new investigators and established investigators, who may be more likely to get funding based on past awarded grants. Thus, this measure may not be representative of the level of competition for new or less established investigators. After peer review, successful grantees go through the award process in which the grantee is notified of an award and assigned a program officer who manages the distribution and monitoring of the award. Grant awards can range from a few thousand to tens of millions of dollars and depend on numerous factors including the type of grant (known at NIH as the "grant mechanism"), the institution, the structure of the proposal (individual vs. center), the period of time that the award is assigned, and many more. The oldest and most common mechanism applied for is the R01, which cannot exceed $250,000 annually in direct costs, unless a special request is made (United States Department of Health and Human Services & National Institutes of Health, 2010). Other common grant types are K mechanisms, which represent training grants, fellowships, and early stage investigator-initiated research; U mechanisms, which are cooperative agreements; and R21 exploratory grants.

Funding and Scientific Accomplishments

Funding of the NIH has been integral to the progression of innovative scientific discovery. President Obama recently released his 2012 budget proposal, in which he requested a 745 million dollar increase to the NIH budget. When added to the existing budget, the total request for the NIH is 32 billion dollars. Considering the 3.5% rate of inflation, the proposed amount is roughly equivalent to flat funding (Brown, 2011).

The latest scientific accomplishments of NIH-funded projects were highlighted in a recent status report (United States Department of Health and Human Services, 2011). Many of these accomplishments have been in the area of HIV/AIDS, with the development of antiretrovirals (ARVs) to treat the disease as one of the most notable. One large-scale study involved an ARV microbicide for women and found a 50% protection rate against HIV transmission. Future research focuses for the NIH in the field of HIV/AIDS will be on vaccine development and efficacy.

Advances in technology are key to NIH's research success. For example, because of NIH-sponsored research, high-resolution imaging is becoming a critical part of studying disease and disease progression. Findings of a study conducted by the National Lung Society suggest that low-dose CT screening can decrease lung-cancer deaths by 20% among heavy smokers. Another technological innovation becoming increasingly important in the treatment of disease is stem cells. Researchers recently found that mouse embryonic and induced pluripotent stem cells can be used to generate new hair cells, which has implications for hearing loss and deafness in humans. Finally, influenza vaccines have been improved to protect against multiple strains of the virus, which has helped science progress toward the goal of eradicating the virus worldwide.

Future Changes and Directions

There are many areas in the health sciences that are under-researched, including pain control, drug delivery, vaccines, medical informatics, and more that may lead to future initiatives and structural changes. The NIH is currently developing translational research initiatives by creating a clinical and translational research institute, which would help facilitate the process of translating clinical findings to practical applications such as drug development and other health improvements. Another example of a structural change to the NIH is the recent vote by the Scientific Management Review Board to merge the National Institute on Drug Abuse (NIDA) and the National Institute on Alcohol Abuse and Alcoholism (NIAAA) (Mcmanus, 2010). These proposals indicate that the NIH is

responsive to changes in science and attempts to accommodate these changes through reorganization and constant refinement both at the structural level and the scientific level.

Major Impact on the Field

The NIH is the largest source of funding for medical research in the world. Major contributions to various scientific fields linked directly to NIH funding cannot be understated. For example, the increase in lifespan in the United States from 47 to 78 between 1900 and 2009 has been attributed to the long history of NIH funding (United States Department of Health and Human Services & National Institutes of Health, 2010). Diseases such as smallpox, measles, polio, and chronic conditions such as diabetes and cancer, which were once death sentences, are now eradicated, curable, preventable, or treatable. In fact, rates of cancer diagnoses and cancer-related deaths have seen significant drops in recent years (United States Department of Health and Human Services, 2008). The field of mental health has seen advances, including the development of more effective antidepressants as well as improvements in the overall treatment for mental health and substance use disorders. The Human Genome Project, an ambitious campaign that began in 1990 and involved researchers from all over the world dedicated to decoding the mysteries of the human genetic code, resulted in the sequencing of the human genome in 2003. This enormous effort has given researchers unique insight into more than 1,800 disease genes and has played an invaluable role in the development of genetic tests (United States Department of Health and Human Services & National Institutes of Health, 2011). Prevention research has made immense strides due to NIH funding and has resulted in a better understanding of measures that can be taken to reduce the risk of heart disease, stroke, dementia, and many other adverse health conditions.

More than 80 Nobel Prizes have been presented to NIH-funded projects. However, scientific advances resulting from NIH-supported projects extend well beyond the ones receiving special recognition, and often the true impact of such projects cannot be gauged until long after their completion. Additionally, despite major cuts to NIH funding, the latest advances in comparative effectiveness research offer a better understanding of how to best utilize and distribute funds to maximize the long-term impact that projects will be able to have in their respective fields. Each day, NIH-funded projects address the challenges of a changing health climate facing chronic disease, the resurgence of old diseases, and emergence of new, and progress toward a healthier society both across the United States and worldwide.

Cross-References

- ▶ National Cancer Institute
- ▶ National Institute of Diabetes and Digestive and Kidney Diseases

References and Readings

Brown, D., (2011). *Budget 2012 and CDC*. [Updated February 14, 2011; cited April 25, 2011] Available from http://voices.washingtonpost.com/44/2011/02/budget-2012-nih-and-cdc.html

Mcmanus, R. (2010). Board recommends merger of NIDA, NIAAA. *NIH Record*, *62*(20). [Updated October 1, 2010; cited April 22, 2011] Available from http://nihrecord.od.nih.gov/newsletters/2010/10_01_2010/story1.htm

NIH home page. www.nih.gov/

United States Department of Health and Human Service & National Institutes of Health. (2009). *NIH success rate definition*. Bethesda, MD. [Updated February, 2009 cited April 27, 2011]. Available from: http://report.nih.gov/UploadDocs/NIH_Success_Rate_Definition.pdf

United States Department of Health and Human Services. (2010a). *Office of the director, major components*. Bethesda, MD: National Institutes of Health. [Updated October 26, 2010; cited February 26, 2011]. Available from http://www.nih.gov/icd/od/offices.htm

United States Department of Health and Human Services. (2010b). *Grant application basics*. Bethesda, MD: National Institutes of Health. [Updated Nov 16, 2010; Cited Feb 26, 2011]. Available from http://grants.nih.gov/grants/grant_basics.htm

United States Department of Health and Human Services. (2010c). *National Institutes of Health: Research portfolio online reporting tools*. Bethesda, MD. [Updated December 14, 2010; cited April 28, 2011].

Available from http://report.nih.gov/award/success/Success_ByIC.cfm
United States Department of Health and Human Services. (2011). *National Institutes of Health overview by institute*. [Cited April 25, 2011] Available from http://officeofbudget.od.nih.gov/pdfs/FY12/COPY%20of%20NIH%20BIB%20Chapter%202-9-11-%20FINAL.PDF
United States Department of Health and Human Services & National Institutes of Health: Office of Extramural Research. (2009). *Description of the NIH guide for grants and contracts*. Bethesda, MD. [Updated 2009 June 26; cited 2011 April 25]. Available from http://grants.nih.gov/grants/guide/description.htm
United States Department of Health and Human Services, National Institutes of Health, & National Cancer Institute. (2008). *NCI Cancer Bulletin*, *5*(8). [Updated December 2, 2008; cited August 20, 2011] Available from http://www.cancer.gov/aboutnci/ncicancerbulletin/archive/2008/120208/page10
United States Department of Health and Human Services & National Institutes of Health. (2010). *About the National Institutes of Health*. Bethesda, MD. [Updated October 27, 2010; cited February 26, 2011]. Available from http://www.nih.gov/about
United States Department of Health and Human Services & National Institutes of Health. (2011). *NIH fact sheets: Human genome project*. [Cited 2011 August 24] Available from http://report.nih.gov/NIHfactsheets/ViewFactSheet.aspx?csid=45
United States Department of Health and Human Services & National Institutes of Health. *Types of grant programs*. (2010). [Updated February 18, 2010; cited April 28, 2011]. Available from http://grants.nih.gov/grants/funding/funding_program.htm
United States Department of Health and Human Services & National Institutes of Health: Office of Extramural Research. (2011). *Peer review process*. Bethesda, MD. [Updated April 14, 2011, cited April 25, 2011]. Available from http://grants.nih.gov/grants/peer_review_process.htm
Wadman, M., (2010, August 12). One year at the helm. *Nature*, *466*. [Cited February 26, 2011]. Available from http://www.nih.gov/about/director/articles/nature_08122010.pdf

National Occupational Classification

▶ Job Classification

National Statistics Socioeconomic Classification

▶ Job Classification

Natural Killer Cell Activity

Riyad Khanfer[1], Benjamin I. Felleman[2] and G. Alan Marlatt[3]
[1]School of Sport and Exercise Sciences, The University of Birmingham, Edgbaston, Birmingham, UK
[2]Seattle Pacific University, Seattle, Washington, USA
[3]University of Washington, Seattle, Washington, USA

Definition

Natural killer (NK cells) form an important arm of the innate immune system (non specific immunity). They develop in the bone marrow and circulate in the blood. They are called NK cells because they are always active naturally and can kill before they encounter any antigens. They have important roles in eliminating virally infected cells without having specificity against any particular viruses and act as early component of the host response to viral infections. NK cells also play role in fighting cancer cells in the body. NK cells are considered part of the group of lymphocytes; however, they have neither antigen receptor nor antibody receptor, and they are larger in size than T and B lymphocytes.

Upon NK cells activation, they produce cytotoxic granules which contain many molecules like perforins, TNF-α, lymphotoxin-β, INF-α, and many granzymes. NK cells kill virally infected cells by secreting these cytotoxic granules after recognizing the infected cells by lacking MHC molecule (major histocompatability complex) class I, which is not present in the virally infected cells. Also, these granules contain other molecules like TNF (tumor necrosis factor) which can activate apoptosis through death domain of TNF receptor of the target cell; also, NK cells kill by apoptosis via Fas ligand – Fas receptor mechanism upon engagement of the NK cell receptor with the target cell receptor.

NK cells have a mechanism of checking cells by detecting the amount of MHC I protein on the cell

surface; cells which express enough amount of MHC I will escape the killing by NK cells, and cells with reduced amount of MHC I will be targeted and killed by NK cells via programmed cell death after targeted by the cytotoxic granules. NK cells can be activated in response to interferons and macrophage-derived cytotoxins.

NK cells secrete cytokines such as interferon γ and interleukin 12 which help in CD4 T lymphocytes differentiation. NK cells are among the first to encounter cancer cells of the innate immunity components, and they secrete interferon γ which recruit other component of the immune system.

To control their cytotoxic activities, NK cells have two types of receptors on their surface: the first set are activating receptors that induce killing by NK cells and called KAR (killer activating receptors), while the second set of receptors are inhibitory which inhibit NK cells activation and therefore prevent killing to the normal host cells (killer inhibitory receptors KIR), and the KIRs are specific for MHC class I molecule.

Also, NK cells kill cancer cells as well as viral-infected cells by detecting an important specific protein expressed often at the surface of these cells as a result of cellular physiological stresses, such as postneoplastic transformation or viral infection. NK cells express complementary cell surface receptor for the above stress proteins which strongly activate NK cells and the production of the NK cell cytotoxic granules.

Summary: *Specific features of NK cells function and killing*
Part of innate immunity
Kill cancer and virus-infected cells
Killing without specificity
Have two types of receptors: KAR to kill and KIR to avoid killing
Secrete some cytokines like IL12 and IF-γ
Produce cytotoxic molecules to kill target cells by apoptosis
Screen target cells by lack of MHC I expression
Recognize stress proteins on physiologically stressed cells

Cross-References

- ▶ Immune Function
- ▶ Macrophages

References and Readings

Delves, P. J., Martin, S. J., Burton, D. R., & Roitt, I. M. (2006). *Roitt's essential immunology* (11th ed.). Malden, MA: Blackwell.

Janeway, C. A., Travers, P., Walport, M., & Shlomchik, M. J. (2005). *Immunobiology* (6th ed.). New York: London Garland Science.

Levinson, W. (2006). *Review of medical microbiology and immunology* (9th ed.). New York: McGraw-Hill Medical.

Weinberg, R. (2007). *The biology of cancer*. New York: Garland Science.

NCS

- ▶ National Children's Study

Needle Exchange Programs

Hansel Tookes
Epidemiology and Public Health, Miller School of Medicine, University of Miami, Miami, FL, USA

Synonyms

NEPs; SEPs; Syringe exchange programs

Definition

In 2006, injection drug users (IDUs) accounted for 12% of the 56,300 new human immunodeficiency virus (HIV) infections in the USA (Hall et al., 2008). In 2007, 15% of the 43,000 new hepatitis B virus (HBV) infections and 44% of the 17,000 new hepatitis C virus (HCV) infections in the USA were among IDUs (Guardino et al., 2010). IDUs are susceptible to infection via sharing injection equipment and high-risk sexual behavior. While the National Institute on Drug Abuse (NIDA) recommends use of a new, sterile syringe each injection, many IDUs contract these viral infections through the sharing of

contaminated syringes. In response to these viral epidemics, syringe exchange programs (SEPs) have been implemented in many countries including the United States (USA) (Mathers et al., 2010). These programs allow IDUs to exchange contaminated syringes for sterile syringes, with the goal of reducing the likelihood of IDUs' sharing used syringes.

The first SEPs in the USA opened in the late 1980s in Tacoma, Washington, followed shortly by programs in San Francisco, California; Portland, Oregon; and New York, New York (Hagan, Des Jarlais, Purchase, Reid, & Friedman, 1991). The North American Syringe Exchange Network (NASEN) estimates that there are currently 184 SEPs operating in the USA (Guardino et al., 2010). In December of 2009, President Barack Obama signed a bill authorizing the use of federal dollars for SEPs. This signing marked the end of a 21-year-long Congressional ban on SEPs instituted in 1988 at the height of the AIDS epidemic (Kerlikowske & Crowley, 2010).

A 1997 Report to Congress by the then Secretary of Health and Human Services Donna Shalala established SEPs as an effective component of a comprehensive strategy to prevent HIV and other blood-borne infectious diseases (Shalala, 1997). Many studies have shown that SEPs help to reduce the sharing of syringes among IDUs, and IDUs have reported no increased unsafe disposal in areas where SEPs were providing more syringe coverage (Bluthenthal, Anderson, Flynn, & Kral, 2007; Neaigus et al., 2008; Watters, Estilo, Cark, & Lorvick, 1994). Further, SEPs provide a range of preventive care services. Most SEPs services include HIV/AIDS counseling and testing, HCV counseling and testing, condom distribution, referral to substance abuse treatment, alcohol swabs, and safe-injection education. Additional services are often available including primary medical care, tuberculosis screening, and comprehensive hepatitis screening services including HAV and HBV vaccination (Guardino et al., 2010).

SEPs help refer IDUs to treatment and do not encourage drug use among IDUs or recruit new users (Vlahov & Junge 1998). SEPs also help IDUs use sterile syringes and share less, reducing risk of blood-borne illness (Des Jarlais et al., 1994; Heimer, Khoshnood, Bigg, Guydish, & Junge, 1998). HIV incidence among IDUs has decreased by 80% from before SEPs (1988–1990) to their maximum coverage (2003–2006) (Hall et al., 2008). These data are of high public health significance because in 2010, the Departments of State and Health and Human Services have issued policy guidance for US and global partners in the President's Emergency Plan for AIDS Relief (PEPFAR) in the implementation of SEPs, but SEP coverage has yet to increase domestically. Despite the evidence, IDUs have limited coverage with SEPs offered in only 36 states, the District of Colombia, and Puerto Rico (Guardino et al., 2010). However, in 2011, the Centers for Disease Control and Prevention will issue guidance on SEPs to all US Health Departments, which will include recommendations relevant to syringe exchange programs.

Cross-References

- ▶ Centers for Disease Control and Prevention
- ▶ HIV Infection
- ▶ HIV Prevention

References and Readings

Bluthenthal, R. N., Anderson, R., Flynn, N. M., & Kral, A. H. (2007). Higher syringe coverage is associated with lower odds of HIV risk and does not increase unsafe disposal among syringe exchange program clients. *Drug and Alcohol Dependence, 89*(2–3), 214–222.

Daniels, D., Grytdal, S., & Wasley, A. (2009). Surveillance for acute viral hepatitis-United States, 2007. *MMWR, 58*(SS-3), 1.

Des Jarlais, D. C., Freidman, S. R., Sotrheran, J. L., Wenston, J., Marmor, M., Yancovitz, S. R., et al. (1994). Continuity and change within an HIV epidemic: Injection drug users in New York City, 1984–1992. *JAMA: The Journal of the American Medical Association, 271*, 121–127.

Guardino, V., Des Jarlais, D. C., Arasteh, K., Johnston, R., Purchase, D., Solberg, A., Lansky, A., & Lentine, D. (2010). Syringe exchange programs—United States, 2008. MMWR 59, 1488–1491. Accessed August 12, 2010, from http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5945a4.htm

Hagan, H., Des Jarlais, D. C., Purchase, D., Reid, T., & Friedman, S. R. (1991). The Tacoma syringe exchange. *Journal of Addictive Diseases, 10*(4), 81–88.
Hall, H. I., Song, R., Rhodes, P., et al. (2008). Estimation of HIV Incidence in the United States. *JAMA: The Journal of the American Medical Association, 300*, 520–529.
Heimer, R., Khoshnood, K., Bigg, D., Guydish, J., & Junge, B. (1998). Syringe use and reuse: Effect of needle exchange programs in three cities. *JAIDS, 18* (Suppl. 1), AS37–AS44.
Kerlikowske, G., & Crowley, J. S. (2010). *Expanding access to evidence-based services for injection drug users*. Accessed July 26, 2010, from www.Whitehouse.gov (Posted 7.16.10).
Mathers, B. M., Degenhardt, L., Ali, H., Wiessing, L., Hickman, M., Matick, R. P., et al. (2010). HIV prevention, treatment and care services for people who inject drugs: A systematic review of global, regional and national coverage. *Lancet, 375*(9719), 1014–1028.
Neaigus, A., Zhao, M., Gyarmathy, V. A., Cisek, L., Friedman, S. R., & Baxter, R. C. (2008). Greater drug injecting risk for HIV, HBV and HCV infection in a city where syringe exchange and pharmacy syringe distribution are illegal. *Journal of Urban Health, 85*(3), 309–322.
Shalala, DE. (1997, February 18). *Needle exchange programs in America: Review of published studies and ongoing research*. Report to the Committee on Appropriations for the Departments of Labor, Health and Human Services, Educations and Related Agencies.
Vlahov, D., & Junge, B. (1998). The role of needle exchange programs in HIV prevention. *Public Health Reports, 113*(Suppl. 1), 75–80.
Watters, J. K., Estilo, M. J., Cark, G. L., & Lorvick, J. (1994). Syringe and needle exchange as HIV/AIDS prevention for injection drug users. *JAMA: The Journal of the American Medical Association, 271*(2), 115–120.

Negative Affect

Deborah M. Stringer
Department of Psychology, University of Iowa, Iowa City, IA, USA

Synonyms

Emotional distress; Negative emotion

Definition

Negative affect is a broad concept that can be summarized as feelings of emotional distress (Watson, Clark, & Tellegen, 1988); more specifically, it is a construct that is defined by the common variance between anxiety, sadness, fear, anger, guilt and shame, irritability, and other unpleasant emotions. A variety of converging evidence suggests that negative affect is largely statistically independent from positive affect (e.g., Watson, 1988), but it is also clear that there exists a dimension called pleasantness-unpleasantness that has relations to both negative and positive mood terms (e.g., happiness and sadness). Some workers (e.g., Russell & Carroll, 1999) take the existence of the bipolar pleasantness-unpleasantness factor as evidence that negative affect and positive affect form a single dimension.

Negative affect and the dispositional tendency toward negative affect (called neuroticism, negative affectivity, or negative emotionality) are a large component of many forms of psychopathology including mood disorders, anxiety disorders, and personality disorders. Negative affect has also been associated with such important areas in behavioral medicine as coping with illness or caregiving, blood pressure and heart rate, perceived stress, drinking motives, tobacco smoking, drug use, disordered eating, self-reported sleep quality, and cardiovascular disease.

Cross-References

- Negative Affectivity
- Negative Thoughts
- Neuroticism
- Positive Affect
- Positive Affect Negative Affect Scale (PANAS)
- Stress

References and Readings

Russell, J. A., & Carroll, J. M. (1999). On the bipolarity of positive and negative affect. *Psychological Bulletin, 125*, 3–30. doi:10.1037//0033-2909.125.1.3.
Watson, D. (1988). The vicissitudes of mood measurement: Effects of varying descriptors, time frames, and

response formats on measures of positive and negative affect. *Journal of Personality and Social Psychology, 55*, 128–141. doi:10.1037//0022-3514.55.1.128.
Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*, 1063–1070. doi:10.1037//0022-3514.54.6.1063.

Negative Affectivity

Johan Denollet
CoRPS – Center of Research on Psychology in Somatic diseases, Tilburg University, Tilburg, The Netherlands

Synonyms

Neuroticism

Definition

Negative affectivity (NA) is a broad personality trait that refers to *the stable tendency to experience negative emotions* (Watson & Clark, 1984). Individuals who are high in NA are more likely to report negative affective mood states across time and regardless of the situation. They also report more somatic symptoms and have an attention bias toward adverse stimuli or potentially threatening situations (Watson & Pennebaker, 1989).

NA is closely related to *neuroticism* (Costa & McCrae, 1987) as one of the broad trait domains in the Five Factor Model of personality. Because NA is centrally defined by the tendency to experience negative affect (Watson & Pennebaker, 1989), the label NA is used here to designate dysphoric individual differences that are relatively stable over time. From a cognitive point of view, individuals who are high in NA may take a gloomy view of things and are inclined to be worrying. From an affective point of view, symptoms of depressed mood are often accompanied by other negative emotions like anxiety and anger (Watson & Clark, 1984).

There are a number of reasons why it is important to account for individual differences in NA in clinical research and practice. First, *psychological risk factors tend to cluster together within individuals*; clustering of these factors, in turn, elevates the risk for adverse outcomes. NA is a personality trait that predisposes to clustering of negative emotions as a psychological risk factor (Denollet, 2005; Suls & Bunde, 2005). Second, individuals who are high in NA may *scan the world for signs of impending trouble* and seem to focus their attention on adverse stimuli (Watson & Pennebaker, 1989). This may explain why NA or neuroticism has been associated with more reactivity to stressful events and with more negative appraisals of interpersonal stressors. Third, NA may be related to *difficulties in coping with life stress* (Depue & Monroe, 1986) and is associated with an increased risk of affective disorder (Watson, Clark, & Harkness, 1994) and symptoms of both emotional and somatic distress (Watson & Pennebaker, 1989).

Some have argued that neuroticism or NA is associated with complaints of chest pain but not actual heart disease (Costa & McCrae, 1987). However, one should not overlook potential associations between NA and *decrements in physical health*. There is a substantial overlap among the various negative affective dispositions (anger, anxiety, and depression) that have been associated with an increased risk of cardiovascular disease. This suggests that a general disposition toward negative emotions – the core of NA – may be important as a potential determinant of adverse health outcomes (Denollet, 2005; Suls & Bunde, 2005).

Broad and stable personality traits such as NA may carry with them much potential for scientific and clinical purposes. NA taps into chronic attributes of individuals and has many referent attributes and therefore has much predictive and explanatory power. NA can be reliably *assessed with the 7-item NA measure of the DS14* (Denollet, 2005), a scale that was specifically designed to assess this stable tendency to experience negative emotions.

Cross-References

- ▶ Neuroticism
- ▶ Type D Personality

References and Readings

Costa, P. T., & McCrae, R. R. (1987). Neuroticism, somatic complaints, and disease: Is the bark worse than the bite? *Journal of Personality, 55*, 299–316.

Denollet, J. (1991). Negative affectivity and repressive coping: Pervasive influence on self-reported mood, health, and coronary-prone behavior. *Psychosomatic Medicine, 53*, 538–556.

Denollet, J. (2005). DS14: Standard assessment of negative affectivity, social inhibition, and type D personality. *Psychosomatic Medicine, 67*, 89–97.

Depue, R. A., & Monroe, S. M. (1986). Conceptualization and measurement of human disorder in life stress research: The problem of chronic disturbance. *Psychological Bulletin, 99*, 36–51.

Suls, J., & Bunde, J. (2005). Anger, anxiety, and depression as risk factors for cardiovascular disease: The problems and implications of overlapping affective dispositions. *Psychological Bulletin, 131*, 260–300.

Watson, D., & Clark, L. A. (1984). Negative affectivity: The disposition to experience aversive emotional states. *Psychological Bulletin, 96*, 465–490.

Watson, D., Clark, L. A., & Harkness, A. R. (1994). Structures of personality and their relevance to psychopathology. *Journal of Abnormal Psychology, 103*, 18–31.

Watson, D., & Pennebaker, J. W. (1989). Health complaints, stress, and distress: Exploring the central role of negative affectivity. *Psychological Reviews, 96*, 234–254.

Negative Cognitions

- ▶ Negative Thoughts

Negative Emotion

- ▶ Negative Affect

Negative Emotionality

- ▶ Neuroticism

Negative Relationship

- ▶ Inverse Relationship

Negative Religious Coping

- ▶ Religious Coping

Negative Social Interaction

- ▶ Social Conflict

Negative Thoughts

Louise C. Hawkley
Department of Psychology, University of Chicago, Chicago, IL, USA

Synonyms

Anger; Anxiety; Avoidance; Catastrophizing/Catastrophic thinking; Depression; Hostility; Loneliness; Negative affect; Negative cognitions; Neuroticism; Perceived stress; Pessimism; Rumination; Worry

Definition

Negative thoughts are cognitions about the self, others, or the world in general that are characterized by negative perceptions, expectations, and attributions and are associated with unpleasant emotions and adverse behavioral, physiological, and health outcomes.

Negative thoughts are cognitive components of negative psychosocial variables such as depressive symptoms, anxiety, loneliness, and hostility. Depressive cognitions, for instance, include thoughts of hopelessness, helplessness,

and diminished self-worth. Anxiety is a negative affective state or trait that is accompanied and perpetuated by worry and rumination. Loneliness is a negative state or trait that is characterized by perceptions of social threat, negative expectations in social interactions, and lower self-esteem. Hostility is characterized by derogation of others and thoughts of aggression and revenge. Negative thoughts are often intrusive and unwanted and require effort to suppress.

Negative thoughts are assumed to play a major role, together with related negative emotions, in the associations of negative psychosocial variables with poorer health behaviors, greater physiological reactivity, poorer health, and higher rates of mortality. Negative thoughts tend to co-occur with each other, and their combined contributions may exceed those of any particular negative thought. Alternatively, specific negative thoughts may exert unique effects on health-related outcomes. The effects of loneliness on systolic blood pressure, for example, are independent of the effects of negative thoughts related to perceived stress, depressive symptoms, poor social support, and hostility (Hawkley, Thisted, Masi, & Cacioppo, 2010). On the other hand, worrying and pessimism tend to be correlated, and each has shown a positive prospective association with risk for coronary heart disease (Kubzansky et al., 1997; Kubzansky, Sparrow, Vokonas, & Kawachi, 2001), but it is not known whether the effects of worrying and pessimism on disease risk are independent of each other or confounded with each other. Mechanistic models of health risk employ negative thoughts as precursors to behaviors that have health consequences. For instance, low self-esteem has been shown to prospectively predict health problems through its effect on the quality of social bonds (Stinson et al., 2008).

Neuroticism is a personality trait that is defined as a propensity for negative thoughts and experiences of many kinds and has been associated with mortality (Shipley, Weiss, Der, Taylor, & Deary, 2007). Because neuroticism is associated with a variety of specific negative thoughts, studies sometimes employ neuroticism as a covariate to determine the extent to which health effects are attributable to a specific negative psychosocial variable or negative thought as opposed to negativity more generally.

Cross-References

- ▶ Happiness and Health
- ▶ Psychological Factors
- ▶ Psychosocial Predictors
- ▶ Psychosocial Variables
- ▶ Unipolar Depression

References and Readings

Hawkley, L. C., Thisted, R. A., Masi, C. M., & Cacioppo, J. T. (2010). Loneliness predicts increased blood pressure: Five-year cross-lagged analyses in middle-aged and older adults. *Psychology and Aging, 25*, 132–141.

Kubzansky, L. D., Kawachi, I., Spiro, A., III, Weiss, S. T., Vokonas, P. S., & Sparrow, D. (1997). Is worrying bad for your heart? A prospective study of worry and coronary heart disease in the normative aging study. *Circulation, 95*, 818–824.

Kubzansky, L. D., Sparrow, D., Vokonas, P., & Kawachi, I. (2001). Is the glass half empty or half full? A prospective study of optimism and coronary heart disease in the normative aging study. *Psychosomatic Medicine, 63*, 910–916.

Shipley, B. A., Weiss, A., Der, G., Taylor, M. D., & Deary, I. J. (2007). Neuroticism, extraversion, and mortality in the UK health and lifestyle survey: A 21-year prospective cohort study. *Psychosomatic Medicine, 69*, 923–931.

Stinson, D. A., Logel, C., Zanna, M. P., Holmes, J. G., Cameron, J. J., Wood, J. V., et al. (2008). The cost of lower self-esteem: Testing a self- and social-bonds model of health. *Journal of Personality and Social Psychology, 94*, 412–428.

Neighborhood-Level Studies

- ▶ Built Environment

Neoplasm of the Prostate

- ▶ Cancer, Prostate

NEPs

▶ Needle Exchange Programs

Nerve Cell

▶ Neuron

Nerve Damage

▶ Diabetic Neuropathy

Nested Case-Control Study

▶ Nested Study

Nested Study

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Nested case-control study

Definition

A nested case-control study is one that is "nested" within a cohort study.

In many cohort studies, all subjects provide a wide range of information at the time of recruitment, e.g., results from a physical examination, answers to multiple questionnaires, blood and urine samples, and results from imaging techniques. Because of the large numbers of subjects in these studies and the cost of analyzing some biological samples, some of these resources are often not analyzed in detail at the time of collection, but are stored for future use. The nested case-control study is performed using subjects who develop the disease of interest in due course, and control subjects who are selected from those who were disease-free at the time the case subjects (those who developed the disease) were diagnosed.

The appropriate data sets and samples are then retrieved and analyzed for these two subsets (cases and controls) of the original cohort recruited into the study. This approach maintains the major advantage of a cohort study in that the exposure data were collected before the development of the disease in the case subjects, while requiring the analysis (and associated costs) for only those who become entered into the nested case-control study (Webb, Bain, & Pirozzo, 2005).

Cross-References

▶ Case-Control Studies
▶ Cohort Study

References and Readings

Webb, P., Bain, C., & Pirozzo, S. (2005). *Essential epidemiology: An introduction for students and health professionals*. Cambridge, UK: Cambridge University Press.

Neurobehavioral Assessment

▶ Neuropsychology

Neurocognitive Assessment

▶ Neuropsychology

Neuroendocrine Activation

Wiebke Arlt and Ana Vitlic
School of Sport & Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

Synonyms

Flight-or-fight response; Stress cascade; Stress response

Definition

Neuroendocrine activation is activation of both neuronal and endocrine pathways in situations when either internal or external factors act in a way that disturbs the body's homeostasis.

Description

From its very origin, life has been exposed to a highly dynamic environment. In order to survive, everything, from the simple primordial ribonucleic acid (RNA) molecules to the highly structured organisms that exist in the present world, had to learn how to fight these changes and eventually benefit from them. Nevertheless, no matter how beneficial these changes proved to be in the end, first encountered, they usually provoked a physiological response, commonly known as stress. Often, very hostile environments forced more complex organisms, like vertebrates, to develop a response that Walter Cannon (1914) first introduced as a "fight-or-flight" response, which describes a number of physiological changes involved in regulation of the body's response to stimuli. This response aims to maintain body homeostasis, the mechanism that keeps internal environment in the body as constant and balanced as possible (Pacák & Palkovits, 2001).

One of the key features in this adaptive response is neuroendocrine activation (Miller & O'Callaghan, 2002), which includes activation of two systems, the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system (SNS). Neuroendocrine activation can be triggered in two ways: as a feedback signal from peripheral receptors that have recognized the altered homeostasis or by the direct simulation from an activated brain regulatory center (Vigas & Jezová, 1996). Even though different stressors activate different pathways and circuits within an organism, they will always result in the series of neural and endocrine adaptations known as the stress cascade (Miller & O'Callaghan, 2002). The key site involved in neuroendocrine activation is the hypothalamus (Barrett, 2005). Activation of both HPA axis and SNS starts from this very small but extremely important part of diencephalon. The hypothalamus consists of large number of nuclei and fiber tracts situated in three prominent features: supraoptic (anterior) region, tuberal or middle region, and mammillary or posterior region. From its posterolateral nuclei, the hypothalamus establishes strong, direct connections with autonomic nuclei in the brain stem and spinal cord. In this way, this part of central nervous system (CNS) structure controls SNS functions (Barrett). At the same time, and as a response to stress, the hypothalamus releases a neuropeptide called corticotrophin-releasing hormone (CRH) (Griffin & Ojeda, 2004), which stimulates the anterior pituitary gland to release another hormone, adrenocorticotropic hormone or ACTH, into general circulation. ACTH in turn stimulates the adrenal cortical cells of the adrenal glands to synthesize and release species-specific glucocorticoids into blood. The tight control of these glucocorticoids (mainly cortisol in humans) is sustained via negative feedback that controls and, in the end, terminates the release of CRH (Griffin & Ojeda). The necessity for this very subtle regulation of HPA axis at different levels is well represented by adverse health consequences caused by its dysregulation (Tsigos & Chrousos, 2002). For example, increased HPA axis activity and the hyperproduction of cortisol can lead to melancholic depression which can be accompanied by atherosclerosis, suppression of immune cells, infectious diseases, deposition of visceral

fat, and enhanced resistance to autoimmune/inflammatory disease (Tsigos & Chrousos). Hypo-production of cortisol, on the other hand, will lead to the state of the relative resistance to infectious but increased susceptibility to inflammatory and autoimmune diseases, and, in its more severe form, a serious condition known as Addison's disease (Griffin & Ojeda, 2004). Clearly, one of the very important consequences of HPA axis activation is immunosuppression (Nance & Meltzer, 2003) that includes changes in leukocyte trafficking and function, decreased production of inflammatory cytokines, and inhibition of their effects on target tissues. However, although glucocorticoids can express variable effects on different systems, resulting sometimes in activation and sometimes in their suppression, their primary goal is always to act as the guardians of body homeostasis (Munck, Guyre, & Holbrook, 1984). Therefore, it is not stress that glucocorticoids fight against, it is altered homeostasis that triggers their secretion and activation.

Stress triggers the activation of both pituitary gland and adrenal cortex, as well as specialized "neuroendocrine tissue" in the hypothalamus, but it can also act via sympathetic neurons responsible for presence of the circulating catecholamines, adrenaline, and noradrenaline, (epinephrine and norepinephrine) secreted from adrenal medulla (Barrett, 2005). This is known as the sympatho-adrenal-medullary axis or SAM axis/system. Stress can also cause an integrated response that originates entirely within the central nervous system, where activation of cortical and hypothalamic brain centers acts via postganglionic sympathetic neurons, innervating the smooth muscles of blood vessels, heart, skeletal muscles, kidney, gut, and other organs (Barrett). In other words, both short-term physiological changes (such as acute exercise) and chronic pathophysiological disorders (chronic heart failure) will lead to CNS-mediated SNS activation (Goldstein, 1987). SNS activation involves the release of noradrenaline at the end of the postganglionic neurons, but through its preganglionic fibers of splanchnic nerves, it also regulates hormone secretion from adrenal medulla (Barrett, 2005). The cells of this endocrine gland, chromaffin cells, synthesize and secrete adrenaline, and, to a lesser extent, noradrenaline. The presence of these catecholamines in the blood stream (e.g., in response to the exercise-induced stress) will lead to cognitive arousal, cardiac stimulation and vasoconstriction of peripheral blood vessels with preserved skeletal muscle blood flow, and relaxed bronchial smooth muscle, increasing oxygen delivery to the exercising muscle, and it will also affect gastrointestinal, renal, endocrine, and other systems (Barrett). On the other hand, a rise in plasma glucocorticoid levels activates different hormones, prostaglandins and other arachidonic acid metabolites, lymphokines, and bioactive peptides which in turn challenge homeostasis through different physiological mechanisms again affecting endocrine, renal, nervous, as well as the immune systems. However, unlike components of HPA axis, secretion of adrenal medullary hormones is not regulated by endocrine feedback loop, but it is controlled by CNS instead (Barrett).

In sum, activation of the autonomic and neuroendocrine system during stress has been developed as a protective mechanism in "flight-or-fight" situations where it serves to mobilize the metabolic resources necessary to support the requirements of the organism (Havel & Taborsky, 2003). The problem arises when neuroendocrine activation exceeds the body's metabolic requirements, typical in situations of chronic psychological stress, large amounts of caffeine uptake, as well as the pathological condition of chronic myocardial infarction. One example of this negative effect of neuroendocrine activation in humans today is stress-induced hyperglycemia (Havel & Taborsky). Clearly, neuroendocrine activation during stress through sympathoadrenomedullary system triggers the release of neuropeptides such as adrenaline and noradrenaline and, via activation of HPA axis, causes an increase in secretion of cortisol and the pancreatic hormone glucagon (Havel & Taborsky), with a simultaneous decrease in insulin production. As the pancreatic hormone glucagon raises blood glucose levels, and insulin acts as its antagonist, this neuroendocrine pattern will

cause an increase in hepatic glucose production in situations that demand increased muscular activity (Havel & Taborsky). In other words, increased physical activity present in the "flight-or-fight" response will increase the demand for the glucose consumption and effectively balance higher glucose production, therefore maintaining glucose plasma level (Havel & Taborsky). Among modern types of stress, exercise-induced stress is the only one that parallels somatic motor activation present during "flight-or-fight" response (Havel & Taborsky). Other common types of stress such as hypoxia, trauma, myocardial infarction, etc., will trigger neuroendocrine activation and secretion of glucose-mobilizing hormones without adequate glucose utilization and therefore will lead to the state of hyperglycemia (Havel & Taborsky).

Caffeine intake (Lane, Pieper, Phillips-Bute, Bryant, & Kuhn, 2002) can also mimic stress-induced activation of neuroendocrine system and lead to a rise in plasma levels of adrenaline, noradrenaline, as well as glucocorticoids such as cortisol. Similar to stress situations, it not only enhances cardiac output, diastolic blood pressure and heart rate, as well as skeletal muscle blood flow but can also potentiate already stress-induced increases in cardiac output and plasma concentration of adrenaline and cortisol (Lane et al., 2002).

One of the important protective roles of neuroendocrine activation is sustaining circulation in vital organs (such as heart, brain, kidney), in the situations of extensive volume loss, e.g., hemorrhage (Swedberg, 2002). In order to sustain its role, neuroendocrine activation causes tachycardia, constriction of peripheral blood vessels, and volume expansion. Unfortunately, what is beneficial in one situation can become detrimental in other pathological conditions such as chronic heart failure (Swedberg). Direct consequences of chronic neuroendocrine activation in this case are sodium and water retention that leads to congestion, vessel constrictions that will increase demand of oxygen, and toxic cellular effects, all of which combined together may cause cardiac dysfunction and higher morbidity and death in chronic heart failure patients (Swedberg).

Hence, neuroendocrine activation is an adaptive response to changing and demanding external and internal environment, and another example of the complex structure of living organisms, so carefully and subtly regulated that exposure to any new phenomenon, can turn this strong protector into a very dangerous enemy.

Cross-References

- ▶ ACTH
- ▶ Adrenal Glands
- ▶ Hypothalamus

References and Readings

Barrett, E. J. (2005). The adrenal gland. In W. F. Boron & E. L. Boulpaep (Eds.), *Medical physiology: A cellular and molecular approach* (pp. 1049–1065). Philadelphia: Elsevier.

Becker, K. L. (2001). *Principles and practice of endocrinology and metabolism*. Philadelphia: Lippincott Williams and Wilkins.

Cannon, W. B. (1914). The emergency function of the adrenal medulla in pain and the major emotions. *American Journal of Physiology, 33*, 356–372.

Goldstein, D. S. (1987). Stress-induced activation of the sympathetic nervous system. *Baillière's Clinical Endocrinology and Metabolism, 1*, 253–278.

Griffin, J. E., & Ojeda, S. R. (2004). *Textbook of endocrine physiology*. New York: Oxford University Press.

Havel, P. J., & Taborsky, G. J., Jr. (2003). Stress-induced activation of the neuroendocrine system and its effects on carbohydrate metabolism. In D. Porte, R. S. Sherwin, A. Baron, M. Ellenberg, & H. Rifkin (Eds.), *Ellenberg and Rifkin's diabetes mellitus* (pp. 129–150). New York: McGraw-Hill Professional.

Lane, J. D., Pieper, K. F., Phillips-Bute, B. G., Bryant, J. E., & Kuhn, C. M. (2002). Caffeine affects cardiovascular and neuroendocrine activation at work and home. *Psychosomatic Medicine, 64*, 593–603.

Miller, D. B., & O'Callaghan, J. P. (2002). Neuroendocrine aspects of the response to stress. *Metabolism, 51*, 5–10.

Munck, A., Guyre, P. M., & Holbrook, N. J. (1984). Physiological functions of glucocorticoids in stress and their relation to pharmacological actions. *Endocrine Reviews, 5*, 25–44.

Nance, D. M., & Meltzer, J. C. (2003). Interactions between the adrenergic and immune systems. In J. Brienenstock, E. J. Goetzel, & M. G. Blennerhassett (Eds.), *Autonomic neuroimmunology* (pp. 15–34). New York: Taylor & Francis.

Pacák, K., & Palkovits, M. (2001). Stressor specificity of central neuroendocrine responses: Implications for stress-related disorders. *Endocrine Reviews, 22*, 502–548.

Swedberg, K. (2002). Importance of neuroendocrine activation in chronic heart failure. Impact on treatment strategies. *European Journal of Heart Failure, 2*, 229–233.

Tsigos, C., & Chrousos, G. P. (2002). Hypothalamic-pituitary-adrenal axis, neuroendocrine factors and stress. *Journal of Psychosomatic Research, 53*, 865–871.

Vigas, M., & Jezová, D. (1996). Activation of the neuroendocrine system during changes in homeostasis during stress conditions. *Bratislava Medical Journal, 97*, 63–71.

Neuroendocrine Theory of Aging

Emil C. Toescu
Division of Medical Sciences, The University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Hormone theory of aging

Definition

The neuroendocrine hypothesis of aging proposes that aging results from the functional perturbations, both in neuronal control and in endocrine output, of the hypothalamic-pituitary-adrenal axis. These perturbations result in dysfunction in the activity of various endocrine glands and their target organs. The other consequence is a developing imbalance in hormonal cross-communication between various endocrine axes.

Description

The neuroendocrine system has two central components: the hypothalamus, a structure that is part of the central nervous system (CNS), and the pituitary gland, situated in an anatomical bone depression at the base of the skull, just behind the crossing of the optical nerves (optic chiasm). The two components, forming the hypothalamo-pituitary (HP) axis are extensively linked, though a stalk that contains both vascular and neuronal connections.

The activity of the system is regulated both by neurotransmitters and neuropeptides secreted locally in the CNS and by circulating hormones. Together, these factors control the secretion by the hypothalamus of hormonal factors that induce or inhibit the downstream release of hormones from the pituitary gland. In addition to this central role in the control of the endocrine system, the hypothalamus has also an important role in the control and integration of activities in the sympathetic and parasympathetic branches of the autonomic nervous system, thus influencing a wide array of essential physiological functions such as heart beat, blood pressure, and vascular reactivity or glucose metabolism. By changes in neuronal activity in specific discrete nuclei, the hypothalamus also regulates the food intake and the distribution and regulation of fat metabolism, in response to peripheral appetite-inducing, orexigenic (ghrelin), or appetite-reducing, anorexigenic (e.g., leptin) agents.

Aging is associated with multiple endocrinological changes, including a decrease in estrogens in women (menopause) and testosterone in men (andropause), dehydroepiandrosterone (DHEA) and DHEA sulfate (adrenopause), as well as decreases in growth hormone (GH) and insulin-like growth factor (IGF-1) (somatopause).

The general form of the neuroendocrine hypothesis of aging proposes that aging results from the functional perturbations, both in neuronal control and in endocrine output, of the HP axis, that result in a dysfunction in the activity of the endocrine glands and their target organs, as well as imbalances in the hormonal and signaling cross communication between the various components of the endocrine axes. A relevant feature of the activity of the neuroendocrine system is that the secretion of many of the hormone-releasing factors controlling individual endocrine

systems is cyclical, integrated mainly in diurnal (24 h), but also in monthly or longer cycles, through activity in the hypothalamic suprachiasmatic nucleus (SCN). Thus, the neuroendocrine hypothesis of aging proposes that with aging, through yet to be defined mechanisms, the activity of the body clock is perturbed resulting in a gradual and progressive dys-synchronization of the hormonal output. Similar disruptions in SCN activity affect other processes showing a diurnal cycle, such as the sleeping.

Several examples can illustrate this effect of aging on the endocrine system and the resulting functional effects. Thus, with increasing age, there is a decreased secretion of hypothalamic gonadotrophin-releasing hormone (GnRH) and a significant change in the pulsatile rhythm of its secretion that results in a decreased and more erratic secretion of luteinizing hormone (LH). This desynchronization, together with the decrease in the ovarian secretion of regulatory factors, leads, over a period of time, to the loss of reproductive cycles in females. The associated decrease in number of ovarian follicles contributes also to the decline in estrogen levels in aged women, leading to some of the of age-associated changes in postmenopausal women, such as the atrophy of the secondary reproductive tissues, reductions in bone density, or alterations in various cognitive functions. Decreased secretion of GnRH in males results in changes in LH and consecutive decrease in androgen levels, with corresponding loss of skeletal muscle and reproductive functions.

In many instances, the term "neuroendocrine theory of aging" is used in a much more restricted meaning and refers to the consequences of the age-related changes in the hypothalamo-pituitary-adrenocortical gland (HPA) axis. At the top of this axis, the parvocellular neurons in the paraventricular nucleus of the hypothalamus secrete corticotrophin-releasing hormone (CRH) that stimulates the anterior pituitary secretion of the adrenocorticotropic hormone (ACTH). The target organs for this latter hormone are the adrenal glands (situated above each kidney), where it activates the secretion of the glucocorticoids (GCs; cortisol, in humans, corticosterone, in rodents) from specific cortical cells in the cortex of the gland. The GCs have a wide range of effects – some are related to glucose metabolism, inducing through various mechanisms increases in glucose concentration in the blood, while others are related with the immune response, up-regulating overall the expression of anti-inflammatory proteins and decreasing the levels of pro-inflammatory proteins. In addition to these effects, and very relevant to the mechanisms linking glucocorticoids with the aging process, the GCs hormones play a vital role in regulating the stress response. Together with other stress-sensitive systems (e.g., the sympathetic response mediated by secretions from the medullar part of the same adrenal gland), GCs prepare the body for adaptation by mobilizing energy stores, suppressing nonessential physiological systems (e.g., feeding, reproduction), and generating behavioral responses to stimuli perceived as stressful. In the various target organs, including the brain, GCs act through two types of receptors. The glucocorticoid receptor (GR) is expressed throughout the brain, with relatively higher density in the hippocampus and prefrontal cortex. The mineralocorticoid receptor (MR) has a more restricted distribution, mainly in the hippocampus. The existence of both types of receptors in hippocampus is very relevant since the hippocampus, while being a central node in the networks controlling learning and memory, is also exerting an inhibitory effect on the hypothalamic release of CRH and, thus, can exert significant control on the HPA axis.

Two interrelated concepts are important for understanding this formulation of the neuroendocrine theory of aging, particularly in respect to brain aging: (1) chronic stress is associated with increases in circulating GC concentrations and (2) such increases in GCs induce an enhanced vulnerability of neurons to a variety of neuro- and excito-toxic agents. Thus, in a simple scheme, the decrease in the number of GRs and MRs observed with increasing age would contribute to an impaired regulation of the HPA axis, enhanced basal levels of circulating GCs, neuronal death and hippocampal atrophy, and cognitive impairment (the glucocorticoid hypothesis of brain aging).

Experimental evidence indicates that, in fact, the age-dependent changes in HPA axis activity are of a much subtler nature. With age, there are variable changes in the effects of GCs (cortisol) on ACTH secretion or of ACTH on cortisol secretion, while the capacity of the adrenal gland to produce GCs is not affected. Whereas the physiological cortisol circadian rhythmicity is maintained in essence, a significant age-related increase in the peak cortisol levels in physiological aging and an advance shift in the onset of the circadian cortisol rise result in an overall elevated serum GC level. With the important role of the GCs in mediating the stress response, one of the important functional consequences of these changes in the activity of the HPA axis is an increased responsiveness and an impaired ability to terminate stress responses, resulting in prolonged GCs exposures.

Further difficulties in tracing a simple causal relationship between dysregulation in the HPA axis, aging, and behavioral and cognitive impairment have been generated by recent data indicating that not all types of cognition are impaired, such that both encoding and consolidation of emotional learning is, in fact, facilitated by GCs or that in certain conditions, such as the caloric restriction model in rodents, increased GCs levels can be present while cognition performance is better than in the aged-matched control groups on a normal diet. Such results led to the recent proposal that the relationship between GCs, aging, and cognition represents a divergent imbalance between an age-dependent increase in GCs efficacy on some cell types, such as neurons in the hippocampus, enhancing the catabolic processes, and a decreased efficacy on other cell types, resulting in a decreased protective, anti-inflammatory response.

It is still unknown whether the age-associated reduction in hippocampal volume is the primary event leading to elevated levels of cortisol or the other way around, elevated levels of cortisol causing hippocampal atrophy. It is also possible that both events reflect a broader syndrome, often observed during aging, called the metabolic syndrome, and characterized by reduced glucose tolerance, hypertension, obesity, and elevated levels of cortisol. In addition, the actual role of circulating GCs in controlling the aging process is further complicated by the fact that the concentrations of active GC forms (cortisol, in humans, and corticosterone, in rodents) that interact with the intracellular receptors are controlled not only by the circulating levels of GCs but also by powerful pre-receptor mechanisms in the cytosol, such as the enzyme 11b-hydroxysteriod dehydrogenase I (11-HSD I), that is able to convert inactive forms of GCs (cortisone) into active forms (cortisol). Recent experiments indicate that modulation of 11-HSD I expression and activity has effects both on the aging phenotype and on the age-associated cognitive changes.

An important line of support for a neuroendocrine theory of aging comes from studies in the last few decades on the powerful effect of caloric restriction (CR) in delaying aging and increasing life span, in species ranging from yeast and worms to mice, rats, and monkeys (the results in humans are promising but not yet conclusive). From an evolutionary viewpoint, the effect of CR appears to be explained by organisms having evolved adaptation mechanisms in their neuroendocrine systems to maximize survival during periods of food shortage. Although CR induces wider endocrine modifications (fall in thyroid hormones (T_3), increased circulating corticosterone (in rodents), with little change in the secretion of the central regulator factors on the HPA axis, and decreased gonadal hormones), the proposed central mechanism of action of CR is through insulin signaling. It is known that aging is associated with increases in insulin resistance and adiposity, and it is becoming clear that long-time exposure to insulin resistance accelerates biological aging. For example, in diabetes, there is early onset of certain diseases of aging, such as dementia, as well as signs of general body aging such as frailty. Chronic stress may accelerate these age-related metabolic changes. Stress is related to obesity, especially abdominal obesity, and insulin resistance in both animal and human models.

Lifting the veil as to the endocrine mechanisms involved, the positive effects of CR are reproduced, to a large extent, in animal models

that have spontaneous or genetically engineered reductions in the activity of the GH/IGF-1 axis, starting with the initial reports of antiaging effects of hypophysectomized animals, more than 50 years ago. The nature of the processes involved was discovered by genetic studies, initiated in worms (*C. elegans*), showing that mutations in some genes in this pathway confer resistance to environmental stress, enhanced resistance to starvation, and, most importantly, extended longevity. Many of these same genes are conserved in humans: the insulin/insulin-like growth factor-I (IGF-I) peptide and *daf-2* gene (in worms) are homologs of the human insulin and IGF-I receptor, while *age-1* (worms) is related to a conserved phosphoinositol-3-kinase that responds to insulin receptor activation, and *daf-16* (worms) is a homolog of a human transcription factor.

The endocrine mechanisms of the antiaging effects of CR highlight also some of the subtleties of the complex set of processes that regulate the process of normal aging and the resultant potential pitfalls in designing and offering rejuvenation cures. Insulin/IGF-1 signaling is one of the most crucial cellular pathways regulating protein synthesis, glucose metabolism, and cellular proliferation and differentiation. The somatotropic axis in mammals, having the growth hormone (GH) as the major peripheral hormone, can stimulate the expression and secretion of IGF-1 in the liver and modulate the metabolism of several target tissues directly via different GH receptors. Several endocrine mutants, expressing either elevated or reduced levels of GH/IGF-1 hormones, highlight the significance of this axis in the regulation of growth and body size since the deficiencies in the function of GH/IGF-1 axis invariably lead to decreased anabolic capacity and reduced growth. The fact that the GH circulating levels are reduced with age could thus account for many of the features of the aging phenotype and form one face of the so-called GH/IGF-1 paradox of aging. The other face of it is the demonstration of the significant antiaging effects of reducing IGF/insulin signaling, as exemplified by the CR process. To date, the paradox does not have a satisfactory resolution, and it is difficult to decide whether reduced activity in the GH/IGF-1 axis should be viewed as a cause or a simple effect of aging. Furthermore, it can be argued that the decline in GH secretion with age represents a protective mechanism against insulin resistance and/or cancer or even an activation of survival mechanisms rather than simply reflecting a progressive failure of the hypothalamus-somatotrope axis. One way to reduce the conceptual tension generated by this neuroendocrine paradox is to invoke one of the most powerful evolutionary theories of aging, the antagonistic pleiotropy theory, which argues that, from an evolutionary perspective, aging results from the action of a number of physiological and/or homeostatic systems that exert several effects (pleiotropic), some of which being positive and beneficial at early stages of individual development, while others being negative and detrimental at later stages of development. Against this backdrop, opinions about the potential use and benefits of GH or GH secretagogues as antiaging agents differ widely and are highly controversial. Overall, data from well-designed clinical studies reported to date indicate unfavorable risk–benefit ratio for treatment of normal elderly subjects with GH. Prescribing GH to individuals who are not GH deficient in an attempt to slow aging is not supported by the available evidence, is not included among its approved uses, and, in the US, is specifically disallowed.

The same cautious view should be taken in respect to the adrenal sex steroid, dehydroepiandrosterone (DHEA) proposed as another antiaging fountain of youth hormone. With age, there is a large and abrupt decrease of estrogens in postmenopausal women and a slower and gradual decrease of testosterone in men. In a similar fashion, DHEA and its sulfate ester also fall progressively with age. The fact that the changes in DHEA correlate well with other age-induced modifications, such as increased fat mass and visceral adiposity, reduced bone mineral density, reduced muscle mass, and increased frailty, led to proposals for DHEA treatments or even sex hormones, in elderly individual otherwise

endocrinologically well. However, well-controlled clinical trials have repeatedly failed to show significant improvements in either the biological parameters tested or in the overall quality of life reporting.

Cross-References

- ▶ Hypothalamic-Pituitary-Adrenal Axis
- ▶ Neuroendocrine Activation

References and Readings

Bartke, A. (2009). The somatotropic axis and aging: Mechanisms and persistent questions about practical implications. *Experimental Gerontology, 44*(6–7), 372–374.

Brown-Borg, H. M. (2009). Hormonal control of aging in rodents: The somatotropic axis. *Molecular and Cellular Endocrinology, 299*(1), 64–71.

Chahal, H. S., & Drake, W. M. (2007). The endocrine system and ageing. *The Journal of Pathology, 211*(2), 173–180.

Dumitriu, D., Rapp, P. R., McEwen, B. S., & Morrison, J. H. (2010). Estrogen and the aging brain: An elixir for the weary cortical network. *Annals of the New York Academy of Sciences, 1204*, 104–112.

Hertoghe, T. (2005). The "multiple hormone deficiency" theory of aging: Is human senescence caused mainly by multiple hormone deficiencies? *Annals of the New York Academy of Sciences, 1057*, 448–465.

Landfield, P. W., Blalock, E. M., Chen, K. C., & Porter, N. M. (2007). A new glucocorticoid hypothesis of brain aging: Implications for Alzheimer's disease. *Current Alzheimer Research, 4*(2), 205–212.

Levay, E. A., Tammer, A. H., Penman, J., Kent, S., & Paolini, A. G. (2010). Calorie restriction at increasing levels leads to augmented concentrations of corticosterone and decreasing concentrations of testosterone in rats. *Nutrition Research, 30*(5), 366–373.

Stewart, P. M. (2006). Aging and fountain-of-youth hormones. *The New England Journal of Medicine, 355*(16), 1724–1726.

Weinert, B. T., & Timiras, P. S. (2003). Invited review: Theories of aging. *Journal of Applied Physiology, 95*(4), 1706–1716.

Neurogenetics

- ▶ Neurogenomics

Neurogenomics

Ornit Chiba-Falek
Duke University Medical Center, Durham, NC, USA

Synonyms

Neurogenetics

Definition

Neurogenomics is the interface of neurobiology and genome sciences. It is the study of how the genome as a whole contributes to the evolution, development, structure, and function of the nervous system. Neurogenomics research employs genetic strategies, including investigations of the genome sequence and products (transcriptomes and proteomes), to identify genes involved in the nervous system and to understand their gene-product function and the biological mechanisms through which they contribute to brain development, function, plasticity, and disease. A major goal in neurogenomics is the isolation of genes linked to neurological diseases such as Alzheimer's and Parkinson's diseases. Outcomes of such research programs will improve the ability to diagnose neurological disease even before it strikes and will advance the development of novel therapeutic targets to prevent and/or halt the progression of these diseases.

Description

Trends toward large-scale interdisciplinary research projects advance the neurosciences and provide firm foundation the vigorous exploration of the frontier between neurobiology and genome sciences. Neurogenomics field includes, but is not limited to, research of genes that cause neurological disorders; molecular mechanisms through which disease genes act; animal models

and in vitro techniques for studying pathways of gene function; genetically based studies of neuronal patterning, migration, connectivity, and cognitive/behavioral function; and the genetic basis of normal neural development and function. These studies promote also the development of preclinical disease biomarkers, gene-based therapeutics for neurological disorders, and pharmaceuticals targeted to specific gene products.

Genetic methodologies are having a rapidly increasingly impact on studies of the normal and diseased nervous system. The recent advances in genomic and other high-throughput "omic" technologies have allowed an expansion from single-gene to genome-wide analyses, providing a step forward toward a global understanding of neurological and neuropsychiatric disorders. To date, more than 200 genes have been identified that cause or contribute, in various extents, to neurological disorders. Examples of recent milestone advances are the following: (1) A meta-analysis of genome-wide association studies in Parkinson's disease identified 11 Parkinson's risk loci that surpassed the threshold for genome-wide significance. Six were previously identified loci (*MAPT, SNCA, HLA-DRB5, BST1, GAK*, and *LRRK2*), and five were newly identified loci (*ACMSD, STK39, MCCC1/LAMP3, SYT11*, and *CCDC62/HIP1R*). (2) Large Alzheimer's GWAs and replication studies reported genome-wide significant association with the strongest most established genetic risk factor for sporadic late-onset Alzheimer's disease, the *APOE* genomic region, and with three novel loci (*CLU*, *CR1*, and *PICALM*). (3) Using comparative genomics approach, it was found that rare structural variants (CNV) disrupt multiple genes in neurodevelopmental pathways in schizophrenia.

Furthermore, the development of new model organisms (such as the *C. elegance*, honeybee, *Drosophila*, and zebra fish) for neuroscience research is being accelerated by extensive genome sequencing and the application of comparative and functional genomics technologies.

These modern technologies also impact the availability of information and data sharing. Resources for neurogenomics research – such as tissue and information registries, gene expression and function atlases of the brain of animal models, human brain transcriptomes, and whole genome genotypes from neurological affected subjects and control – have been rapidly developed and are becoming publicly available for investigators to use.

Cross-References

- ▶ Alzheimer's Disease
- ▶ Gene
- ▶ Genetics
- ▶ Genome-Wide Association Study (GWAS)
- ▶ Genomics
- ▶ Parkinson's Disease: Psychosocial Aspects

References and Readings

Boguski, M. S., & Jones, A. R. (2004). Neurogenomics: At the intersection of neurobiology and genome sciences. *Nature Neuroscience, 7*, 429–433.

Gibbs, J. R., van der Brug, M. P., Hernandez, D. G., Traynor, B. J., Nalls, M. A., Lai, S. L., et al. (2010). Abundant quantitative trait loci exist for DNA methylation and gene expression in human brain. *PLoS Genetics, 6*, e1000952.

Harold, D., Abraham, R., Hollingworth, P., Sims, R., Gerrish, A., Hamshere, M. L., et al. (2009). Genome-wide association study identifies variants at CLU and PICALM associated with Alzheimer's disease. *Nature Genetics, 41*, 1088–1093.

International Parkinson Disease Genomics Consortium. (2011). Imputation of sequence variants for identification of genetic risks for Parkinson's disease: a meta-analysis of genome-wide association studies. *Lancet, 377*(9766), 641–649.

Lein, E. S., Hawrylycz, M. J., Ao, N., Ayres, M., Bensinger, A., Bernard, A., et al. (2007). Genome-wide atlas of gene expression in the adult mouse brain. *Nature, 445*, 168–176.

Walsh, T., McClellan, J. M., McCarthy, S. E., Addington, A. M., Pierce, S. B., Cooper, G. M., et al. (2008). Rare structural variants disrupt multiple genes in neurodevelopmental pathways in schizophrenia. *Science, 320*, 539–543.

Whitworth, A. J., Wes, P. D., & Pallanck, L. J. (2006). Drosophila models pioneer a new approach to drug discovery for Parkinson's disease. *Drug Discovery Today, 11*, 119–126.

Neuroimaging

Elliott A. Beaton
Department of Psychiatry and Behavioral Sciences and the M.I.N.D. Institute, University of California-Davis, Sacramento, CA, USA

Synonyms

Brain imaging; Computerized tomography (CT); Diffuse optical imaging (DOI); Event-related optical imaging (EROI); Functional magnetic resonance imaging (fMRI); Imaging; Magnetic resonance imaging (MRI); Positron emission tomography (PET)

Definition

Neuroimaging broadly refers to the relatively noninvasive technologies and techniques for localizing, measuring, and visualizing central nervous system function and structure. Common neuroimaging methodologies include magnetic resonance imaging (MRI), positron emissions tomography (PET), and computerized tomography (CAT/CT).

Description

Neuroimaging refers to a collection of techniques used to noninvasively view structure and function of living brain tissue. The methods used to visualize brain tissue have evolved significantly over the last several decades from using x-ray technologies to the more recent and increasingly ubiquitous (nuclear) magnetic resonance imaging.

Contrast X-Rays and Computerized Axial Tomography (CAT/CT)

X-ray photography creates images by passing x-rays through the body and onto a photographic plate by taking advantage of variation in x-ray radiation absorption of different tissues. Certain molecules and denser materials absorb more radiation and thus less reaches the photographic plate. This method is excellent for seeing skeletal bones that appear on the photographic plate with a high degree of contrast compared to surrounding tissues. It is less useful for tissues that do not strongly differ in x-ray radiation absorption such as parts of the brain. One method to get around this is to introduce a radiopaque agent to increase contrast by differentially absorbing x-rays. This allows for the visualization of the cerebral ventricular and circulatory systems. Pneumoencephalography involves injecting air into the cerebral ventricular system to briefly displace cerebral spinal fluid (CSF), and cerebral angiography involves injecting a radiopaque dye into a cerebral artery. These methods are limited in the information they produce but can be used to examine general brain atrophy, damage, or displacement of blood vessels.

The next step in the evolution of x-ray imaging of the living brain was the introduction of computed tomography (CT) which is sometimes referred to as computerized axial tomography (CAT). However, CT is more appropriate because "axial" merely refers to the plane of image acquisition, and images can just as easily be acquired in the coronal or sagittal planes. CT utilizes an x-ray detector rather than a photographic plate. The x-ray source and detector are mounted opposite one another on a rotating ring inside a tube that encircles the person being scanned. The CT scanner captures numerous images of the brain from several angles as the x-ray source and detector rotate around the head. These images are then reconstructed by a computer to make three-dimensional multislice images of the living brain. The brighter and darker areas of the images are described as "hyperdense" and "hypodense," respectively, with grayish components of the images as "isodense." Water and CSF appears almost black, white matter darker than gray matter, and skull as nearly white. Variation in image intensity is more carefully delineated in Hounsfield units (HU) with water having an HU of 0, CSF and HU between 8 and 18, gray matter and white matter HU = 37–41 and 30–34, respectively, and bone

HU = 600–2,000. CT scans have the benefit of being relatively inexpensive and having very fast acquisition times, making them particularly valuable tools for detecting recent brain trauma and intracranial lesions in emergency situations. Limitations of CT scanning include poorer contrast between brain tissue types, and the number of CT scan any one person can have at a given time is limited because of safety requirements to limit exposure to x-ray radiation. Furthermore, while CT can be used to visualize brain structure, it is not useful for measuring brain function while engaging in a process or activity. Other methods allowing for accurate localization of brain function include positron emissions tomography (PET) and functional magnetic resonance imaging (fMRI). Imaging equipment that combine CT and PET technologies in one package are now commonly available and increase information yield and utility with the practical benefit of taking up less space than dedicated CT and PET scanners.

Single-Photon/Positron Emissions Tomography (SPECT/PET)

Positron emissions tomography (PET) and single-photon emission computerized tomography (SPECT) are used to image brain activity. This method also uses radiation and radiation detectors, but rather than shooting an x-ray through the material to be imaged, PET utilizes radiolabeled tracers in the form of chemicals that have specific actions within the brain. For example, fluorine-18-labeled 2-fluoro-2-deoxy-D-glucose (18 F-FDG) is a commonly used radiotracer. When 18 F-FDG is injected into the carotid artery, it is rapidly taken up by metabolically active neurons during an experimental task as it very similar to glucose. However, it cannot be metabolized like glucose and thus accumulates in active brain regions where it slowly breaks down. The radioactive label (or ligand) gives off photons (i.e., S*P*ECT) as a result of a nuclear process where a proton in the nucleus is converted into a neutron, neutrino, and a positron (i.e., *P*ET). Both the neutrino and the positron are then ejected from the nucleus. The kinetic energy of the ejected positron both varies and declines at a rate that depends on the nature of the surrounding material. When an ejected positron meets an electron, it creates an annihilation reaction where the electron and the positron turn into two photons that travel in opposite directions (180°) of each other. These photons are measured as a line by two of a series of scintillation detectors mounted in opposition from one another. The images created by the PET scanner are not images of the brain itself; rather, they are images created from the relative distributions of detected amounts of radioactivity in brain regions of interest.

PET is powerful methodology that can be used to study hemodynamics, drug action localization, receptor function, metabolism, and even molecular processes including DNA synthesis. PET is particularly valuable in detecting disease processes that may be evident as metabolic variation but not are yet manifested as anatomical abnormality that could be detected using CT or MRI. However, PET images can be effectively combined with CT or MRI images, providing accurate localization of accumulated radioactivity. PET is also advantageous in that radiation exposure is relatively limited. The primary limitation of PET is the necessity for local access to a cyclotron to produce radiotracers. The radiotracers have a very short half-life and thus must be made in close physical proximity to the PET scanner and utilized quickly. The limitations of PET and CT have led to a significant increase in application of methods that do not utilize hard radiation like x-rays or radiolabels that are expensive to produce. Structural and functional magnetic resonance imaging (sMRI and fMRI, respectively) and the recent emergence of near infrared spectroscopic imaging (NIRSI) allow for detailed analyses of both brain structure and function in the living brain.

Magnetic Resonance Imaging (MRI)/ Functional Magnetic Resonance Imaging (fMRI)

Magnetic resonance imaging (MRI) methods produce images of the brain and other bodily regions that are high in both contrast and resolution. Although some MRI methods utilize contrast agents, MRI does not expose patients or study participants to ionizing radiation. Rather,

this technique utilizes a very powerful homogeneous and stable electromagnetic field.

This brief description of how MRI works is limited to "classical"/Newtonian physics, but quantum mechanical descriptions are available elsewhere. Protons are found in all of the nuclei of the atoms that make up the body, but conventional MRI utilizes hydrogen protons. Hydrogen protons spin randomly with their magnetic moments "pointing" in random directions until they are in the influence of the strong magnetic field of the MRI scanner where they all align in parallel with the direction (z-axis) of the external field generated by the electromagnet. Application of a radiofrequency (RF) pulse is applied to the z-axis aligned hydrogen protons with an excitation/receiver coil. As a result of absorbed energy from the RF pulse, the hydrogen protons move or "flip" into a higher energy state that is antiparallel to the z-axis toward the x-y plane. With the removal of the RF pulse, the hydrogen protons "relax" or move back into alignment with the external electromagnetic field along the z-axis and release the absorbed energy form the RF pulse as electromagnetic waves that are detected by the excitation/receiver coil and other magnetic gradient coils in three dimensions.

Static contrast methodologies are used to generate anatomical images of the brain. Depending on the type of RF pulse applied, the images highlight different types of tissue or fluids. Static contrast between tissue types is achieved by three properties of protons in tissues: (1) the proton density (i.e., how many hydrogen protons are in the region), (2) proton relaxation times along the z-axis (i.e., the longitudinal relaxation time or T1), and (3) proton relaxation times along the x-y plane (i.e., the transverse relaxation time or T2). Motion contrasts detect dynamic properties of protons in tissues and fluids to generate images of blood flow, capillary irrigation, perfusion, and diffusion of water.

Functional MRI (fMRI) refers to MRI methodologies that estimate brain activity. Brain slices are repeatedly imaged over time, allowing for statistical contrast of experimental manipulations. The most common is blood oxygen level-dependant (BOLD) fMRI. BOLD fMRI methods exploit changes in levels of oxygen in the blood that result from the metabolic demands of brain tissue during neural activity. Active brain tissue utilizes oxygen, and the change from an oxygenated state to a deoxygenated state can be detected because deoxygenated blood is paramagnetic. Other methods include perfusion or dynamic-contrast MRI, which measures changes in blood volume using an injected paramagnetic contrast agent such as gadolinium, or magnetic resonance spectroscopy (MRS), which measures localized levels of brain metabolites. There is also diffusion MRI that measures diffusion coefficients of water in brain tissue. Diffusion tensor imaging (DTI) examines the water diffusion coefficients in neighboring voxels to estimate the shapes and directions of white matter tracts.

MRI possesses advantages over CT and PET including very high-resolution images that can be acquired without ionizing radiation. In most MRI procedures, no contrast agent is needed, and the procedures are completely noninvasive. MRI still requires significant safety procedures though. The magnet is always active, and any objects that are susceptible to magnetism can become dangerous projectiles within the boundaries of the field. Furthermore, patients and study participants must be screened for metallic objects or medical devices such as pacemakers in and on their bodies.

Diffuse Optical Imaging or Tomography (DOI/DOT) and Near Infrared Spectroscopy (NIRS)

Diffuse optical imaging (DOI) and near infrared spectroscopy (NIRS) are relatively new applications for measuring relative changes in blood volume and oxygenation via hemoglobin levels as a proxy for cellular metabolism. These methods exploit changes in the properties of near IR light projected through tissue in the absorptive spectra and light scattering properties of water, oxygenated hemoglobin, and deoxygenated hemoglobin. Like BOLD fMRI, DOI measures the hemodynamic response as blood flows to the active tissue supplying oxygen to satisfy the metabolic needs of neurons in the active

region. Changes in the way that light moves through brain tissue from the IR source to the IR detector can be computationally modeled, and blood flow to particular brain regions can be examined based on the placement of the IR source and detectors.

Modeling how light moves through the various tissues of the head is a complex process that contributes to DOI and NIRS limitations. One method of simplifying the model is to assume the brain region being scanned is essentially "flat" and that the tissues do not differ in their optical properties. However, anatomical MRI can be combined with DOI/NIRS to provide a better model of absorption and scattering of light with bone and other head tissues. Advantages of this technology include a high degree of portability, rapid data acquisition, relative low cost, and complete noninvasiveness. The primary disadvantages result from the lack of robust spatial resolution and that imaging is limited to surface and near-surface brain tissue.

References and Readings

Azar, F., & Intes, X. (Eds.). (2008). *Translational multimodal optical imaging*. Norwood, MA: Artech House.

Christian, P. E., & Waterstram-Richm, K. M. (Eds.). (2012). *Nuclear medicine and PET/CT technology and techniques* (7th ed.). St. Louis, MO: Mosby.

Hanson, S. J., & Bunzl, M. (Eds.). (2010). *Foundational issues in human brain mapping*. Cambridge, MA: The MIT Press.

Huttel, S. A., Song, A. W., & McCarthy, G. (2008). *Functional magnetic resonance imaging* (2nd ed.). Sunderland, MA: Sinauer Associates.

Jezzard, P., Mathews, P. M., & Smith, S. S. (2001). *Functional MRI: An introduction to methods*. New York: Oxford University Press.

Jiang, H. (2010). *Diffuse optical tomography*. Boca Raton, FL: CRC Press/Taylor and Francis LLC.

Mettler, F. A., & Guiberteau, M. J. (2006). *Essentials of nuclear medicine imaging* (5th ed.). Philadelphia: Saunders/Elsevier.

Romans, L. (2011). *Computed tomography for technologists: A comprehensive text*. Baltimore: Wolters Kluwer Health/Lippincot Williams and Wilkins.

Wahl, R. L., & Beanlands, R. S. B. (Eds.). (2009). *Principles and practice of PET and PET/CT* (2nd ed.). Philadelphia: Lippincott Williams and Wilkins/Wolters Kluver.

Neuroimmunology

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

This term refers to the interdisciplinary field merging neurology, immunology, and aspects of neuroscience. It is a scientific and clinical domain. Scientifically, neuroimmunology tries to understand the bidirectional links between the nervous and immune systems, and their implications to illnesses. Clinically, various "classical" neuroimmune diseases (e.g., multiple sclerosis – MS) and recently more diseases are being recognized for their influence by both the nervous and immune systems, including cancer and coronary heart disease. The biological underpinnings of neuroimmunology include the descending pathways from the brain to the immune system, manifested by innervation of lymph nodes, effects of stress hormones on immunity, and the presence of neurotransmitter receptors on immune cells (Dantzer, Konsman, Bluthé, & Kelley, 2000). In parallel, ascending pathways include the vagus nerve, expressing receptors for interleukin-1, brain regions lacking the blood–brain barrier (BBB), and a "domino-like" effect of prostaglandins, an end point of inflammation, on both sides of the BBB (Dantzer et al., 2000; Tracey, 2009). MS, for example, represents an inflammatory autoimmune insult on nerves, which eventually leads to the episodic and degenerative characteristic of this disease (Compston & Coles, 2002). Understanding neuroimmune interactions has been pivotal for developing treatments for MS. Another extraordinary example is the work of Schwartz and colleagues who demonstrated that due to the "immune privilege" status of the brain, closed brain injuries may not undergo immune protection, while in contrast, following a homing intervention of T cells to the brain, recovery is accelerated (Schwartz & Moalem, 2001). The relevance of neuroimmune interactions to other diseases has

recently been claimed by researchers in relation to coronary heart disease (Gidron, Kupper, Kwaijtaal, Winter, & Denollet, 2007) and cancer (Gidron, Perry, & Glennie, 2005), based on multiple converging evidence. Ongoing scientific efforts may hopefully reveal the clinical implications of such neuroimmune associations for such and other diseases.

Cross-References

▶ Neuroimmunomodulation

References and Readings

Compston, A., & Coles, A. (2002). Multiple sclerosis. *The Lancet, 359*, 1221–1231.

Dantzer, R., Konsman, J. P., Bluthé, R. M., & Kelley, K. W. (2000). Neural and humoral pathways of communication from the immune system to the brain: Parallel or convergent? *Autonomic Neuroscience, 85*, 60–65.

Gidron, Y., Kupper, N., Kwaijtaal, M., Winter, J., & Denollet, J. (2007). Vagus-brain communication in atherosclerosis-related inflammation: A neuroimmunomodulation perspective of CAD. *Atherosclerosis, 195*, e1–e9.

Gidron, Y., Perry, H., & Glennie, M. (2005). The Vagus may inform the brain about sub-clinical tumours and modulate them: An hypothesis. *The Lancet Oncology, 6*, 245–248.

Schwartz, M., & Moalem, G. (2001). Beneficial immune activity after CNS injury: Prospects for vaccination. *Journal of Neuroimmunology, 113*, 185–192.

Tracey, K. J. (2009). Reflex control of immunity. *Nature Reviews Immunology, 9*, 418–428.

Neuroimmunomodulation

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

This term refers to the modulating role of the nervous system in relation to immune functions. This modulation reflects part of the bidirectional communication between the nervous system and the immune system. Neuroimmunomodulation is possible due to existence of receptors for neurotransmitters (e.g., norepinephrine, acetylcholine) on immune cells and due to innervation of lymph nodes by sympathetic nervous system (SNS) fibers (Felten et al., 1984). These innervating fibers influence the trafficking and proliferation of immune cells, all evidence for neuroimmunomodulation. Another more recently discovered form of neuroimmunomodulation includes the one by the vagus nerve, where its descending (efferent) branches inhibit cytokine synthesis in peripheral monocytes, via the alpha-7 nicotinic acetylcholine receptor (Tracey, 2009). The neuroimmunomodulating role of the vagus may have clinical implications since the inflammatory response is in the core of the etiology of severe chronic diseases such as cancer and coronary heart disease. Thus, vagal activity is hypothesized to possibly modulate the progress of such diseases (Gidron, Kupper, Waijtaal, Winter, & Denollet, 2007; Gidron, Perry, & Glennie, 2005), a matter under current investigation. Neuroimmunomodulation is also manifested by the differential effects of the cerebral hemispheres on peripheral immunity. Studies have shown that the left hemisphere has immune-potentiating effects while the right hemisphere has immunosuppressive effects (Davidson, Coe, Dolski, & Donzella, 1999; Meador et al., 2004). These effects were found in animals and humans and were found to be mediated by the sympathetic response, since blocking beta-adrenergic receptors abolished differences in immunity between the hemispheres. Here too, the neuroimmunomodulatory effects of the hemispheres may have clinical roles since a shift from left to right hemisphere activity during stressful periods correlated with more reported illness (Lewis, Weekes, & Wang, 2007). Furthermore, in a matched prospective design, people with right hemisphere lateralization were at significantly higher risk of reporting the common cold than those with left hemispheric lateralization, independent of confounders (Gidron, Hall, Wesnes, & Bucks, 2010). Neuroimmunomodulation has

a central role in behavior medicine, by possibly explaining how psychological factors influence the risk of disease, since the SNS, vagal nerve activity, and hemispheric lateralization are each related to psychological factors as well as to immunity and risk of certain illnesses. Research is only at the beginning of understanding these neuromodulatory links and of possibly utilizing them in the service of preventing or treating diseases.

Cross-References

- ▶ Immune Responses to Stress
- ▶ Neuroimmunology
- ▶ Psychoneuroimmunology

References and Readings

Davidson, R. J., Coe, C. C., Dolski, I., & Donzella, B. (1999). Individual differences in prefrontal activation asymmetry predict natural killer cell activity at rest and in response to challenge. *Brain, Behavior, and Immunity, 13*, 93–108.

Felten, D. L., Livnat, S., Felten, S. Y., Carlson, S. L., Bellinger, D. L., & Yeh, P. (1984). Sympathetic innervation of lymph nodes in mice. *Brain Research Bulletin, 13*, 693–699.

Gidron, Y., Hall, P., Wesnes, K. A., & Bucks, R. S. (2010). Does a neuropsychological index of hemispheric lateralization predict onset of upper respiratory tract infectious symptoms? *British Journal of Health Psychology, 15*, 469–477.

Gidron, Y., Kupper, N., Waijtaal, M., Winter, J., & Denollet, J. (2007). Vagus-brain communication in atherosclerosis-related inflammation: A neuroimmunomodulation perspective of CAD. *Atherosclerosis, 195*, e1–e9.

Gidron, Y., Perry, H., & Glennie, M. (2005). The vagus may inform the brain about sub-clinical tumours and modulate them: An hypothesis. *The Lancet Oncology, 6*, 245–248.

Lewis, R. S., Weekes, N. Y., & Wang, T. H. (2007). The effect of a naturalistic stressor on frontal EEG asymmetry, stress, and health. *Biological Psychology, 75*, 239–247.

Meador, K. J., Loring, D. W., Ray, P. G., Helman, S. W., Vazquez, B. R., & Neveu, P. J. (2004). Role of cerebral lateralization in control of immune processes in humans. *Annals of Neurology, 55*, 840–844.

Tracey, K. J. (2009). Reflex control of immunity. *Nature Reviews Immunology, 9*, 418–428.

Neurological

Yori Gidron
Faculty of Medicine and Pharmacy,
Free University of Brussels (VUB), Jette,
Belgium

Definition

Neurological refers to any process or disorder involving the nervous system or its components. The nervous system includes the central and peripheral nervous systems (CNS, PNS, respectively). These systems are composed of neuronal fibers (axons and dendrites) which transmit neurological information to and from neurons, cell bodies that process neurological information, as well as other types of cells (e.g., astrocytes, microglia). The CNS, the "headquarters" of the body, processes and regulates multiple bodily responses and psychological responses to external and internal stimuli. These numerous neurological processes are carried out by various levels of the CNS including the brain stem (basic vital signs), limbic system (memory, sensory, autonomic, immune, hormonal, and emotional processes), and the cortex (executive function and information processing, overall modulation of multiple systems). Neurological disorders are a heterogeneous group of disorders that involve abnormal functioning of the nervous system, PNS or CNS, or both. These could be manifested motorically (as in Parkinson's disease and stroke), behaviorally (as in Alzheimer's disease), or in neurophysiological tests (as in epilepsy or stroke). Neurological diseases caused by genetic mutations are often congenital (e.g., congenital insensitivity to pain with anhydrosis), developmental disorders of the nervous system (e.g., spina bifida), neurodegenerative diseases (e.g., Alzheimer's disease), diseases due to cerebral ischemia (e.g., stroke), injuries to the PNS or CNS, seizures (e.g., epilepsy), cancers (e.g., glioma), and infections (e.g., meningitis). These disorders are treated by

neurologists, physicians who specialize in neurological disorders, their etiology and treatment. A major consequence of neurological disorders is their psychosocial impact, both on the patient and on his or her social environment. Often, the social stigma associated with certain neurological problems is quite severe (de Boer, 2010) and has the potential to isolate patients. This is of great importance for behavior medicine and includes the assessment of the health-related quality of life affected by such conditions. The accumulating evidence linking neuroimmune processes (e.g., inflammation, vagal nerve modulation) to peripheral and central pathways (Tracey, 2009) and to diseases reveals the importance of the interdisciplinary nature of the neurologist's work, encompassing basic biology, medicine, and behavioral sciences.

Cross-References

- ▶ Neuroimaging
- ▶ Neuron

References and Readings

de Boer, H. M. (2010). Epilepsy stigma: Moving from a global problem to global solutions. *Seizure, 19*, 630–636.

Tracey, K. J. (2009). Reflex control of immunity. *Nature Reviews Immunology, 9*, 418–428.

Neuromuscular Diseases

Robert J. Gatchel and Matthew T. Knauf
Department of Psychology, College of Science, The University of Texas at Arlington, Arlington, TX, USA

Synonyms

Neuromuscular disorders

Definition

Neuromuscular disease is a broad term that encompasses many different specific diseases and ailments that, in general, impair the functioning of the muscles, the neuromuscular junction, and/or the peripheral nervous system (LaDonna, 2011; National Institute of Neurological Disorders and Stroke [NINDS], 2011).

Description

Many different specific diseases fall under the broad spectrum of neuromuscular diseases. Because it would be impossible to list each and every disease in this brief section, the most prevalent ones are included here: (1) motor system disorders (e.g., amyotrophic lateral sclerosis, Parkinson's disease), (2) central nervous system disorders (e.g., multiple sclerosis), (3) muscular dystrophies (e.g., Steinert's, Duchenne, Becker), (4) autoimmune disorders (e.g., myasthenia gravis), (5) neuropathies (acquired or inherited), and (6) hereditary muscular disorders (e.g., spinal muscular atrophy). There are also numerous other diseases that fall under each of the above categories. For example, muscular dystrophies include a group of more than 30 diseases, but the most common are myotonic (i.e., Steinert's, which primarily affects adults) and Duchenne (which primarily affects children) [NINDS, 2011]. Neuromuscular diseases may present symptoms at any time during an individual's lifetime, some may be present while an individual is an infant or child, but others may not be present until mid- to later portions of an individual's lifetime. Reported overall prevalence rates for all individuals that may be affected by an inherited neuromuscular disease at sometime during their life (infancy to adulthood) are approximately 1 in 3,500 individuals (Emery, 1991).

Many of the neuromuscular diseases are chronic and/or inherited, as well as progressive in nature, and individuals with these disorders tend to require specialized care in dealing with them (LaDonna, 2011; Seesing, Drost, van der Wilt, & van Engelen, 2011). In addition,

adjustment of the current treatment may be required if new symptoms arise at any time or if the existing symptoms begin to aggregate over time (Seesing et al., 2011). Many of the common symptoms of neuromuscular disease may include fatigue, muscle weakness, loss of motor control, and spasticity. Recent studies have shown that chronic pain may also be considered a common symptom of neuromuscular disease (Engel, Kartin, Carter, Jensen, & Jaffe, 2009; Hoffman et al., 2005; Jensen et al., 2005; Tiffreau et al., 2006). It has been reported that the rates for adults with neuromuscular diseases who have chronic pain range from 70% to 96% of those affected (Engel et al., 2009). One interesting point that should be noted is that chronic pain is not only present for adults with neuromuscular disease but is also present in children with neuromuscular diseases (Engel et al., 2009). It has been reported that the rate for children with neuromuscular diseases who have chronic pain is over 70% (Engel et al., 2009).

In addition to the physical impact of neuromuscular disease, there may also be a psychosocial impact as well. While individuals with neuromuscular diseases may experience a lower quality of life due to the effects of the disease, there is evidence that there are some activities, such as employment, recreational physical activity, and social outlets (peer support), that may improve quality of life (LaDonna, 2011). Also, the more independence that individuals with neuromuscular diseases are able to maintain, (i.e., the more activities that individuals can do by/for themselves), the better their quality of life (Abresch et al., 1998).

Once an individual has been diagnosed with having a neuromuscular disorder, the appropriate treatment plan may begin. While there is currently no "cure" for neuromuscular diseases and current treatments are limited (LaDonna, 2011), various treatments aimed at managing the diseases have included (1) drug therapy, (2) referral to specialists for potential surgical intervention (e.g., stimulator implants), (3) patient and familial education and counseling, (4) massage therapy, (5) acupuncture, (6) biofeedback or relaxation training, (7) chiropractic manipulation, (8) nerve blocks, and (9) hypnosis (Jensen, Abresch, Carter, & McDonald, 2005). Drug therapies may include giving the patient immunosuppressive drugs, analgesic medication (e.g., nonsteroidal anti-inflammatory, narcotic), muscle relaxants, anticonvulsants, and/or antidepressants. Any one of the treatments listed may be used to manage the neuromuscular disease itself, and they may also be employed to manage the pain that is associated with neuromuscular disease. Patients may go to many different specialists, ranging from occupational/physical therapists to physiatrists to surgeons, depending on the severity of the disease. Patient and familial education may be done individually or with family present, and patients and family members also have the option to go to counselors and support groups.

Currently, there is little evidence to suggest that any one treatment provides either significant and/or permanent reprieve of pain, as patients with neuromuscular disease who reported using any one or combination of pain treatments still report some pain (Jensen et al., 2005). Because there is currently no cure for the diseases, disease management approaches must be employed. It is important to attempt to identify the cause of the neuromuscular disease and any pain associated with the disease in order to provide the best method of management (Jensen et al., 2005). However, research is still needed to examine the etiology of the diseases, and, in addition, more research is needed to examine the underlying mechanisms of the pain that is associated with neuromuscular diseases. If new symptoms arise over the course of the disease, physicians and other specialists may conduct appropriate further testing and adjustment of treatments as warranted.

Cross-References

- ▶ Chronic Pain Patients
- ▶ Multiple Sclerosis: Psychosocial Factors

References and Readings

Abresch, R. T., Seyden, N. K., & Wineinger, M. A. (1998). Quality of life. Issues for persons with neuromuscular

diseases. *Physical Medicine and Rehabilitation Clinics of North America*, *9*(1), 233–248.
Emery, A. E. H. (1991). Population frequencies of inherited neuromuscular diseases – a world survey. *Neuromuscular Disorders, 1*(1), 19–29.
Engel, J. M., Kartin, D., Carter, G. T., Jensen, M. P., & Jaffe, K. M. (2009). Pain in youths with neuromuscular disease. *The American Journal of Hospice & Palliative Care, 26*(5), 405–412.
Hoffman, A. J., Jensen, M. P., Abresch, R. T., & Carter, G. T. (2005). Chronic pain in persons with neuromuscular disorders. *Physical Medicine and Rehabilitation Clinical North America, 16*(4), 1099–1112.
Jensen, M. P., Abresch, R. T., Carter, G. T., & McDonald, C. M. (2005). Chronic pain in persons with neuromuscular disease. *Archives of Physical Medicine and Rehabilitation, 86*, 1155–1163.
LaDonna, K. A. (2011). A literature review of studies using qualitative research to explore chronic neuromuscular disease. *Journal of Neuroscience Nursing, 43*(3), 172–182.
National Institute of Neurological Disorders and Stroke (NINDS). (2011). Muscular dystrophy: Hope through research. Office of Communications and Public Liaison, National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bethesda, MD 20892.
Seesing, F. M., Drost, G., van der Wilt, G. J., & van Engelen, B. G. (2011). Effects of shared medical appointments on quality of life and cost-effectiveness for patients with a chronic neuromuscular disease. Study protocol of a randomized controlled trial. *BMC Neurology, 11*, 106.
Tiffreau, V., Viet, G., & Thévenon, A. (2006). Pain and neuromuscular disease: The results of a survey. *American Journal of Physical Medicine & Rehabilitation, 85*(9), 756–766.

Neuromuscular Disorders

► Neuromuscular Diseases

Neuron

Marijke De Couck
Free University of Brussels (VUB), Jette, Belgium

Synonyms

Nerve cell

Definition

Neurons are the basic building blocks of the nervous system, which includes the brain, the spinal cord, and the peripheral nervous system. These specialized cells are the information-processing units responsible for receiving, processing, and transmitting information by electrical and chemical signaling. Chemical signaling occurs via synapses, specialized connections with other cells.

Structure

Although the morphology of various types of neurons differs in some respects, they all contain four distinct regions with differing functions in the communication of information: the cell body (soma), the dendrites, the axon, and the axon terminals. Most neurons have multiple dendrites, which receive chemical signals from the axon termini of other neurons. Dendrites convert these signals into small electric impulses and transmit them in the direction of the cell body. The cell body contains the nucleus and is the site of synthesis of virtually all neuronal proteins. Almost every neuron has a single axon, which is specialized for the conduction of a particular type of electric impulse, called an action potential, away from the cell body toward the axon terminus. When an action potential reaches a chemical synapse, a neurotransmitter is released into the synaptic cleft. The axon terminals are the small branches of the axon that form the synapses, or connections, with other cells. A single axon in the central nervous system can synapse with many neurons and induce responses in all of them simultaneously (Lodish, Berk, & Zipursky, 2000).

Classification

Neurons can be classified by their morphology and function. Functionally, we can distinguish afferent neurons, efferent neurons, and interneurons. Sensory neurons are afferent neurons that convey information from tissues and organs to the central nervous system. Motor neurons are efferent neurons that transmit signals from the central nervous system to the effector cells. The interneurons connect neurons within specific regions of the central nervous system.

Neurons can also be classified anatomically into unipolar, bipolar, and multipolar neurons. Unipolar neurons are those where the dendrite and axon merge into a single process of the cell body. Bipolar neurons are those where axon and single dendrite are on opposite ends of the soma, while multipolar neurons consist of more than two dendrites.

Functions

Neurons connect to each other to form networks. Neurotransmitters are used to carry the signal across the synapse to other neurons (Lodish et al., 2000). The shape, structure, and connectivity of nerve cells are important aspects of neuronal function. Genetic and epigenetic factors that alter neuronal morphology or synaptic localization of pre- and postsynaptic proteins contribute significantly to neuronal output and function and may underlie clinical states. Furthermore, neuronal loss is one of the major pathological hallmarks of neurodegenerative disorders including Alzheimer's disease (AD), Parkinson's disease, Huntington's disease, and amyotrophic lateral sclerosis. In daily mental functioning and psychiatric conditions, the pathways and amounts of neurotransmitters crossing between neurons influence and represent at the neuronal level such conditions. Examples include reduced serotonin in depression and aggression. Numerous studies have shown that neuronal stimulation can also influence behavior. For example, a study in rats showed that the stimulation of one single neuron in the somatosensory cortex can change the animal's detection behavior (Houweling & Brecht, 2008). Thus, the neuron is the basic unit of the nervous system, and understanding its multiple functions is of pivotal significance in health and disease.

References and Readings

Houweling, A. R., & Brecht, M. (2008). Behavioural report of single neuron stimulation in somatosensory cortex. *Nature, 451*, 65–68.

Lodish, H., Berk, A., Zipursky, S. L., et al. (2000). *Molecular cell biology* (4th ed.). New York: WH Freeman.

Neuronal Nitric Oxide Synthase (nNOS)

▶ Nitric Oxide Synthase (NOS)

Neuropeptide Y (NPY)

Mustafa al'Absi
University of Minnesota Medical School, University of Minnesota, 235 School of Medicine, Duluth, MN, USA

Synonyms

Hormones

Definition

Neuropeptide Y (NPY) is a 36-amino acid hormone expressed in multiple sensory, cerebral, and autonomic pathways. It is a highly conserved peptide reflecting its important role in the body.

In 1982, NPY was isolated from the hypothalamus, and early studies to characterize specific locations and functions of this neuropeptide documented the presence of NPY neurons within the paraventricular nucleus (PVN) of the hypothalamus. Subsequent experiments using a technique called in situ hybridization also found the highest cellular levels of NPY messenger RNA within the arcuate nucleus of the hypothalamus. Research has also demonstrated the presence of NPY in other areas within the brainstem, monoaminergic neurons, and GABAergic neurons in the cortex. Peripherally, NPY is present in sympathetic nerve fibers and innervated organs including the blood vessels, heart, gastrointestinal tract, thyroid gland, and sensory nerves.

Several receptors are targeted by NPY and these receptors are widely distributed throughout the body. These include subtypes Y1 and Y5 receptors which mediate effects of NPY on appetite stimulation, and receptors Y2 and Y4 which mediate NPY effects on appetite reduction.

The wide distribution of these receptors results in various effects on multiple central and peripheral functions. These effects can be a result of direct NPY action or indirect though influencing other neurotransmitters, such as norepinephrine and glutamate.

NPY Functions

NPY plays a significant role in a number of physiological and behavioral processes. It is involved in regulating emotions, energy balance, and cognitive functions. NPY increases food intake while also increasing rate and proportion of nutrients storage as fat. It may also interrupt pain processing leading to analgesic effects. Administering NPY by injecting it directly into the PVN leads to increases in the release of corticotropin-releasing hormone (CRH). Because CRH is involved in regulating various psychological and stress-related processes, it is thought that NPY is indirectly involved in these processes.

NPY and Appetite Regulation

The involvement of NPY in appetite regulation has received significant attention over the last 20 years, with animal research documenting NPY orexigenic effect. Studies on rats and using multiple methodologies, including immunochemical and in situ hybridization techniques, have shown that activation of NPY neurons increases food intake. Injecting the NPY agonist dexamethasone into the third ventricle or the hypothalamus increases appetite. Consistent results were also found when using antagonists that block NPY receptor. The blocking effect led to reduction in NPY neuron activity and reduction in food intake. When the receptors that inhibit the release of NPY (called autoreceptor Y2) were activated in the arcuate nucleus reduction in appetite was noted. Blocking these autoreceptors also led to the reverse effect of increased food intake. Studies that used genetically obese rats have also been conducted to investigate the role of NPY and found that NPY contributes to obesity through direct and indirect pathways. For example, both NPY mRNA and NPY release are high in obesity animal models. Factors that contribute to obesity, such as glucocorticoids, also increase NPY release. Other hormones that block NPY, such as leptin, have also been found to reduce appetite and reduce risk for obesity.

NPY and Emotion Regulation

NPY is involved in the regulation of emotional and affective behaviors. It also influences cognition functions and pain perception. For example, several of the NPY hypothalamic functions have been found to be dysregulated in depression. Hypothalamic-pituitary-adrenal (HPA) axis functions and disruption of appetite regulation in addition to disrupted circadian rhythm occur in depression and have been attributed to certain abnormalities of NPY functions. When NPY is administered into the hypothalamic PVN, it increased ACTH and corticosterone. It is also known that NPY fibers directly innervate CRH-producing neurons within PVN, and injection of NPY increases CRH levels. Patients with depression show decreased levels of NPY in CSF. Similar observation was found in suicide victims. Pharmacological treatment for depression using tricyclic antidepressants and treatment using electroconvulsive shocks were associated with increased NPY in multiple areas of the brain. Research also suggests that depressive symptoms associated with withdrawal from a stimulant like cocaine may be related to reduced levels of NPY caused by chronic drug use.

Studies related to anxiety have also shown a negative association between the level of anxiety and NPY levels in cerebrospinal fluid. Observational research using electrophysiological and behavioral approaches in multiple models of anxiety has demonstrated anxiolytic effects of NPY when administered intracerebroventricularly. This research has also been advanced toward pinpointing specific structures that are involved in this effect of NPY and has shown that localized microinjection into the central nucleus of the amygdala was specifically effective in producing NPY anti-anxiety effects. NPY anxiolytic effect in the amygdala appears to be mediated by Y1 receptor. Considering that this

location is where multiple stress-response factors and neurochemical pathway also interact suggests that NPY plays a significant role in regulating the stress response.

Studies both in humans and in animals indicate the possibility that NPY is directly involved in regulating the stress response and emotional reactivity. In humans, observational studies have indicated that NPY may have anxiolytic effects and may help in coping with stress by facilitating speedy recovery after exposure to intense stressors. Related to this, one study in veterans with posttraumatic stress disorder (PTSD) measured plasma NPY and compared them with veterans with no PTSD. The study found that veterans who did not suffer from PTSD had higher NPY levels than those with PTSD, suggesting that NPY may be a marker of the ability to recover from stress. On the other hand, studies in both mice and monkeys have demonstrated that exposure to repeated stress leads to increased NPY release. This is a similar effect to that seen when the animal ingests a high fat, high sugar diet. It is possible that the increased NPY concentrations contribute to increased fat buildup, especially around the waist and abdomen, and the effects of chronic stress on metabolism function are mediated by NPY.

Cross-References

- ▶ Anxiety
- ▶ Appetite
- ▶ Depression
- ▶ Leptin
- ▶ Stress

References and Readings

Allen, Y. S., Adrian, T. E., Allen, J. M., Tatemoto, K., Crow, T. J., Bloom, S. R., et al. (1983). Neuropeptide Y distribution in the rat brain. *Science, 221*, 877–879.

Morales-Medina, J. C., Dumont, Y., & Quirion, R. (2010). A possible role of neuropeptide Y in depression and stress. *Brain Res, 1314*, 194–205.

Yehuda, R., Brand, S., & Yang, R. K. (2006). Plasma neuropeptide Y concentrations in combat exposed veterans: Relationship to trauma exposure, recovery from PTSD, and coping. *Biol Psychiatry, 59*, 660–663.

Neuropsychological Assessment

▶ Neuropsychology

Neuropsychology

Richard Hoffman
Academic Health Center, School of Medicine-Duluth Campus University of Minnesota, Duluth, MN, USA

Synonyms

Brain-behavior relationships; Neurobehavioral assessment; Neurocognitive assessment; Neuropsychological assessment

Definition

Neuropsychology is a subdivision of the field of psychology that is concerned with the investigation of brain-behavior relationships. The broad category of neuropsychology can be further divided into experimental neuropsychology and clinical neuropsychology, with this latter specialty definable as an applied science focusing on the behavioral sequelae and manifestations of brain dysfunction.

Description

Neuropsychology is currently one of the fastest growing scientific specialty areas in the field of psychology, and the subspecialty area of human clinical neuropsychology is of direct relevance to behavior medicine practitioners as well as physicians in clinical practice, especially neurologists and neurosurgeons. Neuropsychologists in clinical practice use a variety of specially constructed tests and test batteries to assess the functional effects of brain trauma, infection, neoplasias, and structural changes to the brain (including

Neuropsychology, Table 1 Domains of brain-behavior functioning in neuropsychological assessment

Orientation and general mental status
Estimation of premorbid status
Potential for malingering/symptom validity testing
Sensory deficits and paresthesias
Motor and gait disturbances
Psychomotor activity and speed of information processing
Processing efficiency and reaction time
Response inhibition
Visual search and visuomotor scanning
Psychomotor efficiency
Speed of information processing
Motor speed
Grip strength
Disorders of perceptual function/apraxias
Attention and concentration
Mental speed
Visual attention
Auditory attention
Divided attention/visual monitoring and visual sequencing
Vigilance and sustained attention
Selective attention/freedom from distractibility
Working memory
Verbal abilities and language skills
Disorders of language functions/aphasias
Receptive language
Expressive language
Object naming/anomias
Verbal fluency
Phonemic fluency
Semantic fluency
Learning and memory
Visual learning and immediate memory
Verbal learning and immediate memory
Visual delayed memory
Verbal delayed memory
Long-term declarative memory and remote memory
Nondeclarative and procedural memory
Implicit memory
Incidental memory
Semantic and episodic autobiographical memory
Recognition and working memory speed
Spatial memory
Visual perception
Spatial cognition
Visuospatial skills and construction skills
Drawing skills
Visual discrimination
Visuospatial construction
Visuospatial integration
Facial recognition matching
Executive functions and conceptual skills
Metacognitive functions
Cognitive flexibility
Abstract reasoning/concept formation
Planning and sequencing ability
Judgment
Decision making
Novel problem solving
Overall intellectual abilities
Crystallized intelligence
Fluid intelligence
Academic achievement skills
Emotional functioning, personality, and affect
Instrumental activities of daily living/competencies
Compensatory strategies

vascular abnormalities and strokes) as well as degenerative central nervous system disorders, cortical and subcortical dementias, and progressive disorders of the nervous system, such as multiple sclerosis, Huntington's disease, and Parkinson's disease. Specialized neuropsychological test batteries have been constructed to investigate the neurocognitive effects alcohol and drugs of abuse, assess the functional effects of epilepsy and diabetes, and assess the effects of neurotoxins and hypoxia. In the field of neuropsychiatry, the neuropsychology of schizophrenia and the mood and affective disorders has been extensively studied and neuropsychological impairments have been identified that are independent of medication effects or the psychiatric illnesses themselves.

Modern clinical neuropsychology is very much a hybrid specialty, with influences from – and ties to – neurology, neurosurgery, neuroimaging (especially functional neuroimaging), neuropsychiatry, cognitive psychology, neuroscience, and clinical psychology as well as the significant advances that have been made in experimental neuropsychology, particularly in the areas of memory and learning. This is

Neuropsychology, Table 2 Common diagnostic questions that neuropsychological assessment can help answer

Does the patient have an impaired memory and, if so, what does that mean for that patient and for the treating physicians?
Is dementia present and, if so, what kind of dementia and how severe is it?
Can the patient understand and follow health-care provider instructions regarding their medical care?
What is the patient capable of doing vocationally and can the patient return to work following a neurological injury? What components need to be included in the patient's rehabilitation plan? Is the patient a candidate for a cognitive rehabilitation program and what should be included in this program?
Does the patient have only psychological or psychiatric problems, or do they have brain problems as well?
What problems remain for the patient following successful physical recovery from a head injury, stroke, or other brain injury?
What is the patient's capacity in a legal sense (can they drive a car safely, manage their money adequately, participate fully in legal matters such as criminal or civil trials, can they maintain professional licensure)?
What are the cognitive and functional effects of progressive illnesses as time progresses (dementias, brain tumors, progressive deteriorating disorders such as Huntington's, etc.)?
What is normal aging versus neurological disease/dementia?
Is the temporal lobe epilepsy patient a candidate for resection surgery?
Does the patient have specific learning disabilities/attention deficit disorder? What needs to be included in educational programming and follow-up?
Is the patient in need of a conservator or guardian? Are there neuropsychological deficits that require supervision, additional support, or placement out of the home (such as nursing home placement)?

reflected in the broad range of functional domains that are examined in neuropsychological assessment, as noted in Table 1.

Specialized neuropsychological test batteries have been developed for child and pediatric populations in addition to the adult and aged adult populations, and neuropsychological assessment is great value in the assessment of developmental and learning disorders of childhood. Child and pediatric neuropsychology has close ties to both child neurology and behavioral pediatrics, in addition to child psychiatry and contemporary learning models in child psychology.

Neuropsychology is a relatively new area of scientific inquiry. Sir William Osler first used the term "neuropsychology" in 1913, but neuropsychology as an emerging scientific discipline can trace its roots to the contemporary psychology, experimental/cognitive psychology, and neurology of the mid-1930s. In 1935, Ward Halstead at the University of Chicago – in close collaboration with neurologists and neurosurgeons – carefully observed the behavior of brain-damaged patients and constructed a battery of ten functional tests or neuropsychological tests designed to provide a comprehensive assessment of the functional impairment seen in individuals with known injury to the brain. This was the first true battery of neuropsychological tests, later refined by Ralph Reitan at the University of Indiana (working closely with neurosurgery colleagues), culminating in the Halstead-Reitan neuropsychological test battery, which is still in use today. A contemporary of Halstead's in Russia, the neurologist and psychoanalyst A.R. Luria, was carefully studying aphasia in the 1930s and later did extensive single-case studies in the 1940s of the effect of brain injury on the behavior of individuals, many who were injured in World War II. The legacy of Luria is reflected in contemporary neuropsychological assessment that emphasizes an individualized, hypothesis-testing approach to neuropsychological assessment, including careful observation of the qualitative responses of the patient examinee.

There are currently a wide variety of specialized neuropsychological test batteries that have been constructed to examine specific diseases or disorders, many of which are designed to be administered following one or more cognitive screening tests. There are also dozens of single neuropsychological tests designed to assess very specific areas of brain functioning. Many of the

newer test batteries are hypothesis driven and symptom specific such that individual patients with the same medical diagnosis or disorder may be given different component subtests depending upon their specific presenting symptoms and performance on the initial tests given (the flexible battery approach). There also remain several fixed neuropsychological test batteries where all patients – or all patients with a given diagnosis or disorder – are given the same tests. Irrespective of whether a fixed or flexible battery approach is employed, the performance of the examinee is customarily compared relative to normative data of individuals without neurological disorder matched by age, gender, and education in order to arrive at reliable and valid conclusions. The earliest neuropsychological test batteries were designed to accurately and efficiently assess the type and extent of specific brain problems and the area or areas of the brain affected and were quite successful in doing so. At the present time, neuropsychological assessment can extend localization information already available from neuroimaging studies and also address in addition many more issues related to treatment planning, educational planning, and the impact of brain problems on a variety of tasks of daily living, some of which are listed in Table 2.

Cross-References

- ▶ Cognitive Function
- ▶ Cognitive Impairment
- ▶ Neurological

References and Readings

Armstrong, C., & Morrow, L. (Eds.). (2010). *Handbook of medical neuropsychology*. New York: Springer.
Davis, A. (Ed.). (2010). *Handbook of pediatric neuropsychology*. New York: Springer.
Feinberg, T., & Farah, M. (Eds.). (2003). *Behavioral neurology and neuropsychology* (2nd ed.). New York: McGraw-Hill.
Grant, I., & Adams, K. M. (Eds.). (2009). *Neuropsychological assessment of neuropsychiatric and neuromedical disorders*. New York: Oxford University Press.
Kolb, B., & Whishaw, I. Q. (2008). *Fundamentals of human neuropsychology* (6th ed.). New York: Worth.
Kreutzer, J., DeLuca, J., & Caplan, B. (2011). *Encyclopedia of clinical neuropsychology*. New York: Springer.
Lezak, M. D., Howieson, D., & Loring, D. W. (2004). *Neuropsychological assessment* (4th ed.). New York: Oxford University Press.
Marcotte, T. D., & Grant, I. (Eds.). (2010). *Neuropsychology of everyday functioning*. New York: Guilford Press.
McCarthy, R.A., & Warrington, E.K. (in press). *Cognitive neuropsychology: a clinical introduction* (2nd ed.). London: Elsevier (Academic Press).
Strauss, E., Sherman, M. S., & Spreen, O. (2006). *A compendium of neuropsychological tests: Administration, norms, and commentary*. New York: Oxford University Press.

Neurotensin

Yori Gidron
Faculty of Medicine and Pharmacy,
Free University of Brussels (VUB), Jette,
Belgium

Definition

Neurotensin (NT) is a multipotent 13-amino acid neuropeptide, originally found in the hypothalamus of cattle. It plays multiple physiological and pathological roles since it affects the nervous, cardiac, immune, and gastrointestinal systems, among others. NT is found in the central nervous system (CNS) and in the gastrointestinal system. It seems to play a role as an endogenous antipsychotic peptide and a role in colorectal cancer (Mustain, Rychahou, & Evers, 2011). In cancer, NT and its receptor neurotensin receptor 1 have been found to play oncogenic roles. They mark prognosis in breast, lung, and head and neck cancers. Furthermore, NT seems to play a role in tumor growth as it acts as a growth factor as well as in metastasis (Dupouy et al., 2011). These have important implications for drug development in the treatment of cancers as well. Dysregulation of NT also plays a role in schizophrenia and in the sensitizing reaction toward drugs, thus playing a possible role in addiction

as well. Given this knowledge, NT can be a target in the treatment of such diseases (Caceda, Kinkead, & Nemeroff, 2006). NT is closely related to the dopaminergic system and has thus been studied in relation to diseases related to dopamine including Parkinson's disease. NT is widely distributed in the CNS and is projected in numerous circuits including the mesocorticolimbic circuit, implicated in pain and in the stress response. Given its multipotent roles in regulation of behavior and in onset of diseases, it requires further research in behavior medicine as well.

Cross-References

► Addictive Behaviors

References and Readings

Cáceda, R., Kinkead, B., & Nemeroff, C. B. (2006). Neurotensin: Role in psychiatric and neurological diseases. *Peptides, 27*, 2385–2404.

Dupouy, S., Mourra, N., Doan, V. K., Gompel, A., Alifano, M., & Forgez, P. (2011). The potential use of the neurotensin high affinity receptor 1 as a biomarker for cancer progression and as a component of personalized medicine in selective cancers. *Biochimie, 93*, 1369–1378.

Mustain, W. C., Rychahou, P. G., & Evers, B. M. (2011). The role of neurotensin in physiologic and pathologic processes. *Current Opinion in Endocrinology, Diabetes, and Obesity, 18*, 75–82.

Neurotic Anger, Subcategory of Anger

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

This term refers to a type of anger which mainly reflects the affective component of anger. Neurotic anger (or neurotic hostility) is contrasted with antagonistic anger (or antagonistic hostility), which refers mainly to the behavioral component of anger (Dembroski & Costa, 1987). Neurotic anger includes feelings of anger and is associated with the personality dimension of neuroticism-emotional stability, with neuroticism being the tendency to attend to, experience, and report negative affect. In contrast, antagonistic anger refers mainly to the agreeable-antagonism dimension of personality and includes disagreeable behavior, argumentativeness, rudeness, being evasive, and lack of cooperation. This distinction is of great importance in behavior medicine because studies have shown that mainly antagonistic, but not neurotic anger/hostility, is the element of anger/hostility predictive of cardiovascular reactivity to stress and of coronary artery disease (Felsten, 1996). Furthermore, some studies even suggest that neuroticism (which includes neurotic anger) may be a factor unrelated to survival (Costa & McCrae, 1987). The latter authors suggested that neuroticism (and neurotic anger) is related mainly to self-reported but not to objective indices of health, primarily because self-reported indices are biased by perceptions and reporting of physical and emotional symptoms, strongly related to neuroticism. These perceptions are thought to involve less physiological overactivity, while antagonistic anger/hostility is thought to involve greater physiological reactivity, explaining its prediction of cardiac disease (Felsten, 1996). It has been suggested that use of objective measures of disease could partly solve this issue as well as use of measures of antagonistic anger/hostility. On the other hand, clinically, while individuals with neurotic anger may not always be more ill on objective measures, they suffer from excessive distress, for which they require psychological assistance.

Cross-References

► Anger-in
► Anger-out
► Coronary Artery Disease
► Neuroticism

References and Readings

Costa, P. T., & McCrae, R. R. (1987). Neuroticism, somatic complaints, and disease: Is the bark worse than the bite? *Journal of Personality, 55*, 299–316.

Dembroski, T. M., & Costa, P. T., Jr. (1987). Coronary prone behavior: Components of the type A pattern and hostility. *Journal of Personality, 55*, 211–235.

Felsten, G. (1996). Five-factor analysis of Buss-Durkee hostility inventory neurotic hostility and expressive hostility factors: Implications for health psychology. *Journal of Personality Assessment, 67*, 179–194.

Neuroticism

Leigh A. Sharma
Department of Psychology, University of Iowa, Kenosha, WI, USA

Synonyms

Negative affectivity; Negative emotionality

Definition

Neuroticism, broadly defined, refers to an individual's tendency to experience negative affect or negative emotional states (e.g., anger, anxiety, hostility, sadness); those high in neuroticism react more strongly to negative stimuli (e.g., threat, frustration, loss) than those low in neuroticism. Structural models of personality establish that neuroticism is linked to Digman's alpha, composed of negative emotionality and disinhibition, in a two-factor model; negative affectivity/negative emotionality splits from disinhibition in three- and four-factor models and emerges as neuroticism in a five-factor model (Markon, Krueger, & Watson, 2005). Most broad personality measures include a neuroticism scale (e.g., measures of the five-factor model of personality), and as early as 1984, authors were demonstrating the high levels of relations among these scales (Watson & Clark, 1984). Neuroticism scores tend to peak in adolescence and decline moderately through adulthood (Roberts & Mroczek, 2008), and evidence demonstrates that females tend to score slightly but significantly higher than males (Costa, Terracciano, & McCrae, 2001).

Neuroticism is a dimension of normal personality, though it is linked to several psychological phenomena and behavioral tendencies. Evidence demonstrates neuroticism acts as a nonspecific vulnerability to the internalizing disorders (e.g., anxiety, depression) and that it can account for a portion of the comorbidity among them (Malouff, Thorsteinsson, & Schutte, 2005). Further, after controlling for sociodemographic variables, neuroticism has been linked to higher rates of smoking, drinking alcohol, and using illegal drugs; poor physical health; lower quality of life; and mortality from all causes (Lahey, 2009).

Cross-References

- ▶ Negative Affect
- ▶ Negative Thoughts
- ▶ Personality

References and Readings

Costa, P. T., Terracciano, A., & McCrae, R. R. (2001). Gender differences in personality traits across cultures: Robust and surprising findings. *Journal of Personality and Social Psychology, 81*, 322–331.

Lahey, B. B. (2009). Public health significance of neuroticism. *American Psychologist, 2009*, 241–256.

Malouff, J. M., Thorsteinsson, E. B., & Schutte, N. S. (2005). The relationship between the five-factor model of personality and symptoms of clinical disorders: A meta-analysis. *Journal of Psychopathology and Behavioral Assessment, 27*, 101–114.

Markon, K. E., Krueger, R. F., & Watson, D. (2005). Delineating the structure of normal and abnormal personality: An integrative hierarchical approach. *Journal of Personality and Social Psychology, 88*(1), 139–157.

Roberts, B. W., & Mroczek, D. (2008). Personality change in adulthood. *Current Directions in Psychological Science, 17*, 31–35.

Watson, D., & Clark, L. A. (1984). Negative affectivity: The disposition to experience aversive emotional states. *Psychological Bulletin, 96*(3), 465–490.

Neurotransmitter

Susan Dorsey
School of Nursing, University of Maryland, Baltimore, MD, USA

Definition

A neurotransmitter is a naturally occurring (endogenous) chemical messenger that passes a message from one neuron to the next by being released into the synaptic gap from the axon of the presynaptic neuron and then attaching itself to the receptor on the dendrite of the postsynaptic neuron.

Neurotransmitters allow neurons to pass on both excitatory and inhibitory messages when they fire. Excitatory messages mean that the next neuron is more likely to fire having received the message, while inhibitory messages mean that the next neuron is less likely to fire.

Some chemicals act as both neurotransmitters and hormones. Neurotransmitters can only influence neurons that come into contact with by traveling over the very small gaps at synapses. In contrast, hormones are secreted by endocrine glands into the blood stream, which carries then around the body and enables them to affect cells that are distant from their point of origin. Another major difference is that neurotransmitters exert their influence for a very brief time: reuptake or chemical decomposition takes place very quickly. In contrast, once released into the blood stream, hormones remain present, and hence active, for much longer.

Cross-References

- ▶ Central Nervous System
- ▶ Dopamine
- ▶ Epinephrine
- ▶ Serotonin

New Drug Development

▶ Pharmaceutical Industry: Research and Development

NHIS

▶ National Health Interview Survey

Nicotine

Motohiro Nakajima and Mustafa al'Absi
University of Minnesota Medical School, University of Minnesota, 235 School of Medicine, Duluth, MN, USA

Synonyms

Cigarette; Smoking; Tobacco

Definition

Nicotine is a psychostimulant alkaloid that is addictive and heavily used in cigarettes and other tobacco-related products. It is primary metabolized by the liver to cotinine, and the average half-life is 2 h.

Description

Upon administration, nicotine binds to nicotinic cholinergic receptors (nAChRs), ligand-gated ion channels composed of several subunits, that are located in central and peripheral nervous systems. Stimulation of nAChRs by nicotine leads to the release of various neurotransmitters. Of those, dopamine secreted in the mesolimbic area and the frontal cortex mediates pleasurable experience and rewarding pathways linked

to the positive reinforcement of nicotine (Benowitz, 2010). Other neurotransmitters, such as norepinephrine, acetylcholine, glutamate, serotonin, beta-endorphin, and gamma-aminobutyric acid (GABA), are also released in response to nicotine and mediate its effects on attention, tension reduction, and appetite suppression. These symptoms are commonly cited motivators of tobacco use by smokers. Nicotine activates the sympathetic nervous system which leads to the release of epinephrine from the adrenal medulla, eventually increasing heart rate, blood pressure, and cardiac output. Nicotine excites paraventricular nucleus in the hypothalamus leading to the release of corticotrophin-releasing factors (CRF), causing the release of adrenocorticotropin (ACTH) from the anterior pituitary, promoting the production of cortisol in the adrenal cortex (i.e., hypothalamic-pituitary-adrenocortical (HPA) axis). Nicotine-induced activation of cardiovascular and HPA functions are very similar to those changes observed during stress. Nicotine modulates pain perception: It induces analgesia via endogenous opioid system. Nicotine may also play a role in stress-induced analgesia through increased activity in the cardiovascular system. Taken together, acute exposure to nicotine activates multiple central nuerochemical regulatory systems that influence psychological and physiological processes and behavior. Pharmacological effects of nicotine play an important role in the development of nicotine dependence and addiction.

Repeated exposure to nicotine leads to tolerance or neuroadaptation to the effects of nicotine. Desentization of nAChRs in the central system may contribute to abstinence-related withdrawal symptoms and craving. Nicotine withdrawal is associated with negative affect including anger, tension, depression, difficulty in concentration, impatience, insomnia, and restlessness (Hughes, 2007). Absence of nicotine may also be linked to reduction of cardiovascular and adrenocortical activity. These symptoms typically peak within a few days of nicotine abstinence and may last for 2–4 weeks and are powerful determinants of smoking lapse and relapse (al'Absi, Hatsukami, & Davis, 2005).

Maintenance of nicotine intake may result from the combination of positive and negative reinforcement. Smokers may take up cigarettes to enhance subjective mood and pleasure (positive reinforcement) but also consume them to maintain levels of nicotine in the body to prevent withdrawal symptoms (negative reinforcement). Certain environmental cues associated with smoking may facilitate subsequent smoking behaviors due to conditioning. Genetic component may also contribute to these processes. Furthermore, chronic nicotine use may be related to structural and functional changes in various stress and emotion-regulations systems as well as endogenous pain modulation mechanisms, which may lead to increased withdrawal symptoms and risk for early smoking relapse.

Cross-References

- ▶ Cortisol
- ▶ Dopamine
- ▶ Heart Disease and Smoking
- ▶ Smoking Behavior
- ▶ Stress

References and Readings

al'Absi, M., Hatsukami, D., & Davis, G. L. (2005). Attenuated adrenocorticotropic responses to psychological stress are associated with early smoking relapse. *Psychopharmacology, 181*, 107–117.

Benowitz, N. L. (2010). Nicotine addiction. *The New England Journal of Medicine, 362*, 2295–2303.

Hughes, J. R. (2007). Effects of abstinence from tobacco: valid symptoms and time course. *Nicotine & Tobacco Research, 9*, 315–327.

Nicotine Dependence and Nicotine Addiction

- ▶ Cessation Intervention (Smoking or Tobacco)

Nicotine Patch

Jed E. Rose
Department of Psychiatry, Duke Center for Nicotine & Smoking Cessation Research, Durham, NC, USA

Definition

Nicotine skin patches are drug delivery systems that are worn on the skin and provide the user with a controlled dose of nicotine to assist in smoking cessation, by replacing the nicotine previously obtained from cigarettes. The ability of nicotine to permeate intact skin has been known for decades, but the first published study exploring the therapeutic potential of transdermal nicotine to ameliorate tobacco withdrawal symptoms appeared in 1985 (Rose, Herskovic, Trilling, & Jarvik, 1985). Previous work in the 1970s had developed transdermal patches for the delivery of medications such as scopolamine and nitroglycerin. Theoretically, transdermal drug administration has the advantages over oral delivery in providing a more uniform blood level of a therapeutic agent and avoiding first-pass liver metabolism. However, other theoretical considerations initially argued against the efficacy of providing a steady level of nicotine in treating tobacco dependence, since it was widely believed that smokers were addicted to rapid bolus delivery of nicotine from inhaled smoke, which could not be duplicated using a nicotine patch. Nonetheless, transdermal nicotine has been shown to be efficacious in aiding smoking cessation, approximately doubling success rates over placebo. In 1991, nicotine patches were approved for marketing in the United States as a prescription drug, and they have been available over the counter since 1996. Despite well-established efficacy (Stead, Perera, Bullen, Mant, & Lancaster, 2008), however, long-term success rates (1 year following treatment) are often only 10–15%.

The standard nicotine patch dose is 21 mg/day, similar to that of a pack of cigarettes; however, the delivery is more uniform than with cigarettes. After 6–8 weeks of wearing 21 mg/day patches, weaning doses are often recommended providing 14 mg/day for 2–4 weeks and 7 mg/day for 2–4 weeks. Combination therapy, entailing adding other forms of nicotine replacement, such as nicotine polacrilex (nicotine chewing gum) or nicotine lozenge, to supplement nicotine patch treatment, increases success rates beyond treatment using the nicotine patch alone.

Interestingly, the mechanism of action of nicotine patch therapy has still not been fully elucidated. One hypothesis holds that successful abstinence is facilitated by nicotine replacement due to the alleviation of nicotine withdrawal symptoms. However, while withdrawal alleviation is clearly obtained from nicotine replacement using nicotine patches, other mechanisms may be more important, such as the attenuation of the rewarding effects of cigarette smoking. When a cigarette is smoked after nicotine blood levels have been elevated from wearing nicotine patches, cigarettes are rated less enjoyable (Levin et al., 1994). Moreover, the odds ratio of preventing the first "lapse" following an attempt to quit smoking is only marginally increased by nicotine patch treatment. In contrast, the progression from an initial lapse to a relapse is markedly reduced (Shiffman et al., 2006). These results suggest that nicotine patch treatment helps smokers remain abstinent largely by affecting their reaction to the first cigarette smoked in a way that discourages the subsequent reuptake of smoking.

In a similar vein, studies in the last several years have shown that initiating nicotine patch treatment before a target quit date, while smokers temporarily continue to smoke, significantly enhances efficacy (Rose, Herskovic, Behm, & Westman, 2009). Thus, as the rewarding effects of smoking are attenuated, smoking behavior declines, and smokers are better able to succeed in quitting when they reach their target quit date. Although not yet approved in the United States, pre-cessation use of nicotine

patch has been approved in Australia, Canada, and the United Kingdom.

Cross-References

- Nicotine
- Smoking Behavior

References and Readings

Levin, E. D., Westman, E. C., Stein, R. M., Carnahan, E., Sanchez, M., Herman, S., et al. (1994). Nicotine skin patch treatment increases abstinence, decreases withdrawal symptoms and attenuates rewarding effects of smoking. *Journal of Clinical Psychopharmacology, 14*, 41–49.

Rose, J. E., Herskovic, J. E., Behm, F. M., & Westman, E. C. (2009). Pre-cessation treatment with nicotine patch significantly increases abstinence rates relative to conventional treatment. *Nicotine & Tobacco Research, 11*(9), 1067–1075.

Rose, J. E., Herskovic, J. E., Trilling, Y., & Jarvik, M. E. (1985). Transdermal nicotine reduces cigarette craving and nicotine preference. *Clinical Pharmacology and Therapeutics, 38*, 450–456.

Shiffman, S., Scharf, D. M., Shadel, W. G., Clark, D. B., Gwaltney, C. J., Paton, S. M., et al. (2006). Analyzing milestones in smoking cessation: Illustration in a nicotine patch trial in adult smokers. *Journal of Consulting and Clinical Psychology, 74*(2), 276–285.

Stead, L. F., Perera, R., Bullen, C., Mant, D., & Lancaster, T. (2008) Nicotine replacement therapy for smoking cessation. *Cochrane Database of Systematic Reviews 2008* (*1*), Art. No.: CD000146. DOI:10.1002/14651858.CD000146.pub3.

Night-Shift Workers and Health

Alyssa Haney[1] and Michele L. Okun[2]
[1]Department of Psychiatry, School of Medicine, University of Pittsburgh, Pittsburgh, PA, USA
[2]Sleep Medicine Institute and Department of Psychiatry, School of Medicine, University of Pittsburgh, Pittsburgh, PA, USA

Synonyms

Grave yard shift

Definition

Shift work refers to a job schedule in which employees work hours other than the standard hours of 8 a.m. to 5 p.m. or a schedule other than the standard workweek – Monday through Friday in the USA.

Description

Human beings are by nature a diurnal species with a natural tendency among adults to sleep at night and be most active during the day. Society is largely structured to facilitate such tendencies, with most social interactions, familial gatherings and work hours during the daylight hours. However, there is often occasion to diverge from these norms especially for work purposes. Today's society is driven by a 24 h mentality which demands that over 8.6 million people (in the USA) perform shift work (Kryger, Roth & Derent, 2005).

Circadian factors are a primary determinant of one's ability to cope with shift work. Humans are biologically wired to sleep during the night and be most active during the day. Certain biological processes determine the body's natural sleep-wake cycle, particularly the secretion of melatonin in response to the light-dark cycle. Such processes are not easily changed so as to shift the natural cycle. Some evidence indicates that sleep phase adjustment for a night worker, as measured by urine melatonin, can take as long as 5–6 days (90 min phase delay per day) before melatonin onset is readjusted to the new schedule.

In addition to endogenous determinants of the sleep cycle, there are other factors known as zeitgebers (time givers) that contribute to one's perceived normal sleep-wake cycle. Such zeitgebers include social factors like meal times and social gatherings and natural factors like daylight and night. These timing cues help orient the body to a certain time schedule and to encourage humans to be active during the day. However, for a night-shift worker who must be awake during the night, these zeitgebers can work

against an individual's inclination to remain awake at night resulting in dysregulated immune, hormonal, and cardiovascular function, thereby contributing to poor health.

Domestic factors also play a big role in sleep and subsequent health of the shift worker. For the night worker, social, familial, and societal obligations often impinge on sleep time since they occur in opposition to the night worker's schedule. Moreover, many shift workers have families and homes to care for that often require significant attention during daylight hours. Not only do these domestic factors inhibit a shift worker from obtaining adequate sleep during the day but they often impact the quality of the sleep he/she does get. Several consequences can arise from poor coping with a shift-work lifestyle, including a predisposition to depression and anxiety (Akerstedt, 2003).

Not surprisingly, shifting of one's sleep to the daytime hours can have negative consequences (Akerstedt, 2003). Not only is shift work (and daytime sleep) in contradiction to most of the biological system's circadian rhythms, but it is a strong predictor in the development of chronic sleep disorders. For instance, shift workers have been found to have sleep maintenance insomnia as opposed to sleep onset insomnia, which results in a constant state of sleep deprivation. Surveys in Europe and in the USA have found that night workers get approximately 10 h less sleep per week than day workers. Akerstedt and colleagues found that there is a significant loss of Stage 2 and REM sleep among shift workers. Although a night worker's sleep loss can be partially attenuated by sleeping longer on days off, they are unable to "repay the debt" and still experience chronic sleep deprivation and its consequences.

Health consequences of shift work include cardiovascular disease, diabetes, and obesity. There are also associations with stomach problems and ulcers, increased depression, and risk for injury or accidents. Many of these adverse outcomes stem from dysregulation of metabolic, digestive, and immune processes that maintain alignment with the circadian rhythm. While important and often necessary for many people to perform shift work, it is imperative to understand and appreciate the significant toll that shift work can have on health and social relationships (Akerstedt, 1990).

Cross-References

- ▶ Cardiovascular Disease
- ▶ Metabolic Syndrome
- ▶ Sleep Deprivation

References and Readings

Akerstedt, T. (1990). Psychological and psychophysiological effects of shift work. *Scandinavian Journal of Work, Environment & Health, 16*(Suppl. 1), 67–73.

Akerstedt, T. (2003). Shift work and disturbed sleep/wakefulness. *Occupational Medicine, 53*(2), 89–94.

Kryger, M., Roth, T., & Derent, W. (2005). *Principles and practice of sleep medicine*. Philadelphia: Elsevier Saunders.

van Drongelen, A., Boot, C. R., Merkus, S. L., Smid, T., & van der Beek, A. J. (2011). The effects of shift work on body weight change – A systematic review of longitudinal studies. *Scandinavian Journal of Work, Environment & Health, 37*(4), 263–275.

Nitric Oxide Synthase (NOS)

Sarah Aldred
School of Sport and Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

Synonyms

Endothelial nitric oxide synthase (eNOS); Inducible nitric oxide synthase (iNOS); Neuronal nitric oxide synthase (nNOS)

Definition

Nitric oxide synthase (NOS) is an enzyme which catalyzes the formation of nitric oxide (NO).

NO is a free radical gas, which acts as a vasodilator, and is formed from the amino acid arginine. NOS comprises a family of enzymes which were first described in 1989. NOS is present in the human body in three distinct isoforms: neuronal NOS (nNOS) found predominantly in neuronal tissue, inducible NOS (iNOS) being inducible in a wide range of cells and tissues, and endothelial NOS (eNOS) which was first found in vascular endothelial cells.

Description

Three distinct genes for the human NOS isoforms exist, with 51–57% homology between the human isoforms in primary amino acid sequence. The similarity in genomic structure indicates a common ancestral NOS gene. The isoforms are classified by the tissue in which they were originally found, although it is now known that expression of these enzymes also occurs in other cell types, such as cardiac muscle, skeletal muscle, and blood platelets. The NOS enzymes are dimeric in their active form, and function is facilitated by the cofactors NADPH (nicotinamide adenine dinucleotide phosphate-oxidase) and tetrahydrobiopterin (BH_4). Other cofactors are calmodulin/calcium, heme, flavin mononucleotide (FMD), and flavin adenine dinucleotide (FAD). A summary of the reaction catalyzed by NOS can be written as:

$$\mathrm{L-arginine} + 2\mathrm{NADPH} + \mathrm{O_2} = \mathrm{L-citrulline} + \mathrm{NO} + 2\mathrm{NAPD^+}$$

The enzyme consists of two domains, an oxygenase and a reductase domain, which both have catalytic activities. Binding sites for heme and BH_4 reside on the oxygenase domain and are linked to the reductase domain through a binding site for calmodulin. The binding sites for FAD and FMD are also found on the reductase domain. To allow production of NO, the enzyme must be fully coupled through BH_4. Deficiencies in any of the cofactors can influence NO biosynthesis.

iNOS and nNOS are soluble and found predominantly in the cytosol, while eNOS is a membrane-associated protein, docked to regions of the plasma membrane called calveolae.

nNOS

nNOS was initially identified in neuronal cells, but has since been identified in the many other cells including those of the gastrointestinal tract. It has a role in NO production for sphincter relaxation and blood flow and modulates the response to glutamate.

iNOS

iNOS as its name suggests is inducible. It is calcium insensitive, in contrast to the calcium-sensitive isoforms nNOS and eNOS. iNOS is linked with the immune response. Previous research in this field has identified that iNOS is expressed as a result of inflammation, but its role in inflammatory disease is a complex one. iNOS has also been implicated in attenuation or suppression of inflammatory tissue injury. In addition, NO forms part of the nonspecific immune defense mechanism against invading microorganisms. NO and superoxide ions are produced in response to microorganisms and react together to form the reactive ion peroxynitrite which, with NO, has the ability to react with and kill invading cells.

eNOS

eNOS is present in the cells of the endothelium, and its main function is to regulate blood flow and blood pressure. eNOS is activated by the pulsatile flow of blood causing a small release of NO with every heart beat. When intracellular levels of calcium increase, eNOS detaches from the plasma membrane and becomes activated. The requirement for calcium is significantly reduced if the enzyme becomes phosphorylated on one of its serine residues, and phosphorylation is now recognized as a common mechanism for activation of eNOS.

More details can be found in Packer (1996).

Cross-References

▶ Oxidative Stress

References and Readings

Packer, L. (Ed.). (1996). *Nitric oxide, part A: Sources and detection of NO; NO synthase: Sources and detection of NO; NO synthase pt. A (methods in enzymology)* (Vol. 268). San Diego: Academic Press.

Nocebo and Nocebo Effect

Magne Arve Flaten
Department of Psychology, University of Tromsø, Tromsø, Norway

Synonyms

Anxiety; Context effect; Expectancy

Definition

A nocebo is any inert substance, procedure, apparatus, or similar that alone has no effect in the body. The nocebo effect is the psychological and/or physiological response to the nocebo when it is administrated with a suggestion that the substance, procedure, apparatus, or similar will increase pain or unpleasantness, or otherwise harm the individual.

Description

Placebo comes from the Latin word "nocere" which means "I will harm" and has been used in medicine to describe treatments that worsen or induce symptoms in the patient, without having a specific effect on the symptom.

A nocebo effect may occur when a substance, procedure, or other stimulus is administrated to a person together with a suggestion that this will increase pain or unpleasantness, or in some way harm the person. The nocebo effect occurs whether or not the substance or procedure is harmful or painful. Thus, a substance or procedure that induces a placebo effect may, when administrated with the opposite suggestion, induce a nocebo effect (Flaten, Simonsen, & Olsen, 1999). The nocebo effect may consequently be considered the opposite of the placebo effect.

Nocebo effects have mostly been studied in the field of pain. The effect may be observed as an increase in pain report to a painful stimulus after suggestion that the pain will increase, although the stimulus is kept at constant levels. The nocebo effect may also be observed as the induction of pain by a non-painful stimulus that is administrated with information that it will induce pain (Colloca, Sigaudo, & Benedetti, 2008). Nocebo effects have also been observed in asthmatic patients and in nausea, but are not well researched outside the field of pain.

Brain imaging studies have shown that nocebo effects are associated with further increases in brain regions activated by painful stimulation, i.e., a painful stimulus activates certain brain regions (often the anterior cingulate cortex and insula), the activation of which have been found to be further increased by expectation that the pain will become more intense. These effects are the opposite of those observed during placebo analgesia.

One mechanism underlying nocebo effects is most likely anxiety. Information that a procedure or substance will induce pain or may harm induces anxiety, which in turn increases pain. Administration of an anxiolytic reduces nocebo hyperalgesia. Furthermore, blockade of cholecystokinin, a hormone associated with anxiety, has been found to block completely nocebo hyperalgesia (Benedetti, Lanotte, Lopiano, & Colloca, 2007).

Nocebo effects are clinically relevant for diagnostic or therapeutic medical procedures. Injections of anesthetics, e.g., may be made painful, or more painful, if the information provided by health personnel performing these procedures induces nervousness or anxiety in the patient (Varelman, Pancaro, Capiello, & Camann, 2010).

Cross-References

- ▶ Functional Magnetic Resonance Imaging (fMRI)
- ▶ Pain

References and Readings

Benedetti, F., Lanotte, M., Lopiano, L., & Colloca, L. (2007). When words are painful – unraveling the mechanisms of the nocebo effect. *Neuroscience, 147*(2), 260–271.

Colloca, L., Sigaudo, M., & Benedetti, F. (2008). The role of learning in nocebo and placebo effects. *Pain, 136* (1–2), 211–218.

Flaten, M. A., Simonsen, T., & Olsen, H. (1999). Drug-related information generates placebo and nocebo responses that modify the drug response. *Psychosomatic Medicine, 61*(2), 250–255.

Varelman, D., Pancaro, C., Capiello, E. C., & Camann, W. R. (2010). Nocebo-induced hyperalgesia during local anesthetic injection. *Anesthesia and Analgesia, 110*(3), 868–870.

Noise-Related Hearing Loss

► Hearing Impairment (Noise Pollution Related)

Nonadherence

► Adherence

Noncoding RNA

► RNA

Noncommercial Advertising

► Social Marketing

Noncompliance

► Adherence
► Unintentional Nonadherence

Nonexperimental Designs

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Observational designs; Observational studies; Observational study

Definition

There are two fundamental types of study design: experimental and nonexperimental (Piantadosi, 2005). Experimental designs (discussed in their own entry) involve a series of measurements (observations) made under conditions in which the influences of interest are controlled by the research scientist. Nonexperimental studies are often called observational studies, but this term is inaccurate; it does not definitively distinguish between nonexperimental studies and experimental studies, in which observations are also made.

The term "nonexperimental" is not a relative quality judgment compared with experimental. This nomenclature simply distinguishes methodological approaches. In some cases, nonexperimental studies are the only type of medical study that can legitimately be used. If one wishes to examine the potentially negative health impact of a specific influence, such as exposure to nicotine via smoking cigarettes or living close to an environmental toxin, it is not appropriate for the research scientist to exert control over the influence of interest by asking some individuals to smoke or to live in a certain location. Rather, the research scientist makes use of naturally occurring cases of individuals who have and have not smoked and individuals who live close to and far from an environmental toxin to examine a potential relationship between the influence of interest and a specific health outcome.

Case-control studies and cohort studies are examples of nonexperimental designs.

Cross-References

- ▶ Case-Control Studies
- ▶ Cohort Study
- ▶ Experimental Designs

References and Readings

Piantadosi, S. (2005). *Clinical trials: A methodologic perspective* (2nd ed.). Hoboken, NJ: Wiley.

Rothman, K. J., Greenland, S., & Lash, T. L. (2008). Types of epidemiologic studies. In K. J. Rothman, S. Greenland, & T. L. Lash (Eds.), *Modern epidemiololgy* (3rd ed.). Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins.

Nonidentical Twins

- ▶ Dizygotic Twins

Non-insulin-Dependent Diabetes Mellitus

- ▶ Type 2 Diabetes Mellitus

Non-Q Wave Myocardial Infarction

- ▶ Acute Myocardial Infarction

Non-REM Sleep

Salvatore Insana
Western Psychiatric Institute and Clinic, Pittsburgh, PA, USA

Synonyms

N1, N2, N3; Quiet sleep; Sleep stages 1, 2, 3, and 4

Definition

Sleep can be measured with polysomnography (PSG) (see review, Keenan & Hirshkowitz, 2011). Polysomnographically measured sleep can be classified into distinct behavioral categories. Non-Rapid Eye Movement (NREM) sleep is a distinct sleep behavior classification. NREM sleep is a term used to describe, as the name suggests, sleep stages that are not Rapid Eye Movement (REM) sleep. NREM sleep includes sleep stages N1, N2, and N3 as defined by the 2007 American Academy of Sleep Medicine (AASM) sleep scoring manual (Iber, Ancoli-Israel, Chesson, & Quan, 2007). NREM sleep is otherwise known as the combination of sleep stages 1, 2, 3, and 4 as identified by the Rechtschaffen and Kales "classic criteria" (Rechtschaffen & Kales, 1968).

General descriptions of AASM scoring criteria for each NREM sleep stage are briefly stated. N1 is characterized by the expression of slow rolling eye movements, decreased muscle tone, and low-amplitude, mixed-frequency brain activity (4–7 Hz). N2 is characterized by K-complexes (a negative vertex sharp brain wave immediately followed by a positive wave that lasts $\geq$0.5 s) and sleep spindles (a train of brain waves with high frequency [11–16 Hz] and low amplitude that last $\geq$0.5 s). N3 is characterized by slow brain waves that are high amplitude (>75 V), low frequency (0.5–2 Hz), and occur throughout $\geq$20% of a given epoch. N3 sleep includes stages 3 and 4 sleep in the Rechtschaffen and Kales' classification and is also frequently called "delta" or "slow-wave" sleep.

NREM sleep has been observed among all mammals (Siegel, 2009). The function of human sleep, and particularly NREM sleep, is complex and unknown (e.g., Rector, Schei, Van Dongen, Belenky, & Krueger, 2009; Siegel, 2009). Theories about the function of each NREM sleep stage are briefly mentioned. N1 serves as the transitional stage from wake to sleep. N2 components (spindles and K-complexes) are thought to integrate new memories and regulate arousal from sleep (Halász, 2005; Tamminen, Payne, Stickgold, Wamsley, Gareth, & Gaskell, 2010). N3, or rather

the amount of N3, is thought to reflect the homeostatic sleep drive, or one's escalating need for sleep with increasing time awake (Dijk, Brunner, Beersma, & Borbély, 1990). NREM sleep appears central to memory formation; specifically, NREM sleep and REM sleep complement each other to process and consolidate various types of memory (Diekelmann & Born, 2010).

Human development: From birth to approximately 6 months post-term, infant sleep patterns are classified into either REM or NREM; during this period, these classifications are also referred to as active sleep and quiet sleep, respectively (Grigg-Damberger et al., 2007). Following 6 months post-term, infant NREM sleep patterns become more complex, and then can be categorized into N1, N2, and N3 sleep stages – in addition to REM sleep. Across the life span, generally, the percentages of N1 and N2 sleep increase, whereas N3 and REM sleep decrease (Ohayon, Carskadon, Guilleminault, & Vitiello, 2004).

Cross-References

- ▶ Polysomnography
- ▶ REM Sleep
- ▶ Sleep
- ▶ Sleep Architecture

References and Readings

Diekelmann, S., & Born, J. (2010). The memory function of sleep. *Nature Reviews: Neuroscience, 11*, 114–126.

Dijk, D. J., Brunner, D. P., Beersma, D. G., & Borbély, A. A. (1990). Electroencephalogram power density and slow wave sleep as a function of prior waking and circadian phase. *Sleep, 13*, 430–440.

Grigg-Damberger, M., Gozal, D., Marcus, C. L., Quan, S. F., Rosen, C. L., Chervin, R. D., et al. (2007). The visual scoring of sleep and arousals in infants and children. *Journal of Clinical Sleep Medicine, 3*, 201–240.

Halász, P. (2005). K-complex, a reactive EEG graphoelement of NREM sleep: An old chap in a new garment. *Sleep Medicine Reviews, 9*, 391–412.

Iber, C., Ancoli-Israel, S., Chesson, A., Quan, S. F., & American Academy of Sleep Medicine. (2007). *The AASM manual for the scoring of sleep and associated events: Rules, terminology and technical specifications* (1st ed.). Westchester, IL: American Academy of Sleep Medicine.

Keenan, S., & Hirshkowitz, M. (2011). Monitoring and staging human sleep. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5th ed., pp. 1602–1609). St. Louis, MO: Elsevier.

Ohayon, M. M., Carskadon, M. A., Guilleminault, C., & Vitiello, M. V. (2004). Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: Developing normative sleep values across the human lifespan. *Sleep, 27*, 1255–1273.

Rechtschaffen, A., & Kales, A. (1968). *A manual of standardized, techniques and scoring system for sleep stages in human subjects*. Washington, DC: NIH Publication No. 204, US Government Printing Office.

Rector, D. M., Schei, J. L., Van Dongen, H. P., Belenky, G., & Krueger, J. M. (2009). Physiological markers of local sleep. *European Journal of Neuroscience, 29*, 1771–1778.

Siegel, J. M. (2009). Sleep viewed as a state of adaptive inactivity. *Nature Reviews Neuroscience, 10*, 747–753.

Tamminen, J., Payne, J. D., Stickgold, R., Wamsley, E. J., & Gareth Gaskell, M. (2010). Sleep spindle activity is associated with the integration of new memories and existing knowledge. *Journal of Neuroscience, 30*, 14356–14360.

Nonseminoma

- ▶ Cancer, Testicular

Nonsteroidal Anti-inflammatory Medications (NSAIDs)

- ▶ Anti-inflammatory Medications

Nonverbal Communication

Elizabeth Galik
School of Nursing, University of Maryland, Baltimore, MD, USA

Synonyms

Body language; Kinesics

Definition

Nonverbal communication is a complex system of communication that involves sending and receiving messages without the use of words. Nonverbal communication consists of a combination of (1) body movement (kinesics), such as gestures, posture, stance, and facial expressions; (2) touch (haptics); (3) eye contact and gaze, (4) bodily use of physical space, position, and proximity (proxemics), and nonverbal vocalizations that convey emotions or attitudes (paralanguage), such as laughing, crying, whistling, speech hesitancy, and tone. Research has indicated that 50–90% of social meaning is transmitted through nonverbal communication (Knapp & Hall, 2007). Functions of nonverbal communication include expression of emotion and attitude, communication of interpersonal relationships, support or contradiction of verbal communication, reflection of personality, and performance of social rituals, such as greetings and farewells (Argyle, 1988; Argyle & Hinde, 1972). Nonverbal communication is particularly influenced by the function of culture (Agliati, Vescovo, & Anolli, 2006). An understanding of nonverbal communication can be applied widely in a variety of fields such as medicine, business, politics, psychology, education, and the criminal justice system.

References and Readings

Agliati, A., Vescovo, A., & Anolli, L. (2006). A new methodological approach to nonverbal behavior analysis in cultural perspective. *Behavioral Research Methods, 38*(3), 364–371.

Argyle, M. (1988). *Bodily communication* (2nd ed.). New York: Methuen.

Argyle, M., & Hinde, R. A. (1972). *Nonverbal communication*. Oxford: Cambridge University Press.

Buck, R. (1984). *The communication of emotion*. New York: Guilford Press.

Buck, R., & Duffy, R. (1980). Nonverbal communication of affect in brain damaged patients. *Cortex, 16*, 351–362.

Buck, R., & Van Lear, C. A. (2002). Verbal and nonverbal communication: Distinguishing symbolic, spontaneous, and pseudo-spontaneous nonverbal behavior. *Journal of Communication, 52*, 522–541.

Ekman, P., & Friesen, W. V. (1969). Nonverbal leakage and cues to deception. *Psychiatry, 32*, 88–105.

Ekman, P., & Friesen, W. V. (1975). *Unmasking the face*. Englewood Cliffs, NJ: Prentice-Hall.

Knapp, M. L., & Hall, J. A. (2007). *Nonverbal communication in human interaction* (5th ed.). Wadsworth: Thomas Learning.

Ross, E. (1981). The aprosodias: Functional-anatomic organization of the affective components of language in the right hemisphere. *Archives of Neurology, 38*, 561–569.

Norepinephrine/Noradrenaline

Sabrina Segal
Department of Neurobiology and Behavior, University of California, Irvine, CA, USA

Definition

Norepinephrine/noradrenaline is a catecholamine that is released as a stress hormone in the peripheral sympathetic nervous system via the adrenal medulla and initiates the fight or flight response. It is released as a neurotransmitter in the central sympathetic nervous system via noradrenergic neurons.

Norepinephrine is synthesized from dopamine by dopamine beta-hydroxylase through a series of enzymatic steps, beginning with tyrosine hydroxylase. This enzyme converts tyrosine to L-dihydroxyphenylalanine (L-DOPA), which in turn is transformed to dopamine by dopa-decarboxylase and packaged into storage vesicles. The membrane-bound enzyme, beta-hydroxylase, converts dopamine to norepinephrine within storage vesicles.

Cross-References

▶ Catecholamines
▶ Norepinephrine/Noradrenaline
▶ Sympatho-adrenergic Stimulation

References and Readings

Cannon, W. B. (1914). The interrelations of emotions as suggested by recent physiological research. *The American Journal of Psychology, 25*(2), 256–282.

Christensen, N. J. (1982). Catecholamines and essential hypertension. *Scandinavian Journal of Clinical and Laboratory Investigation, 42*, 211–215.
Chrousos, G. P., & Gold, P. W. (1992). The concepts of stress and stress system disorders. Overview of physical and behavioral homeostasis. *Journal of the American Medical Association, 267*(9), 1244–1252.
Clayton, E., & Williams, C. (2000). Adrenergic activation of the nucleus tractus solitaries potentiates amygdale norepinephrine release and enhances retention performance in emotionally arousing and spatial memory tasks. *Behavioral Brain Research, 112*, 151–158.
Coull, J. T. (1994). Pharmacological manipulations of the alpha2-noradrenergic system: Effects on cognition. *Drugs & Aging, 5*, 116–126.
Kalat, J. W. (1992). *Biological psychology* (4th ed.). Belmont, CA: Wadsworth Publishing Company.
van Stegeren, A. H. (2008). The role of the noradrenergic system in emotional memory. *Acta Psychologica, 127*(3), 532–541.

Norms

Chad Barrett
Department of Psychology, University of Colorado Denver, Denver, CO, USA

Synonyms

Social norms

Definition

In the context of psychological assessment, norms refer to sets of scores from well-defined samples that are used for standardizing and interpreting assessments. Norms are derived from sampling one or more groups of individuals and obtaining the distribution of scores on a particular assessment for each group. These sets of scores make it possible to interpret the scores of individuals who are assessed. The score of individual test takers can be compared to the scores from the group to which they belong. This provides a basis for comparison and makes it possible to interpret a person's score. For example, if a high school student obtains a certain score on an aptitude test, there is no way to interpret their score without comparing it to the scores of other high school students. If only 5% of their peers scored better than them, this would indicate that they demonstrated exceptional aptitude on the test relative to their peers. However, if 95% of their peers scored better than them, this would indicate that they demonstrated a diminished aptitude relative to their peers. Given that norms are essential for interpretation, it is important that norms be based on samples that are sufficiently representative of the populations they represent and large enough to minimize standard error (Kaplan & Saccuzzo, 2008; Kline, 2000).

Norms, also called social norms, refer to rules or standards of expected behavior that are shared by members of a certain group. Social norms shape individual group members' perceptions about the acceptability of certain behaviors (injunctive norms) as well as the type, and frequency, of behaviors that other group members perform (descriptive norms). A person's perceptions of social norms are often constructed by observing the behavior of other group members, by communicating with other group members, and by exposure to normative messages through media outlets and from explicit or implicit instruction (Dubios, 2003; Hechter & Opp, 2005). Social norms have been found to influence a variety of health-related behaviors, such as eating and exercise habits, alcohol consumption, smoking, drug use, and seeking medical help (Berkman & Glass, 2000; Borsari & Carey, 2003; McNeill, Kreuter, & Subramanian, 2006; Shaikh, Yaroch, Nebeling, Yeh, & Resnicow, 2008; Sorensen et al., 2007). According to the social norms approach, individuals who engage in unhealthy behaviors often overestimate the extent to which others engage in those unhealthy behaviors and underestimate the extent to which others engage in healthy behaviors. Such misperceptions predict the likelihood of engaging in unhealthy or healthy behaviors. Many interventions that focus on correcting such misperceptions by providing normative feedback have been successful in correcting such misperceptions and in promoting healthier behaviors (Berkowitz, 2003, 2004).

Cross-References

- ▶ Assessment
- ▶ Psychometrics

References and Readings

Berkman, L. F., & Glass, T. (2000). Social integration, social networks, social support, and health. In L. F. Berkman & I. Kawachi (Eds.), *Social epidemiology* (pp. 137–173). New York: Oxford University Press.

Berkowitz, A. D. (Ed.). (2003). *The social norms resource book*. Little Falls, NJ: PaperClip Communications.

Berkowitz, A. D. (2004). *The social norms approach: Theory, research, and annotated bibliography*. Unpublished manuscript. Retrieved November 22, 2011 from http://www.alanberkowitz.com/articles/social_norms.pdf

Borsari, B., & Carey, K. B. (2003). Descriptive and injunctive norms in college drinking: A meta-analytic integration. *Journal of Studies on Alcohol, 64*, 331–341.

Dubios, N. (Ed.). (2003). *A sociocognitive approach to social norms*. New York: Routledge.

Emmons, K. M., Barbeau, E. M., Gutheil, C., Stryker, J. E., & Stoddard, A. M. (2007). Social influences, social context, and health behaviors among working-class, multi-ethnic adults. *Health Education & Behaviour, 34*, 315–334.

Hechter, M., & Opp, K. (Eds.). (2005). *Social norms*. New York: Russell Sage.

Kaplan, R. M., & Saccuzzo, D. P. (2008). *Psychological testing: Principles, applications, and issues*. Belmont, CA: Cengage Learning.

Kline, P. (2000). *The handbook of psychological testing* (2nd ed.). London: Routledge.

McNeill, L. H., Kreuter, M. W., & Subramanian, S. V. (2006). Social environment and physical activity: A review of concepts and evidence. *Social Science & Medicine, 63*, 1011–1022.

Shaikh, A. R., Yaroch, A. L., Nebeling, L., Yeh, M. C., & Resnicow, K. (2008). Psychosocial predictors of fruit and vegetable consumption in adults a review of the literature. *American Journal of Preventative Medicine, 34*, 535–543.

Sorensen, G., Stoddard, A. M., Dubowitz, T., Barbeau, E. M., Bigby, J., Emmons, K. M., et al. (2007). The influence of social context on changes in fruit and vegetable consumption: Results of the healthy directions studies. *American Journal of Public Health, 97*, 1216–1227.

Nosocomephobia

- ▶ Hospital Anxiety

Nosocomial Medical Errors

- ▶ Iatrogenic Conditions

NSTEMI

- ▶ Acute Myocardial Infarction

Null Hypothesis

- ▶ Hypothesis Testing

Numerical Information

- ▶ Data

Numerical Representation of (Biological, Psychological, Behavioral) Information

- ▶ Data

Nurses' Health Study

William Whang
Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Definition

The Nurses' Health Study is a long-term cohort study that has collected information about cardiovascular and other disease risk factors, in addition to lifestyle factors, since its beginning in 1976 (Belanger, Hennekens, Rosner, & Speizer, 1978). At the onset, 121,701 female

registered nurses, aged 30–55 years, completed a questionnaire about their medical history and disease risk factors. The cohort has been followed up every 2 years with mailed questionnaires that update exposure information and inquire about newly diagnosed medical illnesses. Numerous analyses involving NHS data have been performed, including investigation of the impact of smoking cessation on long-term risks of coronary artery disease (Kawachi et al., 1994) and relationship between phobic anxiety and coronary heart disease events (Albert, Chae, Rexrode, Manson, & Kawachi, 2005).

References and Readings

Albert, C. M., Chae, C. U., Rexrode, K. M., Manson, J. E., & Kawachi, I. (2005). Phobic anxiety and risk of coronary heart disease and sudden cardiac death among women. *Circulation, 111*, 480–487.

Belanger, C. F., Hennekens, C. H., Rosner, B., & Speizer, F. E. (1978). The nurses' health study. *The American Journal of Nursing, 78*, 1039–1040.

Kawachi, I., Colditz, G. A., Stampfer, M. J., et al. (1994). Smoking cessation and time course of decreased risks of coronary heart disease in middle-aged women. *Archives of Internal Medicine, 154*, 169–175.

Nutrient Intake

► Nutrition

Nutrition

Steven Gambert
Department of Medicine, School of Medicine, University of Maryland, Baltimore, MD, USA

Synonyms

Dietary requirements; Nutrient intake

Definition

Nutrition is the process by which living organisms obtain nutrients and use them for proper growth and development, cellular metabolism, and repair. It is a process that involves the ingestion, digestion, absorption, transport, assimilation, and excretion of essential nutrients.

Description

Nutrition is the process by which living organisms obtain nutrients and use them for proper growth and development, cellular metabolism, and repair. It is a process that involves the ingestion, digestion, absorption, transport, assimilation, and excretion of essential nutrients. In order to insure an adequate "nutrition," there are a variety of macro and micro-nutrients that must be obtained. While the body has the ability to utilize certain nutrients obtained through nutrition to provide the building blocks for cellular growth, maturation, and repair, some are essential and must be part of one's nutrition. This is illustrated by the fact that although animal and plant protein contain 20 amino acids, 9 are considered "essential" and not able to be produced by the body itself.

Recommended Dietary Allowances are established by the Food and Nutrition Board of the National Research Council and represent the nutrient allowances thought to be necessary for the maintenance of good health (National Research Council, NRC, 1989). Malnutrition is the state associated with either an under or over intake of essential components of one's nutritional requirements including calories and specific nutrients such as protein, fat, minerals, and vitamins. Examples include obesity (an excess intake of calories), marasamus (a deficiency in caloric intake), kwashiorkor (a deficiency in protein intake), among other examples. Certain individuals will require intake above the minimum daily requirement of certain nutrients. One example is calcium. The RDA is 800–1,500 mg of elemental calcium daily. Individuals who are breast-feeding, pregnant, or who have metabolic bone disease should be at the higher range of intake.

Each individual utilizes nutrients differently based on genetic and environmental factors. Muscle has a higher metabolic utilization than fat and individuals with more muscle have a higher caloric requirement to maintain their body weight. Catecholamines and thyroid hormone play a role in our ability to up and downregulate our metabolism as well as the type of food we consume. For example, individuals who "nibble," eating multiple meals throughout the day are metabolically different from those who "gorge" (one meal a day) their dietary intake even at the same level of caloric consumption . Eating only one meal a day (gorger) causes the body to function the rest of the day as it is starving and metabolically downregulating metabolism. Not all foods are also handled the same in the body with only 78% of protein utilized by the body due to the increase in metabolic work needed to break down the protein bonds as compared to fat and carbohydrate that are utilized by the body at 99% and 98%, respectively.

Nutritional assessment may be done by assessing a variety of measures including body mass index. This is a calculated value that is determined by taking the weight in kilograms divided by the height in meters squared. A value of 19–25 is considered to be normal and values over 30 are considered to be obese. Another measure of nutritional status is pre-albumin with values of 20 or greater indicating adequate protein stores. A questionnaire (mini-nutritional assessment) can provide information to predict who is at risk of malnutrition (Kaiser et al. 2009).

Cross-References

- ▶ Carbohydrates
- ▶ Nutrition Data System for Research (NDSR)
- ▶ Obesity

References and Readings

Kaiser, M. J., et al. (2009). Validation of the nutritional assessment short form (MNA8-SF): A practical tool for identification of nutritional status. *The Journal of Nutrition, Health & Aging, 3*, 782–788.

National Research Council (NRC). (1989). Diet and health: Implications for reducing chronic disease risk. *Report of the Committee on Diet and health, Food, and Nutrition Board* (750 pp). Washington, DC: National Academy Press.

Nutrition Data System for Research (NDSR)

Lisa Harnack
Division of Epidemiology and Community Health, School of Public Health, University of Minnesota, Minneapolis, MN, USA

Definition

Nutrition Data System for Research (NDSR) is a Windows-based dietary analysis program designed for the collection and analyses of 24-h dietary recalls and the analysis of food records, menus, and recipes. The system is developed and maintained by the University of Minnesota Nutrition Coordinating Center (ncc.umn.edu).

Description

Overview of NDSR

NDSR is a software application that may be used to quantify the food and nutrient intake of individuals, analyze the nutrient composition of menus, and calculate the nutrient composition of food recipes. Consequently, the program has both research and practice applications in the arena of behavioral medicine. Examples of its use include collection and analysis of 24-h dietary recalls from study participants to evaluate the effect of a nutrition intervention on food and nutrient intake. In clinical settings, NDSR is used to analyze food records kept by patients as a behavioral monitoring tool. Institutions such as hospitals, prisons, and colleges use NDSR in planning nutritionally balanced menus. The program is also used to calculate the nutrient content

of recipes for publication in health promotion pamphlets, cook books, etc.

Food and Nutrient Database

The University of Minnesota Nutrition Coordinating Center (NCC) Food and Nutrient Database serves as the source of food composition information in the software. This database includes over 18,000 foods, including 7,000 brand-name products. Ingredient choices and preparation methods provide more than 160,000 food variants. Values for 162 nutrients, nutrient ratios, and other food components are generated from the database. Also, food group assignments are provided so that intake of food categories such as fruits and vegetables may be calculated. Missing nutrient values are kept to a minimum by obtaining nutrient composition information from a variety of sources and utilizing standardized imputation procedures (Schakel, Sievert, & Buzzard, 1988; Schakel, Buzzard, & Gebhardt, 1997; Westrich, Buzzard, Gatewood, & McGovern, 1994). The database is updated annually to reflect marketplace changes and new analytic data.

24-H Dietary Recall Collection and Analysis

Dietary recall data gathered by interview are entered directly into NDSR. The software searches for foods and brand products by name. Sophisticated search algorithms locate the food, and the interview prompts standardize requests for more detail. Dietary intake data gathered by interview are governed by the multiple-pass approach interview methodology. Four distinct passes provide multiple opportunities for the participant to recall and clarify food intake.

Dietary supplement use may be assessed in conjunction with collection of 24-h dietary recalls using the Dietary Supplement Assessment Module included in NDSR. Use of all types of dietary supplements and nonprescription antacids is queried in the module. The database linked with the module includes over 2,000 dietary supplement products. A "missing product" feature in the software allows the user to add products to the database. The coding of foods and dietary supplements occurs automatically as data are entered. Calculation of nutrients occurs immediately and yields data per ingredient, food, meal, and day in report and analysis file formats.

Food Record, Menu, and Recipe Analysis

Data entry windows tailored for direct entry of food records, menus, and recipes are provided in NDSR. The coding of foods occurs as data are entered. Calculation of nutrients occurs simultaneously, allowing for generation of reports and analysis files immediately following completion of data entry.

Background and History

NDSR was developed and is maintained by the University of Minnesota Nutrition Coordinating Center (NCC). NCC was established in 1974 by the National Heart, Lung, and Blood Institute (NHLBI) to support the food-coding and nutrient-analysis needs of two historically significant national collaborative research programs – the Multiple Risk Factor Intervention Trial (MRFIT) and the Lipid Research Clinics (LRC). For these studies, a standardized mainframe computer-based food-coding and nutrient-analysis system was created by NCC in collaboration with NHLBI and outside experts in nutrition, statistics, computer science, and education (Dennis, Ernst, Hjortland, Tillotson, & Grambsch, 1980). This system was designed for in-house use, with NCC staff responsible for using it to code foods for nutrient calculations.

By 1977, NCC services were made available to other researchers studying the impact of diet and nutrition on various health conditions, including cardiovascular disease, cancer, hypertension, obesity, diabetes, age-related eye disease, and acquired immune deficiency syndrome (HIV/AIDS). In 1988, NCC released Nutrition Data System (NDS), a DOS-based software program designed to provide a standardized interview and direct data entry for collection of dietary intake (Feskanich, Sielaff, Chong, & Bartsch, 1989). For the first time, coding of foods and amounts was computerized, providing immediate calculation of nutrient data. The software was developed for distribution to researchers for use on their own computers. A user manual, technical support, and

training were among the services developed by NCC to support those using NDS.

Since 1998, NCC has worked to keep NDSR up-to-date with computer hardware and software advances, and dietary intake assessment methodological improvements. In addition, major expansion to the nutrients and foods in the NCC Food and Nutrient Database has been made to keep the database current, with the ever-expanding food marketplace and the growing number of nutrients and other food components of interest to researchers and of importance to human health.

Cross-References

- ▶ Fat, Dietary Intake
- ▶ Nutrition
- ▶ Nutritional Supplements

References and Readings

Dennis, B., Ernst, N., Hjortland, M., Tillotson, J., & Grambsch, V. (1980). The NHLBI nutrition data system. *Journal of the American Dietetic Association, 77*, 641–647.

Feskanich, D., Sielaff, B., Chong, K., & Bartsch, G. (1989). Computerized collection and analysis of dietary intake information. *Computer Methods and Programs in Biomedicine, 30*, 47–57.

Nutrition Data System for Research. http://www.ncc.umn.edu/products/ndsr.html.

Schakel, S., Buzzard, M., & Gebhardt, S. (1997). Procedures for estimating nutrient values for food composition databases. *Journal of Food Composition and Analysis, 10*, 102–114.

Schakel, S., Sievert, Y., & Buzzard, M. (1988). Sources of data for developing and maintaining a nutrient database. *Journal of the American Dietetic Association, 88*, 1268–1271.

Westrich, B., Buzzard, M., Gatewood, L., & McGovern, P. (1994). Accuracy and efficiency of estimating nutrient values in commercial food products using mathematical optimization. *Journal of Food Composition and Analysis, 77*, 223–239.

Nutritional Supplement

- ▶ Nutritional Supplements

Nutritional Supplements

Steven Gambert
Department of Medicine, School of Medicine, University of Maryland, Baltimore, MD, USA

Synonyms

Dietary supplement; Food supplement; Nutritional supplement

Definition

A nutritional supplement is a product available in any form that is intended to provide nutrients that are considered to either be lacking or insufficient in the diet. These may contain one of more of the following: vitamins, minerals, fiber, fatty acids, amino acids, and herbs or other botanicals. It is intended for ingestion in pill, capsule, tablet, powder, or liquid form and is not to be used as a conventional food or as a primary form of meal.

Description

A nutritional supplement is a product available in any form that is intended to provide nutrients that are considered to either be lacking or insufficient in the diet (US Food and Drug Administration). These may contain one of more of the following: vitamins, minerals, fiber, fatty acids, amino acids, and herbs or other botanicals. It is intended for ingestion in pill, capsule, tablet, powder, or liquid form and is not to be used as a conventional food or as a primary form of meal. While various countries regulate nutritional supplements in different ways, in the United States, nutritional supplements are regulated by the Food and Drug Administration (FDA) as a category of foods, not drugs, and are not regulated by the National Academy of Sciences that provides the Recommended Dietary Allowances (RDA's) (National Research Council, 1989). Anyone wishing to market a dietary/nutritional supplement that

contains a "new dietary ingredient" as defined as "a vitamin, mineral, herb or other botanical (Goldman, 2001), amino acid, or dietary substance for use by man to supplement the diet by increasing total dietary intake or a concentrate, metabolite, constituent, extract, or combination of any of these must notify the FDA prior to marketing and receive approval as per the provisions of the Dietary Supplement and Health Education Act of 1994. Nutritional supplements must not be used for diagnosis, treatment, cure, or prevention of any disease. In 2007, the FDA implemented a 'current good manufacturing practices' policy to ensure that nutritional supplements are produced in a 'quality manner,' do not contain contaminants or impurities, and are accurately labeled." There are a wealth of products available that are characterized as nutritional supplements with a majority of persons in the USA reporting intake of at least one. While some nutritional supplements may have benefits based on limited studies, many have not been proven to have clinically significant benefits (Blendon, DesRoches, Benson, Brodie, & Altman, 2001).

Cross-References

► Nutrition

References and Readings

Blendon, R. J., DesRoches, C. M., Benson, J. M., Brodie, M., & Altman, D. E. (2001). Americans views on the use and regulation of dietary supplements. *Archives of Internal Medicine, 161*(6), 805–810.

Goldman, P. (2001). Herbal medicines today and the roots of modern pharmacology. *Annals of Internal Medicine, 135*, 594–600.

National Research Council. (1989). *Recommended dietary allowances* (10th ed.). Washington, DC: National Academy Press.

US Food and Drug Administration. (2012). *Dietary supplement information*. Retrieved from http://www.cfsan.fda.gov/dms/supplmnt.html

O

Obesity

Chad D. Jensen[1], Amy F. Sato[2] and Elissa Jelalian[3]
[1]Department of Psychology, Brigham Young University, Provo, UT, USA
[2]Department of Psychology, Kent State University, Kent, OH, USA
[3]Department of Psychiatry, Rhode Island Hospital, Brown Medical School, Providence, RI, USA

Synonyms

Overweight

Definition

The prevalence of obesity has increased dramatically in the USA and worldwide over the past 30 years. Obesity and its consequences represent a significant public and individual health concern. Generally, obesity results when there is a sustained energy imbalance such that caloric intake (i.e., through food and beverages) exceeds caloric output (i.e., body functions, physical activity). In adults, obesity is defined as a body mass index (kg/m^2) of 30 or higher and overweight is defined as a BMI between 25.0 and 29.9. In children and adolescents, obesity is defined as a BMI at or above the 95th percentile for age and gender, and overweight is defined as a BMI at or above the 85th percentile. Current estimates suggest that 32% of men and 35% of women in the USA meet criteria for obesity (Flegal, Carroll, Ogden, & Curtin, 2010). Similarly, almost 17% of US children and adolescents (ages 2–19) are obese and 32% of youth are either overweight or obese (Ogden, Carroll, Curtin, Lamb & Flegal, 2010). Although increases in prevalence may have slowed over the past 10 years, researchers estimate that if the trajectory of obesity prevalence continues at present rates, almost half of US adults will meet criteria for obesity by 2020 (Stewart, Cutler & Rosen, 2009).

Obesity confers significant risk for individual medical, psychological, and social risks. Deleterious health effects strongly associated with obesity include cardiovascular disease, type 2 diabetes, certain types of cancers, sleep apnea, asthma, joint problems, depression, and suboptimal quality of life. Additionally, obesity represents a sizeable economic burden on society. The direct costs of medical care for adult obesity-related illness have been estimated to exceed $147 billion annually (Finkelstein, Trogdon, Cohen & Deitz, 2009). Moreover, estimates suggest that increases in obesity-related illness explain 27% of the increase in overall health-care spending in the USA between 1987 and 2001 (Thorpe, Florence, Howard, & Joski, 2004). Direct costs of childhood and adolescent obesity-related illness are estimated at 14.1 billion for prescription, outpatient, and emergency costs (Trasande & Chatterjee, 2009) in

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

addition to 237.6 million for inpatient treatment (Trasande, Liu, Freyer, & Weitzman, 2009).

Given the high prevalence of obesity in children and adults, prevention and treatment approaches have received increasing attention. Individual and group interventions for obesity have adopted behavioral (i.e., "lifestyle"), pharmacological, and surgical approaches (Wilfley, Tibbs, Van Buren, Reach, Walker & Epstein 2007; Wadden, Webb, Moran & Bailer, 2012). Establishing a healthy eating plan and attaining recommended levels of physical activity are key lifestyle components for both obesity prevention and treatment.

Cross-References

- ▶ Diabesity in Children
- ▶ Nutrition
- ▶ Physical activity

References and Readings

Finkelstein, E. A., Trogdon, J. G., Cohen, J. W., & Dietz, W. (September/October 2009). Annual medical spending attributable to obesity: Payer-and service-specific estimates. *Health Affairs*, *28*(5), w822–w831.

Flegal, K. M., Carroll, M. D., Ogden, C. L., & Curtin, L. R. (2010). Prevalence and trends in obesity among US adults, 1999–2008. *JAMA: The Journal of the American Medical Association*, *303*(3), 235–241.

Ogden, C. L., Carroll, M. D., Curtin, L. R., Lamb, M. M., & Flegal, K. M. (2010). Prevalence of high body mass index in US children and adolescents, 2007–2008. *JAMA: The Journal of the American Medical Association*, *303*(3), 242–249.

Stewart, S. T., Cutler, D. M., & Rosen, A. B. (2009). Forecasting the effects of obesity and smoking on U.S. life expectancy. *N England Journal of Medicine*, *361*(23), 2252–2260.

Thorpe, K. E., Florence, C. S., Howard, D. H., & Joski, P. (2004). The impact of obesity on rising medical spending. *Health Affairs*, *23*, 480–486.

Trasande, L., & Chatterjee, S. (2009). The Impact of Obesity on Health Service Utilization and Costs in Childhood. *Obesity*, *17*, 1749–1754.

Trasande, L., Liu, Y., Fryer, G, & Weitzman, M. (2009). Effects of childhood obesity on hospital care and costs, 1999–2005. *Health Affairs*, *28*, w751–w760.

Wadden, T. A., Webb, V. L., Moran, C. H., & Bailer, B. A. (2012). Lifestyle modification for obesity. *Circulation*, *125*(9), 1157–1170.

Wilfley, D. E., Tibbs, T. L., Van Buren, D. J., Reach, K. P., Walker, M. S., & Epstein L. H. (2007). Lifestyle interventions in the treatment of childhood overweight: A meta-analytic review of randomized controlled trials. *Health Psychol*ogy, *26*, 521–532.

Obesity in Children

Elizabeth R. Pulgaron[1] and Alan M. Delamater[2]
[1]Department of Pediatrics, University of Miami, Miami, FL, USA
[2]Department of Pediatrics, University of Miami Miller School of Medicine, Miami, FL, USA

Synonyms

Overweight children

Definition

Obesity in children is defined as having a body mass index (BMI) at or above the 95th percentile compared to peers of the same age and gender.

Description

BMI is calculated from height and weight and plotted on a growth chart according to the child's age and gender. Obesity occurs when over time more calories are consumed than expended. Many factors affect obesity including poor eating habits, lack of exercise, family history, and psychosocial problems. Very few cases of obesity can be attributed to a specific disease.

Recent estimates indicate that nearly 10% of infants and toddlers and 12% of children and adolescents aged 2 through 19 years meet criteria for obesity in the United States (Ogden, Carroll, Curtin, Lamb, & Flegal, 2010). Correlates of childhood obesity include genetics, environment, metabolism, behavior, culture, and SES. Children from ethnic minority backgrounds and lower

socioeconomic status are at an increased risk of being obese. Obesity has a strong familial component: if a child's parent is obese, there is a 50% chance that the child will be obese; if both parents are obese, the likelihood of the child being obese increases to 80% (Whitaker, Wright, Pepe, Seidel, & Dietz, 1997). Obese children have a high likelihood of remaining obese as adults (Freedman et al., 2005).

The consequences of being obese are potentially severe, including higher risk for cardiovascular disease, hypertension, dyslipidemia, stroke, impaired glucose tolerance, type 2 diabetes, cancer, breathing problems, joint problems, and fatty liver disease. In addition to health consequences, there are also psychosocial and emotional consequences of being obese. Obese children often experience bullying and teasing in school which is associated with poor self-esteem and low self-confidence.

Cross-References

- ▶ Body Mass Index
- ▶ Overweight

References and Readings

Freedman, D. S., Khan, L. K., Serdula, M. K., Dietz, W. H., Srinivasan, S. R., & Berenson, G. S. (2005). The relation of childhood BMI to adult adiposity: The Bogalusa heart study. *Pediatrics, 115*, 22–27.

Ogden, C. L., Carroll, M. D., Curtin, L. R., Lamb, M. M., & Flegal, K. M. (2010). Prevalence of high body mass index in US children and adolescents, 2007–2008. *Journal of the American Medical Association, 303*(3), 242–249.

Whitaker, R., Wright, J., Pepe, M., Seidel, K., & Dietz, W. (1997). Predicting obesity in young adulthood from childhood and parental obesity. *The New England Journal of Medicine, 337*, 869–873.

Obesity Treatment

- ▶ Lifestyle Changes

Obesity: Causes and Consequences

Chad D. Jensen[1], Amy F. Sato[2] and Elissa Jelalian[3]
[1]Department of Psychology, Brigham Young University, Provo, UT, USA
[2]Department of Psychology, Kent State University, Kent, OH, USA
[3]Department of Psychiatry, Rhode Island Hospital, Brown Medical School, Providence, RI, USA

Description

Obesity results from a complex interplay of biologic, environmental, genetic, and psychosocial factors that influence appetite, satiety, and food storage in the form of body fat. At a fundamental level, obesity results from a sustained positive energy balance; that is, consuming more energy than one expends over an extended period of time. Weight gain results from excess caloric intake, and weight loss results from greater caloric expenditure than intake. Thus, excessive caloric intake and insufficient physical activity are the primary contributors to obesity.

Biologic Influences

Research has demonstrated that energy balance is highly influenced by a complex biological system. This system balances the amount of fat in the body, partially by regulating the unconscious drive to eat (Friedman, 2009). This drive to eat is adaptive in times of food scarcity but has become problematic given the relative abundance of high-calorie foods and limited physical activity participation in today's society.

Genetic Contributors

Although the dramatic increase in obesity is not genetic in origin, genes interact with environmental factors to give individuals in the same environment significantly different risks of becoming obese. Estimates of the heritability of obesity – the percentage of variability due to

genetic factors – range from 70% to 80%, indicating that genetics play a clear role in increasing one's vulnerability to becoming obese (Walley, Asher, & Froguel, 2009).

Perinatal Influences

Recent research has also highlighted the important contribution of the perinatal environment to development of obesity. Studies have demonstrated that severely obese mothers are much more likely to have obese children than previously obese mothers who lost weight prior to conception (Kral et al., 2006). These results highlight the significance of gene-environment interactions: Epigenetic influences play a critical role in one's likelihood of becoming obese.

Environmental Factors

There is increasing recognition of the extent to which environmental factors contribute to the rise in obesity. Indeed, American society has been characterized as an *obesogenic environment* that promotes excess eating and physical inactivity (Swinburn, Egger, & Raza, 1999). Energy expenditure has decreased for most individuals in the developed world, largely because employment primarily consists of sedentary work, energy-saving devices limit physical exertion, and transportation requires little physical effort. In concert with decreases in physical activity, access to highly palatable, inexpensive food that requires little or no preparation has increased dramatically. These foods are often high in caloric content and contain high amounts of fat and sugar. Additionally, the food industry promotes consumption of these foods through aggressive advertising and promotion, a practice that has been shown to increase their consumption (Kumanyika et al., 2008).

Research has consistently demonstrated that attributes of the "built" environment (i.e., buildings such as homes, schools, and shopping centers, as well as the ways in which these buildings are configured within space) also contributes to the prevalence of obesity. Individuals who live in walkable neighborhoods with access to recreation facilities are more likely to be physically active and less likely to be obese. Residents of communities with ready access to healthful foods tend to have healthier diets and lower incidence of obesity. Furthermore, environmental characteristics that contribute to obesity affect low-income and racial minorities disproportionately, contributing to higher levels of obesity among these groups (Sallis & Glanz, 2009). Fast-food restaurants in particular have been identified as a key contributor to the rise in obesity, and these restaurants are more prevalent in low-income neighborhoods, a characteristic thought to partially explain the higher incidence of obesity among the poor (Powell, Auld, Chaloupka, O'Malley, & Johnston, 2007).

Consequences of Obesity

A broad spectrum of medical, psychological, social, and behavioral consequences result from obesity. These consequences have been shown to arise from one of two factors: the increased mass of fat in the body of the obese individual and the risks resulting from metabolic changes associated with excess fat.

Consequences Due to Metabolic Changes

One of the most well-known risks associated with metabolic changes arising from excess fat is the higher incidence of type 2 diabetes mellitus. Associated conditions include insulin insensitivity and metabolic syndrome, a concurrence of elevated blood pressure, cholesterol, centrally deposited fat, and elevated blood sugar. Cardiovascular disease including hypertension is also a frequent consequence of obesity. Estimates suggest that as many as 50% of obese individuals suffer from hypertension. Excess body weight also increases the risk of heart failure. Increased cardiac work resulting from higher body mass raises the likelihood of cardiomyopathy and heart failure in obese individuals. Obese individuals are also at risk for nonalcoholic fatty liver disease, a constellation of liver abnormalities associated with obesity. Certain forms of cancer, including colorectal, prostate, and female reproductive and breast cancers, are more prevalent among obese individuals (Bray, 2004).

Consequences Due to Increased Fat Mass

Medical comorbidities associated with increased fat mass include osteoarthritis, cartilage and bone

metabolism problems independent of weight bearing effects, and skin pigmentation and stretch marks. Obstructive sleep apnea risk is also greatly elevated in obese individuals.

Equally concerning are the psychosocial consequences associated with obesity. Obese individuals experience significant social stigmatization, including difficulty obtaining employment, education, and health care in addition to difficulties establishing and maintaining social relationships. Obese children are also more likely to have academic problems and to experience social difficulties, such as peer victimization and increased loneliness. Furthermore, obese individuals are more likely to report poor quality of life, depression, and disordered eating (Bean, Stewart, & Olbrisch, 2008).

Obesity Shortens Life

The detrimental effects of obesity combine to shorten life for individuals with this condition. Although estimates of life-shortening effects vary, obesity has been shown to shorten life on a population level. Indeed, the rise in rates of obesity has overwhelmed the positive effects of reduced rates of smoking on a population level (Stewart, Cutler, & Rosen, 2009). Individuals with obesity (BMI 30–39.9) have a reduced median survival rate of 2–4 years, while for those with extreme obesity (BMI 40–45), survival is reduced by 8–10 years (Prospective Studies Collaboration, 2009).

Conclusion

In summary, obesity is caused by energy imbalance resulting from excess caloric input relative to expenditure. The increased rate of obesity observed in the USA over the last three decades has been attributed primarily to an interaction between biologic risk for obesity and an *obesogenic environment* that provides limited opportunity for physical activity and ready access to calorie-dense foods. There are a number of medical and psychosocial consequences associated with obesity, including cardiovascular disease, joint problems, type 2 diabetes, depression, social stigmatization, poorer quality of life, and abbreviated life expectancy. These consequences underscore the importance of prevention and intervention efforts to ameliorate the negative consequences of obesity.

Cross-References

- ▶ Nutrition
- ▶ Physical Inactivity

References and Readings

Bean, M. K., Stewart, K., & Olbrisch, M. E. (2008). Obesity in America: Implications for clinical and health psychologists. *Journal of Clinical Psychology in Medical Settings, 15*, 214–224.

Bray, G. A. (2004). Medical consequences of obesity. *Journal of Clinical Endocrinology and Metabolism, 89*, 2583–2589.

Friedman, J. M. (2009). Causes and control of excess body fat. *Nature, 459*, 340–342.

Kral, J. G., Biron, S., Simard, S., Hould, F. S., Lebel, S., Marceau, S., et al. (2006). Large maternal weight loss from obesity surgery prevents transmission of obesity to children who were followed for 2 to 18 years. *Pediatrics, 118*, 1644–649.

Kumanyika, S. K., Obarzanek, E., Stettler, N., Bell, R., Field, A., Fortmann, F. A., et al. (2008). Population-based prevention of obesity: The need for comprehensive promotion of healthful eating, physical activity, and energy balance. A scientific statement from American Heart Association Council on epidemiology and prevention, interdisciplinary committee for prevention. *Circulation, 18*, 428–464.

Olshansky, S. J., Passaro, D. J., Hershow, R. C., Layden, J., Carnes, B. A., Brody, J., et al. (2005). A potential decline in life expectancy in the United States in the 21st century. *New England Journal of Medicine, 352*, 1138–1145.

Powell, L. M., Auld, M. C., Chaloupka, F. J., O'Malley, P. M., & Johnston, L. D. (2007). Association between access to food stores and adolescent body mass index. *American Journal of Preventive Medicine, 33*(4S), S301–307.

Prospective Studies Collaboration. (2009). Body-mass index and cause-specific mortality in 900,000 adults: Collaborative analyses of 57 prospective studies. *The Lancet, 373*, 1083–1096.

Sallis, J. F., & Glanz, K. (2009). Physical activity and food environments: Solutions to the obesity epidemic. *Millbank Quarterly, 87*, 123–154.

Stewart, S. T., Cutler, D. M., & Rosen, A. B. (2009). Forecasting the effects of obesity and smoking on US

life expectancy. *New England Journal of Medicine, 361*(23), 2252–2260.
Swinburn, B., Egger, G., & Raza, F. (1999). Dissecting obesogenic environments: The development and application of a framework for identifying and prioritizing environmental interventions for obesity. *Preventative Medicine, 29*, 563–570.
Walley, A. J., Asher, J. E., & Froguel, P. (2009). The genetic contribution to non-syndromic human obesity. *Nature Reviews Genetics, 10*, 431–442.

Obesity: Prevention and Treatment

Amy F. Sato[1], Chad D. Jensen[2] and Elissa Jelalian[3]
[1]Department of Psychology, Kent State University, Kent, OH, USA
[2]Department of Psychology, Brigham Young University, Provo, UT, USA
[3]Department of Psychiatry, Rhode Island Hospital, Brown Medical School, Providence, RI, USA

Description

Obesity prevention and treatment approaches have been developed for individuals across the developmental continuum, from childhood through adulthood. The basic goal of obesity prevention and treatment approaches is to shift energy balance (i.e., calories consumed versus calories expended) in order to reduce the presence of, or risk for, excess weight.

Obesity Prevention

Obesity prevention efforts include attention to both promoting healthy dietary intake and achieving adequate physical activity. The NIH Obesity Research Task Force outlines three levels of obesity prevention (US Department of Health and Human Services, 2004). Primary prevention is aimed at all individuals regardless of weight or obesity risk status. Secondary prevention seeks to prevent further weight gain in individuals who are already overweight or obese. The goal of tertiary prevention is to prevent complications due to co-occurring health problems (e.g., type 2 diabetes) in individuals who are already overweight or obese. A staged, or tiered, approach to obesity prevention and treatment has been recommended for child and adolescent overweight and obesity, with the appropriate level of intervention determined based on degree of overweight and presence of comorbidity (see Spear et al., 2007). With respect to youth, it is recommended that children ages 6 and older be screened for obesity, and then referred to multicomponent behavioral interventions to promote improvement in weight status (US Preventive Services Task Force, 2010). Obesity prevention programs are aimed at fostering a healthy lifestyle (e.g., consumption of fruits and vegetables, minimizing sugar-sweetened beverages, obtaining 60 min daily physical activity, and limiting screen time).

Environmental factors, such as the high cost of healthy foods and lack of access to safe places to exercise, are key contributors to the increased prevalence of obesity (Hill & Peters, 1998; Sallis & Glanz, 2009). For this reason, obesity prevention efforts are also aimed at community and population level change (Khan et al., 2009). Environmental- and policy-level changes can be divided into six categories aimed to (1) promote availability and affordability of healthy food/beverages (e.g., improve geographic availability of supermarkets in underserved areas), (2) support choice of healthy foods and beverages (e.g., restrict availability of high-calorie, high-fat, high-sugar, and high-sodium content foods and beverages in public service areas), (3) encourage breastfeeding (e.g., support for breastfeeding in the workplace), (4) encourage physical activity/limit sedentary activity in youth (e.g., require physical education in schools), (5) create safe communities that increase physical activity through changes in the built environment (e.g., increase access to outdoor recreational facilities), and (6) encourage communities to organize for change (e.g., participate in community coalitions or partnerships to address obesity) (Khan et al., 2009).

Obesity Treatment

The treatment of overweight and obesity in children and adults shares the same fundamental

principal, which is to increase energy expenditure and decrease caloric intake. However, the goal of treatment may differ somewhat depending on age. For adults, obesity treatment seeks to reduce body weight. For children, the goal of obesity treatment may be either reduction of body weight or deceleration of weight gain (Oude Luttikhuis et al., 2009). The most appropriate mode of treatment is based on several considerations, including initial degree of overweight, previous intervention efforts, co-morbid health problems, and age.

Lifestyle Intervention

There is a strong evidence base for multicomponent lifestyle intervention approaches that combine dietary restriction, physical activity prescription (increasing physical activity, limiting sedentary behaviors), and behavioral modification approaches for the treatment of obesity in adults and youth (Oude Luttikhuis et al., 2009; Shaw, O'Rourke, Del Mar, & Kenardy, 2005). Behavioral modification components for adults and youth typically include goal setting, self-monitoring, stimulus control, reinforcement, and problem solving (Jelalian & Hart, 2009; Oude Luttikhuis et al., 2009; Shaw et al., 2005). Research supports including parent involvement as part of lifestyle intervention approaches for pre-adolescent children (Spear et al., 2007).

A comprehensive review of behavioral treatments for overweight and obese adults (18 years and older) revealed that the combination of behavioral or cognitive-behavioral techniques with dietary and exercise interventions was effective in producing short-term and long-term (12-month) weight loss (Shaw et al., 2005). However, maintenance of weight loss following behavioral intervention continues to be poor, with participants typically gradually discontinuing changes in exercise and diet and consequently regaining weight (Perri et al., 2001). For overweight and obese children and adolescents, a comprehensive review of randomized controlled trials utilizing lifestyle intervention found that the combination of dietary intervention, physical activity, and behavior modification components was effective compared to standard care and self-help (Oude Luttikhuis et al., 2009). This is consistent with a previous meta-analysis which found that lifestyle interventions for pediatric obesity yield significant decreases in obesity immediately posttreatment, compared to no treatment or education-only controls (Wilfley et al., 2007).

Pharmacological Intervention

Pharmacological intervention approaches have been utilized for the treatment of obesity in both adults and adolescents (i.e., 12 years or older). In adults, the most commonly studied antiobesity medications have been orlistat and sibutramine. Results of a comprehensive review of these medications in adults suggested that, combined with lifestyle modification, orlistat and sibutramine resulted in greater weight loss than placebo with maintenance of weight loss at long-term follow-up (i.e., 12+ months; Padwal, Li, & Lau, 2004). The majority of the research in adolescents has focused on metformin, orlistat, and sibutramine as antiobesity drugs, with preliminary support for these pharmacologic interventions in adolescents (Delamater, Jent, Moine, & Rios, 2008; US Preventive Services Task Force, 2010). Orlistat is approved by the FDA for use in children ages 12 and older (US Preventive Services Task Force). However, recently, sibutramine was withdrawn from the US and Canadian markets due to concerns arising from research finding that it was associated with an increased risk of cardiovascular events (e.g., myocardial infarction; James et al., 2010). Finally, attrition rates with pharmacological interventions are somewhat high and there is a need for more methodologically rigorous studies of pharmacological treatments for obesity, with longer-term follow-up (Padwal et al., 2004).

Surgical Intervention

Surgical interventions for obesity have increased as the prevalence of obesity and morbid obesity (BMI $\geq$ 40) has increased in adults as well as in children and adolescents. Bariatric surgery, a surgical intervention for obesity, seeks to reduce weight and maintain weight loss via restriction of dietary intake, malabsorption of food, or a combination of these mechanisms (Colquitt, Picot, Loveman, & Clegg, 2009). Gastric bypass (i.e., Roux-en-Y, resectional gastric bypass) and adjustable gastric

banding have been the most commonly performed types of surgery, although other methods are available (e.g., biliopancreatic diversion, sleeve gastrectomy, vertical banded gastroplasty) (Colquitt et al., 2009). A comprehensive review of surgical interventions found that bariatric surgery was more effective in producing short- and long-term weight loss relative to nonsurgical interventions (e.g., dietary intervention) (Colquitt et al.). However, this may be due in part to the fact that most adult and adolescent bariatric surgery patients have a high BMI (i.e., BMI $\geq$ 40), and therefore have more weight to lose (Colquitt et al.; Delamater et al., 2008). Though results are mixed, overall, the available evidence suggests that gastric bypass yields greater weight loss than adjustable gastric banding or vertical banded gastroplasty, but similar to banded gastric bypass and isolated sleeve gastrectomy (Colquitt et al., 2009). Gastric bypass surgery has been found to be efficacious for the treatment of obese adolescents, and preliminary evidence supports the use of adjustable gastric banding in severely obese adolescents, with promising short-term and long-term weight reduction (Delamater et al., 2008).

Summary

A range of obesity prevention and treatment approaches have been implemented in adults as well as in children and adolescents. Prevention and intervention efforts involve attention to achieving a healthy diet and maintaining adequate levels of physical activity and, at the most basic level, seek to shift energy balance toward achieving weight loss or decelerating the rate of weight gain. Behavioral lifestyle interventions have traditionally received the most attention, with support for use in both adult and child populations; however, pharmacologic and surgical interventions have increasingly received support in adults and adolescents.

Cross-References

- ▶ Nutrition
- ▶ Physical Inactivity
- ▶ Weight Loss

References and Readings

Colquitt, J. L., Picot, J., Loveman E., Glegg, A. J. (2009). Surgery for obesity. *Cochrane Database of Systematic Reviews*, *15*(2):CD003641.

Delamater, A. M., Jent, J. F., Moine, C. T., & Rios, J. (2008). Empirically supported treatment of overweight adolescents. In E. Jelalian & R. J. Steele (Eds.), *Handbook of childhood and adolescent obesity* (pp. 221–240). New York: Springer. Chapter 14.

Hill, J. O., & Peters, J. C. (1998). Environmental contributions to the obesity epidemic. *Science, 280*, 1371–1374.

James, W. P., Caterson, I. D., Coutinho, W., Finer, N., Van Gaal, L. F., Maggioni, A. P., et al. (2010). Effects of sibutramine on cardiovascular outcomes in overweight and obese subjects. *The New England Journal of Medicine, 263*(10), 907–917.

Jelalian, E., & Hart, C. N. (2009). Pediatric obesity. In M. C. Roberts & R. G. Steele (Eds.), *Handbook of pediatric psychology* (4th ed., pp. 446–463). New York: Guilford.

Khan, L. K., Sobush, K., Keener, D., Goodman, Lowry, A., Kakietek, J., & Zaro, S. (2009). *Recommended community strategies and measurements to prevent obesity in the United States*. Morbidity and Mortality Weekly Report, 58(RR07), 1–26.

Oude Luttikhuis, H., Baur, L., Jansen, H., Shrewsbury, V. A., O'Malley, C., Stolk, R. P et al. (2009). Interventions for treating obesity in children. *Cochrane Database of Systematic Reviews, 21*(1):CD001872.

Padwal, R., Li, S. K., & Lau, D. C. (2004). *Long-term pharmacotherapy for obesity and overweight*. Issue: Cochrane Database of Systematic Reviews. 3.

Perri, M. G., Nezu, A. M., McKelvey, W. F., Shermer, R. L., Renjilian, D. A., & Viegener, B. J. (2001). Relapse prevention training and problem-solving therapy in the long-term management of obesity. *Journal of Consulting and Clinical Psychology, 69*(4), 722–726.

Sallis, J. F., & Glanz, K. (2009). Physical activity and food environments: Solutions to the obesity epidemic. *The Millbank Quarterly, 87*, 123–154.

Shaw, K. A., O'Rourke, P., Del Mar, C., & Kenardy, J. (2005). *Psychological interventions for overweight or obesity*. Issue: Cochrane Database of Systematic Reviews. 2.

Spear, B. A., Barlow, S. E., Ervin, C., Ludwig, D. S., Saelens, B. E., Schetzina, K. E., et al. (2007). Recommendations for treatment of child and adolescent overweight and obesity. *Pediatrics, 120*(Suppl. 4), S254–288.

US Department of Health & Human Services. (2004). *Strategic plan for NIH obesity research*. Washington, DC: National Institutes of Health.

US Preventive Services Task Force (2010). Screening for obesity in children and adolescents: US preventive services task force recommendations statement. *Pediatrics*. Published online Jan 18, 2010; DOI:10.1542/peds.2009-2037.

Wilfley, D. E., Tibbs, T. L., Van Buren, D. J., Reach, K. P., Walker, M. S., & Epstein, L. H. (2007). Lifestyle interventions in the treatment of childhood overweight: A meta-analytic review of randomized controlled trails. *Health Psychology, 26*(5), 521–532.

Obrist, Paul A

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Biographical Information

Paul A. Obrist was born on June 17, 1931. He received his B.S. degree from Cornell University in 1953 and his Ph.D. from the University of Rochester in 1958. His graduate training included 2 years of clinical psychology at the Veterans Administration and a 2-year teaching assistantship, assisting in the teaching of introductory personality, child, and abnormal psychology. He also had extensive predoctoral research experience, culminating in his thesis research, "An Investigation of the Claim of Autonomic Discrimination Without Awareness and the Relationship of GSR Conditioning to Measures of Skin Conductance."

Obrist held a postdoctoral fellowship at the Fels Research Institute from June 1958 to June 1960. His specialty area was psychophysiology, working under the guidance of John Lacey. He then moved to the University of North Carolina, Chapel Hill, in 1960 as an assistant professor of psychology in the department of psychiatry. He progressed to associate professor in 1965 and full professor in 1970. His primary focus was developing the department's psychophysiology laboratory and guiding research (both chronic dog preparation and young human adults) on the role of behavioral stress in the etiology of hypertension.

Major Accomplishments

From the time of his arrival at UNC-Chapel Hill until his death in 1987, Obrist established and maintained one of the most prominent and productive psychophysiology research laboratories in the world. From his earliest studies, he sought to identify the cardiovascular mechanisms responsible for inducing variations in cardiovascular performance and to understand the functions that these cardiovascular responses serve in the context of behavior. His bringing behavior and physiology together in this manner generated a comprehensive view of cardiovascular psychophysiology.

Obrist was particularly proud of being president of the Society for Psychophysiological Research from 1974 to 1975. Additional honors included being the recipient of the 1985 Award for Distinguished Contributions to Psychophysiology from the Society for Psychophysiological Research and being conferred the honor of fellow by the Academy of Behavioral Medicine Research, the American Association for the Advancement of Science, and the American Psychological Association (Divisions 3, 6, and 38).

Obrist received numerous National Institute for Mental Health (NIMH) and National Heart, Lung, and Blood Institute (NHLBI) grants from 1963 to 1987. He was the recipient of continuous NIMH R01 grant funding from 1963 to 1976 and continuous NHLBI funding from 1976 to 1987. During his career, he published over 100 papers, one authored book, and one edited book.

Many outstanding psychophysiologists (including some who went on to make their mark in the fields of health psychology and behavioral medicine) had the good fortune to be trained in his laboratory. Among his trainees who later became department chairs were James Lawler (University of Tennessee-Knoxville), Kathleen Lawler-Row (East Carolina University), James McCubbin (Clemson University), and Michael T. Allen (University of Mississippi). P. Murali Doraiswamy became head of the Division of Biological Psychiatry at Duke University, where Andrew Sherwood continued his research. Kathy Light joined Obrist's laboratory in 1976, collaborating as a research scientist and assuming directorship of the lab in 1987. The coeditor of this encyclopedia, J. Rick Turner, also trained under Obrist and Light. Light also trained her

own graduate students there, including Susan Girdler who is now professor and director of the university's Stress and Health Research Program.

One of Obrist's central themes was that psychophysiologists (and, by extension, those in behavioral medicine and health psychology) should become better biologists. The depth of biological discussions in this encyclopedia attests to the usefulness of this approach.

Cross-References

- ▶ Cardiovascular Disease
- ▶ Psychophysiology: Theory and Methods

References and Readings

Allen, M. T., Sherwood, A., & Obrist, P. A. (1986). Interactions of respiratory, and cardiovascular adjustments to behavioral stressors. *Psychophysiology, 23*, 532–541.

Langer, A. W., Hutcheson, J. S., Charlton, J. D., McCubbin, J. A., Obrist, P. A., & Stoney, C. M. (1985). On-line minicomputerized measurement of respiratory gas exchange during exercise. *Psychophysiology, 22*, 50–58.

Langer, A. W., Stoney, C. M., McCubbin, J. A., Hutcheson, J. S., Obrist, P. A., & Charlton, J. D. (1985). Cardiopulmonary adjustments during exercise and an aversive reaction time task: Effects of beta-adrenoceptor blockade. *Psychophysiology, 22*, 59–68.

Light, K. C., & Obrist, P. A. (1985). Renal and cardiovascular effects of psychological stress and sodium intake. In E. S. Katkin & S. B. Manuck (Eds.), *Advances in behavioral medicine* (pp. 89–114). Greenwich: JAI Press.

Obrist, P. A. (1962). Some autonomic correlates of serial learning. *Journal of Verbal Learning and Verbal Behavior, 1*, 100–104.

Obrist, P. A. (1963a). Skin resistance levels and the galvanic skin response: Unilateral differences. *Science, 139*, 227–228.

Obrist, P. A. (1963b). Cardiovascular differentiation of sensory stimuli. *Psychosom Medicine, 25*, 450–459.

Obrist, P. A., Grignolo, A., Koepke, J. P., Langer, A. W., & Light, K. C. (1985). Behavioral influences and beta-adrenergic mechanisms: The kidney and sodium retention. In R. B. Williams (Ed.), *Perspectives on behavioral medicine* (Vol. 2, pp. 183–187). New York: Academic Press.

Obrist, P. A., & Light, K. C. (1988). Active-passive coping and cardiovascular reactivity: Interaction with individual differences and types of baseline. In A. Baum, W. Gordon, & J. A. Herd (Eds.), *Proceedings of the academy of behavioral medicine research* (pp. 109–126). New York: Academic Press.

Obrist, P. A., Light, K. C., Langer, A. W., & Koepke, J. P. (1986). Psychosomatics. In M. Coles, E. Donchin, & S. Porges (Eds.), *Psychophysiology: Systems, processes and applications* (pp. 626–645). New York: Guilford Press.

Obrist, P. A., Light, K. C., Sherwood, A., & Allen, M. T. (1986). Behavioral influences on blood pressure control: Implications for the hypertensive process. In W. H. Birkenhager & J. L. Reid (Series Eds.) & R. C. Tarazi, & A. Zanchetti (Vol. Eds.), *Handbook of hypertension: Vol. 8. Section on physiology and pathophysiology of hypertension* (pp. 250–277). Amsterdan: Elsevier/North Holland.

Obrist, P. A., Light, K. C., Sherwood, A., Allen, M. T., Langer, A. W., & Koepke, J. P. (1986). Some working hypothesis on the significance of behavioral-evoked cardiovascular reactivity to pathophysiology. In T. H. Schmidt, T. D. Dembroski, & C. Blumchen (Eds.), *Biological and psychological factors in cardiovascular disease* (pp. 406–417). Berlin: Springer.

Sherwood, A., Allen, M. T., Obrist, P. A., & Langer, A. W. (1986). Evaluation of beta-adrenergic influences on cardiovascular and metabolic adjustments to physical and psychological stress. *Psychophysiology, 23*, 89–104.

Observational Designs

- ▶ Clinical Trial
- ▶ Cohort Study
- ▶ Nonexperimental Designs

Observational Studies

- ▶ Clinical Trial
- ▶ Cohort Study
- ▶ Nonexperimental Designs

Observational Study

- ▶ Clinical Trial
- ▶ Cohort Study
- ▶ Nonexperimental Designs

Obstructive Sleep Apnea

► Sleep Apnea

Occipital

► Brain, Cortex

Occupation

► Job Classification
► Occupational Therapy

Occupational Classification

► Job Classification

Occupational Health

Johannes Siegrist
Department of Medical Sociology, University of Duesseldorf, Düsseldorf, Germany

Synonyms

Organizational health; Work-related health

Definition

"Occupational health" is defined as the field of research and intervention that deals with work- and employment-related influences on people's health and health-related behaviors and their modification. The field of occupational health is broader than the academic discipline of occupational medicine as it includes organizational, psychosocial, and behavioral aspects in addition to the more traditional physical and chemical hazards, thus incorporating research and expertise from social and behavioral sciences.

Description

Generally, behavioral medicine is interested in two approaches toward occupational health. The first approach considers the organizations, companies, and businesses where large population groups can be met recurrently and simultaneously as an ideal setting of implementing programs of behavioral modification. In these programs, health-adverse behaviors such as smoking, unhealthy diet, or lack of physical exercise are targeted with the aim of promoting a healthy lifestyle. These individual- or group-based interventions are often supported by employers and health insurance organizations as they were shown to produce benefits to both employees and employers. The second approach focuses on those aspects of work and employment that directly affect the health of working people. Here, scientific analyses of these associations and interventions based on respective evidence are of central interest.

As the nature of employment and work has changed significantly over the last half century, psychological and socio-emotional demands and threats evolving from an adverse psychosocial environment have become more widespread in all advanced societies (Schnall, Dobson, & Rosskam, 2009). Technological progress and economic growth in the context of globalized markets and trades result in new types of tasks (e.g., information processing, occupations, and professions in the service sector). Chronic exposure to an adverse psychosocial work environment affects the health of working people by triggering negative emotions and by eliciting recurrent stress responses in the organism that may compromise different bodily systems in the long run and increase the susceptibility to stress-related physical and mental disorders (Siegrist & Marmot, 2006).

To measure a health-adverse psychosocial work environment a theoretical model is needed that identifies "toxic" components within complex and diverse work settings at a level of high

generalization. Several such models were proposed, but the following three concepts are considered being the most established as they have been supported by a substantial body of empirical research (Cartwright & Cooper, 2009). First, the "demand-control model" posits that a specific job task profile characterized by high quantitative demands in combination with a low degree of decision latitude or control elicits recurrent stress reactions. These effects are moderated by the presence or absence of social support at work (Karasek & Theorell, 1990). Second, the "effort-reward imbalance model" is based on the basic principle of reciprocity of contractual social exchange. High efforts spent at work that are not reciprocated by adequate rewards trigger recurrent stressful experience. Rewards include salary or wage, promotion prospects and job security, and esteem or appreciation from significant others. Effort-reward imbalance is frequent among employees who have no alternative choice in the labor market or who work in highly competitive jobs. Stressful experience is reinforced among those who are overcommitted to their work (Siegrist, 1996). Third, the "organizational justice model" claims that unfair procedures within organizations and inappropriate interpersonal relationships, in particular within the hierarchies of organizations, are experienced as stressful (Greenberg, 2010).

These complementary theoretical models offer opportunities of health-promoting behavioral interventions at three levels. At the personal level, the individual workers' coping resources are strengthened by different approaches of stress management (e.g., relaxation and meditation techniques, cognitive-behavioral interventions). At the interpersonal or group level, social relationships at work are improved (e.g., communication, leadership, esteem, social support). At the organizational or structural level, interventions target changes in the division of work, work time, work load, task content, promotion prospects, or monetary and non-monetary rewards. Evidence indicates that interventions which combine these levels produce stronger and more sustainable effects than single-level interventions (Bourbonnais, Brisson, & Vézina, 2010; Semmer, 2008). Moreover, integrating health-promoting behavioral change into programs of stress management and organizational change seems particularly promising in an attempt of reducing social inequalities in health where those in lower socioeconomic positions suffer from a higher work-related burden of disease.

Behavioral interventions in occupational health are commonly located at the level of primary prevention where they are directed either at the workforce as a whole or at specific vulnerable subgroups. However, behavioral interventions at the level of secondary prevention are becoming more important as working populations are aging. Improving return to work in chronically ill and disabled people is considered a major challenge of occupational health, particularly with regard to mental health problems. There is a strong business case in terms of sickness absence reduction and productivity gain for introducing such secondary prevention measures where behavioral medicine expertise can be successfully implemented (e.g., individual placement and support models) (Black, 2008).

Despite these promises, occupational health from a behavioral medicine perspective still has to face substantial challenges. So far, only a minority of companies and organizations is committed to promote healthy work. There is a lack of resources, personnel, and management incentives. In view of financial pressures and heavy competition priorities are diverted from long-term concern about workers' health to short-term profit. The impact of legal regulations and labor policies at national level is limited by transnational trade and labor markets in a globalized economy (Blouin, Chopra, & van der Hoeven, 2009). Therefore, diffusion and promotion of excellent research findings and successful models of good practice in the field of behavioral occupational health is needed in an attempt to reduce the burden of work-related disease and disability.

References and Readings

Black, C. (2008). *Working for a healthier tomorrow*. London: TSO.

Blouin, C., Chopra, M., & van der Hoeven, R. (2009). Trade and social determinants of health. *Lancet, 373*, 502–507.

Bourbonnais, R., Brisson, C., & Vézina, M. (2010). Long-term effects of an intervention on psychosocial work factors among health care professionals in a hospital setting. *Occupational and Environmental Medicine*. doi:10.1136/oem.2010.055202.
Cartwright, S., & Cooper, C. L. (Eds.). (2009). *The Oxford handbook of organizational well-being*. Oxford, US: Oxford University Press.
Greenberg, J. (2010). Organizational injustice as an occupational health risk. *The Academy of Management Annuals, 4*, 205–243.
Karasek, R. A., & Theorell, T. (1990). *Healthy work stress, productivity, and the reconstruction of working life*. New York: Basic Books.
Schnall, P. L., Dobson, M., & Rosskam, E. (Eds.). (2009). *Unhealthy work: Causes, consequences cures*. Amityville, NY: Baywood Press.
Semmer, N., (2008). *Stress management and well-being interventions in the workplace. State of Science Review: SR-C6*. Report by the Foresight Project. Government Office for Science, London.
Siegrist, J. (1996). Adverse health effects of high effort-low reward conditions at work. *Journal of Occupational Health Psychology, 1*, 27–41.
Siegrist, J., & Marmot, M. (Eds.). (2006). *Social inequalities in health: New evidence and policy implications*. Oxford, US: Oxford University Press.

Occupational Prestige

► Occupational Status

Occupational Science

Clare Hocking
Faculty of Health and Environmental Sciences, Auckland University of Technology, Auckland, New Zealand

Definition

Occupational science is the systematic study of the things that people do (their occupations). Occupations include everyday activities undertaken to care for self and others, animals and living and inanimate objects; secure shelter and sustenance; raise children; develop and test abilities, learn, and experience a sense of competence; contribute to family and community; create or distribute material wealth; play and enjoy life; pursue interests; celebrate significant life events; express creativity and spirituality; preserve or regain health; and the things people do for rest and restoration. In brief, occupation is everything people do to occupy themselves (Townsend, 1997). Research is undertaken at the level of individuals, groups, or populations, with an emphasis on understanding what occupations mean to people, occupational patterns, the factors that influence people's participation in occupation, and the relationship between occupation and health.

Description

Occupational science is concerned with the nature, performance, and outcomes of the ordinary and extraordinary things people do in their everyday lives. It is an interdisciplinary synthesis of basic knowledge (Yerxa, 1993), which draws from the human sciences, population studies, engineering, economics, ecology, and political sciences (Dickie, 2010).

Occupation

Within occupational science, various definitions of occupation have been proposed. While no consensus is sought, many definitions have included notions of occupation being self-initiated, intentional, and self-directed; meaningful; and organized, goal-directed, or purposeful (Crabtree, 1998; Yerxa et al., 1989). These characteristics, and the fact that occupations are identifiable and named within their cultural context, differentiate occupations from actions (such as brushing hair out of one's eyes) (Yerxa, 1993). Occupations command some level of attention, energy, time, and interest (Gray, 1997), are a means of fulfilling needs or meeting environmental demands, and lead to development and competence (Crabtree, 1998; Yerxa, 1998). They are variously described as discrete chunks of activity or as enfolded – indicating that people might be doing more than one occupation at a time, as in directing

children's play while preparing a meal. Occupations are also considered unique, in that an occupation cannot be repeated exactly as it was done before and each person performs an occupation in his or her own way (AOTA, 1997). Occupations are patterned, in that many occupations occur on a regular and repeated basis, and expected to change over time in response to maturation and over the lifespan.

Occupational Science Perspective on People

Occupational scientists view people as occupational beings (Yerxa et al., 1990), meaning that engaging in occupation is part of the essential nature of being human. Accordingly, occupation is positioned as the mechanism by which people develop and exercise their innate capacities; fulfill basic needs for food, water, and shelter; "adapt to changes in the environment, and flourish as individuals" (Wilcock, 1993, p. 17). Because she considered the need for occupation to be innate, Wilcock argued that lack of engagement in occupation is experienced as a discomfort that warns of a problem, manifesting as boredom, anxiety, depression, loneliness, tiredness, or tension; as a protective energy surge, triggering use of one's capacities; and as a sense of pleasure, satisfaction, or competence, which rewards and prompts engagement in occupation. While these assertions remain largely untested, adoption of the concept of flow into the occupational science literature suggests acceptance of these premises (Csikszentmihalyi, 1993).

Emergence of Occupational Science

Occupational science was founded by scholars at the University of Southern California (Yerxa et al., 1989), who initiated the discipline to "support the practice of occupational therapy" (Yerxa et al., 1990, p. 1) by generating a unique and substantive knowledge base for the profession, and to consolidate occupational therapy's place in academia by demonstrating its field is worthy of scholarly investigation (Yerxa, 1993). The discipline's focus on occupation is described as providing an alternative conceptualization of health to the biomedical focus that predominates in post-industrial societies. Occupational science rejects the mind-body split of physical disability and mental illness (Yerxa, 1993), instead emphasizing that occupation (doing) is a determinant of health and well-being, as well as being the mechanism through which people "learn to be competent, participating masters of their environments" (Yerxa, 1993, p. 3) and experience being, becoming, and belonging (Wilcock, 2006).

Primary Focuses

Occupation is a complex concept. Within the diverse topics addressed, much of the occupational science research has coalesced around several key concerns; the relationship between occupation and health, the meanings occupation holds, and occupation as a means of identity construction.

Large-scale empirical studies to establish the influence occupation has on health have been conducted by researchers from the University of Southern California, targeting lifestyle redesign™ for elders living in the community and for individuals at risk of decubitus ulcers and other health conditions (Clark, Jackson, & Carlson, 2004; Clark & Lawlor, 2009). In the Well Elderly Study, elders who were assisted to incorporate meaningful occupations into their everyday lives achieved global improvements in physical and social functioning, vitality, and mental health. Delivery of the intervention was cost-effective, and participants reported substantially reduced health costs. Smaller-scale studies have investigated the impact of various health conditions on individuals' occupations, explored the concept of occupational balance, and identified patterns of engagement in occupation associated with self-reported stress levels or health status.

Exploration of the relationship between occupation and well-being utilizing a history of ideas methodology has generated theoretical understandings of using occupation to both promote health and well-being, and to prevent illness and disability (Wilcock, 2006). These perspectives align with the Declaration of Alma Ata and the Ottawa Charter, which call for the reorientation of health services toward the pursuit

of health, and point to the need for widespread reform of occupational therapy to address population health (Wilcock & Hocking, 2004).

In addition to the focus on health, multiple small-scale qualitative studies have explored the meaning and experience of engaging in occupation. Perspectives range from meaning making through participation in cultural and mundane occupations (traditional forms of Indian dance, making a cup of tea) to the meaning of specialized occupations, such as teaching in a tertiary setting. Meaning has been explored through studies of participation in solitary and group occupations, such as cake decorating or singing in a choir, in relation to ritual, and from the perspective of making and using objects (Dickie, 2010; Hocking, 2009). An emerging perspective, grounded in Deweyan pragmatism, is that occupation is a transaction between the environment, self, and others. This strand of discussion draws attention to the ways internal and external changes mediate the meanings experienced and conveyed through occupation, and implicitly critiques earlier studies of individuals' engagement in occupation that paid little attention to environmental influences (Dickie, Cutchin, & Humphry, 2006).

A third focus has been the construction of identity and sense of self through occupation (Phelan & Kinsella, 2009), including the ways identity is strengthened or claimed (Christiansen, 1999; Rudam, 2002). Occupational identity is frequently discussed in relation to roles or role loss, gender, ethnicity, group membership, place or living with a chronic illness. In vulnerable groups, including older adults, refugees, migrants, and asylum seekers, restrictions and disruptions in occupation have been associated with identity change or preservation.

Key Concepts in Occupational Science

Occupational Deprivation

Grounded in the belief that "unused skill or capacity . . . can become a disease centre or else atrophy or disappear" (Maslow, cited in Wilcock, 1993, p. 21), Wilcock proposed that insufficient engagement in occupations has detrimental effects on health. Occupational deprivation is the term used to refer to situations where people experience chronic or permanent health problems due to lack of access to the range of occupations required to sustain health, because of cultural, political, economic, or social factors outside of themselves. Examples include the deliberate occupational deprivation of prisoners; sex role stereotypes, and cultural and religious sanctions that curtail women's participation in culturally meaningful occupations; the displacement and containment of refugees and asylum seekers (Whiteford, 2010), and the lack of opportunity to engage in occupation experienced by older people living in care facilities.

Occupational Justice

An aspect of social justice, occupational justice hinges on the equitable distribution of resources "as part of fair, enabling and empowering societies" (Wilcock, 2006, p. 245). Four occupational rights have been proposed, centering on (a) experiencing occupation as meaningful and enriching; (b) participation in occupations that foster development, health and social inclusion; (c) having choice in occupations, at individual or population levels; and (d) deriving fair privileges from participation in diverse occupations (Townsend & Wilcock, 2004). Systematically depriving people of occupation, to the extent that individual and population health are undermined (Stadnyk, Townsend, & Wilcock, 2010), is termed an occupational injustice. Such injustices are thought to manifest at the individual level as chronic stress, fatigue, and immune deficiencies and at a population level as civic disturbance and social disintegration. The concept of occupational justice has been taken up by the World Federation of Occupational Therapists in its Position Statement on Human Rights (n.d.). Related concepts including occupational marginalization and occupational apartheid have been proposed.

Occupational Potential/Possibilities

While occupational potential has been defined as "a vision of future possibilities for engagement in occupations, or structuring of society to enable people to participate as fully as possible"

(Christiansen & Townsend, 2010, p. 421), it is more usually considered to refer to the human potential that emerges through participation in occupation (Asaba & Wicks, 2010). Assumptions include that people naturally strive to develop their potential, employing creativity and constructive means across the life course. Human potential is not seen as predetermined; rather it is responsive to both the person and the social environment, relates to participation in occupation, and emerges over time as human capacities are exercised. The concept of occupational potential relates to the notion of possibilities, viewed as the interaction between experiences, actions, skills, and contexts.

Co-occupation

First published in 1996, the concept of co-occupation refers to those occupations where the shared participation of two or more people is requisite, such as playing on a see-saw, feeding an infant, or playing "peek-a-boo" (Zemke & Clark, 1996). In relation to infants, co-occupation is discussed in terms of promoting development, adaptation, and interaction. Researchers have sought to establish psychological, physiological, and behavioral differences between people engaged in solitary occupations and co-occupations, with some statistically significant differences identified.

Scope of Occupational Science

The scope of the discipline has been described as encompassing the substrates, form, function, and socio-cultural and historical context of human occupation (Clark et al., 1991). The substrates comprise humans' capacity to engage in occupation, including:

- Neuromuscular and other bodily structures, which both give the potential for and delimit human's capacities (e.g., physical strength, dexterity, and reaction times)
- Metabolic functions to maintain homeostasis despite exertion, which manifest as fitness, endurance, recovery times, and so on
- Cognitive and psychological processes such as information processing, memory, and regulation of emotions, which provide the capacity to maintain attention and monitor performance, and to remember, interpret, and apply the culturally accumulated and individually acquired knowledge of occupations
- Learned motor patterns and ways of responding.

Humans' stereoscopic vision, opposable thumbs, upright posture (which frees the hands to manipulate objects), and the size of humans' cerebral cortex (thus, the capacity for language, judgment, problem solving, and self-consciousness) have been identified as particularly important physical characteristics (Wilcock, 2006), which underpin these substrates.

The form of an occupation is its observable features. Depending on the occupation, different characteristics will be important. Those might include the capacities required to carry out the occupation (e.g., sufficient strength to wield a hammer), where the occupation usually takes place, who typically participates in the occupation (age, gender, socioeconomic status, ethnicity), whether the occupation is completed alone or with others, the time of day it is usually performed, how long it takes and the sequence of steps involved, how rigidly the steps or timeframes must be adhered to, the pace or tempo of performance required, and what tools or resources are used.

Other sources have included aspects that are not observable in their definition of an occupational form, such as the cultural meanings the occupation holds, the rules that govern performance, and the cognitive processes involved in monitoring performance (Nelson, 1999).

The function of an occupation is what is achieved by participating in it. There may be a concrete product, such as the vegetables growing in the garden, in addition to individual gains (a child learns about caring for seedlings), group outcomes (a family conserves its financial resources), and societal consequences (horticultural knowledge is preserved and disseminated).

The sociocultural context of an occupation shapes the meanings associated with it. For instance, expectations related to individuals' socioeconomic status, gender, age, and ethnicity influence who does what and how people respond to people acting within and outside of those

norms. The sociocultural context also influences the value placed on different occupations, with playing football being more acceptable to mainstream society than driving a stolen car, and programming a computer likely to be better remunerated than cleaning a bathroom – even though keeping bathrooms clean has an important role in preserving people's health. The historical development of an occupation can influence the meaning attributed to it, as in an occupation attributed the status of a tradition, as well as how widely dispersed it is across different cultures and people of different ages. Making bread and baskets, cleaning, building, weaving, raising crops, and fishing are occupations present in many cultures, albeit using different technologies. Reading a book is a more recent occupation, although its history spans several centuries, that is encouraged in school-aged children and an assumed ability in adults, even if they prefer not to participate. Playing interactive games on a computer has a shorter history, and younger rather than older people tend to have higher levels of proficiency in that occupation.

Critical Perspectives in Occupational Science

A range of critical discourses are emerging in the literature (Whiteford & Hocking, 2012). It is acknowledged that Western assumptions of the centrality of occupations in people's lives, the meanings occupations hold, and individualist rather than collectivist understandings predominate, and indigenous perspectives, such as the things people do to care for the land or in relation to the spiritual world, have not received much attention. One review of the discipline's research has also identified a gender inequity, with "adult, white women without disabilities" (Pierce et al., 2010, p. 209) being the group most studied by presenters at conferences convened by the Society for the Study of Occupation: USA between 2002 and 2007. A similar bias is evident in the Journal of Occupational Science.

In response to such critiques, a derived emic method was developed to generate cross-cultural understandings of occupation (Shordike, Hocking, Pierce et al., 2010), a transactional perspective has been advocated (Dickie et al., 2006), and governmentality has provided a critical lens through which to consider people's occupational choices (Rudman, Huot, & Dennhardt, 2009).

Cross-References

▶ Occupational Therapy

References and Readings

AOTA, (1997). Statement: Fundamental concepts of occupational therapy: Occupation, purposeful activity and function. *American Journal of Occupational Therapy*, *51*, 864–866.

Asaba, E., & Wicks, A. (2010). Occupational potential. *Journal of Occupational Science, 17*, 120–124.

Christiansen, C. (1999). Defining lives: Occupation as identity: An essay on competence, coherence, and the creation of meaning. *American Journal of Occupational Therapy*, *53*, 547–558.

Christiansen, C. H., & Townsend, E. A. (Eds.). (2010). *Introduction to occupation: The art and science of living* (2nd ed.). Upper Saddle River, NJ: Pearson Education.

Clark, F. A., Parham, D., Carlson, M. E., Frank, G., Jackson, J., Pierce, D., et al. (1991). Occupational science: Academic innovation in the service of occupational therapy's future. *American Journal of Occupational Therapy*, *45*, 300–310.

Clark, F. A., Jackson, J., & Carlson, M. (2004). Occupational science, occupational therapy and evidence-based practice: What the well Elderly study has taught us. In M. Molineux (Ed.), *Occupation for occupational therapists* (pp. 200–218). Oxford, England: Blackwell.

Clark, F., & Lawlor, M. C. (2009). The making and mattering of occupational science. In E. B. Crepeau, E. S. Cohn, & B. A. Boyt Schell (Eds.), *Willard and Spackman's occupational therapy* (11th ed., pp. 2–14). Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins.

Crabtree, J. L. (1998). The end of occupational therapy. *American Journal of Occupational Therapy, 52*, 205–214.

Csikszentmihalyi, M. (1993). Activity and happiness: Towards a science of occupation. *Journal of Occupational Science: Australia, 1*(1), 38–42.

Dickie, V., Cutchin, M. P., & Humphry, R. (2006). Occupation as transactional experience: A critique of individualism in occupational science. *Journal of Occupational Science, 13*, 83–93.

Dickie, V. A. (2010). Are occupations processes too complicated to explain? What we can learn by trying. *Journal of Occupational Science, 17*, 195–203.

Gray, J. M. (1997). Application of the phenomenological method to the concept of occupation. *Journal of Occupational Science: Australia, 4*, 5–17.

Hocking, C. (2009). The challenge of occupation: Describing the things people do. *Journal of Occupational Science, 16*, 140–150.
Nelson, D. (1999). Occupational terminology interactive dialogue: Occupational form. *Journal of Occupational Science, 6*, 76–78.
Phelan, S., & Kinsella, A. E. (2009). Occupational identity: Engaging socio-cultural perspectives. *Journal of Occupational Science, 16*, 85–91.
Pierce, D., Atler, K., Baltisberger, J., Fehringer, E., Hunter, E., Malkawi, S., et al. (2010). Occupational science: A data-based American perspective. *Journal of Occupational Science, 17*, 204–215.
Rudman, D. L. (2002). Linking occupation and identity: Lessons learned through qualitative exploration. *Journal of Occupational Science*, *9*, 12–19.
Rudman, D. L., Huot, S., & Dennhardt, S. (2009). Shaping ideal places for retirement: Occupational possibilities within contemporary media. *Journal of Occupational Science*, *16*(1), 18–24. doi:10.1080/14427591.2009.9686637.
Shordike, A., Hocking, C., Pierce, D., Wright-St. Clair, V., Vittayakorn, S., Rattakorn, P., et al. (2010). Respecting regional culture in an international multi-site study: A derived etic method. *Qualitative Research, 10*(3), 333–355. doi:10.1177/1468794109360145.
Stadnyk, R. L., Townsend, E. A., & Wilcock, A. A. (2010). Occupational justice. In C. H. Christiansen & E. A. Townsend (Eds.), *Introduction to occupation: The art and science of living* (2nd ed., pp. 329–358). Upper Saddle River, NJ: Pearson Education.
Townsend, E. (Ed.). (1997). *Enabling occupation: An occupational therapy perspective*. Ottawa: CAOT Publications.
Townsend, E., & Wilcock, A. (2004). Occupational justice. In C. H. Christiansen & E. A. Townsend (Eds.), Introduction to occupation: The art and science of living (pp. 243–273). Upper Saddle River, NJ: Pearson Education.
Whiteford, G. E. (2010). Occupational deprivation: Understanding limited participation. In C. H. Christiansen & E. A. Townsend (Eds.), Introduction to occupation: The art and science of living (2nd ed., pp. 303–328). Upper Saddle River, NJ: Pearson Education.
Whiteford, G. E., & Hocking, C. (Eds.). (2012). Critical perspectives on occupational science: Society, inclusion, participation. Oxford, UK: Wiley-Blackwell.
Wilcock, A. (1993). A theory of the human need for occupation. *Journal of Occupational Science: Australia*, *1*(1), 17–24.
Wilcock, A. A. (2006). *An occupational perspective of health* (2nd ed.). Thorofare, NJ: Slack.
Wilcock, A. A., & Hocking, C. (2004). Occupation, population health and policy development. In M. Molineux (Ed.), *Occupation for occupational therapists* (pp. 219–230). Oxford, England: Blackwell.
World Federation of Occupational Therapists. (n.d.). *Position statement on human rights*. Retrieved from http://www.wfot.org/documents.asp?cat=31
Yerxa, E. J. (1993). Occupational science: A new source of power for participants in occupational therapy. *Journal of Occupational Science: Australia, 1*(1), 3–9.
Yerxa, E. J. (1998). Health and the human spirit for occupation. *American Journal of Occupational Therapy, 52*(6), 412–418.
Yerxa, E. J., Clark, F., Frank, G., Jackson, J., Parham, D., Pierce, D., et al. (1990). An introduction to occupational science: A foundation for occupational therapy in the 21st century. *Occupational Therapy in Health Care, 6*(4), 1–17.
Yerxa, E. J., Clark, F., Jackson, J., Parham, D., Stein, C., & Zemke, R. (1989). An introduction to occupational science: A foundation for occupational therapy in the 21st century. *Occupational Therapy in Health Care, 6*(4), 1–17.
Zemke, R., & Clark, F. (Eds.). (1996). *Occupational science: The evolving discipline*. Philadelphia: F. A. Davis.

Occupational Status

Karen Jacobs[1], Miranda Hellman[2], Jacqueline Markowitz[2] and Ellen Wuest[2]
[1]Occupational Therapy, College of Health and Rehabilitation Science, Sargent College, Boston University, Boston, MA, USA
[2]Boston University, Boston, MA, USA

Synonyms

Employment status; Job prestige; Occupational prestige

Definition

Occupational status is the "collective term encompassing occupational performance components, occupational performance, and occupational role performance" (Jacobs & Jacobs, 2009).

Occupational performance components are "any subsystem that contributes to the performance of an occupation" (such as self-care, leisure, work, education, etc.) (Jacobs & Jacobs, 2009).

Occupational performance is "the act of doing and accomplishing a selected activity or occupation that results from the dynamic transaction among the client, the context, and the activity" (Jacobs & Jacobs, 2009).

Occupational role performance is the "rights, obligations, and expected behavior patterns associated with a particular set of activities or occupations, done on a regular basis, and associated with social cultural roles" (Hillman & Chapparo, 1995).

Overall, occupational status can be thought of as the various roles that a person identifies through participation in occupations. Occupational status may be impacted by other factors such as socioeconomic status. However, in general, it is being engaged in an occupation for wages.

Cross-References

- ▶ Job Demand/Control/Strain
- ▶ Job Performance

References and Readings

Hillman, A., & Chapparo, C. J. (1995). An investigation of occupational role performance in men over sixty years of age, following a stroke. *Journal of Occupational Science, 2*(3), 88–99.

Jacobs, K., & Jacobs, L. (2009). *Quick reference dictionary for occupational therapy* (5th ed.). Thorofare, NJ: SLACK Incorporated.

Occupational Therapist

- ▶ Therapy, Occupational

Occupational Therapy

Carolyn Baum[1], Leeann Carey[2] and Helene J. Polatajko[3]
[1]School of Medicine in St Louis, Washington University, St. Louis, MO, USA
[2]Melbourne Brain Centre, Heidelberg, VIC, Australia
[3]Department of Occupational Science and Occupational Therapy, Graduate Department of Rehabilitation Science Dalla Lana School of Public Health, University of Toronto, Toronto, ON, Canada

Synonyms

Activities of daily living (ADL); Ergotherapy; Health science; Life skills; Occupation; Occupational science; Participation; Rehabilitation; Therapy

Definition

World Federation of Occupational Therapists (WFOT) defines occupational therapy as a "client-centered health profession concerned with promoting health and well being through occupation. The primary goal of occupational therapy is to enable people to participate in the activities of everyday life. Occupational therapists achieve this outcome by working with people and communities to enhance their ability to engage in the occupations they want to, need to, or are expected to do, or by modifying the occupation or the environment to better support their occupational engagement" (World Federation of Occupational Therapists [WFOT], 2010, para. 1)

Description

Occupational therapy, founded in 1917, has become a well-established profession within the

areas of health and rehabilitation with over 60 member countries in the WFOT. Occupational therapists use a variety of interventions including the therapeutic use of everyday activities to enable persons to improve or regain the ability to participate in meaningful and satisfying activities that are necessary to support everyday life. Occupational therapy is applied at individual, group, community, and population levels (Christiansen, Baum, & Bass-Haugen, 2005; Söderback, 2009).

The term "occupation," as used by occupational therapists, refers to more than paid work; it refers to engagement in all life activities and includes activities necessary to care for self and others, education, work, play, leisure, and social participation. The discipline holds a number of key values and assumptions; in particular, it views people as occupational beings, believes in the positive relationship between occupation and health, holds that all people have the need for, and right to, engagement in meaningful occupation of their choice, and experience the health and well-being it brings (Townsend & Polatajko, 2007).

Occupational therapy is an applied discipline with theoretical underpinnings related to occupation and its enablement. The practice of occupational therapy draws from occupational science and neuroscience, as well as the behavioral, biomedical, neurological, psychological, social, and environmental sciences. The integration of knowledge from these diverse sciences and its application to daily life places the profession in a key position to contribute to translation of science to everyday living to improve the human condition (Carey & Baum, 2011).

The Practice of Occupational Therapy

As an applied science, occupational therapy is focused on the role occupation plays in the everyday lives of those who experience difficulties in engaging in their occupations. Occupational therapists become involved when everyday living is compromised or is at risk of being compromised by impairments or potentially disabling conditions. They help people overcome occupational restrictions or limitations by identifying barriers to occupational engagement and by evaluating the process that supports it. Occupational therapy interventions are designed to promote engagement; restore function; and enable important activities, tasks, and life roles. Occupational therapists rely on valid and reliable tools that assist in their inquiry and promote their unique roles as members of a team supporting a person's development or recovery, health, and well-being. Occupational therapy practitioners understand performance and engagement from a holistic perspective and address all aspects of performance when providing interventions.

Occupational therapists use a client-centered approach. They use a detailed analysis of the fit between personal capacity, occupational demands, and environmental demands and support to determine the best approaches to improving capacity and/or modifying occupations and environments to facilitate change and enable performance and engagement (Law, Baum, & Dunn, 2005). Eriksson, Kottorp, Borg, and Tham (2009) describe the limitations to daily occupations caused by disruptions of health and social situation as "occupational gaps" – the gaps that occur between what the individual wants, needs, and is expected to do, and what he or she actually does. The process of enabling change is driven by a variety of approaches to intervention, most especially learning-based models that draw on skills in activity analysis and problem solving.

Occupational therapy is practiced in a wide range of settings, including hospitals, rehabilitation settings, health centers, mental health centers, community centers, homes, workplaces, schools, and residences for seniors and those living with chronic disability. Clients are actively involved in the therapeutic process, and outcomes of occupational therapy are measured in terms of occupational performance and engagement, participation, or well-being (WFOT, 2010). Occupational therapists bridge between the medical perspectives focused on impairment management or recovery and the sociocultural perspective focused on the contexts in which people live their lives. Occupational therapists are committed to promoting inclusion, diversity, independence,

and safety for all recipients in various stages of life, health, and illness; and are guided by an Occupational Therapy Code of Ethics (American Occupational Therapy Association, 2005).

The day-to-day practice of occupational therapy is guided by the self-appointed World Federation of Occupational Therapists (WFOT, 2010). All schools worldwide must meet minimum standards established by WFOT. Many countries have additional standards and some have registration standards that must be met by therapists to be allowed to practice. There are also self-appointed national and international bodies, such as the American, Australian, Canadian, British, and Swedish Associations of Occupational Therapists.

The Science of Occupational Therapy

The science of occupational therapy provides a unique contribution to the understanding of occupational performance and participation in the activities of daily life. Occupational therapy is based on the premise that individuals can support their own health through meaningful and purposeful engagement in occupations. Occupation is a primary source of meaning (Christiansen, 1999; Hasselkus, 2002) and a determinant of life satisfaction (Eriksson et al., 2009). A number of empirical studies have supported occupational engagement as a determinant of health and well-being (Clark et al., 1997; Glass, de Leon, Marottoli, & Berkman, 1999; Herzog, Franks, Markus, & Holmberg, 1998; Horgas, Wilms, & Baltes, 1998; Hultsch, Hertzog, Small, & Dixon, 1999). Absence of engagement in occupation may lead to physiological decline and negatively impact health (Kielhofner, 2004; Townsend & Polatajko, 2007). Importantly, occupation has the potential to be therapeutic.

The science of occupation seeks to understand the mechanisms that support *function* (e.g., postural control, grasp, pinch, problem solving, attention, strategy development, endurance), *activity* (e.g., writing, dressing, feeding, grooming, learning), *participation* (e.g., education, work, community life, leisure), and the *environmental enablers and barriers* that make function, activity, and participation possible (e.g., social support, assistive technology, accommodations). Occupational scientists study the full range of phenomena that contribute to human occupation. From basic mechanisms of body function and structure to sociocultural influences, they address measurement, intervention, and translational and health services research.

Occupational therapy is a discipline as it has an established literature testing the theoretical underpinnings of the profession, such as occupation, as well as empirical studies of the practice using scientific methods (Carey & Baum, 2011). The science of occupational therapy contributes to occupational science, neuroscience, and environmental science and furnishes evidence to support recovery and/or adaptive or compensatory strategies that improve the lives of people, support development, or maintain health and well-being.

Cross-References

- ▶ Activities of Daily Living (ADL)
- ▶ Occupational Science

References and Readings

American Occupational Therapy Association. (2005). Occupational therapy code of ethics. *American Journal of Occupational Therapy, 59*, 639–642.

Carey, L. M., & Baum, C. (2011). Occupational therapy. In N. P. Azari (Ed.), *Encyclopaedia of sciences and religions*. Heidelberg: Springer.

Christiansen, C. (1999). Defining lives: Occupation as identity: An essay on competence, coherence, and the creation of meaning. *American Journal of Occupational Therapy, 53*, 547–558.

Christiansen, C., Baum, C. M., & Bass-Haugen, J. (2005). *Occupational therapy: Performance, participation, and well-being* (3rd ed.). Thorofare, NJ: SLACK.

Clark, F., Azen, S. P., Zemke, R., Jackson, J., Carlson, M., Mandel, D., et al. (1997). Occupational therapy for independent-living older adults: A randomized controlled trial. *Journal of the American Medical Association, 278*, 1321–1326.

Eriksson, G., Kottorp, A., Borg, J., & Tham, K. (2009). Relationship between occupational gaps in everyday life, depressive mood and life satisfaction after

acquired brain injury. *Journal of Rehabilitation Medicine, 41*, 187–194.
Glass, T. A., de Leon, C., Marottoli, R. A., & Berkman, L. F. (1999). Population based study of social and productive activities as predictors of survival among elderly Americans. *British Medical Journal, 319*, 478–483.
Hasselkus, B. (2002). *The meaning of everyday occupation.* Thorofare, NJ: SLACK.
Herzog, A. R., Franks, M. M., Markus, H. R., & Holmberg, D. (1998). Activities and well-being in older age: Effects of self-concept and educational attainment. *Psychology and Aging, 13*, 179–185.
Horgas, A. L., Wilms, H., & Baltes, M. M. (1998). Daily life in very old age: Everyday activities as expression of successful living. *The Gerontologist, 38*, 556–568.
Hultsch, D. E., Hertzog, C., Small, B. J., & Dixon, R. A. (1999). Use it or lose it: Engaged lifestyle as a buffer of cognitive decline in old age? *Psychology and Ageing, 14*, 245–263.
Kielhofner, G. (2004). *Conceptual foundations of occupational therapy* (3rd ed.). Philadelphia, PA: F. A. Davis. Authoritative texts on occupational therapy.
Law, M. C., Baum, C. M., & Dunn, W. (2005). *Measuring occupational performance: Supporting best practice in occupational therapy* (2nd ed.). Thorofare, NJ: SLACK.
Söderback, I. (Ed.). (2009). *International handbook of occupational therapy interventions* (1st ed., Vol. 1). New York: Springer.
Townsend, E., & Polatajko, H. (2007). *Enabling occupation II: Advancing an occupational therapy vision for health, well-being, & justice through occupation.* Ottawa, Canada: CAOT Publications ACE. Authoritative texts on occupational therapy.
World Federation of Occupational Therapists. (2010). *What is occupational therapy?* Retrieved from http://www.wfot.org/information.asp (accessed May 5, 2011).

Odds Ratio

Elizabeth M. Maloney
Formerly of the Viral and Rickettsial Division, Centers for Disease Control and Prevention, Atlanta, GA, USA

Definition

An odds ratio (OR) is a measure of association between an exposure and an outcome (i.e., disease) and is commonly derived from data collected in a case–control study design. When the outcome is rare, the OR is a good estimate of the relative risk of the outcome. The absolute value of the OR varies in size from 0 to infinity; its size is an indication of the magnitude of the association. An OR >1.0 indicates that the likelihood of the exposure is increased in the presence of the outcome. For example, an OR of 3.00 is interpreted as meaning that those with the outcome are three times as likely to be exposed compared to those without the outcome. An OR <1.0 indicates that the likelihood of the exposure is decreased in the presence of the outcome, which is often referred to as a "protective effect." For example, an OR of 0.70 is interpreted as meaning that persons with the outcome are 1.00–0.70, or 30%, less likely to be exposed compared to persons without the outcome. Since a case–control design is cross-sectional in nature, an OR cannot be used to assess causation between an exposure and an outcome.

The precision of the OR is determined based on the computation of a 95% confidence interval (CI) around the OR, and is greatly affected by the sample size. A narrow CI indicates greater precision, and conversely, a wide CI indicates less precision and more variability. The larger the sample size, the greater the precision of the OR. A CI that *does not* include 1.0 indicates that the OR is statistically significant. An unadjusted OR and 95% CIc can be computed using the simple chi-square statistic, or logistic regression analysis if preferred. However, logistic regression analysis is typically used to generate an OR and 95% CI that is adjusted for the effects of one or more confounders.

Office of Family Assistance

► Family Assistance

Oils

► Fat: Saturated, Unsaturated

Oldenburg, Brian

Brian Oldenburg
Department of Epidemiology and Preventive Medicine, Monash University,
Melbourne, VIC, Australia

Biographical Information

Brian Oldenburg was born in Sydney, Australia, on October 23, 1953. He received his Bachelor of Science (Honors in Psychology) degree from University of New South Wales (UNSW) in 1975, and he then worked in a community mental health center in Sydney before living and working in the United Kingdom for 2 years. He returned to Australia in 1978 to the Department of Psychiatry at Prince Henry Hospital and University of NSW, before completing a Masters of Clinical Psychology in 1983. He completed his Ph.D. degree in the UNSW Schools of Medicine and Psychiatry in 1987. He was employed as a lecturer – and then, senior lecturer – in the Department of Public Health, University of Sydney (1987–1994) before becoming professor and head of the School of Public Health at Queensland University of Technology in Brisbane (1994–2006). In 2006, Oldenburg became the inaugural professor and director of International Public Health and Associate Dean International at Monash University in Melbourne, Australia. Since 2006, he has also been a visiting professor at University of Queensland, as well as the Finnish National Public Health Institute (KTL) and also at China's Beijing Centers for Disease Control. He has been a regional director of the Asia Pacific Academic Consortium for Public Health since 2004 and a regular consultant for the World Health Organization since 2003. He was a public health consultant in China for the 5 years leading up to the Beijing Olympic Games in 2008. He also undertook a review for the South African Human Sciences Research Council of their research program on the "Social Aspects of HIV/AIDS and Health" in 2008.

He has chaired grant review panels for Australia's National Health and Medical Research Council and other research organizations since 1990, and he has also served on grant review panels for US NIH. He has served on many national and international committees and advisory boards related to health psychology, behavioral medicine, and public health. He has held senior editorial appointments on *European Journal of Health Psychology*, *International Journal of Behavioral Medicine*, *Health Psychology Review* and *Translational Behavioral Medicine*. He has held senior appointments on many national and international organizations, for example, he has served as secretary, president, local program chair, and congress program chair for the International Society of Behavioral Medicine over the past 20 years. He received the Public Health Recognition Award from Asia Pacific Academic Consortium for Public Health in 2004, and in 2006, he was awarded Lifetime Fellow Membership of US Society for Behavioral Medicine, 2006, for his contribution to "building evidence-based population health interventions that can guide practice and change policy globally." He also has invited membership of the United Kingdom Faculty of Public Health for his achievements in global public health.

Major Accomplishments

Oldenburg's research has made a substantial contribution to evidence development, practice,

and policy in the fields of public health, prevention, and behavioral medicine in Australia and internationally over the last 30 years. He has a very distinguished and internationally recognized track record in the design, implementation, and evaluation of behavioral, psychosocial, and environmental interventions for the prevention and management of chronic disease and the promotion of health and well-being in community, clinical, workplace, school, and other settings. Conducted with one of his academic mentors, Gavin Andrews, his earliest intervention trials conducted in the early 1980s focused on psychosocial and behavioral interventions to improve rehabilitation and health outcomes for patients following myocardial infarction. Conducted with another of his mentors, Graham Macdonald, in the early 1980s, he investigated how psychosocial and behavioral factors were important for treatment adherence and quality of life in people on renal dialysis. His interest in the complex interplay between biological, psychosocial, and behavioral factors in the development, course, and outcomes of chronic conditions is one that has continued throughout his research over the last 30 years. For example, his research team is currently examining the comorbid relationship between negative emotions and both cardiovascular disease and diabetes in order to develop new approaches to interventions.

Since 1990, most of Oldenburg's intervention trials have had a "real-world" focus, and they have emphasized implementation and policy and practice uptake. He has undertaken recent prevention and chronic disease management trials in China, Malaysia, South Africa, and Finland, and his team is currently undertaking a large trial of diabetes prevention in Kerala, India (K-DPP). His interventions have involved both "soft touch" (peer support, socio-behavioral, and environmental interventions) as well as more "high-tech" (ICT) interventions. In collaboration over the past 10 years with Dr. Rob Friedman and other colleagues from Boston University, his research team has developed one of the first automated telehealth programs in the world to improve diabetes management (*Australasian TLC Diabetes*). The results demonstrate that people living with diabetes in the community can significantly improve their self-management of diabetes by using this interactive telehealth program. His research has also focused on chronic disease and socioeconomic health inequalities and social disadvantage as well as interventions with Aboriginal and Torres Strait Islander populations in Australia and disadvantaged people in other countries.

He is the current recipient of a 5-year capacity building and training grant from the US National Institutes of Health (*Millennium Promise Grant Award*) to establish the ASCEND Network in Asia (*Asian Non-Communicable Chronic Disease Research Network)*2010–2015. This program will establish the first comprehensive and cross-country research training program for early career researchers in the prevention and control of chronic diseases in the Asia Pacific Region. He regularly provides significant evidence-based advice to government and nongovernment organizations in Australia and internationally, about how governments and countries can more effectively prevent and manage chronic conditions, thereby improving the health of their people.

Cross-References

- ▶ Behavioral Medicine
- ▶ Cardiac Rehabilitation
- ▶ Diabetes
- ▶ International Society of Behavioral Medicine
- ▶ Population Health
- ▶ Public Health

References and Readings

Absetz, P., Oldenburg, B., Hankonen, N., Valve, R., Nissinen, A., Fogelholm, M., et al. (2009). Type 2 diabetes prevention in the "real world": Three-year results of the GOAL lifestyle implementation trial. *Diabetes Care, 32*, 1418–1420.

Oldenburg, B., & Absetz, P. (2011). Lost in translation: Overcoming the barriers to global implementation and exchange of behavioral medicine evidence. *Translational Behavioral Medicine, 1*, 252–255.

Oldenburg, B., Absetz, P., & Chan, C. (2010). Behavioral interventions for prevention and management of chronic disease. In A. Steptoe, K. Freedland, J. R. Jenning, M. Llabre, S. Manuck, & E. Susman (Eds.), *Handbook of behavioral medicine: Methods and applications* (pp. 969–988). New York: Springer.

Oldenburg, B., Absetz, P., Dunbar, J., Reddy, P., & O'Neil, A. (2011). The spread of diabetes prevention programs around the world: A case study from Finland and Australia. *Translational Behavioral Medicine, 1*(2), 270–282.

Oldenburg, B., De Courten, M., & Frean, E. (2010). The contribution of health psychology to the advancement of global health. In J. Suls, K. W. Davidson, & R. K. Kaplan (Eds.), *Handbook of health psychology* (pp. 397–410). New York: Guilford Press.

Oldenburg, B., & Glanz, K. (2008). Diffusions of Innovation, in Glanz, Rimer and Viswanath. In *Health behavior and health education: Theory, research and practice* (4th ed.). San Francisco: Jossey-Bass.

Oldenburg, B., Perkins, R. J., & Andrews, G. (1985). A controlled trial of psychological intervention in myocardial infarction. *Journal of Consulting and Clinical Psychology, 53*(6), 852–859.

Older Adult

► Elderly

Omega-3 Fatty Acids

Matthew Muldoon
Department of Medicine, University of Pittsburgh, Pittsburgh, PA, USA

Synonyms

Alpha-linolenic acid; DHA; EPA; Fish oil; n-3 fatty acids; Omega-3 polyunsaturated fatty acids

Definition

Fatty acids are fat molecules that are ubiquitous in the diet and all living organisms. The long-chain, omega-3 fatty acids, eicosapentaenoic and docosahexaenoic acids (EPA, DHA) are known to be effective in reducing recurrence of heart disease events and in the treatment of depression. EPA and DHA are being studied in relation to heart disease prevention, various psychiatric disorders, eye health, cancer, immune function, and arthritis.

Description

Basic Science

Biochemistry and Nutritional Sources

Triglycerides are the classic fat molecule and consist of three fatty acids bound to a 3-carbon glycerol backbone. Phospholipids are triglycerides in which a phosphate group has replaced one of the fatty acids. Phospholipids are the primary building block of all cell membranes.

Fatty acids are simple chains of carbons atoms of lengths between 6 and 22 with a carboxylic acid group at one end. Saturated fatty acids have all single bonds between carbon atoms, monounsaturated fatty acids have a single double bond, and polyunsaturated fatty acids (PUFAs) have multiple double bonds. PUFAs are classified as omega-6 when the terminal double bond is present at the sixth carbon atom, and omega-3 when is occurs at the third carbon.

The principle long-chain omega-6 fatty acid is arachidonic acid (AA), and the omega-3 polyunsaturated fatty acids of interest are EPA and DHA. Humans synthesize AA from the essential fatty acid, linoleic acid, and EPA and DHA from the essential fatty acid, alpha-linolenic acid. However, the efficiency of these synthetic steps is limited for EPA and more so for DHA. As a result, EPA and DHA are viewed as "conditionally essential" in the diet. Only seafood has significant quantities of EPA and DHA, and within seafood oily fish such as salmon, trout, tuna, and mackerel are the richest sources of these nutrients. In North America and Europe, median EPA and DHA intake is under 200 mg/day, substantially less than earlier and more primitive cultures.

Functional Roles

The multiple actions of the long-chain, omega-3 fatty acids in biological chemistry establish their importance generally to health and disease and,

therefore, the biological plausibility of various sequelae of nutritional deficiency. Phospholipids are the basic building block of cell and organelle membranes, and most have an AA, EPA, or DHA in sn-2 position of the glycerol backbone. DHA and AA are highly concentrated in brain phospholipids, and DHA edges out AA as the most prevalent PUFA in human brain gray matter and synaptic membranes. As a highly unsaturated fatty acid, DHA increases the fluidity of cell membranes relative to other fatty acids. Fluidity is a physicochemical property of membranes that modulates the location and activity of membrane-bound proteins, including enzymes, ion transporters, and neurotransmitter receptors.

PUFAs are released by phospholipases and then serve as precursors to eicosanoids – families of prostaglandins, thromboxanes, and leukotrienes regulating inflammation, vasomotion, and hemostasis. AA-derivatives are pro-inflammatory while EPA-derived eicosanoids are relatively anti-inflammatory. Increased EPA availability competes with, and reduces the production of, AA-derived eicosanoids. Additionally, EPA and DHA serve as precursors of resolvins which temper inflammation, vasoconstriction, and thrombogenesis. Finally, DHA both affects brain-derived neurotrophic factor production and forms nitro-fatty acids that are reactive electrophilic species which modulate oxidative stress.

Acting through the preceding mechanisms, the long-chain n-3 PUFA exert neurotrophic effects. These include promotion of neurogenesis, dendritic arborization, selective pruning, and myelination. In a complementary fashion, neural tissue degeneration may be reduced by neuroprotective or anti-apoptotic effects of DHA. Logically, the foregoing cellular roles of PUFA may be expected to affect neurotransmission, either generally or selectively. Evidence from laboratory experiments and humans studies indicates that omega-3 fatty acid exposure affects serotonergic and dopaminergic neurotransmission.

Clinical Investigation

Early Brain Development

From the last trimester through the second year of life, the human brain undergoes very rapid growth and during this period DHA accretion is rapid and depends upon maternal delivery across the placebo and via breast milk. Infants of mothers reporting low perinatal maternal fish consumption have low early childhood intelligence and increased risk of suboptimal outcomes for prosocial behavior, fine motor, communication, and social development scores. Similarly, the effects of breast-feeding on IQ may be moderated by genetic variation in fatty acid metabolism. Randomized clinical trials suggest that supplementing the diets of either pregnant mothers or infants with DHA improves cognitive development.

Affective Disorders

Evidence from numerous observational studies links low intake of omega-3 fatty acids with greater risk of major depressive disorder and, more generally, depressive symptomatology. Randomized and placebo-controlled trials of fish oil supplementation indicate efficacy in treating this disorder. Treatment benefit appears after 2–3 months of supplementation and appears to be most robust for EPA. In persons not suffering from major depression, supplementation has not been found to affect mood symptoms.

Cognitive Functioning, Decline, and Dementia

Cross-sectional studies and small randomized trials in children and non-elderly adults using dietary data, blood levels, or supplementation suggest that higher intake enhances cognitive performance. However, absent more strongly designed investigations, it is unclear whether such effects are robust and which aspects of cognitive functioning are affected. A comparative wealth of studies finds omega-3 fatty acids to correlate inversely with age-related cognitive decline. Clinical trials to date have provided only inconsistent evidence that taking a fish oil supplement ameliorates cognitive decline among elderly individuals.

Dementia risk in large cohort studies is greatest in persons with relatively low omega-3 fatty acid intake. However, no clinical trial has tested supplementation effects on dementia incidence, and two trials of disease progression found no benefit.

Nonetheless, omega-3 fatty acids may be protective in persons not carrying the gene conveying risk for Alzheimer's disease, APOE ε4.

Other Psychiatric Disorders

Other major conditions such as schizophrenia and attention-deficit/hyperactivity have also been linked to low consumptions of EPA and DHA. Preliminary evidence of efficacy from supplementation exists, and additional studies are underway.

Public Health

Potential Health Benefits and Risks of Consumption of Fish or Omega-3 Fatty Acid Supplements

As summarized above, a variety of common and serious chronic health conditions are associated with low dietary consumption of the omega-3 fatty acids, EPA, and DHA. To date, clinical trials providing 2–6 months of supplementation have yielded mixed evidence of intervention efficacy, although dose and/or duration may not have been sufficient. National intake levels are low, and long-term diet modification has the potential to affect the incidence of various disorders and, accordingly, have substantial public health benefit.

Risks of fish consumption require consideration. Mercury is toxic to the brain in high doses and may have mild effects on the central and peripheral nervous systems with more modest exposure. Mercury bio-accumulates in aquatic food chains, reaching potentially dangerous concentrations in large, predatory fish with long life spans (e.g., swordfish, shark, and king mackerel). Caution against consumption of these fish during pregnancy is particularly emphasized, whereas consumption of other fish and any shellfish three times a week is widely considered to be safe. PCBs and dioxin are organochlorine compounds produced by commercial and industrial processes. Although they contaminate many foods (e.g., beef, chicken, butter, and fish), their levels are decreasing in recent years and PCBs and dioxin have not been clearly linked to adverse health outcomes in contemporary clinical studies. Fish oil capsules can contain varying amounts of PCBs and dioxins, but typically have no mercury.

Public Health Recommendations

To the extent that EPA and DHA can be synthesized by humans, albeit at low rates, these nutrients are not absolutely required in the diet and this complicates attempts to arrive at consensus regarding dietary recommendations. Nonetheless, a variety of expert bodies have issued recommendations and these generally suggest minimum daily intake of 250–500 mg of EPA and DHA. In order to achieve putative health benefits, persons with lifelong low intake of EPA and DHA consumption may need to increased consumption well above these recommended targets.

Cross-References

- ▶ Cognitive Function
- ▶ Coronary Heart Disease
- ▶ Fat, Dietary Intake
- ▶ Neurotransmitter

References and Readings

Appleton, K. M., Rogers, P. J., & Ness, A. R. (2010). Updated systematic review and meta-analysis of the effects of n-3 long-chain polyunsaturated fatty acids on depressed mood. *American Journal of Clinical Nutrition, 91*(3), 757–770.

Chow, C. K. (2008). *Fatty acids in foods and their health implications* (3rd ed.). Boca Raton, FL: CRC Press.

Harris, W. S., Mozaffarian, D., Lefevre, M., Toner, C. D., Colombo, J., Cunnane, S. C., et al. (2009). Towards establishing dietary reference intakes for eicosapentaenoic and docosahexaenoic acids. *Journal of Nutrition, 139*(4), 804S–819S.

Kidd, P. M. (2007). Omega-3 DHA and EPA for cognition, behavior, and mood: Clinical findings and structural-functional synergies with cell membrane phospholipids. *Alternative Medicine Review, 12*(3), 207–227.

Mozaffarian, D., & Rimm, E. B. (2006). Fish intake, contaminants, and human health: Evaluating the risks and the benefits. *JAMA: Journal of the American Medical Association, 296*(15), 1885–1899.

Omega-3 Polyunsaturated Fatty Acids

▶ Omega-3 Fatty Acids

Oncology (Oncologist)

Yu Yamada
Department of Psychosomatic Medicine, Kyushu University, Fukuoka, Japan

Definition

Oncology is the branch of medicine dealing with the biological and chemical properties of cancer, in addition to its prevention, development, diagnosis, and treatment. An oncologist is a medical professional who practices oncology. In general, oncology mainly consists of three primary disciplines: surgical oncology, radiation oncology, and medical oncology.

Surgical oncology is the field of surgery dedicated to the operative ablation of cancer. Surgery is the oldest treatment for cancer and the only treatment that can cure a patient with cancer, although chemotherapy for hematological malignances has also been known to be an effective cure. Recently, laparoscopy has emerged as a valuable tool in surgical oncology. Radiation oncology is the study of the use of radiation to destroy cancer. Radiation kills cancer cells by radiating the cells with either photons (i.e., x-rays and gamma rays) or particles (i.e., protons and electrons). Medical oncology is a subspecialty of internal medicine concerned with the study of cancer, especially chemotherapy involving treatment with anticancer drugs (see "► Chemotherapy" for more information).

There are other subspecialties within oncology. Gynecologic oncology focuses on cancers of the female reproductive system. Pediatric and geriatric oncology is the branch of medicine that studies benign and malignant tumors in children and the elderly, respectively.

Behavioral medicine may be closely related to psycho-oncology, which is the study of psychological aspects of the treatment and management of patients with cancer. Psycho-oncology addresses two major psychological dimensions of cancer: the emotional reactions of the patients, families, and staff to cancer and its treatment (psychosocial) and the psychological and behavioral factors that influence cancer risk and survival (psychobiological). While great advances have been made in the fields of psycho-oncology in general, further research is needed (1) to improve the assessment and treatment of psychiatric syndromes in cancer patients, (2) to understand the nature of distress in all stages of disease and survival, (3) to assess the needs of special subgroups such as geriatric cancer patients, (4) to understand the mechanisms and implications of cognitive changes associated with cancer treatment, (5) to integrate basic science insights into clinical practice, and (6) to continue the development and study on intervention modalities for the full range of psychological and psychiatric symptoms and syndromes encountered in the oncology setting.

Cross-References

► Cancer, Types of

References and Readings

Breitbart, W. S., & Alici, Y. (2009). Psycho-oncology. *Harvard Review of Psychiatry, 17*(6), 361–376.
DeVita, V. T., & Lawrence, T. S. (2008). *DeVita, Hellman, and Rosenberg's cancer (cancer: principles & practice)*. Philadelphia: Lippincott Williams & Wilkins.
Holland, J. C. (2009). *Psycho-oncology* (2nd ed.). New York: Oxford University Press.

Online Training

► Williams LifeSkills Program

Operant Conditioning

Misato Takada
Department of Health Economics and Epidemiology Research, School of Public Health, The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Synonyms

Instrumental conditioning

Definition

A type of learning in which the probability of a behavior recurring is increased or decreased by the consequences that follow upon occurrence of the behavior. The three-term contingency represents the simplest conceptual model of operant conditioning (Holland & Skinner, 1961).

Description

Operant conditioning applies many techniques and procedures first investigated by E. L. Thorndike (1898), but was later refined and extended by B. F. Skinner (1938). Although operant conditioning is built on the classical conditioning work of Ivan Pavlov (1927), it is distinguished from classical conditioning in that operant conditioning deals with the modification of "voluntary" (operant) behavior. The operant is behavior that acts on the environment to produce a consequence, which is meted out by the environment in response to the operant. This response encourages the operant to either repeat or cease the behavior. Operant conditioning techniques are currently used in clinical therapy, although they are typically applied as part of cognitive behavioral therapy.

Three-Term Contingency

Three-term contingency consists of discriminative stimulus, operant response, and consequences of behavior (reinforcer/punisher). Discriminative stimulus is an antecedent stimulus and is defined as a cue that signals the probable consequence of an operant response. That is, it signals whether the operant response will be reinforced or punished. Certain types of results occur after an organism performs a response to a discriminative stimulus. If the results are advantageous or favorable, the response increases; however, the response decreases when the results are disadvantageous or unfavorable.

In an example of operant conditioning, a hungry pigeon is caged in an operant box, which contains a feeder that can be activated to dispense feed by the pecking of a lighted key. Initially, the pigeon walks around the inside of the box and accidentally pecks at the key, which then releases feed. Although the pigeon does not comprehend the relation between the lighted key and feed, gradually, the frequency that the pigeon moves to the location where the feed is dispensed after pecking the lighted key increases. In this case, the pigeon learns the three-term contingency, consisting of the operant box (discrimination stimulation), pecking a lighted key (operant response), and feed (reinforcer). The change in frequency with which the pigeon pecks the lighted key represents the process of operant conditioning.

Reinforcement

Reinforcement is the process of increasing or sustaining a behavior by its consequences. Two kinds of reinforcement exist: positive and negative reinforcement.

Positive reinforcement occurs when the frequency of a behavior is increased as a result of the presentation of favorable events or outcomes, known as positive reinforcers. This form of conditioning is termed reward training.

Negative reinforcement occurs when the frequency of a behavior is increased because it is followed by the removal of unfavorable events or

outcomes, known as negative reinforcers. This form of conditioning is termed escape training.

Punishment

Punishment is a process by which a behavior is decreased by its consequences. There are two kinds of punishment: positive and negative.

Positive punishment occurs when a behavior is decreased because it is followed by the presentation of unfavorable events or outcomes, which are known as positive punishers. This form of conditioning is termed punishment training.

Negative punishment occurs when a behavior is decreased because it is followed by the removal of favorable events or outcomes, which are known as negative punishers. This form of conditioning is termed omission training.

Although punishment is more effective if combined with reinforcement, as appropriate and alternative behaviors can be learned, it is less effective without reinforcement because it only suppresses inappropriate behavior.

Extinction

Extinction is a process whereby the positive reinforcement of a previously reinforced behavior is discontinued. Organisms may exhibit resistance to extinction, by which a response continues even after the reinforcement ceases. The greater the resistance to extinction, the longer the response will continue.

Notably, extinction may produce adverse side effects; two commonly noted effects are an increase in the frequency of the target response and an increase in aggression.

Shaping

Shaping is a method for conditioning an organism to perform a new behavior. It is well described by its technical name: the method of successive approximations. To approximate something is to get close to it, and successive approximations condition an organism in small steps. Shaping works by starting with whatever the organism can already do and subsequently reinforces with closer and closer approximations to a goal.

Five simple rules for shaping are as follows: (1) Ensure the target behavior is realistic and biologically possible. (2) Specify the entering and target behaviors. (3) Plan a small chain of behavioral changes leading from the entering behavior to the target behavior. (4) If a step proves too large, break it into smaller, more manageable steps. (5) Use reinforcers in small quantities to avoid satiation.

Schedule of Reinforcement

Several types of reinforcement schedules exist. If reinforcement occurs after each desired behavior, the situation is termed continuous reinforcement. However, if reinforcement occurs only after certain desired behaviors, it is referred to as partial reinforcement. A response learned under the latter conditions is more resistant to extinction, a phenomenon called the partial reinforcement effect.

Fixed-ratio schedules are those where a response is reinforced only after a specified number of responses, while variable-ratio schedules occur when a response is reinforced after an unpredictable number of responses. Fixed-interval schedules are those where the first response is rewarded only after a specified amount of time has elapsed. Variable-interval schedules occur when a response is rewarded after an unpredictable amount of time has passed.

The response styles of subjects, whether they are pigeons in an operant box or employees in a workplace, vary based on the schedule used. Other factors being equal, a variable-ratio schedule produces the greatest number of responses from a subject in a given time period, whereas a fixed-ratio schedule fosters rapid learning of the desired response; the number of responses then remains steady, but is lower than those produced by a variable-ratio schedule. Fixed-interval schedules produce relatively few responses overall and a drop in number of responses immediately following reinforcement, although the number increases as the time for reinforcement nears. Variable-interval schedules result in slower learning of the response, followed by a steady number of responses, but produce fewer than those resulting from the fixed-ratio schedule.

Notably, the type of reinforcement schedule will have an impact on how quickly a behavior is extinguished.

Operant Conditioning Therapy

Operant conditioning therapy is a form of behavioral therapy that utilizes the procedures of shaping, token economy, chaining, response cost, time out, and stimulus control.

Token economy uses a token as a reinforcer. Tokens begin as essentially neutral stimuli and are of minor significance in and of themselves. However, as tokens become increasingly associated with the reinforcers for which they are exchanged, the tokens themselves can become mildly reinforcing. Chaining is yet another procedure that is based on shaping, but it is used to condition an entire complex series of different responses, not just one.

Response cost represents the removal of a positive reinforcer after the occurrence of an undesirable response. Time out is the time during which a discriminative stimulus is not available. Stimulus control is the process of controlling discriminative stimulus.

Cross-References

- ▶ Behavior Change
- ▶ Behavior Modification
- ▶ Behavioral Inhibition
- ▶ Behavioral Intervention
- ▶ Behavioral Medicine
- ▶ Behavioral Therapy
- ▶ Cognitive Behavioral Therapy (CBT)

References and Readings

Holland, J. G., & Skinner, B. F. (1961). *The analysis of behavior: A program for self-instruction*. New York: McGraw-Hill.

Mazur, J. E. (2006). *Learning and behavior* (6th ed.). Upper Saddle River, NJ: Prentice Hall.

Mednick, S. A., Higgins, J., & Kirschenbaum, J. (1975). *Psychology: Explorations in behavior and experience*. New York: Wiley.

Pavlov, I. P. (1927). *Conditioned reflexes: An investigation of the physiological activity of the cerebral cortex*. Oxford, UK: Oxford University Press.

Reynolds, G. S. (1975). *A primer of operant conditioning* (Revth ed.). Glenview, IL: Scott Foresman.

Robbins, S. J., Schwartz, B., & Wasserman, E. A. (2001). *Psychology of learning and behavior* (5th ed.). New York: W. W. Norton.

Skinner, B. F. (1938). *The behavior of organisms: An experimental analysis*. New York: Appleton-Century.

Skinner, B. F. (1953). *Science and human behavior*. New York: Macmillan.

Thorndike, E. L. (1898). *Animal intelligence: An experimental study of the associative processes in animals* (Psychological review monograph supplement, Vol. 2, No. 4, Whole No. 8). Lancaster, PA: Macmillan.

Operationalization of Anger

▶ Anger, Measurement

Operative Anxiety

▶ Hospital Anxiety

Opiate Neuropeptides

▶ Endogenous Opioids/Endorphins/Enkephalin

Opiate Peptides

▶ Endogenous Opioids/Endorphins/Enkephalin

Opiate Receptors

▶ Endogenous Opioids/Endorphins/Enkephalin

Opinion Poll

▶ Surveys

Opponent Process

Leah Irish
Department of Psychiatry, School of Medicine, University of Pittsburgh, Pittsburgh, PA, USA

Definition

Opponent process is a general theoretical model applied to several psychophysiological concepts, whereby a conditioned response is followed by its opposite, and this opponent process becomes stronger and more efficient with repeated exposure.

Description

Opponent process is a general theoretical model that has been applied to a number of psychological experiences and their underlying neurological processes. It was initially proposed as a theory of color vision (Hurvich & Jameson, 1957), but was later modified by Solomon and colleagues (1973, 1974, 1980) to apply to emotion and motivation. In a basic opponent process model, a stimulus elicits an unconditioned response referred to as "*a* process." After a latency, a "*b* process" is activated, which opposes the *a* process and reduces its intensity. Over repeated exposures, the strength of the *a* process does not change, but the *b* process becomes stronger and more efficient with a reduced latency period. The theory suggests that it is possible for the *b* process to become a conditioned response to neutral stimuli associated with the *a* process. Therefore, with repeated exposures, it is possible for the *b* process to occur before the *a* process, thus suppressing the experience of *a* altogether.

One classic demonstration of opponent process theory is a study of skydivers performed by Fenz and Epstein (1967). Prior to their first jump, novice skydivers reported feelings of terror (*a* process). After landing safely, the terror subsided and was replaced by feelings of exhilaration (*b* process). After several jumps, the terror became less intense because the feelings of exhilaration were stronger and came on more quickly. Measures of sympathetic nervous system activity (i.e., skin conductance, heart rate, respiration rate) before and after the jump paralleled these findings and further supported the opponent process theory (Fenz & Epstein, 1967). According to opponent process theory, after many jumps, the neutral stimuli associated with skydiving (e.g., the plane, the parachute) may activate feelings of exhilaration that suppress the experience of terror, thus making skydiving a very positive experience for veteran skydivers.

Opponent process theory has been applied most notably to the study of addiction (Koob, Caine, Parsons, Markou, & Weiss, 1997; Koob, Markou, Weiss, & Schuteis, 1993; Solomon & Corbit, 1973). In these models, the pleasurable "high" of drug use (*a* process) is followed by an affective low during withdrawal (*b* process). After repeated use, the highs become shorter and less intense because the lows begin to occur more quickly. This motivates drug users to take more drugs in order to sustain the high, and this pattern leads to tolerance and addiction. These experiences are accompanied by biochemical alterations during and after drug use (Koob et al., 1993). Similar models have been proposed to explain motivation for a variety of behaviors including blood donation, breastfeeding, exercise, and workplace satisfaction. Opponent process theory has also been applied to the affective experience of attachment and separation anxiety.

Although the opponent process theory has been successful at modeling motivation for a variety of behaviors, it is not capable of explaining all human behaviors. For example, the model is notably less effective at explaining motivation for more primary or biological needs. Further, not all motivated behaviors are accompanied by opponent processes. Despite these limitations, the opponent process theory continues to serve as an effective model for conceptualizing conditioning of motivation and emotion.

Cross-References

- ▶ Addictive Behaviors
- ▶ Affect
- ▶ Affect Arousal
- ▶ Sympathetic Nervous System (SNS)

References and Readings

Fenz, W. D., & Epstein, S. (1967). Gradients of physiological arousal in parachutists as a function of an approaching jump. *Psychosomatic Medicine, 29*, 33–51.

Hurvich, L. M., & Jameson, D. (1957). An opponent-process theory of color vision. *Psychological Review, 64*(6), 384–404.

Koob, G. F., Caine, S. B., Parsons, L., Markou, A., & Weiss, F. (1997). Opponent process model and psychostimulant addiction. *Pharmacology Biochemistry and Behavior, 57*(3), 513–521.

Koob, G. F., Markou, A., Weiss, F., & Schuteis, G. (1993). Opponent process and drug dependence: Neurobiological mechanisms. *Neurosciences, 5*, 351–358.

Solomon, R. L. (1980). The opponent-process theory of acquired motivation: The costs of pleasure and the benefits of pain. *The American Psychologist, 35*, 691–712.

Solomon, R. L., & Corbit, J. D. (1973). An opponent-process theory of emotion: II. Cigarette addiction. *Journal of Abnormal Psychology, 81*(2), 158–171.

Solomon, R. L., & Corbit, J. D. (1974). An opponent-process theory of motivation: I. Temporal dynamics of affect. *Psychological Review, 81*(2), 119–145.

Opportunistic Infections

- ▶ AIDS: Acquired Immunodeficiency Syndrome

Optimal Aging

- ▶ Successful Aging

Optimism

- ▶ Dispositional Optimism

Optimism and Pessimism Scale (OPS)

- ▶ Optimism and Pessimism: Measurement

Optimism and Pessimism: Measurement

Ryan Garcia
University of Texas Southwestern Medical Center at Dallas, Dallas, TX, USA

Synonyms

Attributional style questionnaire (ASQ); Expanded attributional style questionnaire (EASQ); Extended life orientation test (E-LOT); Life orientation test (LOT); Optimism and pessimism scale (OPS); Parent-rated life orientation test of children (P-LOT); Revised life orientation test (LOT-R); Youth life orientation test (Y-LOT)

Definition

Measures assessing optimism and pessimism have been developed from two perspectives. The first is from the expectancy perspective which reflects the expectations an individual has about their future. From this perspective, dispositional optimism is described as the tendency for an individual to have positive expectations about the future, whereas dispositional pessimism is the tendency to have negative expectations about the future. The second perspective is based on attributional style. Conceptually, attributional style refers to an individual's habitual manner of explaining the cause of negative events. Explanatory styles can be internal/external, stable/unstable, and global/specific. An optimistic explanatory style is when negative events and their causes are viewed as external, unstable, and specific. A pessimistic explanatory

style is when a negative event and its cause are construed as internal, stable, and global.

Several different measures exist to assess optimism and pessimism from an expectancy perspective, and are primarily derived from the Life Orientation Test (LOT). The current standard for measuring optimism and pessimism in adults is the Revised Life Orientation Test (LOT-R). The LOT-R consists of ten items: three assessing optimism (e.g., "In uncertain times, I usually expect the best."), three assessing pessimism (e.g., "If something can go wrong for me, it will."), and four filler items to disguise the purpose of the test (e.g., "It's easy for me to relax"). In principal component factor analysis, all six items of the LOT-R yielded one factor accounting for 48.1% of the variance, with factor loadings of at least 0.58. The LOT-R has sufficient internal consistency (Cronbach's alpha = 0.78), and test-retest reliability as administered between 4 and 28 months apart (0.68–0.79 respectively). The LOT and LOT-R are considered to be measures of "trait" dispositions while the Optimism Pessimism Scale (OPS) measures "state" dispositions. The Extended Life Orientation Test (E-LOT) is a longer measure comprised of items from the LOT and OPS measures, and was developed specifically to evaluate optimism and pessimism as independent constructs. The Youth Life Orientation Test (Y-LOT) is used in adolescent populations, while the Parent-rated Life Orientation Test of children (P-LOT) is a newer instrument designed to obtain parent's reports of their child's levels of optimism and pessimism.

Attributional measures of optimism and pessimism include the Attributional Style Questionnaire (ASQ) and Expanded Attributional Style Questionnaire (EASQ). The ASQ is comprised of six positive and six negative events, while the EASQ contains only negative events. These measures assess appraisals of internality, stability, and globality. The ASQ attempts to assess attributional style through presenting an individual with 12 events, and asks them the cause of each event if they were to happen to them, and then rate the cause of the event on scales representing internality, stability, and globality. Half of the events are good events (e.g., "You become very rich.") and half are bad events (e.g., "You have been looking for a job unsuccessfully for some time."). The ASQ has shown good internal consistency for good events and bad events (Cronbach's alpha = 0.75 and 0.72, respectively), and test-retest reliability over a 5-week time period (with scores ranging from 0.58 to 0.70).

There has been considerable debate in the literature regarding expectant measures of optimism and pessimism and whether optimism and pessimism should be regarded as a single-dimension or two-dimensional constructs. Research findings are varied, with some studies identifying the LOT and LOT-R as two dimensional (i.e., optimistic and pessimistic items load on two different dimensions during factor analysis), while others support a single-dimension interpretation (i.e., both optimistic and pessimistic items load on one dimension). Given the nature of the literature surrounding this topic, it is prudent to determine which orientation will best capture the construct of interest for the research question posed (e.g., expectant versus attributional), and to carefully consider issues surrounding the interpretation of test findings (e.g., the dimensionality of optimism and pessimism).

Cross-References

- ▶ Attribution Theory
- ▶ Dispositional Optimism
- ▶ Explanatory Style
- ▶ Life Orientation Test (LOT)
- ▶ Neuroticism
- ▶ Pessimism
- ▶ Positive Affect Negative Affect Scale (PANAS)

References and Readings

Chang, E. C., Maydeu-Olivares, A., & D'Zurilla, T. J. (1997). Optimism and pessimism as partially independent constructs: Relationship to positive and negative affectivity and psychological well-being. *Personality and Individual Differences, 23*(3), 433–440.

Dember, W. N., Martin, S., Hummer, M. K., Howe, S., & Melton, R. (1989). The measurement of optimism and

pessimism. *Current Psychology: Research and Reviews, 8*, 102–119.
Ey, S., Hadley, W., Allen, D. N., Palmer, S., Klosky, J., Deptula, D., et al. (2005). A new measure of children's optimism and pessimism: The youth life orientation test. *Journal of Child Psychology and Psychiatry, 46*(5), 548–558.
Lemola, S., Raikkonen, K., Matthews, K. A., Scheier, M. F., Heinonen, K., Pesonen, A. K., et al. (2010). A new measure for dispositional optimism and pessimism in young children. *European Journal of Personality, 24*, 71–84.
Peterson, C., Semmel, A., von Baeyer, D., Abramson, L. Y., Metalsky, G. I., & Seligman, M. E. P. (1982). The attributions style questionnaire. *Cognitive Therapy and Research, 6*, 287–300.
Peterson, C., & Villanova, P. (1988). An expanded attributional style questionnaire. *Journal of Abnormal Psychology, 97*, 87–89.
Rasmussen, H. N., Scheier, M. F., & Greenhouse, J. B. (2009). Optimism and physical health: A meta-analytic review. *Annals of Behavioral Medicine, 37*(3), 239–256.
Scheier, M. F., & Carver, C. S. (1985). Optimism, coping, and health: Assessment and implications of generalized outcome expectancies. *Health Psychology, 4*(3), 219–247.
Scheier, M. F., Carver, C. S., & Bridges, M. (1994). Distinguishing optimism from neuroticism (and trait anxiety, self-mastery, and self-esteem): A reevaluation of the life orientation test. *Journal of Personality and Social Psychology, 67*(6), 1063–1078.
Smith, T. W., Pope, M. K., Rhodewalt, F., & Poulton, J. C. (1989). Optimism, neuroticism, coping, and symptom reports: An alternative interpretation of the Life Orientation Test. *Journal of Personality and Social Psychology, 56*(4), 640–648.

Optimism, Pessimism, and Health

Ryan Garcia
University of Texas Southwestern Medical Center at Dallas, Dallas, TX, USA

Synonyms

Dispositional optimism; Dispositional pessimism

Definition

Optimism is a dispositional variable characterized by a generalized positive expectation about the future, while pessimism reflects a generalized negative expectation about the future. Although optimism and pessimism were originally viewed as occupying opposite ends of a single dimension, they have also been considered as independent variables. However, the dimensionality of these two constructs continues to be an ongoing debate in the literature. Each has been associated with different outcomes. Optimism has been associated with better psychological and physiological health outcomes, and these relationships are thought to be mediated by a coping style characterized by problem and emotion approach coping strategies. In contrast, pessimism has been associated with poorer psychological and physiological health outcomes, which is believed to be mediated by the reliance upon problem and emotion avoidance coping strategies.

Description

Optimism is a dispositional variable that involves a propensity to foster positive expectations for the future. Pessimism, on the other hand, involves a propensity to foster negative expectations for the future. Optimistic and pessimistic beliefs are conceptually based on theories of coping and behavioral self-regulation, and are thought to influence behavior in the service of goal pursuit. Optimism is theorized to provide motivation for sustained goal pursuit, which is demonstrated by continued active and productively guided effort and behavior both in daily living and in stressful situations. Pessimism is thought to undermine active goal pursuit and effort, and to contribute to suboptimal outcomes. Other factors theorized to underlie optimistic and pessimistic coping strategies include confidence, doubt, engagement, and disengagement.

High levels of optimism have been associated with several positive physiological outcomes among adults coping with acute and chronic illness, including cardiac patients, and HIV + and AIDS patients. In cardiac patients, high levels of optimism have been associated with minimal increases in carotid intima thickness (an index of atherosclerosis in the carotid

artery and a marker for heart disease) over a three-year period, and a decreased likelihood for developing coronary heart disease. Following coronary artery bypass surgery, high optimism has been associated with a quicker recovery and a significantly decreased chance of rehospitalization. In HIV + and AIDS patients, high optimism has shown associations with delayed symptom onset, slower disease progression, and decreased mortality.

High optimism has also been associated with psychosocial benefits among patients with chronic illness and those experiencing major life changes. Patients recovering from coronary artery bypass surgery report fewer negative mood states and higher satisfaction with medical care and quality of life after surgery, while adults with type 2 diabetes reported experiencing less depression and anxiety. Similar positive psychosocial outcomes have been found in cancer and HIV + populations, and ante- and postpartum mothers. Overall, pessimism is less beneficial for physiological and psychosocial outcomes in health contexts. Evidence suggests individuals high in pessimism experience more fatigue, depression and anxiety, and poorer outcomes and adjustment following illness and illness recovery. Strong support for the associations observed between optimism and pessimism, and physiological and psychosocial health outcomes was demonstrated in a recent meta-analysis of the literature on coping and health outcomes (see Rasmussen, Scheier, & Greenhouse, 2009).

Coping styles have largely been investigated as mediating the associations between optimism, pessimism, and their observed outcomes. Thus far, research investigating coping styles among optimists and pessimists during times of distress has generally supported that optimists who experience physiological and psychological benefits rely on approach coping strategies, while pessimists rely on avoidant coping strategies. Additional support for these coping styles among optimists and pessimists was shown in a meta-analysis in which general patterns emerged; problem and emotion approach coping strategies appear to characterize the coping styles of those high in optimism, while problem and emotion avoidance coping strategies characterize the coping styles of pessimists (Solberg-Nes & Segerstrom, 2006). Also, perceptions of control have been considered to contribute to the types of coping strategies optimists and pessimists use. Optimists seem to rely on problem approach coping strategies when confronted with controllable stressors and emotion approach coping strategies when dealing with uncontrollable stressors. This is thought to underlie an overall engagement and persistence in dealing with their problems, whether it be directly (e.g., active/instrumental coping or planning) or indirectly through cognitive strategies such as acceptance and positive reinterpretation and growth. On the other hand, pessimists seem to show the opposite response in similar situations and rely more on problem avoidance and emotion avoidance coping strategies such as avoidance, distraction, denial, escapism, or substance use. Pessimistic coping reflects an overall disengagement and withdrawal from dealing with stressors, regardless of their level of controllability. For a more detailed discussion about optimism, pessimism, and coping styles, please refer to Solberg Nes & Segerstrom, 2006.

Cross-References

- ▶ Attribution Theory
- ▶ Dispositional Optimism
- ▶ Explanatory Style
- ▶ Life Orientation Test (LOT)
- ▶ Optimism and Pessimism: Measurement
- ▶ Pessimism
- ▶ Self-Regulation Model

References and Readings

Abela, J. R. Z., Auerbach, R. P., & Seligman, M. E. P. (2008). Dispositional pessimism across the lifespan. In K. S. Dobson & D. J. A. Dozois (Eds.), *Risk factors in depression* (pp. 195–220). San Diego, CA: Academic Press.

Carver, C. S., Scheier, M. F., Miller, C. J., & Fulford, D. (2002). Optimism. In C. R. Snyder & S. J. Lopez (Eds.), *Oxford handbook of positive psychology* (pp. 303–312). New York: Oxford University Press.

Carver, C. S., Scheier, M. F., & Segerstrom, S. C. (2010). Optimism. *Clinical Psychology Review, 30*, 879–889.

Chang, E. C. (Ed.). (2002). *Optimism & pessimism: Implications for theory, research, and practice*. Washington, DC: American Psychological Association.
Folkman, S., & Moskowitz, J. T. (2004). Coping: Pitfalls and promise. *Annual Review of Psychology, 55*, 745–774.
Rasmussen, H. N., Scheier, M. F., & Greenhouse, J. B. (2009). Optimism and physical health: A meta-analytic review. *Annals of Behavioral Medicine, 37*(3), 239–256.
Scheier, M. F., & Carver, C. S. (1985). Optimism, coping, and health: Assessment and implications of generalized outcome expectancies. *Health Psychology, 4*(3), 219–247.
Scheier, M. F., & Carver, C. S. (1987). Dispositional optimism and physical well-being: The influence of generalized outcome expectancies on health. *Journal of Personality, 55*(2), 169–210.
Scheier, M. F., & Carver, C. S. (1992). Effects of optimism on psychological and physical well-being: Theoretical overview and empirical update. *Cognitive Therapy and Research, 16*(2), 201–228.
Scheier, M. F., & Carver, C. S. (2003). Self-regulatory processes and responses to health threats: Effects of optimism on well-being. In J. Suls & K. Wallston (Eds.), *Social psychological foundations of health* (pp. 395–428). Oxford, UK: Blackwell.
Scheier, M. F., Weintraub, J. K., & Carver, C. S. (1986). Coping with stress: Divergent strategies of optimists and pessimists. *Journal of Personality and Social Psychology, 51*(6), 1257–1264.
Skinner, E. A., & Zimmer-Gembeck, M. J. (2007). The development of coping. *Annual Reviews of Psychology, 58*, 119–144.
Solberg Nes, L., & Segerstrom, S. C. (2006). Dispositional optimism and coping: A meta-analytic review. *Personality and Social Psychology Review, 10*(3), 235–251.

Oral Glucose Tolerance Test (OGTT)

Adriana Carrillo
Department of Pediatrics, Miller School of Medicine, University of Miami, Miami, FL, USA

Synonyms

Glucose test

Definition

The OGTT test is used for the diagnosis of prediabetes, diabetes, and rare disorders of carbohydrate metabolism. The OGTT is a procedure in which a standardized load of glucose is provided and plasma glucose levels are drawn at different times over a 2-h period. Insulin levels could be also obtained to determine insulin resistance. Normal values for fasting glucose levels are below 100 mg/dL. Impaired fasting plasma glucose levels are considered between 100 and 125 mg/dL, and fasting plasma glucose levels above 126 mg/dL are considered in the diabetes range. The 2-h glucose level will help in the definitive diagnosis of diabetes. Plasma glucose levels above 200 mg/mL are diagnostic for diabetes mellitus. Levels between 140 and 200 mg/mL at 2 h are considered in the prediabetes range.

Procedure

A diet that includes at least 60% of calories as carbohydrates should be provided at least for 3 days before the test. The test should not be performed if there is a history of a physiological stress such as infection, acute illness, trauma, or surgery over the past 2 weeks. Medications that could affect glucose levels should also be discontinued. Patient should be fasting for at least 10–12 h. A baseline sample is obtained, followed by administration of an oral glucose solution of 1.75 g/kg to a max of 75 g within 5 min. Samples are generally obtained at 30, 60, and 120 min after the glucose administration (Lifshitz, 2007).

Cross-References

▶ Fasting Glucose
▶ Hyperglycemia

References and Readings

Lifshitz, F. (2007). *Pediatric endocrinology* (5th ed., p. 755). New York: Informa Healthcare.

Organ Replacement Therapy

▶ Organ Transplantation: Psychological and Behavioral Aspects

Organ Transplantation: Psychological and Behavioral Aspects

Mary Amanda Dew, Kristen R. Fox and Andrea F. DiMartini
School of Medicine and Medical Center, University of Pittsburgh, Pittsburgh, PA, USA

Synonyms

Organ replacement therapy

Definition

Organ transplantation involves the surgical implantation of an organ or section of an organ into a person whose own organ is failing. The donor organ may come from a deceased individual or, in some cases, from a living donor. Psychological and behavioral aspects of the organ transplantation process encompass transplant patients' emotional responses and mental health, as well as their behavior in adhering to the medical regimen both before and after transplantation. The living donor's psychological response to donating an organ (most commonly for kidney and liver segment transplantation) is also an aspect to consider in the transplantation process.

Description

Individuals with end-stage diseases of the kidney, pancreas, intestines, liver, heart, or lung may receive organ transplantation in order to extend their lives and/or to improve the quality of their lives. Organs typically are transplanted from deceased individuals or, in the case of kidney and liver transplantation, may come from living donors. Organ transplantation is one of a growing range of organ replacement therapies. Dialysis for end-stage kidney disease and mechanical circulatory support systems for end-stage heart disease are examples of alternatives to organ transplantation. Organ transplantation remains the optimal strategy for organ replacement because it is often less economically costly both to the patient and to payers (Medicare, private insurance companies) and because it often provides higher quality of life for a longer time period. However, like other organ replacement strategies, it is associated with numerous stressors that may take a psychological toll on patients and their families (Dew & DiMartini, 2011; Rodrigue, 2001; Trzepacz & DiMartini, 2000). It also has specific behavioral consequences: patients must adhere to a complex, lifelong medical regimen involving multiple medications, routine monitoring of the functioning of the transplanted organ, requirements for medical follow-up care, and permanent lifestyle changes in areas such as diet and exercise. Finally, because some types of organ transplantation are increasingly reliant on living donors, it is important to recognize that these donors also face significant stressors associated with medical and surgical risks of surgery, potential financial issues, and longer-term physical health concerns (Dew, Switzer, DiMartini, Myaskovsky, & Crowley-Matoka, 2007c; Dew & DiMartini, 2011).

In the sections below, we summarize the psychological and behavioral issues that are most prominent for organ transplant patients both before and after organ transplantation. We also describe psychological issues relevant for living donors. Lastly, we comment on interventions that have been developed to treat or prevent psychological or behavioral problems during the transplantation process.

Psychological Factors in Organ Transplant Patients

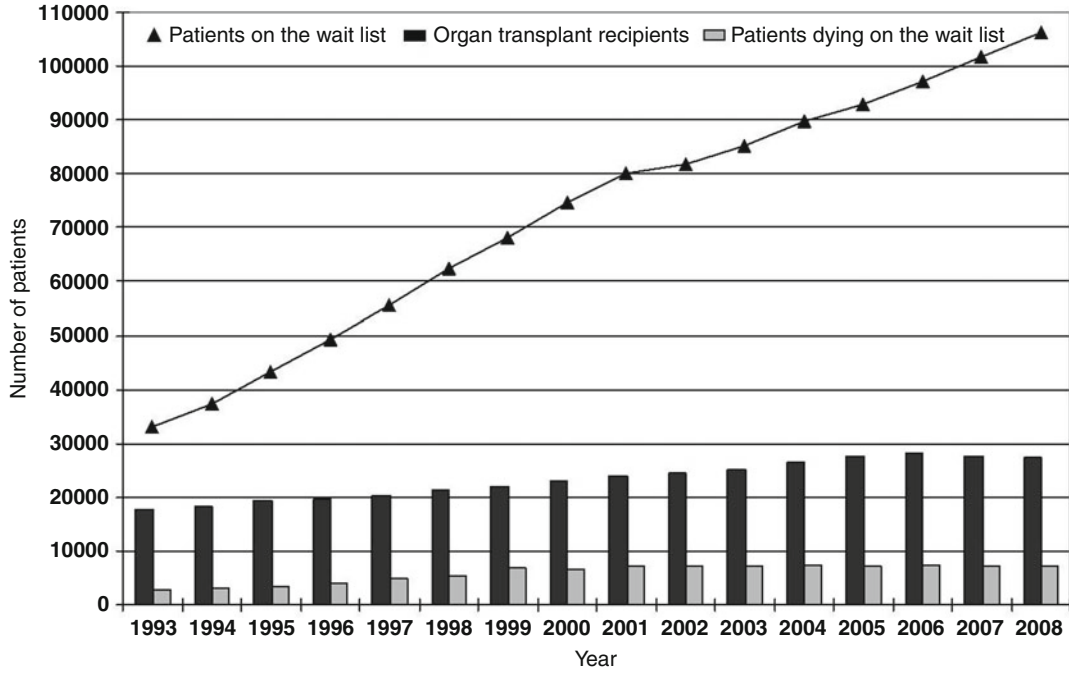
The evaluation for transplant eligibility and the wait for an organ transplant are two of the chief stressors associated with the transplantation process (Craven & Farrow, 1993; Dew, DiMartini, & Kormos, 2007b). These stressors occur in the context of patients' physical morbidity and declines in physical functioning, and this physical decline is a potent chronic stressor as well. The evaluation for transplantation includes both

medical and psychosocial components (DiMartini, Dew, & Crone, 2009; Olbrisch, Benedict, Ashe, & Levenson, 2002). Patients often fear that their health status is too poor for a transplant or that they may be judged to be ineligible for psychological or psychosocial reasons. As part of the evaluation process, patients' psychiatric history, substance use, intellectual functioning, medical adherence history, and psychosocial status are thoroughly reviewed by most transplant programs, and these areas do influence decisions about whether or not to approve patients for transplantation. However, there is substantial variability across transplant programs in whether or not problems in these areas (e.g., current mood disorders, substance abuse history, and current status) are viewed as contraindications to transplantation. In many circumstances, patients are not permanently ruled out for transplantation due to such problems but instead are required to undergo psychological or behavioral interventions, or demonstrate that certain problems are being addressed. For example, many programs require evidence of abstinence from alcohol or drug use for a period of months before an individual will be approved for transplantation. The need to complete interventions in order to be approved for transplantation can be viewed by patients as one more hurdle to complete in the face of their significant medical illness. However, to the extent that they develop stronger coping skills or feelings of control as a result of these interventions, patients may become better prepared for life posttransplant.

In the USA and most other countries, after individuals are approved for transplantation, they are entered into a national wait list for deceased donor organs. Because there are too few organs for the number of individuals in need, the waiting time for transplantation may be months to years (Organ Procurement and Transplantation Network, 2009). Figure 1 shows the number of individuals waiting for transplantation in the USA, as well as the number of organs available for transplantation during each of the past 15 years. It also shows the numbers of patient deaths on the wait list. Thus, in 2008, while 27,281 individuals received transplants, another 7,182 died on the wait list. Fear of not surviving to transplantation is a looming and unfortunately very realistic concern among transplant candidates. Although strategies such as dialysis for end-stage kidney disease and mechanical circulatory support systems for end-stage heart disease can keep patients alive for extended periods during the wait for a transplant, these therapies bring their own risks for health problems and even death. Indeed, the development of complications from these therapies (e.g., infections) can result in an individual being removed from the transplant waiting list. Other stressors during the waiting period for transplant also stem from transplant candidates' declining physical health status: they may have to reduce or give up family and social roles (e.g., parental, household, work, or community responsibilities), and they may eventually become dependent on others for daily care.

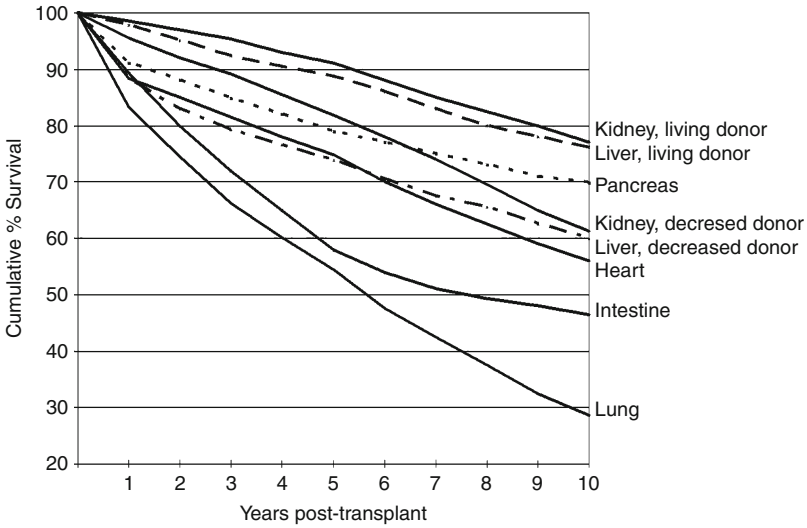
The presence of these powerful stressors during the waiting period explains why patients show high rates of psychological distress and diagnosable episodes of mental illness during this time. Mood and anxiety disorders are the most commonly diagnosed problems in these individuals (Dew & DiMartini, 2011). Transplant candidates show higher rates of these disorders than do other chronic disease populations. Among the chief correlates of psychological distress and disorder in transplant candidates are physical symptom severity and physical functional impairments, as well as poorer social supports. History of psychological disorder is another important risk factor, suggesting the importance of taking steps to prevent new episodes of depression and anxiety as patients face the stressors associated with the evaluation and wait for transplantation.

After transplantation, there is overwhelming evidence that recipients' quality of life and their overall sense of well-being improve (Dew & DiMartini, 2011). In particular, transplant recipients show marked gains in physical functioning. Gains in mental health appear to be somewhat smaller. In the early months posttransplant, recipients typically voice high levels of optimism for the future and great relief that the uncertainties associated with the waiting period are over.



Organ Transplantation: Psychological and Behavioral Aspects, Fig. 1 Size of wait list, number of transplants, and deaths on the wait list, United States (Source: 2002 and 2009 Annual Reports, U.S. Organ Procurement and Transplantation Network and the Scientific Registry of Transplant Recipients (http://optn.transplant.hrsa.org))

Nevertheless, they remain at high risk for mood disorders (including major depression, dysthymia, and adjustment disorders) and anxiety disorders (including posttraumatic stress disorder, panic disorder, generalized anxiety disorder, and adjustment disorders) during the first several years posttransplant. The risk appears to be highest during the first year posttransplant, perhaps because of the major psychosocial adjustments required of patients during this period. For example, they must psychologically incorporate the fact that they have an organ from another person and – in many cases – that the donation was only possible because donor had died. Patients must also adapt to new routines for medical follow-up care and to a new regimen of medications and permanent lifestyle changes related to diet, exercise, limited to no alcohol consumption, and no use of other substances. Patients themselves often comment on the stressfulness of making these psychological and behavioral adjustments. They also report that they have developed significant depression or anxiety as a result of psychosocial stressors related to the financial burdens associated with transplantation (because insurance coverage is often inadequate), attempts to return to former social roles (e.g., returning to employment or parenting activities), and ongoing medical factors related to the transplant. For example, they may develop complications such as infection or they may experience acute rejection (i.e., their bodies may mount an immunologic reaction to the transplanted organ), despite the powerful immunosuppressant medications that they take. Additional personal or dispositional characteristics also appear to increase patients' vulnerability to both distress and diagnosable disorder early posttransplant. These include poorer perceived social supports, use of avoidant coping strategies, a poor sense of mastery or control, and low levels of characteristics such as optimism or hope.



Organ Transplantation: Psychological and Behavioral Aspects, Fig. 2 Patient survival rates by type of organ transplant, United States, 1997–2007 (Source: 2009 Annual Reports, U.S. Organ Procurement and Transplantation Network and the Scientific Registry of Transplant Recipients (http://optn.transplant.hrsa.org))

After the first year posttransplant, the risk for psychological disorders appears to decline. Over the next several years, patients may experience high levels of overall well-being. Their psychological distress levels are low, and this is likely because their physical functional status is at its peak: they have fully recovered from the transplant surgery, and they have been able to resume activities that were impossible during their earlier end-stage organ disease. Most patients express high degrees of appreciation that they were given this "second chance" at life.

There is some evidence that recipients' psychological distress levels begin to rise once again as they enter the later years of posttransplant life expectancy (Dew & DiMartini, 2011). The typical survival time posttransplant varies across the different types of transplantation (Organ Procurement and Transplantation Network, 2009). Survival rates for the major forms of organ transplantation are shown in Fig. 2. In the later years of transplant survivorship, patients are likely to have developed chronic rejection of the transplanted organ, a condition characterized by a progressive deterioration of the organ's function. They are also likely to experience problems secondary to their immunosuppressant medication, including cancers and kidney damage. Some patients may be entered on the wait list for another transplant, with its consequent uncertainties as to whether another organ will become available. Other patients may be ineligible for retransplantation because of conditions such as cancer. All of these stressors emerging during the late-term years increase patients' risk for renewed psychological distress. Research has not yet established whether symptoms of depression or anxiety are more prominent during these later years or whether psychosocial factors related to coping, poor social supports, low levels of optimism, etc., serve as additional risk factors for distress among late-term survivors.

Behavioral Factors in Organ Transplant Patients

Adherence to the medical regimen is the principle behavioral issue for patients (Denhaerynck et al., 2005; Rodrigue, 2001). We noted earlier that patients' medical adherence history is routinely considered during their evaluation for transplantation. Before they are approved for transplantation, patients may be required to demonstrate that they are willing and able to adhere to medical recommendations (including abstaining from substance use if this has been identified as an issue). After transplantation, patients must follow a lifelong regimen that includes (a) taking immunosuppressant and other medications,

(b) attending medical follow-up evaluations and completing required tests, (c) monitoring vital signs and symptoms related to organ function, (d) following lifestyle recommendations regarding diet and exercise, and (e) limiting or abstaining from alcohol use and abstaining from other substance use. This regimen is designed to promote survival and optimal health, and growing evidence shows that nonadherence to its various components is indeed associated with morbidity and mortality posttransplant (Butler, Roderick, Mullee, Mason, & Peveler, 2004).

The majority of transplant recipients are able to maintain adherence to their multifaceted regimen. However, some individuals have difficulty with one or more areas, and this difficulty may begin as soon as they are discharged from the hospital after the transplant. Among the most common areas of nonadherence is medication taking: across many studies, the average rate of nonadherence to immunosuppressant medications is approximately 23% of patients annually (Dew et al., 2007a). Lifestyle and health monitoring requirements are also areas of difficulty, with 19–25% of patients nonadherent annually. Nonadherence to substance use restrictions is less common, occurring in 1–4% of patients annually (Sabaté, 2003). As with other chronic diseases, nonadherence to the posttransplant regimen appears to worsen with time posttransplant.

The correlates and risk factors for nonadherence to the transplant-related medical regimen have been difficult to identify. The World Health Organization has proposed a set of five categories of risk factors for nonadherence to chronic disease treatment regimens (Sabaté, 2003). These categories include demographic factors, health-care system and treatment provider factors (e.g., access to insurance and provider communication skills), disease-related factors (e.g., perceptions of health), treatment-related factors (e.g., medication side effects), and patient-related psychosocial factors (e.g., social supports). There is some evidence in support of each of these categories of factors in transplant recipients, although their impact has often been found to be relatively modest. The most important factor appears to be history of nonadherence before transplant. For example, smoking or alcohol use/abuse before transplant is very strongly associated with posttransplant return to use of these substances, as would be expected given the powerful nature of most addictions. Nevertheless, return to substance use (or abuse) occurs in only a minority – less than 10% – of individuals who receive transplants (Dew et al., 2008).

Psychological Factors in Living Donors

Living donors give an invaluable gift to others. Their gift is all the more remarkable since they face major stressors associated with donation surgery and recovery. They may experience significant pain and discomfort, as well as time lost from work or other responsibilities, financial costs that cannot be reimbursed, and longer-term health risks linked to the donation surgery itself or the loss of organ mass. When a donor has a close emotional connection to the intended recipient, additional stressors arise from watching the patient's health decline gradually or facing a crisis situation of rapid health deterioration.

Like transplant candidates, living donors undergo an extensive medical and psychosocial evaluation (DiMartini, Sotelo, & Dew, 2011; Olbrisch, Benedict, Haller, & Levenson, 2001). Donor candidates may be as fearful as transplant candidates that they will not be approved as a suitable donor. However, other potential donors may find the evaluation stressful because they are ambivalent about or uncertain that they should or want to donate. The evaluation process is designed to ensure that donors do not have major medical or psychological problems that would preclude safe donation, as well as to ensure that they understand the risks associated with donation, are not experiencing coercion or undue pressure from others to donate, and are able to make an informed choice to proceed with the surgery or not. As for transplant candidates, potential living donors may be required to undergo interventions to address any mental health or substance use issues before they are approved as donors.

A chief goal of living donation is to provide needed organs for patients yet protect the living donor from undue harm. This includes protection

from psychological harm. Most donors, in fact, show very favorable quality of life outcomes, and extremely few express any regret associated with donating. The vast majority (up to 95%) also perceive the donation to have been a very gratifying experience that benefitted them personally (by helping them to reframe, e.g., what was important in their lives) (Clemens et al., 2006). Nonetheless, some donors do experience psychological difficulties, including mood and anxiety disorders or heightened nonspecific psychological distress. Some of this distress appears to be linked to ongoing worries about the long-term impact of the donation on donors' physical health, as well as financial strains or family relationships that became strained in the aftermath of the donation. Pre-donation ambivalence about whether or not to donate has also been linked to the occurrence of psychological difficulties postdonation (Dew, Switzer, DiMartini, Myaskovsky, & Crowley-Matoka, 2007c).

Interventions for Psychological or Behavioral Problems During the Transplantation Process

Transplant programs employ strategies found effective in other chronic disease and medically healthy populations in order to address psychological and adherence-related difficulties in organ transplant patients. To care for psychological distress, psychotropic medications may be prescribed, and referrals may be made for individual or group psychotherapy or supportive care. Some programs have attempted to offer brief individual or group psychoeducational programs. There has been little attempt to formally develop and evaluate the effectiveness of interventions in transplant patients due to major constraints that these programs face. For example, many transplant candidates and recipients reside long distances from their transplant center and would not be available to participate in face-to-face interventions except during periods of hospitalization around the time of the transplant. Medical follow-up appointments are generally too brief for intensive interventions. Similar constraints exist for any living donors who develop psychological difficulties after donation because they are even less likely to return to the transplant center after donation.

Some alternatives have been developed that do not require face-to-face contact, including telephone- or internet-based psychotherapeutic or educational strategies or the use of home-based self-monitoring tools. These alternatives have targeted both psychological distress as well as difficulties with adherence. Evaluation of these experimental interventions has yielded promising results in transplant patients, including the abatement of psychological distress and improvements in medical adherence, relative to comparison groups receiving only standard care from the transplant program (De Bleser, Matteson, Dobbels, Russell, & De Geest, 2009; Dew & DiMartini, 2011). Most studies have examined relatively short time periods of well less than 1 year. Whether these interventions' effects would be maintained over longer time periods is an important issue. Work is underway to test brief psychological interventions to prevent psychological distress and associated quality of life decrements in living donors.

Cross-References

- ▶ Adherence
- ▶ Anxiety Disorder
- ▶ Chronic Disease or Illness
- ▶ Depression: Symptoms
- ▶ Depression: Treatment
- ▶ Health Behaviors
- ▶ Health-Related Quality of Life
- ▶ Psychiatric Disorder
- ▶ Psychological Disorder
- ▶ Psychological Factors
- ▶ Psychological Stress
- ▶ Psychosocial Factors
- ▶ Quality of Life
- ▶ Stress
- ▶ Stressor

References and Readings

Butler, J. A., Roderick, P., Mullee, M., Mason, J. C., & Peveler, R. C. (2004). Frequency and impact of nonadherence to immunosuppressants after renal transplantation: A systematic review. *Transplantation, 77*(5), 769–776.

Clemens, K. K., Thiessen-Philbrook, H., Parikh, C. R., Yang, R. C., Karley, M. L., Boudville, N., et al. (2006). Psychosocial health of living kidney donors: A systematic review. *American Journal of Transplantation, 6*(12), 2965–2977.

Craven, J., & Farrow, S. (1993). *Surviving transplantation: A personal guide for organ transplant patients, their families, friends and caregivers* (p. 1993). Toronto: University of Toronto Press.

Cupples, S. A., Dew, M. A., Grady, K. L., De Geest, S., Dobbels, F., Lanuza, D., et al. (2006). Report of the Psychosocial Outcomes Workgroup of the Nursing and Social Sciences Council and the ISHLT. The present status of research on psychosocial outcomes in cardiothoracic transplantation: Review and recommendations for the field. *Journal of Heart & Lung Transplantation, 25*, 716–725.

De Bleser, L., Matteson, M., Dobbels, F., Russell, C., & De Geest, S. (2009). Interventions to improve medication-adherence after transplantation: A systematic review. *Transplant International, 22*(8), 780–797.

Denhaerynck, K., Dobbels, F., Cleemput, I., Desmyttere, A., Schäfer-Keller, P., Schaub, S., et al. (2005). Prevalence, consequences and determinants of nonadherence in adult renal transplant patients: A literature review. *Transplantation International, 18*(10), 1121–1133.

Dew, M. A., DiMartini, A. F., DeVito Dabbs, A., Myaskovsky, L., Steel, J., Unruh, M., et al. (2007a). Rates and risk factors for nonadherence to the medical regimen after adult solid organ transplantation. *Transplantation, 83*, 858–873.

Dew, M. A., DiMartini, A. F., & Kormos, R. L. (2007b). Organ transplantation, stress of. In G. Fink (Ed.), *Encyclopedia of stress* (2nd ed., Vol. 3, pp. 35–44). Oxford, UK: Academic Press (Elsevier).

Dew, M. A., Switzer, G. E., DiMartini, A. F., Myaskovsky, L., & Crowley-Matoka, M. (2007c). Psychosocial aspects of living organ donation. In H. P. Tan, A. Marcos, & R. Shapiro (Eds.), *Living donor transplantation* (pp. 7–26). New York: Informa Healthcare.

Dew, M. A., DiMartini, A. F., Steel, J., DeVito Dabbs, A., Myaskovsky, L., Unruh, M., et al. (2008). Meta-analysis of risk for relapse to substance use after transplantation of the liver or other solid organs. *Liver Transplantation, 14*, 159–172.

Dew, M. A., DiMartini, A. F. (2011). Transplantation. In H. S. Friedman (Ed.), *Oxford Handbook of Health Psychology* (pp. 522–559). NY: Oxford University Press.

Dew, M. A., Dunbar-Jacob, J., Switzer, G. E., DiMartini, A. F., Stilley, C., & Kormos, R. L. (2001). Adherence to the medical regimen in transplantation. In J. R. Rodrigue (Ed.), *Biopsychosocial perspectives on transplantation* (pp. 93–124). New York: Kluwer Academic/Plenum.

DiMartini, A. F., Crone, C., Fireman, M., & Dew, M. A. (2008). Psychiatric aspects of organ transplantation in critical care. *Critical Care Clinics, 24*(4), 949–981.

DiMartini, A. F., Dew, M. A., & Crone, C. C. (2009). Organ transplantation. In B. J. Sadock, V. A. Sadock, & P. Ruiz (Eds.), *Kaplan and Sadock's comprehensive textbook of psychiatry* (9th ed., Vol. 2, pp. 2441–2456). Philadelphia: Lippincott Williams & Wilkins.

DiMartini, A. F., Sotelo, J. L., & Dew, M. A. (2011). Organ transplantation. In J. L. Levenson (Ed.), *The American Psychiatric Publishing textbook of psychosomatic medicine* (pp. 725–758). Washington, DC: The American Psychiatric Press.

Fine, R. N., Becker, Y., De Geest, S., Eisen, H., Ettenger, R., Evans, R., et al. (2009). Nonadherence consensus conference summary report. *American Journal of Transplantation, 9*(1), 35–41.

Kotlyar, D. S., Burke, A., Campbell, M. S., & Weinrieb, R. M. (2008). A critical review of candidacy for orthotopic liver transplantation in alcoholic liver disease. *American Journal of Gastroenterology, 103*, 734–743.

Olbrisch, M. E., Benedict, S. J., Haller, D. L., & Levenson, J. L. (2001). Psychosocial assessment of living organ donors: Clinical and ethical considerations. *Progress in transplantation, 11*(1), 40–49.

Olbrisch, M. E., Benedict, S. M., Ashe, K., & Levenson, J. L. (2002). Psychological assessment and care of organ transplant patients. *Journal of Consulting and Clinical Psychology, 70*(3), 771–783.

Organ Procurement and Transplantation Network and the Scientific Registry of Transplant Recipients (2009). *2009 Annual report, transplant data 1999–2008*. Rockville, MD: Department of Health and Human Services, Health Resources and Services Administration, Healthcare Systems Bureau, Division of Transplantation; Richmond, VA: United Network for Organ Sharing; Ann Arbor, MI: University Renal Research and Education Association. Accessed December 22, 2010 from http://www.ustransplant.org/annual_report/current/default.htm or http://optn.transplant.hrsa.gov/data/annualreport.asp

Rodrigue, J. R. (Ed.). (2001). *Biopsychosocial perspectives on transplantation*. New York: Kluwer Academic/Plenum.

Sabaté, E. (2003) *Adherence to long-term therapies: Evidence for Action*. Geneva: World Health Organization. Accessed April 6, 2012, from http://www.who.int/chp/knowledge/publications/adherence_introduction.pdf.

Simmons, R. G., Marine, S. K., & Simmons, R. L. (1987). *Gift of life: The effect of organ transplantation on individual, family, and societal dynamics*. New Brunswick, NJ: Transaction.

Skotzko, C. E., & Strouse, T. B. (2002). Solid organ transplantation. In M. G. Wise & J. R. Rundell (Eds.), *The American Psychiatric Publishing textbook of consultation-liaison psychiatry: Psychiatry in the medically ill* (9th ed., pp. 623–656). Washington, DC: American Psychiatric Publishing.

Trzepacz, P. T., & DiMartini, A. F. (Eds.). (2000). *The transplant patient: Biological, psychiatric and ethical issues in organ transplantation*. New York: Cambridge University Press.

Organizational Health

► Occupational Health

Organizational Health Promotion

► Worksite Health Promotion

Orleans, C. Tracy

C. Tracy Orleans
Robert Wood Johnson Foundation, Princeton, NJ, USA

Biographical Information

Carole Tracy Orleans (nee Schornagel) was born in Holland, Michigan, and obtained her B.A. degree in Psychology (summa cum laude) from Wellesley College in 1970. She completed her Ph.D. in Clinical Psychology at the University of Maryland, including a clinical internship in Medical Psychology at Duke University Medical Center in 1978.

Orleans is an internationally recognized leader in behavioral medicine, health and behavior, and tobacco control research. In the area of tobacco control, she has contributed to four *Surgeon General's Reports* and, with John Slade, coedited the first text on the management of nicotine addiction. She has led or co-led the development of several effective population-level national tobacco cessation programs which remain in wide use nationally (including *Free & Clear*, the first national proactive telephone quitline, the *Clear Horizons* quit smoking guide for older adults, the *Pathways to Freedom* guide for African American smokers). In addition, she played a leading role in the development of the NCQA's first HEDIS tobacco measure, which has been adopted as a standard metric for national health-care quality improvement, and in early efforts to reduce smokeless tobacco use in Major League Baseball. In 1995, she was named by *Tobacco Control* as one of the 100 most widely cited tobacco control researchers.

Prior to joining the Robert Wood Johnson Foundation's (RWJF) professional staff in 1995, Orleans served as vice president for Research and Development, Johnson & Johnson Applied Behavioral Technologies (1993–1995); associate and full member (professor) and director of Tobacco Control Research at the Fox Chase Cancer Center (1985–1993); and assistant professor of Medical Psychology/Psychiatry at Duke University Medical Center (1978–1984) with an adjunct appointment as research associate with the Cecil B. Sheps Center for Health Policy Research at the University of North Carolina.

Major Accomplishments

As the senior scientist for the RWJF since 1998, Orleans has led numerous national efforts to identify and spread evidence-based clinical, public health (policy/environmental), and health-care systems interventions for health promotion, disease prevention, and chronic disease management. As RWJF's only behavioral medicine specialist, Orleans developed and led the Foundation's first Health and Behavior team, catalyzed the development of its first chronic disease management team, and made significant contributions to its substance abuse prevention, tobacco control, and childhood obesity prevention

teams and initiatives. Her work has advanced RWJF's public policy and health-care systems grant-making in the areas of tobacco control, physical activity promotion, substance abuse prevention and treatment, chronic disease prevention and management, and childhood obesity prevention.

Orleans has developed and/or overseen a broad portfolio of RWJF research grants, initiatives, and competitive research programs, including: Active Living Research, Addressing Tobacco in Managed Care, Bridging the Gap (youth tobacco/substance use/and childhood obesity prevention), Healthy Eating Research, Improving Chronic Illness Care, Substance Abuse Policy Research, and Prescription for Health (multiple risk health behavior change in primary care). She also has directed several RWJF program evaluations (e.g., Health e.Technologies, Reducing Underage Drinking through Coalitions, SmokeLess States), led numerous projects deigned to translate evidence-based behavioral medicine interventions into policy and practice, and helped to organize and co-led four national research "collaboratives" designed to coordinate and strengthen the collective impact of the nation's major tobacco control and childhood obesity prevention research, practice, and policy initiatives, including those funded by the CDC, NIH, RWJF, USDA, HRSA, and Legacy Foundation.

Orleans has been PI or Co-PI on numerous NIH research grants and has authored or coauthored more than 225 publications in the areas of behavioral medicine, tobacco control, health promotion, disease prevention and management, and public health. She has served on many journal editorial boards throughout her career and is currently associate editor for policy for the *American Journal of Preventive Medicine*. In addition, she has been a member of numerous influential national scientific, health policy, and evidence review panels, including:

- The Community Preventive Services Task Force (2009–present)
- The *Healthy People* 2020 Prioritization Sub-Committee (2009–2010)
- Agency for Healthcare Research and Quality (AHRQ) Tobacco Control Clinical Practice Guideline Panel (2006–2008)
- Partnership for Prevention's Expert Panel on Worksite Wellness and Chronic Disease Management (2006–2008)
- The National Commission on Prevention Priorities (2003–present)
- The Cessation Subcommittee, Surgeon General's Interagency Committee on Smoking and Health (2002–2003)
- The Institute of Medicine Committee: Identifying Priority Areas for Healthcare Quality Improvement (2002–2003)
- The US Preventive Services Task Force (1998–2004)
- Several National Cancer Institute Research Advisory Committees including for the Working Well Study (1990–1994), the Tobacco Research Implementation Group (1998–2000), and the Behavioral Science Research Division (1996–1997; 2010).

Orleans was honored to receive several awards and recognitions for her work in behavioral medicine. She is a fellow of the Society of Behavioral Medicine, the American Psychological Association Health Psychology Division, and the Academy of Behavioral Medicine Research. A past-president of the Society of Behavioral Medicine (2000), she has received the Society's Distinguished Service Award (2000, 2006) and its Distinguished Scientist Award (2008). In recognition of career contributions to tobacco control research, Orleans received the 1992 Joseph Cullen Tobacco Memorial Award from the American Society of Preventive Oncology and the 2007 John Slade Award for tobacco control policy research leadership from the Society for Research on Nicotine and Tobacco. In 2006, Orleans was named RWJF's first Distinguished Fellow, and in 2010, she received the American Psychological Association Meritorious Research Service Commendation and the US Department of Health and Human Services Innovators Award, a group award.

Throughout her career, Orleans has been inspired and enriched by opportunities to address important health and health-care problems at the population level and to work with outstanding leaders and colleagues – including at Duke University Medical Center, the Fox Chase Cancer

Center, the Robert Wood Johnson Foundation, and in every professional organization she has been part of. Their example and friendship, and the vital support and inspiration of her family (her husband Jeffrey, their sons Jesse and Alexander, and her mother) are the bedrock of all her professional accomplishments.

Cross-References

- ▶ Health Promotion and Disease Prevention
- ▶ Public Health
- ▶ Tobacco Cessation
- ▶ Tobacco Use

References and Readings

Abrams, D. B., Orleans, C. T., Niaura, R. N., Goldstein, M. G., Prochaska, J. O., & Velicer, W. (1993). Treatment issues in smoking cessation: A stepped-care approach. *Tobacco Control, 2*(Suppl), 17–33.

Brennan, L., Castro, S., Brownson, R. C., Claus, J., & Orleans, C. T. (2011). Accelerating evidence reviews and broadening evidence standards to identify effective, promising and emerging policy and environmental strategies for prevention of childhood obesity. *Annual Review of Public Health, 32*, 199–225.

Glasgow, R., Orleans, C. T., Wagner, E., & Curry, S. (2001). The planned care model: A template for improving chronic illness and preventive care. *Millbank Quarterly, 79*, 579–612.

Houpt, J., Orleans, C. T., George, L. K. & Brodie, H. K. H. (1979). *The importance of mental health services to general health care*. Report prepared for the Institute of Medicine Cambridge: Ballinger Publishing. Precursor to IOM report Health and Behavior.

Levy, D. T., Mabry, P. L., Graham, A. L., Abrams, D., & Orleans, C. T. (2010). Exploring scenarios for dramatically reducing smoking prevalence: A simulation model of the three-part cessation process. *American Journal of Public Health, 100*, 1253–1259.

McKinnon, R. A., Orleans, C. T., Kumanyika, S. K., Haire-Joshu, D., et al. (2009). Considerations for an obesity policy research agenda. *American Journal of Preventive Medicine, 36*, 351–357.

Orleans, C. T. (2000). Promoting the maintenance of health behavior change: Recommendations for the next generation of research and practice. *Health Psychology, 19*, 76–83.

Orleans, C. T., (2001). Efforts to reduce tobacco use among women: Cessation overview. *In 1997 surgeon general's report: Tobacco use among women*. Washington, DC: U.S. Department of Health and Human Services.

Orleans, C. T. (2004). Addressing multiple behavioral health risks in primary care: Broadening the focus of health behavior change research and practice. *American Journal of Preventive Medicine, 27*(2S), 1–4.

Orleans, C. T. (2007). Increasing the demand for and use of effective smoking-cessation treatments: Reaping the full health benefits of tobacco-control science and policy gains – in our lifetime. *American Journal of Preventive Medicine, 33*(6 Suppl.), S340–S348.

Orleans, C. T., Boyd, N. R., Bingler, R., Heller, D., Fairclough, D., McClatchey, M., et al. (1998). A self-help intervention for African American smokers: Tailoring cancer information service counseling for a special population. *Preventive Medicine, 27*, S61–70.

Orleans, C. T., Gruman, J., Ulmer, C., Emont, S. L., & Hollendonner, J. K. (1999). Progress in population-based health promotion: Report card on six behaviors. *American Journal of Health Promotion, 14*, 75–83.

Orleans, C. T., Kristeller, J. L., & Gritz, E. R. (1993). Helping hospitalized smokers quit: New directions for treatment and research. *Journal of Consulting and Clinical Psychology, 61*(5), 778–789.

Orleans, C. T., Resch, N., Noll, E., Keintz, M. K., Rimer, B., Brown, T., et al. (1994). Use of transdermal nicotine in a state-wide prescription plan for the elderly: A first look at "real world" patch users. *Journal of the American Medical Association, 271*, 601–607.

Orleans, C. T., Rimer, B. K. & Salmon, M. A. (1990). Long-term psychological and behavioral consequences and correlates of smoking cessation. In *The health benefits of smoking cessation: A report of the surgeon general* (pp. 532–555, 561–578). Rockville, MD: US Department of Health and Human Services.

Orleans, C. T., Schoenbach, V., Wagner, E. H., Quade, D., Salmon, M. A., Pearson, D., et al. (1991). Self-help quit smoking interventions: Effects of self-help materials, social support instructions and telephone counseling. *Journal of Consulting and Clinical Psychology, 59*(3), 439–448.

Orleans, C. T., & Slade, J. (Eds.). (1993). *Nicotine addiction: Principles and management*. New York: Oxford University Press.

Orleans, C. T., Strecher, V. J., Schoenbach, V. J. & Salmon, M. A. (1988, May). Smoking cessation initiatives for Black Americans. In *The health consequences of smoking: Nicotine addiction*? *A report of the surgeon general*. Rockville, MD: US Department of Health and Human Services.

Schauffler, H., Barker, D. C., & Orleans, C. T. (2001). Medicaid coverage for tobacco dependence treatments. *Health Affairs, 20*, 298–304.

Whitlock, E. P., Orleans, C. T., Pender, N., & Allan, J. (2002). Behavioral counseling interventions for health promotion and disease prevention in health care settings. *American Journal of Preventive Medicine, 22*, 287–324.

Ornish Program and Dean Ornish

Manjunath Harlapur[1] and Daichi Shimbo[2]
[1]Center of Behavioral Cardiovascular Health, Division of General Medicine, Columbia University, New York, NY, USA
[2]Center for Behavioral Cardiovascular Health, Columbia University, New York, NY, USA

Synonyms

Dean Ornish

Definition

Dr. Dean Ornish is currently the president of the Preventive Medicine Research Institute in California and clinical professor of Medicine at University of California, San Francisco.

Description

Dr. Ornish is the founder and president of Preventive Medicine Research Institute in Sausalito, California, and on faculty at the University of California, San Francisco, as a clinical professor of medicine. Dr. Ornish received a medical degree at Baylor College of Medicine and underwent internal medicine training at Massachusetts General Hospital. His major contribution to medicine is his examination of the lifestyle modification/stress reduction approach to cardiovascular prevention and also cancer. He has published several seminal studies in this area. A few of these studies are described here.

In 1990, Ornish and colleagues published results (Ornish et al., 1990) from a landmark but small randomized trial (The Lifestyle Heart Trial) showing that intensive lifestyle changes for 1 year (10% fat vegetarian diet, exercise, stress management, and smoking cessation) was associated with regression in coronary artery disease on angiogram in cardiac patients (N = 28), whereas usual care was associated with coronary artery disease progression (n = 20). None of the patients were taking lipid-lowering medications.

In 1998, Ornish and colleagues published results of a longer follow-up period (i.e., 5 years) of the The Lifestyle Heart Trial (Ornish et al., 1998). The study results were similar to the 1-year results. Patients with coronary disease assigned intensive lifestyle changes had regression in coronary artery disease on angiogram at 5-year follow-up, whereas a group that received usual care had coronary artery disease progression. The group assigned to the intensive lifestyle changes also had a lower cardiac event rate during the 5-year follow-up.

In 2005, Ornish and colleagues (2005) demonstrated that in comparison to usual care group, comprehensive lifestyle changes for 1 year reduced the progression of early, low-grade prostate cancer in 93 men who had not chosen to undergo any conventional cancer treatment.

More recently, in 2008, Ornish and colleagues (2008) showed that comprehensive lifestyle changes for 3 months increased peripheral blood mononuclear cell telomerase activity (an enzyme that inhibits cellular aging) in 30 men with low-risk prostate cancer. This study was not randomized and did not have a control group.

Cross-References

- Lipid Metabolism
- Preventive Medicine Research Institute (Ornish)

References and Readings

Ornish, D., Brown, S. E., Scherwitz, L. W., Billings, J. H., Armstrong, W. T., Ports, T. A., et al. (1990). Can lifestyle changes reverse coronary atherosclerosis? The lifestyle heart trial. *The Lancet, 336*, 129–133.

Ornish, D., Lin, J., Daubenmier, J., Weidner, G., Epel, E., Kemp, C., et al. (2008). Increased telomerase activity and comprehensive lifestyle changes: A pilot study. *The Lancet Oncology, 9*(11), 1048–1057.

Ornish, D., Scherwitz, L., Billings, J., Brown, S. E., Gould, K. L., Merritt, T. A., et al. (1998). Intensive lifestyle changes for reversal of coronary heart disease five-year follow-up of the lifestyle heart trial. *Journal of the American Medical Association, 280*, 2001–2007.

Ornish, D., Weidner, G., Fair, W. R., Marlin, R., Pettengill, E. B., Raisin, C. J., et al. (2005). Intensive lifestyle changes may affect the progression of prostate cancer. *Journal of Urology, 174*(3), 1065–1070. http://www.pmri.org/.

Orth-Gomér, Kristina

Kristina Orth-Gomér
Department of Clinical Neuroscience, Karolinska Institute, Stockholm, Sweden

Biographical Information

Born in Stockholm, Kristina Orth-Gomér was educated with a focus on classic and modern languages at Whitlockska Samskolan, where she obtained baccalaureate in Latin, French, English, and German. After a short period of literature and civilization studies at the University of Lille, France, she began her studies of medicine at the Karolinska institutet, Stockholm. There, she obtained an M.D. in 1972 and a Ph.D. in 1979. She specialized in Internal Medicine in 1980 and later also in Social Medicine/Public Health Sciences.

With an interest in alternative therapies, she spent a semester at the Psychosomatische Kinderklinik, Eppendorf in Hamburg, where she also became involved in the 1968 students movements, which were particularly intense in France and Germany. She married in 1969 and welcomed her first child Johanna in 1973.

Trying to combine motherhood with medicine, Orth-Gomér was appointed an intern in internal medicine at the Seraphimer Hospital, Karolinska institutet. Before that, she served for a year as a research associate at the Cornell Medical School, Human Ecology Laboratory at the New York Hospital, learning from scratch the research methodology of cardiovascular epidemiology and 24-h ambulatory ECG monitoring. She also spent a happy year with her young family in Manhattan, NYC.

Back in Stockholm, as an intern in internal medicine, with regular work hours from 8 to 18 h and night/weekend duties in the emergency room in between, Orth-Gomér did not see much of her daughter. She just barely obtained the admission for her daughter into the day care center, and she learned to appreciate the difficulties of fulfilling several multiple roles in a satisfactory manner at the same time.

Major Accomplishments

Research on Social Support

Her next research position, starting in 1980, offered a solution to the time puzzle. As an associate professor at the National Institute for Psychosocial Medicine (IPM) at the Karolinska Campus in Stockholm and head of the Unit for Social Factors and Health, she worked the same number of hours, but she could choose when and where.

Under the guidance of Lenart Levi, the IPM offered great opportunities to pursue the line of alternative therapies, while Töres Theorell at the IPM focused his research on stress and strain at work – and Torbjörn Akerstedt at IPM concentrated on odd work hours, sleep problems, and circadian rhythms – there was less knowledge about stress and strain during leisure time

activities. To find research outside the work environment, the social network studies of Alameda County offered possibilities for scientific exchange with Lisa Berkman, Len Syme, and Jim House.

A question of considerable interest was the following: Are social networks as crucial for the maintenance of health in Sweden as in the USA? With the help of Statistics Sweden, a Swedish social network instrument, the social interaction index was constructed and applied in 17,400 men and women.

In a 6-year follow-up of the population, it was found that men and women who had more and better social contacts lived longer. Age was a strong confounder and controlling for age was crucial. With increasing age, mortality increased – and social contacts decreased dramatically. The remaining risk was smaller but statistically significant (Orth-Gomer & Johnson, 1987).

In subsequent years, the quality and function of social contacts and their role in health promotion and disease prevention became the focus. A measure of social support with two subscales was constructed: attachment (close emotional ties) and social integration (social contacts in the society). These measures were then applied in populations and patients and almost invariably found to matter for health maintenance.

Behavioral Medicine

Behavioral medicine had evolved as a useful concept in the USA, but was unknown in Europe. Initially, introducing behavioral medicine met with some criticism. To launch another term and another society within this field, beside "psychosomatics," "psychosocial medicine," or "stress research," was thought to be redundant. However, when the Swedish Society of Behavioral Medicine was inaugurated under the auspices of the Swedish Society of Medicine in Stockholm, in the presence of some 300 interested colleagues from medicine and behavioral sciences, it felt natural and appropriate. With the support by Arne Öhman, Karin Harms-Ringdahl, Christina Mats Fredriksson, Gösta Tibblin, and Stephen Weiss, Orth-Gomér served as president of the Swedish Society of Behavioral Medicine, 1986–1990.

A few years later, the International Society of Behavioral Medicine (ISBM) was formed and held its first conference in Uppsala in June 1990. It was a very successful conference, with an excellent faculty, which provided the right framework and starting point for the ISBM. It is not inappropriate to say that the success has been ongoing ever since. During the first years, ISBM expanded rather quickly, and there were many new societies formed. At the end of her term as president elect and president in 1990–1994, Kristina Orth-Gomér reported that from five founding societies (USA, Netherlands Germany, Czech Republic, Sweden), ISBM had grown into 15 societies. The second conference in Hamburg in 1992 and the third conference in Amsterdam in 1994 were equally successful and raised a lot of interest and promise. With the fourth conference in Washington, the international nature of behavioral medicine was settled! Every researcher with a name within the field had gathered to make international behavioral medicine known and respected.

Interdisciplinarity and Integration

Collaboration with the European Society of Cardiology

The first step toward practical interdisciplinarity was taken when introducing behavioral medicine to the European cardiology community. To present scientists and practitioners of both fields and make them understand each other and talk to each other was more difficult than one could imagine.

In February 1999, Orth-Gomér presented her behavioral medicine ideas to the 50 member countries of the European Cardiology Forum at the Heart House in Nice, France. From studies in Canada and the USA, the European cardiologists were aware of the importance of depression – and of the impact of social disadvantage. They wanted to know how to interpret and how to treat their patients. Her handout of two pages with common behavioral medicine knowledge was quickly gone.

Kristina Orth-Gomér was then elected to collaborate with the European Society of Cardiology as a scientist, but she asked to do this as

a representative of ISBM. This led to the formation of a multidisciplinary task force, which included not only behavioral medicine but also diabetes research, nutrition, physiology and rehabilitation, etc. The next step was to integrate this knowledge into the European Guidelines of Cardiovascular Disease Prevention in Clinical Practice (De Backer et al., 2003b).

Having attended the European Cardiovascular Epidemiology meetings for many years and presented her research, Kristina Orth-Gomér knew there was an interest of cardiologists in behavioral medicine methodology. She was included as an ordinary member in the task force for CVD prevention and asked to work on several cardiology committees within the field. This led to ISBM being positioned and recognized as the appropriate partner among all the other psychosocial organizations in Europe.

A behavioral working group was formed which included the most experienced colleagues in the field. This group met at the first European meeting of the American Psychosomatic Society (APS) in Barcelona in 2002. This psychosocial working group described the contribution of behavioral factors to CHD, and they were accepted as such by the medical/cardiological community. A special psychosocial chapter was written and included in the full text of the guidelines (De Backer et al., 2003a).

This was a time of enthusiastic belief in the cross-fertilization that would come out of interdisciplinarity and internationalization.

There was firm resistance within the third task force to recognizing behavioral/psychosomatic processes as in any way being related to heart disease. It was argued that the evidence was just not there. The solution was to avoid the concept of etiology and instead talk about depression, social isolation, and other psychosocial risk factors as "barriers" to health promotion.

"Negative emotions. . .may constitute barriers to preventive efforts, both in patients and in high risk people. . ." (De Backer et al., 2003a). The barrier concept was more easily accepted. Furthermore, only empirical studies that had integrated both medical/cardiological and behavioral/psychosomatic contribution to risk of CHD were cited.

The European guidelines became a turning point, which made the behavioral/psychosomatic map change in Europe. It paved the way for several collaborative interdisciplinary efforts in Europe, Germany, the Netherlands, Scandinavia, and Hungary to name a few countries that saw a profound change in interest and attitude to this field. With the guidelines, the theories and findings in behavioral medicine had been empirically founded and widely accepted (2003).

Women's Cardiovascular Disease

The Stockholm female coronary risk study is a comprehensive study of women's risk factors, mechanisms, and pathways to CHD. It had full financial support from the NIH.

In a community-based case control design, women in Stockholm who were under 65 years and hospitalized at any of the ten coronary care units for an acute coronary event during a 3-year period were included. These 292 women patients were compared to 292 same-aged women, randomly obtained from the public census, who were thus representative of normal Stockholm women of the same age. The first publication (Orth-Gomér et al., 1997) demonstrated that lipoprotein (a), a genetically determined lipid compound, was a risk factor in women, as had previously been found in studies of men. The accompanying editorial observed that "Orth-Gomer et al. made a wise decision in using a case control design. . ., for what is, in women of this age, a rare disease" (Fortman & Marcovina, 1997).

As part of the study protocol, Orth-Gomér developed a flowchart for integrating behavioral with medical risk factors, which has, since then, been used as a model for the first conference on Women, Stress, and Heart Disease held in Stockholm in 1993 (Orth-Gomér, Chesney, Wenger, 1998).

It appears that one important characteristic of the female risk factor profile is the social support provided – or not provided – by family and friends and other members of the social network. This factor was consistently associated with several of the relevant mediators and pathways: with depressive symptoms with disturbed sleep patterns (Leineweber et al., 2003), with low heart

rate variability and with disturbances of clotting and lipid profile (Wamala et al., 1999).

A further line of research was that of progression/regression of coronary artery disease (CAD) as assessed by quantitative coronary angiography. In a subsample of about a 100 women with CHD, two identical coronary angiograms were done with a 3-year interval, after initial hospitalization.

Richard Kirkeeide, from the Texas Medical Center, Houston, came once a year to the angiographic laboratory at the Karolinska in Stockholm to calibrate the lab in order to ensure comparability between the two examinations. The subsequent evaluation of cine films, i.e., blinded comparison of pre- and post-angiograms was performed in Houston. In brief, we found that lack of social support as a psychosocial predictor was consistently related to progression of coronary artery disease. The better support, the slower was the progression (Orth-Gomér et al.,1998). Indeed we found one small group of women who said they had both a good job and a happy marriage. This group showed a widening of the mean luminal diameter of the entire coronary tree – a regression of CAD over the 3 years. The magnitude of the change was about that seen as a result of medication with statins over a similar time period. In fact, this methodology had first been developed for use in statin trials (Wang et al. (2007)).

Conclusion

We now take for granted that women and men share leadership in professional societies. This is so only because of the efforts by an initially small cadre of female researchers who were pioneers in their male-dominated fields. Orth-Gomér has been a leader and pioneer in examining social factors in cardiovascular disease, particularly in women. She has led the Stockholm Female Coronary Risk Study and showed for the first time that marital stress in women increased risk of coronary events (e.g., cardiac death, recurrent MI) threefold, whereas work stress did not increase this risk in women (Orth-Gomer et al. (2000)). Her findings suggest that risk factors for adverse outcome in women are different from those of men. Kristina Orth-Gomér has now gone on to conduct intervention studies (e.g., SWITCHD) that specifically target stress reduction in women with coronary disease, and she has demonstrated a threefold reduction in mortality with this intervention (Orth Gomer et al., 2009). Her work has contributed to the sea change in cardiovascular medicine in the past decade in which it is now accepted that risk factors, clinical manifestations, and interventions in coronary disease differ between women and men. Her many grants, publications, and scientific presentations demonstrate the depth and breadth of her work for behavioral medicine throughout her career.

Cross-References

- ▶ Behavioral Medicine
- ▶ Cardiology
- ▶ Public Health
- ▶ Women's Health

References and Readings

De Backer, G., Ambrosioni, E., Borch-Johnsen, K., Brotons, C., Cifkova, R., Dallongeville, J., et al. (2003a). European guidelines for CVD prevention in clinical practice. Third joint task force of European and other societies. *European Journal of Cardiovascular Prevention and Rehabilitation, 1*, S1–S 78.

De Backer, G., Ambrosioni, E., Borch-Johnsen, K., Brotons, C., Cifkova, R., Dallongeville, J., et al. (2003b). European guidelines on cardiovascular disease prevention in clinical practice. Third joint task force of European and other societies on cardiovascular disease prevention in clinical practice. *European Heart Journal, 24*, 1601–1610.

Fortman, S., & Marcovina, S. M. (1997). Lipoprotein(a), a clinically elusive lipoprotein particle. *Circulation, 95*(2), 295–296.

Leineweber, C., Kecklund, G., Janszky, I., Akerstedt, T., & Orth-Gomer, K. (2003). Poor sleep increases the prospective risk for recurrent events in middle aged women with coronary disease. The Stockholm Female Coronary Risk study. *Journal of Psychosomatic Research, 54*, 121–127.

Orth Gomer, K., Schnekiderman, N., Wang, H., Walldin, C., Blom, M., & Jernberg, T. (2009). Stress reduction prolongs life in women with coronary disease. The Stockholm Women's intervention trial for coronary heart disease (SWITCHD). *Circulation, 2*, 25–32 Cardiovasc Qual Outcomes.

Orth-Gomér, K., Chesney, M., & Wenger, N. K. (1998). *Women, stress, and heart disease* (pp. 1–298). Mahwah: Lawrence Erlbaum.
Orth-Gomér, K., Horsten, M., Wamala, S. P., et al. (1998). Social relations and extent and severity of coronary artery disease. The Stockholm Female Coronary Risk Study. *European Heart Journal, 19*(11), 1648–1656.
Orth-Gomer, K., & Johnson, J. V. (1987). Social network interaction and mortality. A six year follow up of a random sample of the Swedish population. *Journal of Chronic Diseases, 40*(10), 949–957.
Orth-Gomér, K., Mittleman, M. A., Schenck-Gustafsson, K., Wamala, S. P., Eriksson, M., Belkic, K., et al. (1997). Lipoprotein (a) as a determinant of coronary heart disease in young women. *Circulation, 2*, 329–335.
Orth-Gomer, K., Wamala, S., Horsten, M., Schenck-Gustafsson, K., Schneiderman, N., & Mittlemasn, M. (2000). Marital stress worsens prognosis in women with coronary heart disease. The Stockholm Female Coronary Risk study. *Journal of the American Medical Association, 284*(23), 3008–3014.
Wamala, S. P., Mittleman, M. A., Schenck-Gustafsson, K., & Orth-Gomer, K. (1999). Potential explanations for the educational gradient in coronary heart disease: A population based case-control study of Swedish women. *American Journal of Public Health, 89*, 67–72.
Wang, H. X., Leineweber, C., Kirkeeide, R., Svane, B., Schenck-gustafsson, K., Theorell, T., & Orth-Gomér, K. J. (2007). Psychosocial stress and atherosclerosis. Family and work stress accelerate progression of coronary disease in women. The Stockholm Female Coronary Angiography study. *Journal of Internal Medicine, 261*(3), 245–254.

OS

► Oxidative Stress

Osteopenia/Osteoporosis

Eun-Shim Nahm
School of Nursing, University of Maryland, Baltimore, MD, USA

Definition

Osteopenia. Osteopenia is a condition where bone mineral density is lower than normal. It is considered to occur before osteoporosis. However, not every person diagnosed with osteopenia will develop osteoporosis. Osteopenia is not a disease but is a marker for the risk of fractures (Torpy, Lynm, & Glass, 2011).

Osteoporosis. Osteoporosis ("porous bone") is a systemic skeletal disease characterized by decreased bone mass, a loss of microarchitectural bone tissue, and a failure of the bone modulus (Cranney, Jamal, Tsang, Josse, & Leslie, 2007; Gardiner, El Miedany, & Toth, 2007; Osteoporosis Education, 2011). The individuals affected by this illness are susceptible to fracture.

In the United States, an estimated 44 million adults are living with either osteoporosis (ten million) or low bone mass (Osteoporosis Education, 2011; U.S. Dept. of Health & Human Services PHS, 2004). Several risk factors can influence the development of osteoporosis, including age (older age), race (White), family history (heredity/genetics), weight (low body weight/being small and thin), estrogen level (low), rheumatoid arthritis, and personal fracture history (National Osteoporosis Foundation, 2007; U.S. Dept. of Health & Human Services PHS, 2004). Postmenopausal women are at a high risk for osteoporosis because they lose bone mass faster than men as their estrogen level decreases after menopause (Camacho & Miller, 2007; MacLean et al., 2008; The North American Menopause Society, 2010). Although men are less affected than women, osteoporosis is also common among older men as approximately two million male older adults have osteoporosis (Qaseem et al., 2008). Osteoporosis in men, however, remains underdiagnosed and underreported (Qaseem et al., 2008; National Osteoporosis Foundation, 2011a; Rochira et al., 2006; Szulc, 2006).

Bone mineral density (BMD) refers to the amount of matter per square centimeter of bone. The BMD test is the only way to diagnose osteoporosis. This test can predict the chances of fracturing bones. The National Osteoporosis Foundation recommends a bone density test of the hip and spine by a central DXA (dual energy X-ray absorptiometry) machine to diagnose osteoporosis (National Osteoporosis Foundation,

Osteopenia/Osteoporosis, Table 1 Interpretation of T-score (National Osteoporosis Foundation, n.d.)

Category	T-score range
Normal bone density	−1 to +1
Low bone density (osteopenia)	Between −1 and −2.5
Osteoporosis	−2.5 and below

2011b). BMD test results are compared to the ideal BMD of a healthy 30-year-old and reported using T-scores. A score of 0 means the BMD is equal to the norm for a healthy young adult. Differences between the norm and the BMD test results are measured in standard deviations (SDs). The lower the BMD score, the higher the risk for fracture (Table 1).

References and Readings

Camacho, P. M., & Miller, P. D. (2007). *Osteoporosis: A guide for clinicians*. Philadelphia: Lippincott.

Cranney, A., Jamal, S. A., Tsang, J. F., Josse, R. G., & Leslie, W. D. (2007). Low bone mineral density and fracture burden in postmenopausal women. *CMAJ Canadian Medical Association Journal, 177*(6), 575–580.

Gardiner, A., El Miedany, Y., & Toth, M. (2007). Osteoporosis: Not only in women, but in men too. *British Journal of Nursing, 16*(12), 731–735.

MacLean, C., Newberry, S., Maglione, M., et al. (2008). Systematic review: Comparative effectiveness of treatments to prevent fractures in men and women with low bone density or osteoporosis. *Annals of Internal Medicine, 148*(3), 197–213.

National Osteoporosis Foundation (2011). Understanding Osteoporosis. Accessed March 28, 2012, from http://www.nof.org/aboutosteoporosis/prevention/healthyliving

National Osteoporosis Foundation. (2011a). *Prevalence report*. Accessed October 15, 2011, from http://www.nof.org/advocacy/resources/prevalencereport

National Osteoporosis Foundation. (2011b). *Diagnosing osteoporosis*. Accessed May 20, 2011, from http://www.nof.org/aboutosteoporosis/detectingosteoporosis/diagnosing

National Osteoporosis Foundation. (n.d.). *Having a bone density test*. Accessed August 15, 2011, from http://www.nof.org/aboutosteoporosis/detectingosteoporosis/bmdtest

Osteoporosis Education. (2011). *About osteoporosis: Fast facts 2011*. Accessed May 20, 2011, from http://www.nof.org/node/40

Qaseem, A., Snow, V., Shekelle, P., et al. (2008). Screening for osteoporosis in men: A clinical practice guideline from the American College of Physicians. *Annals of Internal Medicine, 148*(9), 680–684.

Rochira, V., Balestrieri, A., Madeo, B., Zirilli, L., Granata, A. R., & Carani, C. (2006). Osteoporosis and male age-related hypogonadism: Role of sex steroids on bone (patho)physiology. *European Journal of Endocrinology, 154*(2), 175–185.

Szulc, P. (2006). Bone density, geometry, and fracture in elderly men. *Current Osteoporosis Reports, 4*(2), 57–63.

The North American Menopause Society. (2010). Management of osteoporosis in postmenopausal women: 2010 position statement of The North American Menopause Society. *Menopause, 17*(1), 25–54. Quiz 55-26.

Torpy, J. M., Lynm, C., & Glass, R. M. (2011). Osteopenia and preventing fractures. *Journal of the American Medical Association, 296*(21), 2644.

U.S. Dept. of Health and Human Services PHS, Office of the Surgeon General. (2004). *Bone health and osteoporosis: A report of the surgeon general*. Accessed August 22, 2011, from http://www.surgeongeneral.gov/library/bonehealth/content.html

Outcome for the Single Case: Random Control Index, Single Subject Experimental Design, and Goal Attainment Scale

Kenneth J. Ottenbacher[1] and Margaret E. Ottenbacher[2]

[1]Division of Rehabilitation Sciences, University of Texas Medical Branch, Galveston, TX, USA

[2]Institute for Translational Sciences, University of Texas Medical Branch, Galveston, TX, USA

Synonyms

Single subject; Single-case experimental, or N of 1 clinical trials; Small-N

Definition

Traditional research and evaluation methods, including randomized clinical trials, are powerful techniques for determining the efficacy of health and behavioral interventions. Randomized clinical trials, however, have practical and ethical

limitations when applied to many research questions important to the field of behavioral medicine (Concato, Shah, & Horwitz, 2000). Alternative methods are needed to fully examine the effectiveness of treatment techniques for individual patients and to document clinical accountability. This entry examines outcomes for the individual or single case.

Description

In this entry, three approaches are discussed that focus on evaluating outcome at the level of the *individual* person or patient: the *Reliability Change Index*, *Single-subject designs*, and *Goal Attainment Scaling*. These approaches are consistent with the client-/patient-centered focus in behavioral medicine. The assumption is made in presenting these procedures that systematic documentation of patient/client performance is the first step in the process of developing an evidence-based science for clinical practice. It is important to note that the procedures described are designed primarily to document clinical change and not to demonstrate causality.

Reliability Change Index (RCI)

The RCI, originally proposed by Jacobson, Follette, and Revenstorf (1984), is a general purpose measure that is useful with outcomes that are expected to remain relatively stable over time. Jacobson et al. (1984) argued that for a change in performance to be clinically significant it must be "statistically" reliable. That is, there must be a way to determine that the change in performance is not due to chance variation or measurement error. To make this determination, Jacobson et al. proposed the use of the *Reliability Change Index* (RCI). To compute the RCI, the following information is required: (a) pre-test score for an outcome measure, (b) the post-test score following treatment, and (c) the standard error of measurement (S_E) for the test. The S_E may be computed from the following formula: $S_E = S_1\ [sq\ root(1 - r)]$ where S_1 equals the standard deviation for the test and r is the reliability coefficient. The RCI is calculated as: $\text{RCI} = (X_2 - \text{X}_1)//S_E$ where X_2 is the post-test score, X_1 is the pre-test score, and S_E is the standard error of measurement as defined above (Jacobson et al.).

In a subsequent article, Jacobson and Truax (1991) revised the *Reliability Change Index* and replaced the S_E with the standard error of difference $S_{diff.}$ The S_E in the original formula is an index of the dispersion of an obtained score about a "true" score. The RCI, however, makes use of two *obtained* scores. The use of the S_E in the RCI formula assumes that the pre-test score is the "true" score and the Post-test score an obtained score (Jacobson & Truax, 1991). This does not reflect the actual situation. The revised formula is $\text{RCI}' = (X_2 - \text{X}_1)//S_{diff}$. The S_{diff} can be computed as: $S_{diff} = S_1[\sqrt{2(S_E)^2}]$. The S_{diff} represents the amount of difference that would be expected between two scores obtained on the same test by the same individual as a function of measurement error alone. This assumption is consistent with obtaining pre- and post-test scores from a person in a clinical setting.

As an example, a student receiving education intervention for low academic performance might have an initial (pre-test) IQ score of 75 on the Stanford Binet test. Following 9 months of specialized instruction, the child's IQ may increase to 85. The S_E for the IQ test is 5 points. Using the revised formula provided above: $\text{RCI}' = (X_2 - \text{X}_1)/S_{diff}$ or $\text{RCI}' = (85 - 75)//7.07 = 1.41$. This value is not statistically significant and indicates the change from 75 to 85 is within the range of measurement error.

Practitioners using the *Reliability Change Index* to document a clinically significant change in an individual client/patient should use the revised method (RCI′) rather than the original formula (Jacobson & Truax, 1991).

Single-Subject Design

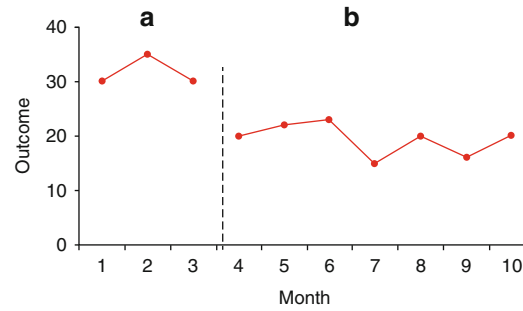
Bloom, Fischer, and Orme (2009) argue that single-system designs, also referred to as single subject, small-N, single-case experimental, or N of 1 clinical trials, represent the preferred method of evaluating clinical change in many behavioral medicine environments. The basic steps followed in using single system designs to assess individual client/patient outcomes are presented below.

Step 1. Specifying the Problem. It is important that the clinical problem be clearly stated and behaviors associated with the problem that can be observed and measured be identified. Traditionally, standardized norm-referenced tests are used to measure research outcomes when comparing persons in treatment versus control groups. Standardized norm-referenced assessments may be of limited value in single system designs where the same person(s) is assessed repeatedly. The ability to measure the person's repeated behavior is essential to implementing the single-system approach. Each outcome can be defined in terms of how often it occurs (frequency) or how long it occurs (duration) and also in terms of qualitatively desirable components (Bloom et al., 2009).

The behavior to be recorded must be clearly defined, and the rater must be familiar with how, when, where, and how long the behavior should be observed and recorded. Procedures have been developed to train raters and observers to determine the consistency and accuracy of their recording (Ottenbacher, 1986).

Step 2. Selecting a Design. The logic of single-system designs rests on a comparison between phases when the intervention is present and when it is not, or when two or more treatments are compared. This comparison is generally made over time within one person (or a small group of participants). The sophistication of single-system designs has increased dramatically over the past two decades (Bloom et al., 2009). The discussion of designs presented here is meant to be illustrative rather than exhaustive. Readers should consult the references for more detailed information.

AB Designs. The AB design is the basic form of single-system design. The A represents the baseline or no treatment condition (phase A). Subsequent interventions are indicated by different letters of the alphabet. If an intervention baseline is repeated, then the same letter is used to represent the repeated phase. For example, Fig. 1 displays the effect of an exercise program on the weight loss of a cardiac patient. Phase A represents the baseline period where the person's weight was recorded on a monthly basis. A program of graded exercise and diet control was initiated at the end of month three and represents start of phase B. The vertical axis of the graph indicates the patient's weight loss in pounds.



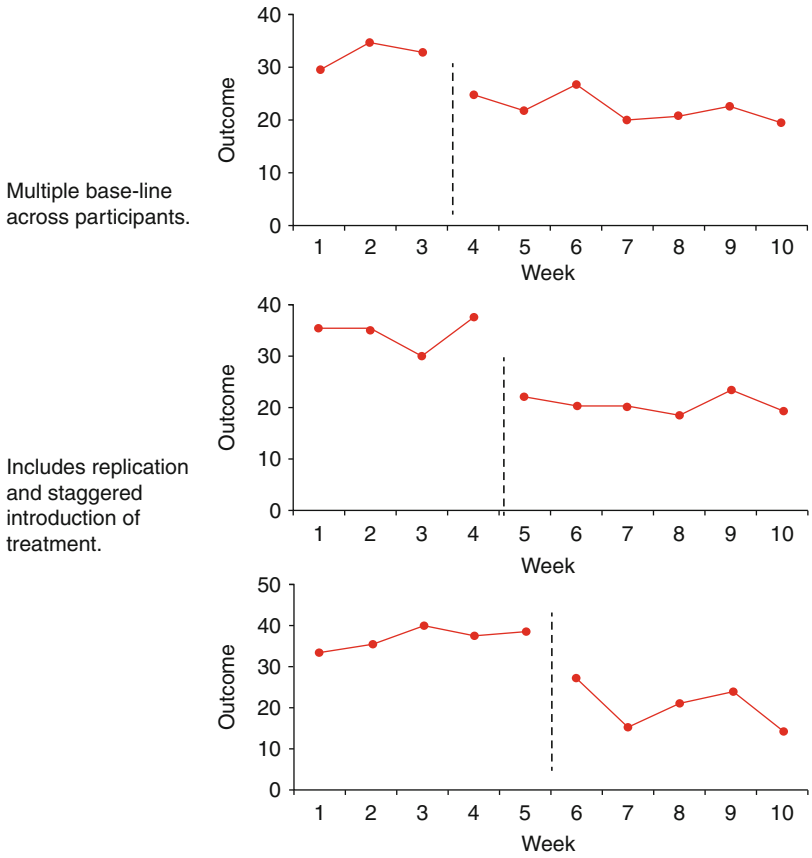
Outcome for the Single Case: Random Control Index, Single Subject Experimental Design, and Goal Attainment Scale, Fig. 1 Example of *AB* design with baseline (*A*) and treatment (*B*) phases

Other design variations are easily developed from this basic design pattern. Bloom et al. (2009) advocate the AB design as the cornerstone for conducting single-system evaluation and research. They contend that the fundamental step in becoming an accountable professional is to start counting with the AB design.

Variations of the AB Design. An extension of the AB design, in which the treatment is withdrawn in the third phase, is the ABA design. The withdrawal of the intervention provides greater confidence in determining the effect of the treatment. With each successive pattern change corresponding to the introduction or withdrawal of the treatment, it becomes increasingly unlikely that the change can be attributed to a coincidence. One disadvantage of the ABA design is that the patient is left in the absence of intervention. Another treatment phase (phase B) may be added to reintroduce the intervention effects resulting in an ABAB design.

Multiple Baseline Designs. In some cases, the treatment or intervention cannot be withdrawn, or withdrawing the treatment may not result in a return to pre-intervention performance levels. In a multiple-baseline design, baseline and intervention data are collected on several individuals for a similar behavior or setting. The most common version of this design is the multiple

Outcome for the Single Case: Random Control Index, Single Subject Experimental Design, and Goal Attainment Scale, Fig. 2 Example of multiple baseline design across three individuals



baseline across participants where data are collected across several different individuals (Bloom et al., 2009). In a multiple baseline design, the intervention phase is introduced in a staggered manner across individuals, behaviors, or settings. Figure 2 illustrates a multiple-baseline across individuals. The intervention is introduced to each participant in a sequential, staggered manner, and the outcome measure recorded repeatedly. The consistently changing performance of each individual as the intervention is introduced strengthens confidence in the relation between the intervention and outcome. The multiple baseline design across individuals is a particularly useful single-system design because it does not require the withdrawal of treatment and has the additional benefit of including replication of the finding across participants.

Graphic analysis and visual inspection are the traditional analytic tools used to present and interpret the results of single-system research (Bloom et al., 2009). Kazdin (1982) has observed that as single-system designs have become popular in applied research areas, there has been an increased emphasis on the need for statistical analysis of the data. The use of statistical procedures with single-system designs is controversial (Kazdin, 1982). Several authorities feel it is inappropriate to use statistical techniques with single-system designs and contend that the application of statistical procedures confuses the issue of clinical and statistical significance. However, Edington (1996) argues that statistical and visual methods should be partners in the analytic endeavor to provide a clearer interpretation of single-system outcome research.

Goal Attainment Scaling (GAS)

The basic idea of GAS is not new. The practice of setting measurable goals for intervention is well

established. Goals should be client/patient-centered and established in relation to the person's home, work, and community environments (Rockwood, Stolee, & Fox, 1993).

The process of establishing goals in GAS is accomplished in series of steps (Kiresuk, Smith, & Cardillo, 1994).

Step 1 is to identify an overall objective in collaboration with the person and his/her family.

Step 2 is to identify specific problem areas that should be addressed. This involves prioritizing areas and then identifying observable, reportable components.

Step 3 is to define specific behaviors or events that will indicate improvement in each of the areas selected in Step 2. The purpose is to provide the operational detail required to make the scale a useful instrument to evaluate performance.

Step 4, the clinician must determine the methodology that will be used to collect the desired information. A plan is developed that will determine how the information will be collected, who will collect it, and in what setting the data will be gathered.

Step 5 involves selecting the expected level of performance. This step relies on professional judgment and realistic appraisal of the client – it simultaneously is one of the strengths and liabilities of GAS. If the expected levels are at variance with actual performance, then the GAS score will reflect this inaccuracy. Step 5 is based on the assumption that experienced practitioners will be able to "predict" treatment outcome with input from the client, family members, and other health care providers. For example, if the clinician and others judge that it is realistic that the client loses 5 lbs in a month as part of a cardiac rehabilitation program, then this is a satisfactory operational criterion for the goal of weight reduction. This operational criterion relies heavily on the practitioner (and others) knowledge of the client, the intervention (e.g., cardiac rehabilitation program), and the environment.

Step 6 involves identifying the most favorable outcome, the least favorable outcome, and intermediate levels of client performance. Kiresuk et al. (1994) suggest that the practitioner develop five levels of performance that range from the most favorable to the least favorable. The most favorable outcome should reflect what the client could accomplish if everything in the program goes smoothly. In the weight loss example, the most favorable outcome may be that the client loses 10 lb in 1 month. The least favorable outcome might be that the client gains 5 lb during the intervention. The expected outcome is to lose 5 lb. Each of the five levels is operationally defined and assigned a numeric value with 0 indicating the expected level of performance and −2 and +2 indicating the least and most favorable outcomes, respectively.

Step 7 involves checking to ascertain whether there are overlapping levels, gaps between levels, or more than one indicator in a problem area. The scale should also be checked to make sure that the definitions of behaviors are clear and that the instructions on how to collect data are not ambiguous. The final step (*Step 8*) is to ascertain the current status of the individual and determine when the client will be evaluated again to document if she or he has progressed. The selection of the time period between evaluations is related to the type of intervention provided, the expected level of performance, and external criteria imposed by third party payers, accrediting agencies and others.

The most commonly used system of scoring relies on the five-point scale of performance (see Step 6) with scores ranging from −2 to +2 for each scale. In this scheme, a rating of −2 is associated with the least favorable outcome; −1 less than expected; 0 expected level of performance; +1 greater than expected outcome; and +2 most favorable outcome. These ratings are used for each of the levels across all goals.

In scoring the GAS, *relative weights* are assigned to each of the goals identified for the client. There is no standard procedure for determining how each goal is weighted. The weighting is (ideally) achieved by consensus among the patient, clinician, family members, and other persons concerned with the patient's performance. The weighing reflects a prioritizing or ranking of

the goals. If four primary goals are prioritized, for example, then the most important goal is given a ranking (weight) of +4 and the least important goal a weight of +1. The weights (rankings) must be determined in the goal planning stage and not in the final assessment phase.

Evaluation Formula. After the intervention has been administered, the weights for the goals and the rating of each level of performance are used to compute a GAS score (Kiresuk & Sherman, 1968). This score represents a numeric index of the client's improvement (or lack of improvement). The formula used to compute the goal attainment score is as follows:

$$T = 50 + \frac{(10\Sigma W_i X_i)}{(1 - r)\Sigma W_i{}^2 + r(\Sigma W_i)^2}$$

X_i represents the outcome score for each behavior (a value from −2 to +2).

W_i represents the weighting for a particular goal.

r-value in the formula reflects the estimated average intercorrelation between outcome scores. Kiresuk et al. (1994) suggest that an r-value of 0.30 can safely be assumed and used as a constant in the formula.

T-value is a standardized score with a mean of 50 and a standard deviation of 10.

A T-score of 50 corresponds to the 0 point on the original profile, that is, the expected level of performance ranging from −2 to +2. A T-score of greater than 50 represents performance above the expected level and a T-score of less than 50 reflects performance below the expected level.

The T-score is a better reflection of client performance than the simple raw score because it combines the outcome scores for all the goals to provide an overall measure of client improvement (or lack of improvement) (Kiresuk & Sherman, 1968). Another advantage of the T-score is that it allows the weighting given to the individual goals to be incorporated into the final outcome.

An advantage of GAS is that it is not bound to any theoretical orientation or type of treatment or outcome measure. Another strength is that goals can be individualized and designed to represent realistic expectations concerning client performance. The GAS strategy actively encourages cooperative person-centered goal setting. Input from clients, family members, and other health service providers is important in establishing and prioritizing (weighting) the goals and in determining realistic levels of expected performance (Kiresuk & Sherman, 1968).

As Rockwood et al. (1993) accurately note "Goal Attainment Scaling can, quite properly, be viewed as an accountability system" (p. 450) it is not a research method. The procedures associated with GAS can, however, be incorporated into many standard research designs (Khan, Pallant, & Turner-Stokes, 2008). If GAS is used in a research context then attention must be devoted to ensuring the internal validity of the investigation by blindly recording and establishing the reliability of the outcome measures. Khan and associates (Khan et al., 2008) provide valuable suggestions and guidelines to practitioners interested in using GAS in a research context.

Implications for Practice and Research

The *Reliability Change Index*, *Single-subject designs* and *Goal Attainment Scaling* can assist practitioners in systematically evaluating change. It may not be realistic to expect behavioral medicine practitioners to complete complex and time-consuming research studies based on experimental procedures or randomized clinical trials. Practitioners do, however, have the opportunity and obligation to assess client/patient performance, document change, and report the results to consumers and colleagues. Increasingly, this obligation includes providing an opportunity for the patient and family to have a role in the assessment, intervention, and evaluation process. *Goal Attainment Scaling* and other patient-centered methods focused on individual outcomes can help improve the evidence-based services provided to persons with disabilities and chronic disease.

Cross-References

▶ Randomized Clinical Trial

References and Readings

Bloom, M., Fischer, J., & Orme, J. G. (2009). *Evaluating practice: Guidelines for the accountable professional* (6th ed.). Boston: Allyn and Bacon.

Concato, J., Shah, N., & Horwitz, R. I. (2000). Randomized, controlled trials, observational studies, and the hierarchy of research designs. *The New England Journal of Medicine, 342*, 1887–1892.

Edington, E. S. (1996). Randomized single-subject experimental designs. *Behaviour Research and Therapy, 34*, 567–574.

Jacobson, N. S., Follette, W., & Revenstorf, D. (1984). Psychotherapy outcome research: Methods for reporting variability and evaluating clinical significance. *Behavior Therapy, 15*, 336–352.

Jacobson, N., & Truax, P. (1991). Clinical significance: A statistical approach to defining meaningful change in psychotherapy research. *Journal of Consulting and Clinical Psychology, 59*, 12–19.

Kazdin, A. E. (1982). *Single-case research designs: Methods for clinical and applied settings*. New York: Oxford University Press.

Khan, F., Pallant, J. F., & Turner-Stokes, L. (2008). Use of goal attainment scaling in inpatient rehabilitation for persons with multiple sclerosis. *Archives of Physical Medicine and Rehabilitation, 89*, 652–659.

Kiresuk, T., & Sherman, R. (1968). Goal attainment scaling: A general method of evaluating general mental health programs. *Community Mental Health Journal, 4*, 443–453.

Kiresuk, T. J., Smith, A., & Cardillo, J. E. (1994). *Goal attainment scaling: Applications, theory and measurement*. Hillsdale, NY: Lawrence Erlbaum.

Ottenbacher, K. J. (1986). *Evaluating clinical change: Strategies for occupational and physical therapists*. Baltimore, MD: Williams & Wilkins.

Rockwood, K., Stolee, P., & Fox, R. A. (1993). Use of goal attainment scaling in measuring clinically important change in the frail elderly. *Journal of Clinical Epidemiology, 46*, 1113–1118.

Outcomes

► Health Outcomes Research

Outpatient Treatment

► Substance Abuse: Treatment

Outreach Educators

► Promotoras

Ovarian Carcinoma

► Cancer, Ovarian

Ovarian Neoplasm

► Cancer, Ovarian

Overweight

Elizabeth R. Pulgaron
Department of Pediatrics, University of Miami, Miami, FL, USA

Synonyms

Excess weight

Definition

Being overweight is defined as being heavier than is healthy given a person's height, gender, and age. This includes having more body fat than is desired. Being classified as an overweight adult consists of having a body mass index (BMI) between 25 and 29.9. BMI is calculated by taking a person's weight in kilograms and dividing it by the square of the person's height in meters. For adults, a BMI of 25 is considered the 85th percentile. A similar calculation is computed to determine a child's BMI; however, gender and age are also added into the equation to account for differences in expected body fat at different

developmental levels according to gender. Although BMI is a generally accepted measure for obesity, it is not diagnostic since it is not a direct measure of body fat. Other measures of body fat distribution include skinfold thickness tests, waist circumference, calculation of waist-to-hip circumference ratios, ultrasound, computed tomography, and magnetic resonance imaging (MRI; CDC, 2009b).

Data from the United States National Health and Nutrition Examination Survey (NHANES) indicate that between 2005 and 2006, nearly 33% of adults 20 years and older were classified as overweight according to their BMI. From that same survey, 34.3% and 5.9% of adults were classified as obese and extremely obese, respectively. Thus, 72.9% of adults in the USA were overweight or obese. Data from 2007 to 2008 indicated that 32% of children aged 2–19 years old met criteria for being overweight according to BMI (Ogden et al., 2010). These rates are much higher than those reported 10 years ago, which were significantly higher than the rates reported 10 years before that. High rates of obesity have been reported in other countries outside of the USA as well.

Being overweight is a result of chronic caloric imbalance, with more calories being consumed than expended each day. Genetics, environment, metabolism, behavior, culture, and SES all play a role in being overweight. The consequences of being overweight may be severe, including but not limited to higher risk for cardiovascular diseases, type 2 diabetes, cancer, hypertension, dyslipidemia, stroke, and sleep apnea (CDC, 2009a). In addition to health consequences, there are also psychosocial and economic consequences of being overweight. These include morbidity and mortality, medical prevention and treatment expenses, and physical and psychological distress. For example, one study conducted by Gortmaker, Must, Perrin, Sobol, and Dietz (1993) followed obese adolescents for 7 years. Results indicated that young obese women completed less years of school, had lower household incomes, experienced higher rates of poverty, and were less likely to be married than women who had not been obese.

Cross-References

- ▶ Body Mass Index
- ▶ Obesity

References and Readings

CDC. (2009a). *Overweight and obesity*. Retrieved January 31, 2011 from http://www.cdc.gov/obesity/causes/health.html

CDC. (2009b). *Overweight and obesity*. Retrieved January 31, 2011 from http://www.cdc.gov/obesity/defining.html

Gortmaker, S. L., Must, A., Perrin, J. M., Sobol, A. M., & Dietz, W. H. (1993). Social and economic consequences overweight in adolescence and young adulthood. *The New England Journal of Medicine, 329*, 1008–1012.

Ogden, C. L., Carroll, M. D., Curtin, L. R., Lamb, M. M., & Flegal, K. M. (2010). Prevalence of high body mass index in US children and adolescents, 2007–2008. *Journal of the American Medical Association, 303*(3), 242–249.

Overweight Children

- ▶ Obesity in Children

Oxidative Stress

Sarah Aldred
School of Sport and Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

Synonyms

OS

Definition

Oxidative stress is an environment where the balance of prooxidant species to antioxidant species is altered in favor of the former. Prooxidant species include free radicals (e.g., the superoxide ion; O_2^-) and nonradical species (e.g., hydrogen peroxide; H_2O_2), which together are classified as reactive oxygen and nitrogen species (RONS). RONS are produced as a consequence of normal cellular oxidation processes. Sources include the mitochondrial electron transport chain, peroxisomes, endothelial or hepatic xanthine oxidase, and leukocytes. RONS have a variety of roles in normal homeostasis, including respiration and cellular signaling: RONS are reported to act as transient signaling molecules in the Ras GDP/GTP cycle and MAP kinase cascades, through modification of protein-bound redox-sensitive thiol groups.

Antioxidants serve to delay or prevent the oxidation of substrates or cellular constituents. They may be endogenous, for example, the enzymes superoxide dismutase, glutathione peroxidase, and catalase, or exogenous and taken in via the diet such as vitamin A, C, and E.

RONS can increase to high levels in some disease processes or where there is antioxidant deficiency and may react with cellular constituents to cause damage, disruption of function, or degradation resulting in an oxidative stress.

Oxidative stress has been implicated in a number of diseases including Alzheimer's disease, vascular dementia, cardiovascular disease, rheumatoid arthritis, and diabetes.

Indices of Oxidative Stress

Proteins are highly susceptible to free radical insults, and such events can lead to irreversible oxidative modification. Free radicals can oxidize a protein's backbone and the side chains of particular amino acids (e.g., lysine, arginine, proline, and threonine), as well as induce protein fragmentation. In all of these instances, carbonyl groups (C═O) may be introduced into the protein's structure. In addition, the reaction of proteins with aldehyde compounds produced by lipid peroxidation (e.g., 4-hydroxynonenal (4-HNE) and malondialdehyde (MDA)) can lead to carbonyl formation.

Peroxynitrite (ONOO-) is a highly potent reactive nitrogen species (RNS) formed during the reaction between nitric oxide ($NO^{\bullet}$) and $O2^{\bullet -}$. In addition to oxidizing proteins, $ONOO^-$ has the ability to nitrate proteins: a nitro group ($-NO_2$) replaces a hydrogen atom at the 3′ position of a tyrosine residue, forming a 3-nitrotyrosine adduct. This posttranslational modification can impact upon protein function.

Polyunsaturated fatty acids (PUFA) are components of cell membranes which are highly susceptible to oxidative damage by free radicals: such damage is referred to as free radical-mediated lipid peroxidation (LPO). The initial step of LPO involves the free radical-mediated abstraction of hydrogen atoms from PUFA to form lipid radicals which react with molecular oxygen to form the highly reactive peroxyl radical. This extremely unstable species can further oxidize lipids to produce new lipid radicals and thereby propagate a chain reaction. LPO can be terminated if lipid radicals (e.g., lipid peroxides) react with themselves to form stable lipid peroxide products, or if lipid-soluble antioxidants (e.g., vitamin E) are available to reduce peroxyl radicals to lipid hydroperoxides.

Damage to DNA in peripheral blood lymphocytes and tissues of the human body is a highly sensitive and specific marker of oxidative stress. The use of DNA repair endonucleases (e.g., formamidopyrimidine glycosylase; FPG) allows the direct quantification of damage caused specifically by oxidative stress. Free radicals, and in particular the hydroxyl radical ($OH^{-\bullet}$), cause sugar and base modifications, strand breaks, and DNA-protein cross-links.

Cross-References

► Antioxidant

References and Readings

Halliwell, B., & Gutteridge, J. (2007). *Free radicals in biology and medicine* (4th ed.). New York: Oxford University Press.

Oxytocin

Laura D. Kubzansky
Department of Society, Human Development, and Health, Harvard School of Public Health, Boston, MA, USA

Synonyms

Pitocin; Syntocinon (synthetic forms)

Definition

Oxytocin is a peptide that is produced in the supraoptic (SON) and paraventricular nuclei (PVN) of the hypothalamus and released into circulation from the magnocellular neurons that extend to the posterior pituitary (peripheral system). It is released into the systemic bloodstream in pulsatile form by sensory and other stimuli. Oxytocin is also produced and released from parvocellular neurons in the PVN that project to numerous areas within the brain. It is a hormone but also functions as a neurotransmitter. Oxytocin is found in both males and females. Circulating levels of oxytocin and the number of oxytocinergic neurons in the hypothalamus are independent of sex, and oxytocin release and receptors are similarly induced and distributed in males and females.

Description

Oxytocin has well-known physiological functions during labor and in milk letdown in humans. However, animal research has also demonstrated effects of oxytocin on a broad range of social and affiliative behaviors, as well as on physiological and developmental outcomes. For example, oxytocin plays an important role in the ability to form normal social attachments and is involved with social recognition and social interaction behaviors for both males and females of a number of animal species (Carter, 2003; Insel & Young, 2001; Uvnas-Moberg, 1998). Administering oxytocin has been shown to facilitate maternal behaviors among rodents such as grooming and licking pups, as well as other affiliative behaviors.

Research in both rodents and humans has found that oxytocin activity is elicited by various conditioned and nonconditioned sexual and social behaviors such as olfactory stimuli, touch, hair stroking, warmth, and pleasant vocal sounds (Depue & Morrone-Strupinsky, 2005). As a result, investigators now posit that positive social behaviors and social bonding, characterized by repeated physical contact, can lead to the release of oxytocin (Uvnas-Moberg, 1998). Additional research in animals and humans suggests that oxytocin is involved in general central nervous arousal, with effects that oppose stress-related activation by inhibiting sympathetic nervous system activity and increasing parasympathetic activity, as well as inhibiting the release of glucocorticoids. Some animal work has also demonstrated that oxytocin seems to shift energy use toward more positive health-promoting internal activities like storing nutrients and increasing the rate of wound healing. These findings have led to strong interest among investigators in behavioral medicine, as to whether research on oxytocin in humans may provide new insights into the neurobiology of resilience and health.

The role of oxytocin in social, mental, and physical aspects of health in humans is an area of active investigation. Preclinical research suggests that oxytocin facilitates bonding and approach behaviors partly by inhibiting stress-related maladaptive cognitive, affective, and biological activation. Because of its potential role in mediating human social behavior, ongoing research is focused on whether oxytocin may lead to improved social and emotional functioning for a range of disorders including autism, schizophrenia, and anxiety or depression (Averbeck, 2010; Bartz & Hollander, 2006). Other studies in nonclinical populations have found that exogenously administered oxytocin reduces fear-related activation in the amygdala, reduces cortisol levels and distress in response to social stress, increases prosocial behaviors, and modulates social memory (Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003;

Heinrichs, Meinlschmidt, Wippich, Ehlert, & Hellhammer, 2004; Kirsch et al., 2005; Kosfeld, Heinrichs, Zak, Fischbacher, & Fehr, 2005). However, effects of oxytocin do not appear to be uniformly positive and prosocial; other research has found that under certain conditions oxytocin may actually enhance negative responses to social stress in humans (Shamay-Tsoory et al., 2009).

Measuring oxytocin and its effects in humans presents some challenges. Oxytocin release into peripheral circulation is pulsatile, and it has a very short half-life. In addition, plasma oxytocin does not easily cross the blood–brain barrier and therefore may not be informative about oxytocin activity and effects in the brain. As a result, research on the role of oxytocin in human health is still in an early period with investigators developing new assays and protocols for addressing these methodological challenges.

As with any biological substrate, oxytocin is likely to have a multiplicity of effects, and potential therapeutic and mechanistic insights will be enhanced as we achieve a greater understanding of the range of effects possible and the conditions under which they occur.

Cross-References

- ▶ Attachment Theory
- ▶ Biomarkers
- ▶ Empathy
- ▶ Gender Differences
- ▶ Resilience
- ▶ Social Relationships
- ▶ Social Stress
- ▶ Social Support
- ▶ Stress

References and Readings

Averbeck, B. B. (2010). Oxytocin and the salience of social cues. *Proceedings of the National Academy of Sciences of the United States of America, 107*(20), 9033–9034.

Bartz, J. A., & Hollander, E. (2006). The neuroscience of affiliation: Forging links between basic and clinical research on neuropeptides and social behavior. *Hormones and Behavior, 50*(4), 518–528.

Carter, C. S. (2003). Developmental consequences of oxytocin. *Physiology and Behavior, 79*, 383–397.

Depue, R. A., & Morrone-Strupinsky, J. V. (2005). A neurobehavioral model of affiliative bonding: Implications for conceptualizing a human trait of affiliation. *The Behavioral and Brain Sciences, 28*(3), 313–350; discussion 350–395.

Heinrichs, M., Baumgartner, T., Kirschbaum, C., & Ehlert, U. (2003). Social support and oxytocin interact to suppress cortisol and subjective responses to psychosocial stress. *Biological Psychiatry, 54*(12), 1389–1398.

Heinrichs, M., Meinlschmidt, G., Wippich, W., Ehlert, U., & Hellhammer, D. H. (2004). Selective amnesic effects of oxytocin on human memory. *Physiology and Behavior, 83*(1), 31–38.

Insel, T. R., & Young, L. J. (2001). The neurobiology of attachment. *Nature Reviews Neuroscience, 2*(2), 129–136.

Kirsch, P., Esslinger, C., Chen, Q., Mier, D., Lis, S., Siddhanti, S., et al. (2005). Oxytocin modulates neural circuitry for social cognition and fear in humans. *Journal of Neuroscience, 25*(49), 11489–11493.

Kosfeld, M., Heinrichs, M., Zak, P. J., Fischbacher, U., & Fehr, E. (2005). Oxytocin increases trust in humans. *Nature, 435*(7042), 673–676.

Shamay-Tsoory, S. G., Fischer, M., Dvash, J., Harari, H., Perach-Bloom, N., & Levkovitz, Y. (2009). Intranasal administration of oxytocin increases envy and schadenfreude (gloating). *Biological Psychiatry, 66*(9), 864–870.

Uvnas-Moberg, K. (1998). Oxytocin may mediate the benefits of positive social interaction and emotions. *Psychoneuroendocrinology, 23*(8), 819–835.

P

P-30 Antigen

► Prostate-Specific Antigen (PSA)

Pain

Michael James Coons[1] and Jeremy Steglitz[2]
[1]Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA
[2]Department of Psychiatry and Behavioral Sciences, Clinical Psychology Division, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Synonyms

Pain management/control; Pain: psychosocial aspects; Pain threshold

Definition

Pain is a noxious sensory phenomenon that provides information to organisms about the occurrence or threat of injury. Pain is also a multidimensional experience that results from a complex interaction between biological and psychological components and is further influenced by behavioral and social factors. The temporal course of pain can range from acute, or time-limited states in response to injury, to chronic states in which pain persists beyond the point of tissue repair or healing. Although the Gate Control Theory (Melzack & Wall, 1967) provides a unified model of pain, a variety of pain subtypes exist that have different underlying mechanisms.

Description

Neurobiology of Pain

A primary function of the nervous system is to communicate information to alert organisms to the experience or threat of injury. Therefore, the noxious qualities of pain function to capture our attention and motivate action to minimize the risk of harm. Nociceptors are a specialized class of primary afferent nerves. Myelinated nociceptors signal sharp pain from heat and pressure stimuli on skin with hair. Unmyelinated nociceptors signal burning and pressure-induced pain on hairless skin. Both unmyleinated and myelinated nociceptors signal pain from chemical stimuli. Injury leads to increased pain sensitivity or hyperalgesia caused by inflammation.

CNS Mechanisms of Pain Modulation

At the level of the spinal cord, pain modulation occurs in the substantia gelatinosa (SG) of the dorsal horn. The SG serves an inhibitory or gating function that modulates pain signal transduction. Pain modulation also occurs in the periaqueductal gray (PAG) and rostral ventromedial medulla (RVM) of the midbrain, which

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

function as a bidirectional relay station. The system is involved in the suppression of ascending responses to harmful stimuli and enhances nociceptive responses. The PAG-RVM system has connections to the hypothalamus and limbic forebrain structures including the amygdala, anterior cingulate cortex, and anterior insula. Consequently, fear, attention, and pain expectancies exert top-down processes that influence pain perception.

Clinical Classifications of Pain

Postoperative Pain and its Management

Acute postoperative pain is a product of a range of physiological mechanisms. It comprises a constellation of sensory, emotional, and psychological experiences that follow surgery and is associated with autonomic, endocrine-metabolic, physiological, and behavioral responses. Considerable progress has been made to understand the functions of the peripheral and central nervous systems, mechanisms of acute postoperative pain, side effects of analgesic drugs, and techniques and interventions in surgical patients. However, improvements in the management of acute postoperative pain are needed. Specifically, the use of acute pain management services may help to ensure that evidence-based analgesic techniques are implemented. Furthermore, improved collaboration would be useful between interdisciplinary care teams including anesthesiologists, surgeons, nurses, and mental health professionals to optimize postoperative pain management.

Deep Somatic Tissue Pain Deep tissue pain encompasses pain from the joints and muscles. The pain is often diffuse and characterized by dull and aching sensations. Major sources of deep somatic tissue pain are inflammatory diseases, trauma, overload, and degenerative diseases. Under normal conditions, these nociceptors are activated by stimuli that can lead to structural damage including overload, twisting, pressure, and ischemic contraction. Under conditions of inflammation or trauma, nociceptors of joint and muscle become increasingly sensitized, particularly to mechanical stimuli.

Two types of spinal cord neurons process nociceptive input from joint and muscle. These neurons show two distinct properties: (1) They converge inputs from the skin and deep tissue, and (2) they are activated by mechanical stimuli. Neurons in the thalamus and cortex process inputs from deep tissue.

Arthritis Osteoarthritis (OA) is a degenerative joint disease that results in pain localized to the joint cartilage and subchondral bone. It affects approximately 30% of adults 75 years and older. OA is classified by the location of the affected joints (e.g., hip or knee) and whether the pain is primary or secondary to other conditions or disease processes. OA pain may occur in response to mechanical stimuli but may be chemically mediated when joint tissues fail to adequately repair. Consequences of OA include loss of articular cartilage, new bone formation in the subchondral region, and formation of new cartilage and bone in the joints and are characterized by pain, stiffness, functional limitations, and impaired quality of life.

Rheumatoid arthritis (RA) is an autoimmune disease that results in significant joint inflammation and pain. It affects nearly 1% of the adult population. RA involves both the small and large joints and is distributed symmetrically. RA may eventually progress to joint failure, which can result in secondary OA. Age, sex, family history, and tobacco use are significant risk factors for RA.

Treatment for arthritis is multidimensional. Components may include education about the disease process, weight loss to minimize joint stress, increased physical activity and/or physical therapy, analgesics, anti-inflammatory drugs, and local steroid or hyaluronic acid injections. Specific to RA, antirheumatic drugs (DMARDs) may be used to reduce inflammation and slow the disease progression. In severe or advanced cases, surgical intervention including joint replacement may be indicated.

Fibromyalgia Syndrome Fibryomyalgia syndrome (FMS) is a persistent pain condition involving the soft tissues. It affects approximately 2% in

the general population and is significantly more prevalent among women than men. Although its etiology is unknown, significant advances have been made in understanding its clinical presentation. Pain is believed to result from central sensitization of somatosensory pathways that induce enhanced pain perception. This includes persistent, diffuse pain to touch at localized areas in soft tissues. Other systemic symptoms include recurrent headaches, dizziness, fatigue, morning stiffness, irritable bowel syndrome, irritable bladder syndrome, insomnia, cognitive dysfunction, depression, and anxiety.

Treatment for FMS is similar to that of other persistent pain conditions and includes education, physical activity and/or physical therapy, cognitive-behavioral therapy, and pharmacological interventions that act on central neural pathways.

Low Back Pain Low back pain (LBP) is defined as pain, muscle tension, or stiffness that is localized between the costal margin and inferior gluteal folds. It also presents with or without radiating leg pain. It can also be classified as specific, or in response to a known pathology (e.g., hernia, fracture), or nonspecific or idiopathic. Nonspecific LBP accounts for 90% of all cases. Physical exercises are currently the only empirically supported intervention to prevent LBP. Many randomized controlled trials and systematic reviews have been published that examine its treatment. Physical activity, nonsteroidal anti-inflammatory drugs (NSAIDs), and muscle relaxants are effective treatments for acute LBP. Physical therapy and CB therapy in the context of interdisciplinary pain treatment programs are recommended for persistent LBP.

Visceral Pain Visceral pain is diffuse. It results in referred pain (pain perceived in sites other than the site of injury or pain stimuli). Unlike somatic nociceptors, the activation of visceral nociceptors does not require tissue damage. Rather, it can be caused by the distension of hollow organs, traction on the mesentery, ischemia, and endogenous chemicals associated with inflammatory processes.

Thorax Pain Angina pectoris is the most frequently occurring form of thorax pain. It is caused by myocardial ischemia but often produces chest pain that is distal to the ischemic event. Cardiac pain is mediated by interplay between autonomic reflexes and peripheral cardiac nerves. The thalamus and hypothalamus transmit cardiac pain to the prefrontal cortex. The occurrence of pain is slow and lacks spatial localization. The clinical presentation of myocardial ischemia can be different in men and women. Pharmacotherapy is the primary intervention for angina pectoris and acute myocardial infarction (MI) including beta-blockers, nitroglycerin, morphine, and antithrombotic agents. Percutaneous transluminal coronary intervention (PCI) and coronary artery bypass graft surgery (CAGB) are surgical interventions that improve coronary blood flow. Other forms of thorax pain include esophageal chest pain, aortic aneurysms, and pulmonary events including embolism.

Abdominal Pain Abdominal pain can be caused by chronic functional disorders or acute life-threatening conditions. Chronic disorders, such as irritable bowel syndrome, account for the majority of diagnoses that account for abdominal pain. Most chronic disorders result in enhanced pain perception in response to visceral stimuli, which results from central pain modulation mechanisms. In contrast, acute abdominal pain is caused by an identifiable stimulus or pathology (e.g., gastric ulcer). Although interventions for acute abdominal pain are highly effective, the development of efficacious treatments for chronic conditions is needed.

Orofacial Pain Orofacial pain is defined as pain experienced in the motor or sensory aspects of the trigeminal nerve system. Pain signals are transmitted to the nucleus caudalis in the medulla and project onward to the thalamus and cortex via the trigeminothalamic tract. Orofacial pain is exacerbated by central convergence, inflammation of the oral mucosa, and central sensitization to pain (increased spontaneous firing of nerves). Prevention strategies include preoperative administration of NSAIDs to block inflammatory

processes and local anesthetics to reduce central sensitization. However, due to common adverse events when using systemic opioids, their outpatient use is limited. The etiology of persistent (unremitting or recurrent) orofacial pain is poorly understood. Consequently, pain management is imprecise, and the majority of therapies are not validated.

Neuropathic Pain Neuropathic pain occurs in response to injury (lesion or dysfunction) of the nervous system. Numerous animal models have been proposed to account for the underlying mechanisms. One of the earliest models originated from the observation that animals attack a limb in which the axons of neurons have been severed following injury. This model suggests that a central sensitization process occurs, such that severed nerves exhibit greater spontaneous firing frequencies than healthy intact nerves. Individuals experiencing neuropathic pain describe their pain as "electric shocks, burning, tingling, itching, and prickling." Examples of neuropathic pain conditions include neuropathies (e.g., peripheral, autonomic) and neuralgias (e.g., trigeminal). Pharmacological treatments may include topical analgesics, tricyclic antidepressants, anticonvulsants, and opioids.

Phantom Limb Pain This is a form of neuropathic pain and refers to perceived pain sensations in the anatomical space of a limb that has been amputated. Although the majority of individuals experience sensations related to the shape, posture, or movement of the missing limb, 60–80% of individuals experience intermittent or persistent pain. Although the specific mechanisms underlying phantom limb pain are not entirely understood, both the peripheral and central nervous systems are involved. Additional treatments may include sodium channel blockers, physical therapy, and transcutaneous electrical nerve stimulation.

Cancer Pain The neurobiology of cancer pain is poorly understood. However, new models have identified mechanisms that both produce and maintain cancer pain. Tumor cells and tumor-associated cells (macrophages, neutrophils, T-lymphocytes) are believed to sensitize primary afferent neurons in the periphery. Findings from these studies may lead to the development of novel therapies that act on these peripheral mechanisms and could improve the quality of life of individuals living with cancer.

Pain Assessment and Intervention Pain is a complex and subjective experience characterized by sensory-discriminative, motivational-affective, and cognitive-evaluative dimensions. Pain can be evaluated using a variety of tools including verbal and numerical self-report scales, visual analogue scales (VAS), self-report measures, behavioral observation, and physiological markers. A combination of these measures is recommended to yield the most valid information about this phenomenon.

Within clinical and research settings, the VAS, the McGill Pain Questionnaire (MPQ), and the Multidimensional Pain Inventory (MPI) are examples of commonly used instruments. Using a VAS, individuals are asked to rate their current pain intensity on a numeric scale that is anchored by 0 (no pain at all) and 10 (the most pain you could imagine experiencing). The McGill Pain Questionnaire was developed Melzack and Wall (Melzack, 1975). It evaluates the specific location(s), intensity, and qualities of pain that individuals currently experience. It contains four domains including sensory, affective, evaluative, and miscellaneous. It can either be clinician-administered or completed independently by patients in a self-report manner. The McGill Pain Questionnaire is a reliable and valid tool to assess the multidimensional nature of pain experience. For research applications where time is limited to obtain information, a short-form McGill Pain Questionnaire (SF-MPQ) is also available. The MPI is a comprehensive self-report instrument designed to evaluate a full range of experiences with persistent pain conditions. It includes dimensions of pain intensity, emotional distress, cognitive and functional adaptation, and social support. It has also been shown to have solid psychometric properties.

Cognitive-Behavioral Approaches to Pain Management In the context of persistent pain conditions, cognitive-behavioral (CB) interventions focus on developing adaptive pain coping skills. These skills are intended to minimize the experience of pain, prevent or minimize pain exacerbations, and limit pain-related disability. A CB intervention addresses the subjective and contextual aspects of pain. First, patients receive education about theories of pain (e.g., Gate Control Theory) to foster an understanding of why pain persists beyond the point of tissue repair. Understanding this conceptual framework provides the foundation for deploying a series of related cognitive and behavioral interventions. Cognitive interventions may include cognitive restructuring to minimize pain catastrophizing, attention diversion and distraction techniques, and problem solving exercises. Behavioral interventions may include relaxation, paced diaphragmatic breathing, goal setting, behavioral activation, and graded exposure to feared physical activities. In the management of persistent pain, CB interventions are often most effective when implemented in the context of interdisciplinary care teams and can be used as an adjuvant to other physical, pharmacological, or surgical therapies.

Cross-References

- ▶ Gate Control Theory of Pain
- ▶ Pain Anxiety
- ▶ Pain Management/Control
- ▶ Pain Threshold
- ▶ Pain: Psychosocial Aspects
- ▶ Pain-Related Fear

References and Readings

Bielefeldt, K., & Gebhart, G. F. (2006). Visceral pain: Basic mechanisms. In S. B. McMahon & M. Koltzenburg (Eds.), *Wall and Melzack's textbook of pain* (pp. 721–736). Philadelphia, PA: Elsevier.

Dahl, J. B., & Kehlet, H. (2006). Postoperative pain and its management. In S. B. McMahon & M. Koltzenburg (Eds.), *Wall and Melzack's textbook of pain* (5th ed., pp. 635–652). Philadelphia, PA: Elsevier.

Dionne, R. A., Kim, H., & Gordon, S. M. (2006). Acute and chronic dental and orofacial pain. In S. B. McMahon & M. Koltzenburg (Eds.), *Wall and Melzack's textbook of pain* (5th ed., pp. 833–850). Philadelphia, PA: Elsevier.

Mantyh, P. W. (2006). Cancer pain: Causes, consequences, and therapeutic opportunities. In S. B. McMahon & M. Koltzenburg (Eds.), *Wall and Melzack's textbook of pain* (5th ed., pp. 1087–1098). Philadelphia, PA: Elsevier.

Melzack, R., & Katz, J. (2006). Pain assessment in adult patients. In S. B. McMahon & M. Koltzenburg (Eds.), *Wall and Melzack's textbook of pain* (5th ed., pp. 291–304). Philadelphia, PA: Elsevier.

Melzack, R., & Wall, P. D. (1967). Pain mechanisms: A new theory. *Science, 150*, 971–979.

Melzack, R. (1975) The McGill Pain Questionnaire: Major properties and scoring methods. *Pain 1*, 275–299

Meyer, R. A., Ringkamp, M., Campbell, J. N., & Raja, S. N. (2006). Peripheral mechanisms of cutaneous nociception. In S. B. McMahon & M. Koltzenburg (Eds.), *Wall and Melzack's textbook of pain* (5th ed., pp. 3–34). Philadelphia, PA: Elsevier.

Nikolasjsen, L., & Straehelin Jensen, T. (2006). Phantom limb. In S. B. McMahon & M. Koltzenburg (Eds.), *Wall and Melzack's textbook of pain* (5th ed., pp. 961–972). Philadelphia, PA: Elsevier.

Ossipov, M. H., Lai, J., & Porreca, F. (2006). Mechanisms of experimental neuropathic pain: Integration from animal models. In S. B. McMahon & M. Koltzenburg (Eds.), *Wall and Melzack's textbook of pain* (5th ed., pp. 929–946). Philadelphia, PA: Elsevier.

Russell, I. J., & Bieber, C. S. (2006). Myofascial pain and fibromyalgia syndrome. In S. B. McMahon & M. Koltzenburg (Eds.), *Wall and Melzack's textbook of pain* (pp. 669–682). Philadelphia, PA: Elsevier.

Scadding, J. W., & Koltzenburg, M. (2006). Painful peripheral neuropathies. In S. B. McMahon & M. Koltzenburg (Eds.), *Wall and Melzack's textbook of pain* (5th ed., pp. 973–1000). Philadelphia, PA: Elsevier.

Schaible, H. G. (2006). Basic mechanisms of deep somatic tissue. In S. B. McMahon & M. Koltzenburg (Eds.), *Wall and Melzack's textbook of pain* (5th ed., pp. 621–635). Philadelphia, PA: Elsevier.

Scott, D. L. (2006). Osteoarthritis and rheumatoid arthritis. In S. B. McMahon & M. Koltzenburg (Eds.), *Wall and Melzack's textbook of pain* (pp. 653–668). Philadelphia, PA: Elsevier.

Sylven, C., & Erikson, E. (2006). Thorax. In S. B. McMahon & M. Koltzenburg (Eds.), *Wall and Melzack's textbook of pain* (5th ed., pp. 737–752). Philadelphia, PA: Elsevier.

Turk, D. C. (2002). A cognitive-behavioral perspective on treatment of chronic pain patients. In R. J. Gatchel & D. C. Turk (Eds.), *Psychological approaches to*

pain management: A practitioner's handbook (pp. 138–158). New York: Guilford.
van Tulder, M. W., & Koes, B. (2006). Low back pain. In S. B. McMahon & M. Koltzenburg (Eds.). *Wall and Melzack's textbook of pain* (5th ed., pp. 699–708). Philadelphia, PA: Elsevier
Wong, H. Y., & Mayer, E. A. (2006). A clinical perspective on abdominal pain. In S. B. McMahon & M. Koltzenburg (Eds.), *Wall and Melzack's textbook of pain* (5th ed., pp. 753–776). Philadelphia, PA: Elsevier.

Pain Anxiety

Michael James Coons
Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Synonyms

Pain-related fear

Definition

Pain anxiety is a future-oriented state of autonomic arousal that is triggered by the anticipation of pain. Similar to the tripartite model of anxiety, it is comprised of cognitive, physiological, and behavioral components. Pain anxiety is an affective manifestation of the autonomic nervous system that occurs in response to the anticipation of pain. Albeit to a lesser degree than that triggered by fear, the sympathetic nervous system becomes activated by the septo-hippocampal brain regions. Pain anxiety is characterized by cognitive, physiological, and behavioral symptoms. Cognitively, individuals become hypervigilant for pain-related cues by internally scanning their body (for internal signs and symptoms of pain) and the environment (for pain-inducing contexts or stimuli). They also anticipate experiencing pain in the future and may often expect their pain to be catastrophic (e.g., "Having to get my tooth fixed at the dentist will be excruciating"). In turn, individuals become motivated to engage in avoidance behaviors to minimize the likelihood of experiencing future pain. According to fear-avoidance models of pain, behavioral avoidance (typically of situations involving physical activity or movement) leads to physical deconditioning and muscle atrophy, which in turn, may result in greater pain intensity. Pain anxiety and related avoidance behavior greatly contributes to the progression of acute to persistent pain.

The most common mode of assessment of pain anxiety involves the administration of well-validated self-report instruments. Several published scales are available that assess the nature and extent of pain-related cognitions (e.g., "When I hurt, I think about pain constantly"), pain-related avoidance (e.g., "I try to avoid activities that cause pain"), and physiological symptoms (e.g., "When I sense pain, I feel dizzy or faint"). Examples of self-report instruments include the Pain Anxiety Symptom Scale (PASS) and the Burn-Specific Pain Anxiety Scale. Alternative modes of assessing pain-related fear include semi-structured clinical interviews and the direct observation of patient behavior.

Cognitive behavior therapy (CBT) is the most well-validated intervention for pain anxiety. Treatment components include psychoeducation and introduction, demonstration, and practice of a variety of adaptive coping skills (i.e., progressive muscle relaxation, diaphragmatic breathing, mental imagery, behavioral activation and pleasant activity scheduling, activity-rest cycling, physical therapy/exercise, problem-solving, cognitive restructuring, and calming self-statements). In treating pain-related fear, special emphasis is placed on graded in vivo exposure with behavioral experiments. These latter components allow individuals to identify situations through which they can gather information to test and challenge their distorted pain-related cognitions.

Cross-References

▶ Pain, Psychosocial Aspects
▶ Pain-Related Fear

References and Readings

Asmundson, G. J. G., Norton, P. J., & Vlaeyen, W. S. (2004). Fear-avoidance models of chronic pain: An overview. In G. J. G. Asmundson, J. W. S. Vlaeyen, & G. Crombez (Eds.), *Understanding and treating fear of pain* (pp. 3–24). New York: Oxford University Press.

de Williams, A. C., & McCracken, L. M. (2004). Cognitive behavioral therapy for chronic pain: An overview with specific reference to fear avoidance. In G. J. G. Asmundson, J. W. S. Vlaeyen, & G. Crobez (Eds.), *Understanding and treating fear of pain* (pp. 293–312). New York: Oxford University Press.

McNeil, D. W., & Vowles, K. E. (2004). Assessment of fear and anxiety associated with pain: Conceptualization, methods, and measures. In G. J. G. Asmundson, J. W. S. Vlaeyen, & G. Crombez (Eds.), *Understanding and treating fear of pain* (pp. 189–211). New York: Oxford University Press.

Pain Anxiety Symptoms Scale (PASS) and Short Version PASS-20

Lance M. McCracken
Psychology Department, Institute of Psychiatry, King's College London, London, UK

Definition

Short self-report questionnaires designed to measure aspects of pain-related anxiety and avoidance for use in clinical assessment and research.

Description

The 40-item Pain Anxiety Symptoms Scale (PASS) (McCracken, Zayfert, & Gross, 1992) and a shorter 20-item version of the same assessment instrument (PASS-20) (McCracken & Dhingra, 2002) measure pain-related anxiety, fear, and avoidance. They were designed for clinical and research purposes and mostly for use with adults with recurrent or chronic pain conditions. However, they have been validated and used in people without identified pain conditions, such as university students and people recruited from community settings (Abrams, Carleton, & Asmundson, 2007). The original PASS was developed at a time during the late 1980s and early 1990s when clinicians and researchers were beginning to focus greater attention on processes of fear and avoidance in relation to chronic pain. Since that time a model of chronic pain referred to as the "fear-avoidance" model has become a popular organizing framework guiding research and treatment development (Vlaeyen & Linton, 2000). The PASS and PASS-20 have been used in research investigating models and mechanisms of pain-related disability, and in treatment process and outcome research (McCracken & Gross, 1998; Vowles & McCracken, 2008). They are also routinely used in clinical practice for analyzing and conceptualizing cases and in treatment-related decision making.

Both versions of the PASS include four subscales that reflect aspects of avoidance behavior (e.g., I will stop any activity as soon as I sense pain coming on), cognitive anxiety (e.g., During painful episodes it is difficult for me to think of anything else besides the pain), fear (e.g., When I feel pain I am afraid that something terrible will happen), and physiological anxiety (e.g., I find it hard to calm my body down after periods of pain). The four subscales are equal length in each version of the measure. All items are rated on a scale from 0 (never) to 5 (always). In the 40-item version five items are reverse-keyed and must be recoded before calculating summary scores. Summary scores for both versions are calculated by summing assigned items and then by summing the subscales to derive an overall score. There are no set cutoffs for interpreting scores from the instruments. One method to facilitate interpretation is to convert raw scores to standard scores or percentile ranks. As an example, a table of raw scores and percentile rank equivalents has been constructed from a large consecutive sample of patients seen at a tertiary care center in the UK ($N = 339$). From this raw scores of 15.0, 33.0, 60.0, and 78.0 from the PASS-20 correspond to the 5th, 25th, 75th, and 95th percentile, respectively.

The PASS and PASS-20 have been extensively validated. They both show very good

internal consistency and temporal consistency (McCracken & Dhingra, 2002; Roelofs et al., 2004) and a factor structure that generally matches the a priori subscale structure (Abrams et al., 2007; Larsen, Taylor, & Asmundson, 1997; Roelofs et al., 2004). Both versions of the instrument demonstrate adequate construct validity in relation to variables such as general anxiety and depression (e.g., Roelofs et al., 2004) and show mostly moderate to large correlations with important criterion variables, such as measures of physical and psychosocial disability (e.g., McCracken & Dhingra, 2002). The original PASS (McCracken & Gross, 1998) and the 20-item version (Vowles & McCracken, 2008) both appear sensitive to the effects of multidisciplinary treatment for chronic pain.

For most uses currently the PASS-20 is preferred as it appears equivalent to the longer version in most important respects. The PASS-20 has been translated into Chinese, Dutch, French, Icelandic, Iranian, Polish, and Spanish, among other languages.

The fear-avoidance model of chronic pain has been a very useful model for understanding pain-related disability and for guiding the current generation of treatment developments for chronic pain. At the same there are other theoretical and treatment developments that appear wider in scope and possibly more progressive than the fear-avoidance model. These developments include contextual approaches within cognitive behavior therapy, approaches such as Acceptance and Commitment Therapy (ACT) (Hayes, Strosahl, & Wilson, 1999) and mindfulness-based approaches. In this work measures such as the PASS-20 remain useful. Other variables that are known to have strong relations with pain-related fear and avoidance are also now frequently studied, such as acceptance of pain and values-based action, among others, and a wider process called psychological flexibility (e.g., Vowles & McCracken, 2008). The point is that the PASS-20 and its focus on fear and avoidance remains relevant and the field is also evolving so that these process are being examined in a broader and well-integrated cognitive behavioral framework. These theoretical and treatment developments may be important to those who are hoping to assess pain-related fear and avoidance and may be seeking to use an instrument such as the PASS-20.

Cross-References

- ▶ Chronic Pain
- ▶ Cognitive Behavior Therapy

References and Readings

Abrams, M. P., Carleton, R. N., & Asmundson, G. J. G. (2007). An exploration of the psychometric properties of the PASS-20 with a nonclinical sample. *The Journal of Pain, 8*, 879–886.

Hayes, S. C., Strosahl, K. D., & Wilson, K. G. (1999). *Acceptance and commitment therapy: An experiential approach to behavior change*. New York: Guilford Press.

Larsen, D. K., Taylor, S., & Asmundson, G. J. G. (1997). Exploratory factor analysis of the Pain Anxiety Symptoms Scale in patients with chronic pain complaints. *Pain, 69*, 27–34.

McCracken, L. M., & Dhingra, L. (2002). A short version of the Pain Anxiety Symptom Scale (PASS-20): Preliminary development and validity. *Pain Research & Management, 7*, 45–50.

McCracken, L. M., & Gross, R. T. (1998). The role of pain-related anxiety reduction in the outcome of multidisciplinary treatment for chronic low back pain: Preliminary results. *Journal of Occupational Rehabilitation, 8*, 179–189.

McCracken, L. M., Zayfert, C., & Gross, R. T. (1992). The Pain Anxiety Symptoms Scale: Development and validation of a scale to measure fear of pain. *Pain, 50*, 67–73.

Roelofs, J., McCracken, L., Peters, M. L., Crombez, G., van Breukelen, G., & Vlaeyen, J. W. S. (2004). Psychometric evaluation of the Pain Anxiety Symptoms Scale (PASS) in chronic pain patients. *Journal of Behavioral Medicine, 27*, 167–183.

Vlaeyen, J. W. S., & Linton, S. J. (2000). Fear-avoidance and its consequences in chronic musculoskeletal pain: A state of the art. *Pain, 85*, 317–332.

Vowles, K. E., & McCracken, L. M. (2008). Acceptance and values-based action in chronic pain: A study of treatment effectiveness and process. *Journal of Consulting and Clinical Psychology, 76*, 397–407.

Pain Management/Control

Michael James Coons
Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Synonyms

Pain: psychosocial aspects

Definition

Pain is a multidimensional phenomenon. Pain management refers to the physiological (i.e., pharmacological, surgical), psychological, and behavioral interventions that are aimed at minimizing pain perception and alleviating pain-related interference and disability.

Description

Historically, pain was conceptualized using a disease model and was considered to be a purely sensory experience resulting from injury, inflammation, or tissue damage. However, limitations to this model became evident after observing diverse responses to pain across individuals despite objectively similar physical stimuli or trauma. In 1965, Melzack and Wall published a seminal paper in *Science* that outlined a revolutionary theory of pain (Melzack & Wall, 1965).

Gate Control Theory

The Gate Control Theory emphasized central neural mechanisms at the level of the spinal cord that modulate afferent signals from peripheral nerves en route to the brain. This sensory modulation and subsequent pain perception is influenced by sensory input, cognitive processing, affective states, neural inhibitory capacities, activities of the stress-regulation system, and subsequent behavioral responses. Therefore, interventions targeting these multiple mechanisms provide opportunity to achieve effective pain management.

Pharmacological and Surgical Interventions

Pharmacological interventions are often the first-line treatments for pain. Systemic analgesics are the focus of both acute and persistent pain management and include nonsteroidal anti-inflammatory drugs (NSAIDs), including acetaminophen (ASA), and opioid analgesics (pure or in combination with NSAIDs). Combination opioid analgesics are typically administered orally; however, other routes of administration include rectal and sublingual. Pure opioids can be administered through the skin (with a transdermal patch), subcutaneously, or intravenously. These latter routes of administration are typically used when adequate pain relief is not achieved with the use of NSAIDs or combination opioids. In cases of neuropathic pain (i.e., pain due to dysfunction of the nervous system, in the absence of tissue damage), antiepileptic medications are often used (e.g., pregabalin). In light of their misuse and abuse potential of opioids, individuals with persistent pain should be evaluated to determine if chronic opioid management is clinically appropriate. Individuals with a history of medical non-adherence or individuals with severe axis I or axis II pathology may require close monitoring if opioid medications are prescribed.

Surgical procedures for pain management range in their degree of invasiveness. Minimally invasive procedures include steroidal injections and nerve blocks (of the peripheral and sympathetic nerves). Maximally invasive procedures include radiofrequency ablations (for the local destruction of nervous tissue), spinal cord stimulator implantation (for the neuromodulation of afferent pain signals), and intrathecal pump implantations (for the direct administration of opioids and other analgesics into the cerebral spinal fluid when pain relief using other modalities has been unsuccessful). Particularly with these more invasive surgical interventions, individuals must adhere to medical recommendations to avoid potentially life-threatening adverse

medical events. Consequently, candidates for these procedures also require careful evaluation and selection by qualified professionals to ensure patient safety.

Cognitive Behavioral Interventions

Cognitive behavioral interventions focus on developing adaptive pain coping skills to minimize the experience of pain, prevent long-term exacerbations in pain, and minimize pain-related disability. For individuals with persistent pain (either intermittent-recurrent pain or unremitting pain), they often become anxious and fearful of the pain experience and avoid engaging in physical activity to minimize the experience of pain. However, over time, this behavioral avoidance contributes to the loss of physical strength, physical deconditioning, and muscular atrophy (i.e., loss of muscle mass) that exacerbates pain perception. In essence, the avoidance of physical activity *because* of pain results in pain intensification. This behavioral avoidance reinforces the fear of pain, resulting in greater anxiety and propensity to avoid physical activity. Furthermore, avoidance of pain often leads to avoidance of activities through which individual's derive meaning and value (e.g., participation in one's profession, spending time with family/friends). Over time, avoidance may contribute to a sense of isolation, undermine one's confidence in their ability to manage their pain, and increase the focus of their attention to the perception of pain. This process contributes the development or exacerbation of depression, the intensification of pain, and pain-related interference and disability.

Components of cognitive behavioral interventions for persistent pain include psychoeducation and introduction, demonstration, and practice of a variety of adaptive coping skills (i.e., progressive muscle relaxation, diaphragmatic breathing, mental imagery, behavioral activation and pleasant activity scheduling, activity-rest cycling, physical therapy/exercise, problem-solving, cognitive restructuring, and calming self-statements). Within a CBT protocol, the relaxation procedures reduce sympathetic arousal to both "close the gate" to minimize the transmission of afferent pain signals to the brain and reduce muscle tension associated with pain. The attention procedures (i.e., mental imagery, pleasant activity scheduling) function to distract individuals from their pain experience and facilitate positive experiences and positive affect. The behavioral interventions (i.e., activity-rest cycling, behavioral activation, physical therapy/exercise) are intended to help individuals learn how to engage in activities despite experiencing pain and to both minimize pain-related interference and maintain their physical conditioning. While initial behavioral engagement often results in pain exacerbations, and over time, it is associated with reductions in pain intensity. In cases where individuals experience high degrees of pain-related fear and anxiety, graded exposure in vivo to physical movement may be required to minimize their affective response. Cognitive interventions (i.e., cognitive restructuring, calming self-statements, problem-solving) are intended to address pain catastrophizing (e.g., "This pain is so horrible I *cannot* do anything"), maladaptive pain beliefs (e.g., "Walking *should not* be painful"), and enable adaptive problem-solving skills. These cognitive behavioral interventions can be effectively delivered in individual or group formats, can be modified for family or system-based interventions, and can be augmented with the use of biofeedback equipment (e.g., superficial electromyography and monitoring of heart rate, respiration rate, peripheral temperature, and skin conductance). These applications are intended to provide individuals with physiological data that is used to help modulate sympathetic nervous system arousal using the coping skills outlined above.

Pain is a complex and multidimensional experience. In cases of persistent pain, individuals may benefit from clinical management by interdisciplinary teams. Although in some cases complete pain relief is not feasible, a combination of the approaches described above can minimize pain-related interference, disability, and the impact and potential for comorbid psychiatric conditions to maximize the quality of life experienced by individuals.

Cross-References

- ▶ Pain
- ▶ Pain Anxiety
- ▶ Pain, Psychosocial Aspects
- ▶ Pain: Psychosocial Aspects
- ▶ Pain-Related Fear

References and Readings

Asmundson, G. J. G., Norton, P. J., & Norton, G. R. (1999). Beyond pain: The role of fear and avoidance in chronicity. *Clinical Psychology Review, 19*, 97–119.

Asmundson, G. J. G., Norton, P. J., & Vlaeyen, J. W. S. (2004). Fear-avoidance models of chronic pain: An overview. In G. J. G. Asmundson, J. W. S. Vlaeyen, & G. Crobez (Eds.), *Understanding and treating fear of pain* (pp. 3–24). New York: Oxford University Press.

Bajwa, Z. H., & Ho, C. (2004). Antiepileptics for pain. In C. A. Warfield & Z. H. Bajwa (Eds.), *Principles and practice of pain medicine* (2nd ed., pp. 649–654). New York: McGraw Hill.

Day, M., & Anderson, S. (2004). Cryoanalgesia and radiofrequency. In C. A. Warfield & Z. H. Bajwa (Eds.), *Principles and practice of pain medicine* (2nd ed., pp. 751–764). New York: McGraw Hill.

DeSio, J. M. (2004). Epidural steroid injections. In C. A. Warfield & Z. H. Bajwa (Eds.), *Principles and practice of pain medicine* (2nd ed., pp. 655–661). New York: McGraw Hill.

Du Pen, S. L., & Du Pen, A. (2004). Neuraxial drug delivery. In C. A. Warfield & Z. H. Bajwa (Eds.), *Principles and practice of pain medicine* (2nd ed., pp. 720–739). New York: McGraw Hill.

Keefe, F. J., Beaupre, P. M., & Gil, K. M. (2002). Group therapy for patients with chronic pain. In R. J. Gatchel & D. C. Turk (Eds.), *Psychological approaches to pain management: A practitioner's handbook* (pp. 234–255). New York: Guilford.

Lamer, L. J. (2004). Intra-articular injections and facet blocks. In C. A. Warfield & Z. H. Bajwa (Eds.), *Principles and practice of pain medicine* (2nd ed., pp. 667–683). New York: McGraw Hill.

Lehmann, L. J. (2004). Peripheral nerve blocks. In C. A. Warfield & Z. H. Bajwa (Eds.), *Principles and practice of pain medicine* (2nd ed., pp. 684–695). New York: McGraw Hill.

Lipman, A. G., & Jackson, K. C. (2004). Opioid pharmacotherapy. In C. A. Warfield & Z. H. Bajwa (Eds.), *Principles and practice of pain medicine* (2nd ed., pp. 583–600). New York: McGraw Hill.

Melzack, R. (1999). From the gate to the neuromatrix. *Pain* (Suppl. 6), *82*, S121-S126.

Melzack, R., & Wall, P. D. (1965). Pain mechanisms: A new theory. *Science, 150*, 971–979.

Simon, L. S. (2004). Nonsteroidal anti-inflammatory drugs. In C. A. Warfield & Z. H. Bajwa (Eds.), *Principles and practice of pain medicine* (2nd ed., pp. 616–626). New York: McGraw Hill.

Turk, D. C. (2002). A cognitive-behavioral perspective on treatment of chronic pain patients. In R. J. Gatchel & D. C. Turk (Eds.), *Psychological approaches to pain management: A practitioner's handbook* (pp. 138–158). New York: Guilford.

Turk, D. C., Meichenbaum, D., & Genest, M. (1983). *Pain and behavioral medicine: A cognitive-behavioral perspective*. New York: Guilford.

Vlaeyen, J. W. S., de Jong, J., Sieben, J., & Crombez, G. (2002). Graded exposure in vivo for pain-related fear. In R. J. Gatchel & D. C. Turk (Eds.), *Psychological approaches to pain management: A practitioner's handbook* (pp. 210–233). New York: Guilford.

Pain Perception

- ▶ Gate Control Theory of Pain

Pain Sensitivity

- ▶ Gate Control Theory of Pain

Pain Threshold

Michael James Coons[1] and Jeremy Steglitz[2]
[1]Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA
[2]Department of Psychiatry and Behavioral Sciences, Clinical Psychology Division, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Definition

Pain threshold is defined as the minimum intensity of a stimulus that is perceived to be painful. Previously, this threshold was believed to be uniform across individuals, such that given intensity of a stimulus was thought to produce a given pain

response. However, it is now understood that the experience of pain is a subjective phenomenon, which is influenced by a complex interaction of biopsychosocial factors.

Historically, Specificity Theory and Pattern Theory posit that pain results from the direct transmission of peripheral stimuli to the brain, and stimulus response occurs in a reproducible relationship. However, limitations to these theories became evident after observing divergent responses to pain across individuals despite objectively similar physical stimuli or trauma. Consequently, Melzack and Wall proposed the Gate Control Theory of pain, which revolutionized our understanding of this phenomenon.

According to this theory, peripheral small diameter nerve fibers (i.e., pain receptors) and peripheral large diameter nerve fibers (i.e., normal receptors) project to the substantia gelatinosa (SG) in the dorsal horn of the spinal cord. The SG serves an inhibitory or gating function that modulates signal transduction. The SG also projects afferent fibers to the first transmission (T) cells. Activation of the T cells "activates the neural system" via the spinothalamic tract to facilitate pain perception.

In part, pain perception is determined by this bottom-up process. In the absence of sensory input, inhibitory neurons in the SG prevent projection neurons from transducing signals to the brain (i.e., maintaining a closed gate). When there is a preponderance of stimulation of pain receptors, the inhibitory neurons in the SG becomes inactivated (opening the gate), permitting the afferent projection neurons to transduce pain signals to the T cells and to the brain. However, pain perception is also influenced by top-down processes that include cognitive processes (e.g., attention), neural inhibitory capacities, affective states, and activities of the stress-regulation system. These processes transduce efferent signals to the dorsal horn in the spinal cord that further modulate the spinal gate and subsequent pain perception. Pain threshold is determined by variation in spinal gate modulation from both bottom-up and top-down processes that facilitate pain perception.

Cross-References

- ▶ Pain
- ▶ Pain Management/Control
- ▶ Pain: Psychosocial Aspects

References and Readings

Melzack, R. (1999). From the gate to the neuromatrix. *Pain* (Suppl. 6), *82*, S121-S126.

Melzack, R., & Wall, P. D. (1965). Pain mechanisms: A new theory. *Science, 150*, 971–979.

McMahon, S. B., & Koltzenburg, M. (Eds.). (2006). Melzack & Wall's Textbook of Pain. London: Elsevier.

Pain, Psychosocial Aspects

Michael James Coons
Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Synonyms

Pain anxiety; Pain management/control; Pain-related fear

Definition

Researchers and clinicians have begun to map the trajectory of pain from acute to persistent states. These outcomes are influenced by a complex interaction of biological, psychological, behavioral, and social components, which can be clustered into intrapersonal factors (factors affecting the level of the individual) and interpersonal factors (factors affecting the interaction between the individual and their environment). These factors influence both pain perception and responses to treatment.

Description

Pain is a universal phenomenon experienced by most individuals. However, pain sometimes

persists beyond a reasonable time during which tissue typically heals following injury. Several researchers have developed and articulated conceptual models to understand how and why for some individuals pain transitions from acute to persistent states.

Fear-Avoidance Models of Pain

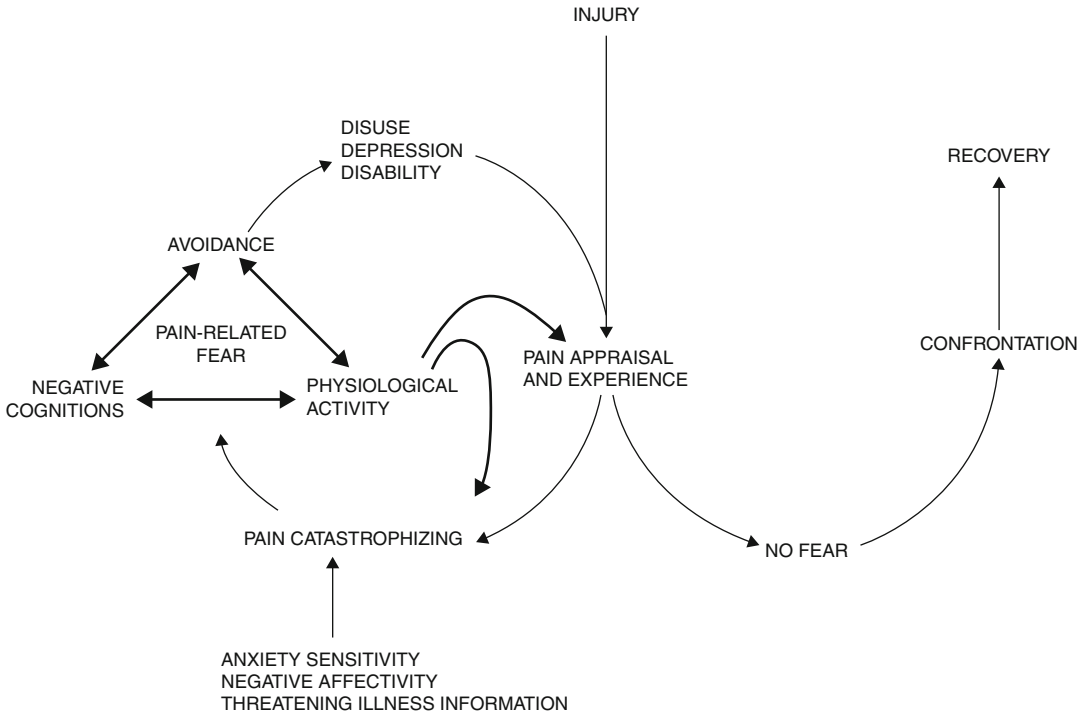
Fear-Avoidance Models posit that following the onset of pain (from injury or disease process), individuals appraise and evaluate their experience (see Fig. 1 Norton & Asmundson, 2003). For those who perceive their pain in a realistic manner, they do not experience excessive fear of pain, are able to engage in activities following a reasonable healing period, and subsequently recover from their experience. However, for others, a more complicated course of recovery ensues triggering a variety of cognitive, physiological, and behavioral symptoms. For those that progress to develop persistent pain (either recurrent intermittent pain or unremitting pain), they interpret their pain to be catastrophic in nature (e.g., "This pain is so excruciating I cannot function"). This overinterpretation triggers a state of autonomic arousal and pain-related fear. This fear is accompanied by a variety of somatic symptoms and negative cognitions about pain that motivates are variety of escape-avoidance behaviors (e.g., prolonged periods of rest, avoidance of physical activity). Over time, the anticipation of experiencing pain in the future motivates continued avoidance of activity, which in turn, leads to loss of physical strength, physical deconditioning, and muscle atrophy. These consequences result in increases in pain intensity, which continues to fuel this fear-avoidance cycle. Several individual difference variables have been identified that seem to place individuals at risk for developing persistent pain and include negative affectivity (i.e., trait experiences of negative emotions) and anxiety sensitivity (i.e., the propensity to experience fear of somatic sensations). Individuals that experience higher levels of these traits have been shown to be at greater risk for developing persistent pain conditions.

Intrapersonal Factors in Persistent Pain

Regardless of the nature of the pain experience (i.e., headache pain; orofacial pain), psychosocial factors have been shown to moderate pain intensity and progression. Specifically, depression, anxiety disorders (e.g., post-traumatic stress disorder, panic disorder, generalized anxiety disorder), substance use disorders, and somatoform pain disorders commonly co-occur with persistent pain conditions and are significantly higher than rates found in the general population. Furthermore, personality disorders also appear to occur more frequently among patients with persistent pain than among the general population; however, precise estimates in the general population are not available. These psychiatric phenomenon may share similar underlying processes to pain (that may account for their co-occurrence), are associated with more negative perceptions of pain, and influence individual's behavioral responses (e.g., are associated with greater pain-related avoidance). It has also been shown that heightened emotional reactivity, particularly when coupled with concurrent psychosocial stressors, further exacerbates negative perceptions of pain and predisposes individuals to pain-related disability. Furthermore, active coping skills (e.g., continuing to engage in activities, distraction from pain) are associated with lower pain intensity and minimize the risk of pain persistence. In contrast, passive coping skills (e.g., pain-related avoidance behavior, reliance on others) are associated with higher pain intensity and increase the risk of pain persistence.

Interpersonal Factors in Persistent Pain

Beyond the individual, several interpersonal factors are associated with pain severity, persistence, and pain-related disability. Within the context of intimate relationships, displays of pain-related behavior solicit responses from others that become reinforced over time and may perpetuate negative outcomes. For instance, an individual with persistent low back pain may display grimacing and guarding behaviors in response to pain experienced while attempting to dress them self. However, their partner may respond by providing physical assistance to help this



Pain, Psychosocial Aspects, Fig. 1 Amended fear-avoidance model of chronic pain (Copyright (2011) by the Association for Behavioral and Cognitive Therapies. Reprinted by permission of the publisher)

individual put on their clothes. Although this physical assistance helps to minimize the pain experienced, and facilitates the timely completion of this task, it reinforces the need for assistance contributing to more significant pain-related disability. Over time, such pain-related behaviors become negatively reinforced (since the assistance provided minimizes the experience of pain), which increases the likelihood of similar future behavioral responses. Furthermore, the need for assistance may undermine an individual's self-efficacy (i.e., their confidence in their ability to manage pain) and contribute to greater pain intensity, more frequent pain-related behavior, physical inactivity, and pain-related disability.

Psychosocial Factors and Treatment Response

Following the progression from acute to persistent pain, psychosocial factors have also been shown to influence responses to a variety of treatments. Across different categorical subtypes (i.e., low back pain; headache pain), the presence of axis I pathology (e.g., depression, anxiety, somatization) is associated with lower response to treatment (i.e., greater frequency and intensity of pain reports, greater perceived functional impairment), and may interfere with individual's ability to engage in the treatment process (e.g., depression interferes with treatment attendance and adherence to interventions). Furthermore, it has been shown that the continued use of maladaptive coping skills (i.e., pain-related avoidance behaviors), positive attitudes and expectations about pain and disability (i.e., pain facilitates the maintenance of supportive relationships; pain prevents the return to unsatisfactory employment), and unresolved worker's compensation/personal injury cases are further associated with lower treatment response. Therefore, a comprehensive assessment of these psychosocial factors is imperative at the outset of any pain-related intervention. This can be accomplished by obtaining a detailed medical and

psychosocial history, conducting semi-structured clinical and diagnostic interviews with both patients and their caregivers, the administration of self-report instruments, and consultation with current and past health-care providers.

Considered together, persistent pain conditions arise from a complex interaction of biological, psychological, social, and environmental factors. Understanding these relationships will help to identify factors that maintain and reinforce persistent pain across time and will provide insight into the development of effective pain management interventions.

Cross-References

- ▶ Pain Anxiety
- ▶ Pain-Related Fear

References and Readings

Asmundson, G. J. G., Norton, P. J., Norton, G. R. (1999). Beyond pain: The role of fear and avoidance in chronicity. *Clinical Psychology Review, 19*, 97–119.

Asmundson, G. J. G., Norton, P. J., & Vlaeyen, J. W. S. (2004). Fear-avoidance models of chronic pain: An overview. In G. J. G. Asmundson, J. W. S. Vlaeyen, & G. Crobez (Eds.), *Understanding and treating fear of pain* (pp. 3–24). New York: Oxford University Press.

Gatchel, R. J., & Dersh, J. (2002). Psychological disorders and chronic pain: Are there cause-and-effect relationships? In D. C. Turk & R. J. Gatchel (Eds.), *Psychological approaches to pain management: A practitioner's handbook* (2nd ed., pp. 30–51). New York: Guilford Press.

Gatchel, R. J., & Epker, J. (1999). Psychosocial predictors of chronic pain and response to treatment. In R. J. Gatchel & D. C. Turk (Eds.), *Psychosocial factors in pain: Critical perspectives* (pp. 412–434). New York: Guilford Press.

Goubert, L., Crombez, G., & Peters, M. (2004). Pain-related fear and avoidance: A conditioning perspective. In G. J. G. Asmundson, J. W. S. Vlaeyen, & G. Crobez (Eds.), *Understanding and treating fear of pain* (pp. 25–50). New York: Oxford University Press.

Keogh, E., & Asmundson, G. J. G. (2004). Negative affectivity, catastrophizing, and anxiety sensitivity. In G. J. G. Asmundson, J. W. S. Vlaeyen, & G. Crobez (Eds.), *Understanding and treating fear of pain* (pp. 91–116). New York: Oxford University Press.

Linton, S. J., & Boersma, K. (2004). The role of fear-avoidance in the early identification of patients risking the development of disability. In G. J. G. Asmundson, J. W. S. Vlaeyen, & G. Crobez (Eds.), *Understanding and treating fear of pain* (pp. 213–235). New York: Oxford University Press.

Norton, P. J., & Asmundson, G. J. G. (2003). Amending the fear-avoidance model of chronic pain: What is the role of physiological arousal? *Behavior Therapy, 34*, 17–30.

Sanders, S. H. (2002). Operant conditioning with chronic pain: Back to basics. In D. C. Turk & R. J. Gatchel (Eds.), *Psychological approaches to pain management: A practitioner's handbook* (2nd ed., pp. 128–137). New York: Guilford Press.

Turk, D. C., & Flor, H. (1999). Chronic pain: A biobehavioral perspective. In R. J. Gatchel & D. C. Turk (Eds.), *Psychosocial factors in pain: Critical perspectives* (pp. 18–34). New York: Guilford Press.

Turk, D. C., & Gatchel, R. J. (1999). Psychosocial factors and pain: Revolution and evolution. In R. J. Gatchel & D. C. Turk (Eds.), *Psychosocial factors in pain: Critical perspectives* (pp. 481–494). New York: Guilford Press.

Pain: Psychosocial Aspects

- ▶ Pain
- ▶ Pain Management/Control

Pain-Related Fear

Michael James Coons
Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Synonyms

Pain anxiety; Pain, psychosocial aspects

Definition

It is an affective manifestation of the fight-or-flight system in response to pain perception. The sympathetic nervous system becomes activated by the amygdala, resulting in cognitive, physiological, and behavioral symptoms. Following pain perception subsequent to injury or pain-inducing stimuli, pain is interpreted catastrophically by inferring more harmful or

life-threatening outcomes to its underlying cause. For instance, when an individual experiences acute pain after rolling over on their ankle, they might infer that the pain is caused by a broken bone, rather than by a sprain. This negative cognitive bias is predisposed by trait-like factors including anxiety sensitivity (i.e., fear of interoceptive experiences) and negative affectivity (i.e., the propensity toward experiencing negative emotions). This cognitive process prompts hypervigilance toward interoceptive cues (e.g., pain or muscle tension consequent to their injury) and illness information in the internal of external environment (continued or exacerbated pain). This motivates a constellation of defensive behaviors (e.g., guarding, rest) that are intended to provide relief or escape from the pain-inducing state. Fear-avoidance models of pain, and the empirical evidence supporting such models, suggest that pain-related fear is a predisposing factor to the development of persistent pain conditions.

Assessment of Pain-Related Fear

The most common mode of assessment of pain-related fear involves the administration of well-validated self-report instruments. Several published scales are available including the Fear of Pain Questionnaire-III (FPQ-III) and the Fear-Avoidance Beliefs Questionnaire (FABQ). These measures assess the nature and extent of pain-related fear by asking individuals to rate how much they fear the pain associated with a variety of situations (e.g., "having someone slam a heavy car door on your hand," "biting your tongue while eating") or the nature and extent of a variety of pain-related beliefs (e.g., "Physical activity might harm my back," "I should not do physical activities that (might) make my pain worse"). Alternative modes of assessing pain-related fear include semi-structured clinical interviews and the direct observation of patient behavior.

Treatment of Pain-Related Fear

Cognitive behavior therapy (CBT) is a mainstay in the treatment of pain-related fear. Treatment components include psychoeducation, and introduction, demonstration, and practice of a variety of adaptive coping skills (i.e., progressive muscle relaxation, diaphragmatic breathing, mental imagery, behavioral activation and pleasant activity scheduling, activity-rest cycling, physical therapy/exercise, problem-solving, cognitive restructuring, and calming self-statements). In treating pain-related fear, special emphasis is placed on graded in vivo exposure with behavioral experiments. These latter components allow individuals to construct situations in which they collect information to test and challenge their distorted pain-related cognitions.

Cross-References

▶ Pain Anxiety
▶ Pain: Psychosocial Aspects

References and Readings

Asmundson, G. J. G., Norton, P. J., & Vlaeyen, W. S. (2004). Fear-avoidance models of chronic pain: An overview. In G. J. G. Asmundson, J. W. S. Vlaeyen, & G. Crombez (Eds.), *Understanding and treating fear of pain* (pp. 3–24). New York: Oxford University Press.

de Williams, A. C., & McCracken, L. M. (2004). Cognitive behavioral therapy for chronic pain: An overview with specific reference to fear avoidance. In G. J. G. Asmundson, J. W. S. Vlaeyen, & G. Crobez (Eds.), *Understanding and treating fear of pain* (pp. 293–312). New York: Oxford University Press.

McNeil, D. W., & Vowles, K. E. (2004). Assessment of fear and anxiety associated with pain: Conceptualization, methods, and measures. In G. J. G. Asmundson, J. W. S. Vlaeyen, & G. Crombez (Eds.), *Understanding and treating fear of pain* (pp. 189–211). New York: Oxford University Press.

Palliative Care

Satoru Iwase and Chica Mori
Department Of Palliative Medicine,
The University of Tokyo Hospital, Bunkyo-ku,
Tokyo, Japan

Synonyms

Hospices; Palliative medicine; Supportive care; Terminal care

Definition

The World Health Organization (WHO) defines palliative care as "an approach that improves the quality of life of patients and their families facing the problems associated with life-threatening illness, through the prevention and relief of suffering by means of early identification and impeccable assessment and treatment of pain and other problems, physical, psychological and spiritual" (World Health Organization, 2002).

Description

The goal of palliative care is to achieve the best possible quality of life for both the patients and their families.

Palliative care:

- Is a multidisciplinary task and uses a team approach by physicians, nurses, psychologists, social workers, and other health professionals to address the needs of patients and their families
- Offers a support system to help patients and their families in the community
- Is applicable at any age and at any stage in the course of illness, in conjunction with other therapies such as chemotherapy or radiation therapy which may prolong life
- Provides relief from distressing symptoms such as pain, shortness of breath, depression, drowsiness, and nausea
- Alleviates the adverse side effects, such as relieving the nausea related to chemotherapy
- Influences positively in the course of illness (Temel et al., 2010)

Dying is a normal event; however, many people feel uncomfortable discussing their own death as well as the death of someone close. A book "Hagakure," written by a samurai warrior, who was keenly aware of the events of the day, teaches us how to cope with spiritual pain within the framework of our own insight. "There is something to be learned from a rainstorm. When meeting with a sudden shower, you try not to wet and run quickly along the road. But doing such things as passing under the eaves of houses, you still get wet. When you are resolved from the beginning, you will not be perplexed, though you still get the same soaking. This understanding extends to everything" (Yamamoto, 2005).

Perception of physical, psychological, and spiritual pain may differ in various countries with different cultures and religions; nonetheless, we all aim for the same goal in palliative care.

Cross-References

▶ Religion/Spirituality
▶ World Health Organization (WHO)

References and Readings

Temel, J. S., Greer, J. A., Muzikansky, A., Gallagher, E. R., Admane, S., Jackson, V. A., et al. (2010). Early palliative care for patients with metastatic non-small-cell lung cancer. *New England Journal of Medicine*, *363*(8), 733–742. Retrieved August 19, 2010, from www.nejm.org

World Health Organization. (2002). *WHO definition of palliative care*. Retrieved September 28, 2009, from http://www.who.int/cancer/palliative/definition/en/

Yamamoto, T. (2005). Hagakure: The book of the samurai (W. S. Wilson, Trans.). Tokyo: Kodansha International.

Palliative Medicine

▶ Palliative Care

Panic Attack

Michael James Coons
Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Synonyms

Panic disorder

Definition

Panic attacks are a state of sympathetic nervous system arousal that results in a discrete episode of intense fear or discomfort in the absence of objective danger. This fear is accompanied by a host of somatic and cognitive symptoms. Symptoms include tachycardia (i.e., racing heart), sweating, palpitations, trembling, dyspnea (i.e., shortness of breath), feelings of being smothered or feelings of choking, nausea, chest pain, abdominal distress, dizziness, light headedness, derealization or depersonalization, numbness or tingling in the face or extremities, chills or hot flushes, fear of "going crazy," fear of losing control, or fear of death from such an episode. Individuals must report experiencing at least 4 of the 13 possible somatic and cognitive symptoms. These aforementioned symptoms typically peak in intensity over a short period of time (i.e., 10 min or less). If individuals report experiencing excessive fear but manifest fewer than four symptoms, it is considered to be a limited-symptom panic attack. Panic attacks are classified into three main subtypes: cued panic attacks (i.e., panic attacks can occur in response to a specific situation or event), uncued panic attacks (i.e., panic attacks that occur "out of the blue" in the absence of a discernable trigger), or situationally predisposed panic attacks (i.e., panic attacks that occur immediately on exposure to or in anticipation of a specific situational cue or trigger). The experience of at least two *uncued* panic attacks is a prerequisite for the diagnosis of panic disorder. However, panic attacks can occur in the context of any other anxiety disorder (e.g., generalized anxiety disorder, social anxiety disorder) when cued by situational events or triggers (e.g., in response to excessive worry; during a social interaction). The possible consequences of such episodes make panic attacks of interest to behavioral medicine.

Cross-References

► Panic Disorder

References and Readings

American Psychiatric Association. (2000). *Diagnostic and statistical manual for mental disorders (Revised)* (4th ed.). Washington, DC: APA.

Antony, M. M., & Swinson, R. P. (2000). *Phobic disorder and panic in adults: A guide to assessment and treatment*. Washington, DC: American Psychological Association.

McCabe, R. (2001). Panic disorder and agoraphobia: A brief overview and guide to assessment. In M. M. Antony, S. M. Orsillo, & L. Roemer (Eds.), *Practitioner's guide to empirically based measures of anxiety* (pp. 87–94). New York: Kluwer Academic/Plenum.

Panic Disorder

Michael James Coons
Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Synonyms

Panic attack

Definition

According to the Diagnostic and Statistical Manual for Mental Disorders, Fourth Edition (DSM-IV-TR), panic disorder (PD) is an anxiety disorder that is defined by the experience of recurrent (two or more) uncured panic attacks. Following these attacks and for a period of at least 1 month, individuals must report experiencing concern about either having additional attacks, concern about the potential implications of having panic attacks (e.g., death), or significantly change their behavior because of the experience of panic attacks (e.g., avoidance of certain situations). In this context, panic attacks cannot occur in response to the physiological effects of a substance (e.g., caffeine, marijuana), cannot be due to a general medical condition, and cannot be better accounted for by another mental disorder

(e.g., generalized anxiety disorder). PD can occur in isolation, or in the presence of agoraphobia (i.e., anxiety about particular places or situations in which individuals fear experiencing a panic attack). According to the DSM-IV-TR, PD occurs in approximately 1–2% of the general population. The onset of PD typically occurs between late adolescence and mid-30s. Researchers have shown that anxiety sensitivity (i.e., an individual difference variable involving the fear of anxiety-related somatic symptoms) is a robust predictor of the development of PD. However, PD (with or without agoraphobia) is diagnosed more commonly among women than among men.

Cognitive Behavioral Models

Theoretical models of PD suggest that among individuals who are predisposed to being fearful of somatic cues (i.e., having high levels of anxiety sensitivity), they catastrophically misinterpret physiological sensations when they occur (e.g., "my heart is racing, I *must* be having a heart attack"). Such distorted cognitions motivate a series of maladaptive behaviors (e.g., escaping the current situation to prevent the catastrophic outcome, avoiding situations because of the fear of experiencing a future panic attack, not leaving the house alone in the event of a panic attack). Over time and across situations, these escape-, avoidance-, and safety-seeking behaviors become negatively reinforced, resulting in a reliance on engaging in these behaviors to either prevent a panic attack from occurring, or in minimizing the potential (perceived) negative and catastrophic outcome(s). Together, these maladaptive cognitions and behaviors contribute to greater sympathetic arousal that results in a host of somatic symptoms (e.g., racing heart, dyspnea, nausea, shaking/trembling), which perpetuates the cycle of panic.

Assessment

Prior to commencing any pharmacological or psychological intervention, it is essential to establish a differential diagnosis to determine if PD is the most appropriate diagnosis. This can be accomplished through the administration of structured clinical interviews, the completion of various self-report instruments, and if necessary, a thorough medical evaluation. The Structured Clinical Interview for DSM-IV (SCID-IV) and the Anxiety Disorders Interview Schedule IV (ADIS-IV) are the two "gold standard" clinical interviews for the anxiety disorders. These interviews are modeled after diagnostic criteria from the DSM-IV and assess the presence of all anxiety disorders, along with a variety of potentially comorbid axis I conditions (e.g., mood disorders, substance use disorders, somatoform disorders, psychotic disorders, and adjustment disorders). In conjunction with structured clinical interviews, a variety of well-validated self-report instruments are available to assess the nature, extent, and risk for PD symptoms. These include the Anxiety Sensitivity Index, Revised (ASI-R), the Panic Disorder Severity Scale (PDSS), and the Agoraphobic Cognitions Questionnaire (ACQ). If there is potential for a patient's panic symptoms to be caused by an underlying medical condition (e.g., atrial fibrillation), a thorough medical evaluation is required before a diagnosis of PD can be established.

Treatment of PD

Efficacious treatments for PD include both pharmacological and psychological interventions. Both selective serotonin reuptake inhibitors (SSRIs) and benzodiazepines are used in the treatment of PD. However, it is recommended that benzodiazepines be prescribed using a scheduled dose, as opposed to an "as needed" basis (i.e., PRN). This helps to minimize the risk of the benzodiazepine use becoming a maladaptive safety behavior that may exacerbate PD-related cognitive distortions. Cognitive behavior therapy (CBT) remains as the front-line psychological intervention for PD. Treatment components include psychoeducation (around the fight-or-flight system, CBT model of panic), identification and restructuring of cognitive distortions, and graded exposure (cognitive, in vivo, and interoceptive). Recent technological advancements permit the augmentation of exposure-based interventions with virtual reality equipment.

Cross-References

▶ Panic Attack

References and Readings

American Psychiatric Association. (2000). *Diagnostic and statistical manual for mental disorders* (Revised 4th ed.). Washington, DC: Author.
Antony, M. M., & Swinson, R. P. (2000). *Phobic disorder and panic in adults: A guide to assessment and treatment*. Washington, DC: American Psychological Association.
Taylor, S. (2000). *Understanding and treating panic disorder: Cognitive-behavioral approaches*. New York: Wiley.

Paradoxal Sleep

▶ REM Sleep

Parallel Group Design

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Independent treatments group design

Definition

A parallel group design is an experimental study design in which each subject is randomized to one of two or more distinct treatment/intervention groups. Those who are assigned to the same treatment are referred to as a treatment group.

While the treatments that these groups receive differ, all groups are treated as equally as possible in all other regards, and they complete the same procedures during the study. This parallel activity on the part of the groups of individuals is captured in the term "parallel group design."

The term controlled study is often heard in this context. One group will receive the treatment of interest and another group a control treatment, against which responses during and at the end of the treatment intervention are compared. Going one step further, the term concurrently controlled study makes clear that the different groups take part in their respective treatment arms at the same time. If all of the subjects in one treatment group completed their participation first, and then all of the other subjects completed their participation at some later time, it is quite possible that other factors could confound the results.

Cross-References

▶ Crossover Design
▶ Randomization

Parasympathetic

▶ Autonomic Balance
▶ Heart Rate Variability

Parasympathetic Nervous System (PNS)

Michael Richter[1] and Rex A. Wright[2]
[1]Department of Psychology, University of Geneva, Geneva, Switzerland
[2]College of Arts and Sciences, Department of Psychology, University of North Texas, Denton, TX, USA

Definition

The parasympathetic nervous system (PNS) is one of two main branches or subsystems of the autonomic nervous system (ANS). It originates in

the brain stem and sacral spinal cord and commonly – but not always – yields peripheral adjustments that are complementary to those produced by its counterpart, the sympathetic nervous system (SNS).

Description

The parasympathetic nervous system is one of two main branches or subsystems of the autonomic nervous system, the physical system responsible for nonconsciously maintaining bodily homeostasis and coordinating bodily responses. Working with the second main branch, the sympathetic nervous system, the parasympathetic nervous system regulates a wide range of functions such as blood circulation, body temperature, respiration, and digestion. Parasympathetic activation commonly leads to adjustments on organs and glands that are complementary to those produced by sympathetic activation and suitable for low activity and bodily restoration ("rest and digest" as opposed to "fight and flight"). Examples of low activity and restorative adjustments are constriction of blood vessels in the lungs, increased gastric secretion, and decreased heart rate and contraction force. Although parasympathetic adjustments tend to complement sympathetic adjustments, they do not always. For example, both parasympathetic nervous system arousal and sympathetic nervous system arousal increase salivary flow, although to different degrees and yielding different compositions of saliva.

Basic functional units of the parasympathetic nervous system are preganglionic and postganglionic neurons. Preganglionic neurons have cell bodies in the brainstem or sacral spinal cord and axons that extend to cell bodies of postganglionic neurons. Postganglionic neurons have cell bodies that are clustered in so-called ganglia and relatively short axons that innervate target organs and glands.

The major neurotransmitter of the parasympathetic nervous system is acetylcholine. It is the neurotransmitter of all preganglionic and postganglionic neurons. Stimulation of the cholinergic receptors of the nicotinergic subtype located on the cell bodies of the postganglionic neurons by acetylcholine leads to an opening of nonspecific ion channels. This opening permits the transfer of potassium and sodium ions, which depolarizes the postganglionic cell and initiates an action potential in the postganglionic cells. Muscarinic cholinergic receptors are located on target organs and glands. Stimulation of muscarinic receptors by acetylcholine activates G-proteins, which trigger the effector response via a second-messenger pathway. Specific effects depend on the innervated visceral structure. For instance, activation of the muscarinic receptors of the heart muscle leads to reduced heart rate and heart contraction force. Stimulation of muscarinic receptors of the salivary glands increases salivary flow.

In working jointly with the sympathetic nervous system, the parasympathetic nervous system does not function in an all-or-none fashion, but rather activates to different degrees. Depending on the affected visceral structure and situation, it may be more or less active than the sympathetic nervous system. Shifts in the magnitude of sympathetic and parasympathetic influence can occur locally within a single visceral structure (e.g., the eye) or across visceral structures, with local shifts occurring to meet highly specialized demands (e.g., a change in ambient light) and global shifts adapting the body to large-scale environmental changes (e.g., the appearance of a substantial physical threat). Autonomic control is maintained by structures in the central nervous system that receive visceral information from an afferent (incoming) nervous system. A key central nervous system structure is the hypothalamus, which integrates autonomic, somatic, and endocrine responses that accompany different organism states.

Cross-References

- ▶ Acetylcholine
- ▶ Adrenaline
- ▶ Autonomic Activation
- ▶ Autonomic Balance

- ▶ Autonomic Nervous System (ANS)
- ▶ Epinephrine
- ▶ Sympathetic Nervous System (SNS)

References and Readings

Berne, R. M., Levy, M. N., Koeppen, B. M., & Stanton, B. A. (2004). *Physiology* (5th ed.). St. Louis, MO: Mosby.

Cacioppo, J. T., & Tassinary, L. G. (1990). *Principles of psychophysiology: Physical, social, and inferential elements*. New York: Cambridge University Press.

Cacioppo, J. T., Tassinary, L. G., & Berntson, G. G. (2000). *Handbook of psychophysiology* (2nd ed.). New York: Cambridge University Press.

Ganong, W. F. (2005). *Review of medical physiology* (22nd ed.). New York: McGraw-Hill.

Levick, J. R. (2009). *An introduction to cardiovascular physiology* (5th ed.). London: Hodder.

Paraventricular Nucleus

- ▶ Hypothalamus

Parent-Child Concordance

- ▶ Family Aggregation

Parent-Rated Life Orientation Test of Children (P-LOT)

- ▶ Optimism and Pessimism: Measurement

Parietal

- ▶ Brain, Cortex

Parkinson's Disease

- ▶ Parkinson's Disease: Psychosocial Aspects

Parkinson's Disease: Psychosocial Aspects

Shawn McClintock, Matthieu Chansard and Mustafa M. Husain
Department of Psychiatry, The University of Texas Southwestern Medical Center at Dallas
Columbia University/New York State Psychiaric Institute, Dallas, TX, USA

Synonyms

Degenerative parkinsonism; Parkinsonism; Parkinson's disease; PD; Secondary parkinsonism

Definition

Parkinson's disease (PD) is the second most common neurodegenerative disorder and is characterized by motoric symptoms of resting tremor, rigidity, bradykinesia, and gait disturbance. The psychosocial aspects of PD involve the interaction of PD symptomatology, psychological development and function, personal relationships, and environmental factors.

Description

Parkinson's disease (PD) is a common neurodegenerative disorder that affects approximately between 500,000 and a million Americans of all races and ethnic groups, and 0.3% (5 million) of the world's population. Pathologically, PD is an inexorably progressive disorder of unknown cause in which neurons of the substantia nigra progressively degenerate resulting in greater degrees of brain dopamine deficiency. In addition, a number of other neuronal pathways degenerate including cholinergic, noradrenergic, and serotonergic pathways. Primary motor manifestations of PD include resting tremor, bradykinesia (e.g., slowed motor ability), rigidity, and gait disturbance. Important clinical features to establish the diagnosis of PD include

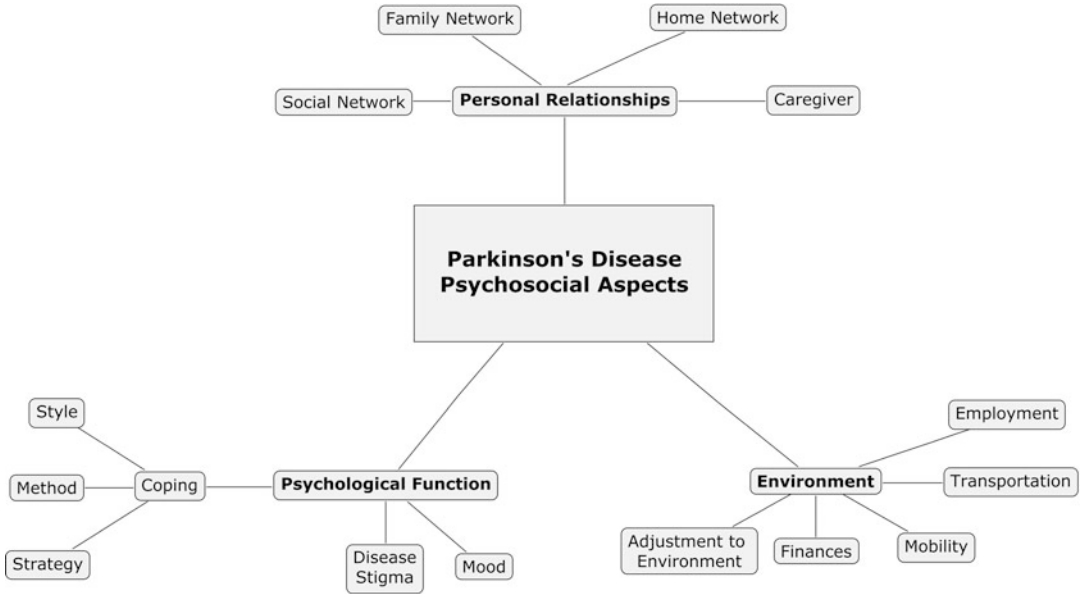
asymmetric symptom onset and responsiveness to levodopa, a commonly used medication to treat PD symptoms. Due to a combination of endogenous and exogenous factors, a significant proportion of patients with PD also suffer from comorbid medical and psychiatric illnesses. This can include pain, insomnia, autonomic dysfunction, as well as sensory and cognitive difficulties.

There has been a primary use of a biomedical approach to inform PD that has focused on the physical, neurological, and medical PD symptoms. A biopsychosocial approach may inform other domains, particularly psychosocial function. Psychosocial aspects of PD revolve around three broad domains including (1) personal relationships, (2) psychological function, and (3) environmental factors (see Fig. 1). These domains are all impacted by the progression and severity of PD symptomatology and age. As patients progress through different disease stages, psychosocial aspects will be relatively affected. There are approximately five stages of PD. In stage 1, the PD symptoms are minimal and may have some impact on activities of daily living, though by stage 2, the PD symptoms are more noticeable and begin to interfere with routine physical tasks. At stage 3, the PD symptoms may be more severe and impede most physical activities. At stages 4 and 5, the PD symptoms may be of such severity that the person is unable to live independently. Thus, greater PD disease severity is associated with greater adverse impact on psychosocial functions. For instance, late-stage PD decreases mobility and communication, thus limiting patients' ability to care for themselves and resulting in greater reliance upon others for activities of daily living. Decreased processing speed, medical problems, and other normal effects of aging exacerbated by PD factors limit self-care behaviors, which further impact psychosocial functioning.

Personal relationships are a tremendous resource for patients with PD. These relationships include many integrated networks of family and friends, and most importantly, caregivers. The size and quality of the social network as well as the subjective viewpoint of the patient all determine a social network's ability to assist patients with PD. A larger and higher quality network may be able to provide more resources than one that is small and of poor quality. The network's quality can be determined by its ability to provide resources for the PD patient in terms of physical and emotional support. Adequate relationship networks are essential to patient well-being, and patients should be encouraged to engage in activities that foster and enhance supportive relational networks. Importantly, should a professional caregiver be unavailable, family and friends may serve dual roles as caregivers, which can complicate the interpersonal relationship. The role of the caregiver may be minimal at the early stages of PD, but increases proportionately to the disease stage, as does stress and strain. Thus, it is important for caregivers to practice healthy stress management techniques.

The psychological functions domain includes mood and affect, personal view of self, and coping. There is a complex interaction between PD and psychological functions. For example, intact psychological functions before the onset of PD can help mitigate the onset of psychiatric illnesses such as depression and decreased self-worth. On the other hand, PD has been associated with an increase in depression and decrease quality of life. Regarding personal perception, patients may be burdened by disease stigma and see themselves as impaired, incapable to care for themselves, and less worthy than others. These negative personal perceptions can be changed with therapeutic management, which can then have positive impacts on overall health. Coping functions can be subdivided into coping style (active, passive), coping method (problem-solving, emotional focused), and coping strategies (cognitive, behavioral, cognitive-behavioral). Adaptive coping functions can help minimize the impact of PD symptoms, decrease poor health burden, and increase psychosocial function. Patients and caregivers should work together when implementing coping strategies in order to ensure that they are in sync and achieve maximal benefit.

The environmental domain includes areas that involve patients with PD to interact with others. This includes such areas as finances, employment and occupational performance, and



Parkinson's Disease: Psychosocial Aspects, Fig. 1 Figure 1 shows three global domains of psychosocial aspects relevant to persons with Parkinson's disease (PD) including psychological function, personal relationships, and the environment. These three domains, independently or collectively, may be impacted by the progression and severity of PD symptomatology

transportation and mobility. Physical PD symptoms (e.g., tremor, postural instability) can affect safety as well as employment and occupational performance. Patients may be unable to perform certain job duties due to PD symptoms or may be embarrassed by some symptoms such as tremors. This can impede work performance for those who are employed or limit others from seeking employment. Mobility and transportation difficulties can decrease self-reliance and increase dependency on others, which can then impact psychological functions. The adverse impact on environmental factors is related to the age of PD onset. Early PD onset tends to have a more marked adverse impact on multiple domains including employment, marital status, and quality of life. Some patients may view early PD onset as premature aging, with profound negative psychosocial consequences.

Parkinson's disease impacts not only motoric function but also psychosocial function. Given its progressive, degenerative nature, it can negatively affect psychosocial domains of personal relationships, psychological function, and environmental factors. These domains are interrelated and are further associated with PD disease severity, age, and age of illness onset. A biopsychosocial approach to therapeutic management will help to inform these domains.

Cross-References

▶ Coping
▶ Family Social Support

References and Readings

Ellgring, H., Seiler, S., Perleth, B., Frings, W., Gasster, T., & Oertel, W. (1993). Psychosocial aspects of Parkinson's disease. *Neurology, 43*(Suppl. 6), S41–S44.

Imke, S. C. (2010). *Psychosocial care for Parkinson patients and care partners*. New York: Springer.

Olanow, C. W., Stern, M. B., & Sethi, K. (2009). The scientific and clinical basis for the treatment of Parkinson disease. *Neurology, 72*(Suppl. 4), S1–S136.

Tagliati, M., Guten, G., & Horne, J. (2007). *Parkinson's disease for dummies*. Hoboken, NJ: Wiley.

Parkinsonism

► Parkinson's Disease: Psychosocial Aspects

Paroxetine

► Selective Serotonin Reuptake Inhibitors (SSRIs)

Partial Sleep Deprivation

► Sleep Restriction

Participation

► Occupational Therapy

Participation Bias

► Bias

Participation Restrictions

► Disability

Participatory Research

Sheryl Zimmerman
School of Social Work, The University of North Carolina at Chapel Hill, Chapel Hill, NC, USA

Synonyms

Community-based participatory research

Definition

Participatory research is an approach to research that emphasizes equitable involvement and shared decision making of community members, organizational representatives, and researchers in all aspects of the research process, ranging from the choice of research question through the interpretation, dissemination, and application of results (Israel, Eng, Schulz, & Parker, 2005). While there has been longstanding recognition that meaningful, ongoing collaboration between communities and researchers is essential to the design and conduct of research that will ethically address community concerns and translate research findings into sustainable public health gains, there has been a resurgence of interest in the participatory approach to health research due to the confluence of two trends. First, researchers have faced the disconcerting fact that many promising findings published in the academic literature are never translated into behavior change by the target populations and therefore do not result in health improvement. Second, potential participants have grown tired of being viewed as the "subjects" of research, and some feel that there has been little benefit to their communities in return for their participation even while they recognize the need for information-gathering. This convergence has led to a restriking of the power balance between the observers and the observed, and the promotion of participatory research.

Cross-References

► Community-Based Participatory Research

References and Readings

Israel, B. A., Eng, E., Schulz, A. J., & Parker, E. A. (Eds.). (2005). *Methods in community-based participatory research for health*. San Francisco: Josey-Bass.

Lantz, P. M., Israel, B. A., Schultz, A. J., & Reyes, A. (2005). *Community-based participatory research: Rationale and relevance for social epidemiology*. San Francisco: Josey-Bass.

Minkler, M., & Wallerstein, N. (Eds.). (2003). *Community based participatory research for health*. San Francisco: Josey-Bass.

Passive Coping Strategies

Linda Carroll
Department of Public Health Sciences, University of Alberta, Edmonton, AB, Canada

Synonyms

Avoidance; Helplessness

Definition

Coping is the set of intentional, goal-directed efforts people engage in to minimize the physical, psychological, or social harm of an event or situation (Lazarus & Folkman, 1984; Lazarus, 1999). There are many different theoretical and empirical frameworks for understanding coping and many different ways of classifying coping strategies, but one such classification is "passive coping." Passive coping refers to feeling of helplessness to deal with the stressor and relying on others to resolve the stressful event or situation (Zeidner & Endler, 1996). Those engaging in passive coping relinquish to others the control of the stressful situation and of their reaction to that situation, or allow other areas of their lives to be adversely affected by the stressful event or situation (Field, McCabe, & Schneiderman, 1985). This reliance on external resources is contrasted with "active coping," in which the individual is relying upon their own resources to cope with the stressor. Passive coping is associated with depression and poor psychological adjustment, as well as a poor outcome.

Passive coping generally involves avoidance, withdrawal, and wishful thinking. Examples of passive coping strategies are such cognitions as "it's awful and I feel that it overwhelms me," "I pray to God it won't last long," and "I know someday someone will be here to help me and it will go away for awhile." Behavioral examples of passive coping strategies include talking (complaining) to others about the situation either to ventilate feelings, get sympathy or elicit their help, withdrawing from social and other activities, or relying on medication to cope with the situation. Catastrophization (e.g., thinking "it's terrible and I think it's never going to get better") is sometimes considered a passive coping strategy.

Cross-References

- ▶ Active Coping
- ▶ Coping

References and Readings

Field, T., McCabe, P. M., & Schneiderman, N. (1985). *Stress and coping*. Hillsdale, NJ: Erlbaum.
Lazarus, R. S. (1999). *Stress and emotion: A new synthesis*. New York: Springer.
Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal and coping*. New York: Springer.
Moos, R. H. (1986). *Coping with life crises: An integrated approach*. New York: Plenum Press.
Zeidner, M., & Endler, N. S. (1996). *Handbook of coping: Theory, research, applications*. New York: Wiley.

Passive Smoking

- ▶ Secondhand Smoke

Past Smokers

- ▶ Ex-Smokers

Pastors

- ▶ Williams LifeSkills Program

Pathophysiology

Michael Witthöft
Psychologisches Institut Abteilung Klinische Psychologie und Psychotherapie, Johannes Gutenberg Universität Mainz, Mainz, Germany

Definition

Pathophysiology (consisting of the Greek origin words "pathos" = suffering; "physis" = nature, origin; and "logos" = "the study of") refers to the study of abnormal changes in body functions that are the causes, consequences, or concomitants of disease processes. Studies of pathophysiology are concerned with the investigation of *biological* processes that are directly related to disease processes of physical, mental, or psychophysiological conditions and disorders (e.g., alterations in the endocrine system, in certain neurotransmitters, or inflammatory parameters related to the activity of the immune system). Thus, pathophysiological research aims at identifying biological markers and mechanisms for predicting and explaining disease processes in terms of *etiology* and *pathogenesis*. Pathophysiology is formally considered as a subdiscipline within physiology.

Description

The fundamental aim of the domain of pathophysiology is to unravel the altered biological (i.e., physical and chemical) processes in our organism that precede, accompany, or follow certain disorders or diseases. In this regard, pathophysiological research aims at identifying factors and mechanisms that are relevant for answering questions of *why* and *how* certain disorders and diseases develop (i.e., questions about *etiology* and *pathogenesis*). Pathophysiological mechanisms of mental, physical, and psychophysiological disorders are rather complex. Yet, many pathophysiological findings and models are incomplete, preliminary, and speculative. However, research into the biological mechanisms of conditions in behavioral medicine is growing rapidly.

Methods and Designs in Pathophysiology Research

Since *pathophysiology* is mainly concerned with indentifying objective, biological factors that are relevant for certain disease processes, *quantitative methods* and *experimental* research designs are typically used. Current examples of research methods used in *pathophysiological* research in behavioral medicine are brain imaging techniques (i.e., (functional) magnetic resonance or positron emission tomography) to explore altered patterns of brain morphology and neural activity associated with certain disorders. Another example of pathophysiological research represents studies that aim at quantifying the amount of stress hormones (e.g., corticosteroids) in the blood and saliva (either in a resting state or after acute stress induction). In addition, *electroencephalography* and *electrocardiography* are typical diagnostic tools in pathophysiology research. In addition to studies with human patients and control participants, animal models (e.g., mouse and rat models) are also routinely used to test predictions of *pathophysiological* hypotheses relevant for our understanding of complex medical, psychological, and psychophysiological conditions in humans (e.g., stroke, schizophrenia).

Examples of Pathophysiological Research

As examples of *pathophysiological* investigations and findings relevant to the field or *behavioral medicine*, three examples from the realm of *obesity*, *chronic pain*, and *stress-related disorders* will be outlined briefly.

Pathophysiology of Obesity: The Metabolic Syndrome

Obesity is associated with an increased risk to develop numerous chronic and life-threatening diseases (e.g., cardiovascular diseases, type-2 diabetes, stroke). Several *pathophysiological* factors have been identified that frequently co-occur with obesity and that are suspected to mediate between *obesity* and severe medical diseases. In this regard, the following physiological

abnormalities have been identified and termed "the metabolic syndrome" (Cornier et al., 2008): abdominal obesity, insulin resistance, dyslipidemia (i.e., abnormal amount of lipids in the blood), and hypertension. Among these four defining features, abdominal obesity and insulin resistance appear as the core factors in the *pathophysiology* of the *metabolic syndrome*. The *etiology* of the *metabolic syndrome* has to be considered as complex and multifactorial and is still widely unknown. As interventions aiming at treating and preventing the *metabolic syndrome*, lifestyle modifications and weight loss (e.g., via increased physical activity) are recommended.

Pathophysiology of Chronic Pain

Pain is considered as a highly adaptive sensation that effectively signals dangers in terms of threats to our body integrity and helps to avoid injuries. *Pain* is the result of a complex interplay between neurobiological and psychological processes, both in the peripheral and central nervous system. Although acute *pain* is highly adaptive, *chronic pain* typically lacks this adaptive purpose. Regarding chronic pain conditions, the mechanism of "central sensitization" (Woolf, 2011) has been proposed to account for the phenomenon of ongoing pain in the absence of sufficient "objective" *nociceptive* stimulation. Accordingly, changes in the excitability of spinal cord neurons are responsible for reductions in pain thresholds, and prolonged neuronal responses to certain stimuli explain why stimuli that are generally considered as non-noxious and non-painful become pain-eliciting stimuli in patients with *chronic pain*. The concept of *central sensitization* rests on the principle of *neural plasticity* of the nociceptive system and a cascade of molecular and biochemical processes has been observed to be involved in *central sensitization* (e.g., Latremoliere & Woolf, 2009).

Pathophysiology of Stress Related Disorders

Ongoing and severe stress represents a threat to one's mental and physical well-being. Regarding the *pathophysiology* of the stress response, a chronic state of uncontrollable, stressful life circumstances has been linked to alterations in the function of the hormonal stress system. Qualitatively different alterations are thereby observed in certain mental and psychophysiological disorders: In the *pathophysiology* of depressive disorders, the hormonal stress system (in terms o the *hypothalamic-pituitary-adrenocortical* system; *HPA*) has been observed to be hyperactive which is reflected in increased *cortisol* secretion of the adrenal glands (e.g., Müller & Holsboer, 2004). This *hypercortisolism* is the direct consequence of increased secretion of the corticotrophin releasing hormone (CRH) (from the paraventricular nucleus of the hypothalamus) and the release of the adrenocorticotropic hormone (ACTH) via stimulation of the anterior pituitary gland (Ehlert, Gaab, & Heinrichs, 2001). The *hypercortisolism* observed in depression is most likely attributable to dysfunctions in HPA-axis feedback mechanisms that are responsible for the downregulation of the cortisol release (via CRH and ACTH). It has to be acknowledged that the phenomenon of *hypercortisolism* only occurs in certain subtypes of depression (e.g., melancholic depression).

Interestingly, the opposite phenomenon of a reduced activity of the *HPA* system resulting in a state of *hypocortisolism* is observed in patients with atypical depression, and patients with complex stress-related disorders. Regarding the latter group of disorders, reduced levels of cortisol indicative of lower HPA-axis reactivity have been observed in patients with *posttraumatic stress disorder*, *chronic fatigue syndrome*, *chronic pain disorders*, *fibromyalgia*, *irritable bowel syndrome* and other *functional somatic syndromes* and *somatoform disorders*. Childhood traumas have also been linked to *hypocortisolism* as evidence for early life stress-induced dysregulation of the *HPA* axis (Heim et al., 2009). Moreover, *hypocortisolism* was detected in people with rheumatoid arthritis and asthma.

Cross-References

- ▶ Functional Magnetic Resonance Imaging (FMRI)
- ▶ Functional Somatic Syndromes
- ▶ Homeostasis

- ▶ Inflammation
- ▶ Psychopathology
- ▶ Somatoform Disorders

References and Readings

Cornier, M.-A., Dabelea, D., Hernandez, T. L., Lindstrom, R. C., Steig, A. J., Stob, N. R., et al. (2008). The metabolic syndrome. *Endocrine Reviews, 29*, 777–822.

Ehlert, U., Gaab, J., & Heinrichs, M. (2001). Psychoneuroendocrinological contributions to the etiology of depression, post-traumatic stress disorder, and stress related bodily disorders: The role of the hypothalamus-pituitary-adrenal axis. *Biological Psychology, 57*, 141–152.

Heim, C., Ehlert, U., & Hellhammer, D. H. (2000). The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders. *Psychoneuroendocrinology, 25*, 1–35.

Heim, C., Nater, U., Maloney, E., Boneva, R., Jones, J. F., & Reeves, W. C. (2009). Childhood trauma and risk for chronic fatigue syndrome. *Archives of General Psychiatry, 66*, 72–80.

Latremoliere, A., & Woolf, C. J. (2009). Central sensitization: A generator of pain hypersensitivity by central neural plasticity. *The Journal of Pain, 10*, 895–926.

Lautenbacher, S., & Fillingim, R. B. (2004). *Pathophysiology of pain perception*. New York, NY: Kluwer Academic/Plenum.

Müller, M., & Holsboer, F. (2004). Hormones, stress and depression. In C. Kordon, R.-C. Gaillard, & Y. Christen (Eds.), *Hormones and the brain*. Berlin, NY: Springer.

Steptoe, A. (2010). *Handbook of behavioral medicine – methods and application*. New York: Springer.

Woolf, C. J. (2011). Central sensitization: Implications for the diagnosis and treatment of pain. *Pain, 152*, 2–15.

Patient Adherence

- ▶ Adherence

Patient Care

- ▶ Patient-Centered Care

Patient Compliance

- ▶ Adherence

Patient Control

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

This is a central issue in behavior medicine, since it relates to models of stress, to patient behaviors and outcomes, and has vast clinical implications. Patient control (PC) can reflect both subjective or perceived control, as well as objective control. The perceived control can be understood as one's subjective appraisal of the ability to influence outcomes in a situation. Perceived control reflects a secondary appraisal process in general stress models (Lazarus & Folkman, 1984; Taylor, 1995). In contrast, objective control reflects the externally determined and externally validated level of control over a situation. Thus, objective PC is accurate, while subjective PC refers to subjective levels of control, and thus, could also be inaccurate. Subjective PC is a crucial predictor of health behaviors in the theory of planned behavior, showing a relation to behavior either directly or via intentions. For example, subjective PC has been shown to be important in choice over food types (Lawrence & Barker, 2009), of relevance to overweight. Subjective PC is strongly related to the broader concept of self-efficacy, the belief that one can carry out a certain behavior despite the existence of barriers. Subjective PC could be affected by objective control, but also by past experiences with similar or different stressful situations. Overgeneralizing lack of control from an uncontrollable to a controllable situation reflects the core of "learned helplessness," which has vast implications for multiple outcomes including depression (Abramson et al., 1989) and possibly even acceleration of tumors (Palermo-Neto, de Oliveira Massoco, & Robespierre de Souza, 2003). In pain patients, subjective PC is positively correlated with engagement in activity, of clinical relevance to daily functioning (Chiros & O'Brien, 2011). Subjective PC can

also be an important moderating variable in the detrimental effect of various factors on health or well-being. For example, Tovbin, Jean, Schnieder, Granovsky, and Gidron (2003) found that low albumin was related to poorer quality of life in dialysis patients, but only in those with low, but not high, subjective control. Objective PC is important in "patient-controlled analgesia" (PCA), where patients control the amount and timing of receiving analgesics during treatment for pain. A meta-analysis of 55 studies found that PCA led to less pain and greater patient satisfaction than did conventional analgesic regiments given by a health professional (Hudcova, McNicol, Quah, Lau, & Carr, 2006). Importantly, the subjective and objective aspects of PC are interrelated: In patients receiving PCA, those with an external locus of control had more pain and less satisfaction with this treatment, while an internal locus of control (which also reflects greater perceived control) was predictive of lower pain and greater satisfaction from PCA (Johnson, Magnani, Chan, & Ferrante 1989). Taken together, PC is an important predictor of patient outcomes, and can be a moderator of the effects of other factors on health outcomes. It is thus of great importance to assess and consider PC in behavior medicine research and clinical practice.

Cross-References

- ▶ Perceived Control
- ▶ Theory of Planned Behavior

References and Readings

Abramson, L. Y., Metalsky, G. I., & Alloy, L. B. (1989). Hopelessness depression: A theory-based subtype of depression. *Psychological Review*, *96*, 358–372.

Chiros, C., & O'Brien, W. H. (2011). Acceptance, appraisals, and coping in relation to migraine headache: An evaluation of interrelationships using daily diary methods. *Journal of Behavioral Medicine, 34*, 307–20.

Hudcova, J., McNicol, E., Quah, C., Lau, J., & Carr, D. B. (2006). Patient controlled opioid analgesia versus conventional opioid analgesia for postoperative pain. *Cochrane Database of Systematic Reviews, 18*, CD003348.

Johnson, L. R., Magnani, B., Chan, V., & Ferrante, F. M. (1989). Modifiers of patient-controlled analgesia efficacy. I. Locus of control. *Pain, 39*, 17–22.

Lawrence, W., & Barker, M. (2009). A review of factors affecting the food choices of disadvantaged women. *Proceedings of the Nutrition Society, 68*, 189–194.

Lazarus, R. S., & Folkman, S. (1984). *Stress, Appraisal, and Coping*. New York: Springer.

Palermo-Neto, J., de Oliveira, M. C., & Robespierre de Souza, W. (2003). Effects of physical and psychological stressors on behavior, macrophage activity, and Ehrlich tumor growth. *Brain, Behavior, and Immunity, 17*, 43–54.

Taylor, S. E. (1995). *Health psychology* (3rd ed.). New York: McGraw-Hill.

Tovbin, D., Jean, T., Schnieder, A., Granovsky, R., & Gidron, Y. (2003). Psychosocial correlates and moderators of QOL in hemodialysis. *Quality of Life Research, 12*, 709–717.

Patient Education

- ▶ Diabetes Education
- ▶ Health Education

Patient Privacy

- ▶ Confidentiality

Patient Protection

- ▶ Health Insurance Portability and Accountability Act (HIPAA)

Patient-Centered Care

Cassie Cunningham
College of Public Health, University of Iowa, Liberty, IA, USA

Definition

Patient-centered care is a term that is becoming widely used in medical practice. It is typically

described in the context of patient-practitioner communication. In contrast to provider-centered care, which places control and decision-making power almost solely in the hands of the health-care provider, is patient-centered. Patient-centered care promotes active participation on the part of the patient decisions regarding their health and health care. Moreover, patient-centered care requires practitioners to provide care concordant with the patient's values as well as account for the patient's desire for information provision and for shared decision-making responsibilities. Patient-centered care has been shown to be associated with increased patient satisfaction and adherence and may also enhance the relationship between the patient and the health-care provider.

References and Readings

Mead, N., & Bower, P. (2000). Patient-centredness: A conceptual framework and review of the empirical literature. *Social Science Medicine, 51*, 1087–1110.

Stewart, M. (2001). Towards a global definition of patient-centered care. *British Medical Journal, 233*, 444–445.

Patient-Reported Outcome

Hiroe Kikuchi
Department of Psychosomatic Research, National Institute of Mental Health, National Center of Neurology and Psychiatry, Tokyo, Japan

Definition

Patient-reported outcomes (PROs) are responses to questions or statements about their perceptions or activities, such as symptoms, capabilities, or performance of roles or responsibilities (Revicki, Hays, Cella, & Sloan, 2008). These responses are typically measured by self-completed questionnaires and combined in some way to create summary scores that can be used to measure concepts such as physical, psychological, or social functioning and well-being, or symptom burden or severity. Symptoms can be rated based on frequency, severity, duration, degree of bother, or impact on patient activities. PROs are increasingly accompanying the traditional clinical ways of measuring health and the effects of treatment on the patient, both nationally and internationally, in order to make a more comprehensive evaluation. According to this context, The Patient-Reported Outcome Information System (PROMIS®) initiative, funded by National Institutes of Health (NIH), began in 2004 with six primary research sites and a statistical coordinating center in the USA. The aims of PROMIS® is to use measurement science to create a state-of-the-art assessment system for self-reported health and to provide clinicians and researchers access to a national resource for precise and efficient measurement of patient-reported symptoms, functioning, and health-related quality of life, appropriate for patients with a wide variety of chronic disease conditions (Cella et al., 2010). In 2010, a second round of PROMIS® funding was provided by the NIH, expanding the network to 13 researchers at 12 research sites. In addition, the US Food and Drug Administration (FDA) 2006 draft guidance on "Patient-Reported Outcome Measures: Use in Medical Product Development to Support Labeling Claims" has engendered wide discussion about PRO domains that should be endpoints in clinical trials (Cleeland, Sloan, & ASCPRO Organizing Group, 2010). In the guidance, reducing the severity and impact of symptoms is considered as a natural intervention endpoint for cancer, a condition associated with considerable symptom burden. Because symptoms are best described by patients who have them, PROs as measures of treatment effectiveness or the differences among treatments provide essential information about the efficacy and toxicity of a treatment and its effects on function. The FDA guidance provides a framework for addressing such issues as clinical significance, study design, and statistical methods. However, there are some problems to be solved. In the guidance, no set of recommended approaches for assessing specific symptoms by patient report in clinical trials exists, other than for pain. Recommendations

about the best approach for evaluating responsiveness and determining minimally important differences for PRO instruments are still needed (Revicki et al., 2008). With regard to PROMIS®, there is also criticism that PROMIS® appears to ignore the International Classification of Function (ICF) sponsored by the World Health Organization (WHO). ICF certainly does not represent the gold standard, but it is a system that, through the weight of WHO endorsement, will be used for a long time to come (Boers, 2010).

Cross-References

- ▶ Cancer, Types of
- ▶ National Institutes of Health
- ▶ Pain

References and Readings

Boers, M. (2010). Standing on the promises: First wave validation reports of the patient-reported outcome measurement information system. *Journal of Clinical Epidemiology, 63*, 1167–1168.

Cella, D., Riley, W., Stone, A., Rothrock, N., Reeve, B., Yount, S., et al. (2010). Initial adult health item banks and first-wave testing of the patient-reported outcomes measurement information system network: 2005–2008. *Journal of Clinical Epidemiology, 63*, 1179–1194.

Cleeland, C. S., Sloan, J. A. & ASCPRO Organizing Group. (2010). Assessing the symptoms of cancer using patient-reported outcomes (ASCPRO): Searching for standards. *Journal of Pain and Symptom Management 39*:1077–1085.

Revicki, D., Hays, R. D., Cella, D., & Sloan, J. (2008). Recommended methods for determining responsiveness and minimally important differences for patient-reported outcomes. *Journal of Clinical Epidemiology, 61*, 102–109.

Patients

- ▶ Care Recipients

Pavlovian Conditioning

- ▶ Classical Conditioning

Paxil®

- ▶ Selective Serotonin Reuptake Inhibitors (SSRIs)

PCP

- ▶ Primary Care Physicians

PD

- ▶ Parkinson's Disease: Psychosocial Aspects

Pediatric Psychology

- ▶ Child Development

Pediatric Quality of Life Inventory (PedsQL)

Melissa A. Alderfer[1] and Meghan L. Marsac[2]
[1]Division of Oncology, The Children's Hospital of Philadelphia, Philadelphia, PA, USA
[2]The Center for Injury Prevention and Research, The Children's Hospital of Philadelphia, Philadelphia, PA, USA

Synonyms

PedsQL 4.0

Definition

The Pediatric Quality of Life Inventory or PedsQL™, now in its fourth version, is a series of assessment instruments designed to measure the health-related quality of life of children.

The authors of the measure conceptualize health-related quality of life as the physical, psychological, and social functioning of the child. The PedsQL 4.0 provides an opportunity for the assessment of both overall (generic) quality of life as well as disease-specific quality of life.

The PedsQL 4.0 Generic Core Scales are appropriate for assessing health-related quality of life in both healthy and chronically ill children. The four scales making up this generic battery include Physical Functioning (8 items), Emotional Functioning (5 items), Social Functioning (5 items), and School Functioning (5 items). From these four core scales, three standardized summary scores can be calculated: a Total Quality of Life Score, a Physical Health Summary Score (based on the physical functioning items), and a Psychosocial Health Summary Score (combining emotional, social, and school items). In addition to these generic core scales, condition-specific modules have been developed for children with arthritis, asthma, brain tumors, cancer, cardiac conditions, cerebral palsy, diabetes, end-stage renal disease, neuromuscular disorders, and rheumatological diseases.

On each of the PedsQL 4.0 scales, the respondent is asked to indicate how much of a problem each item has been in the past month with response options of: 0 = never; 1 = almost never; 2 = sometimes; 3 = often; and, 4 = almost always. Item scores are reverse coded, linearly converted to a 100-point scale, and averaged to form scale and summary scores with higher scores indicating better quality of life. Parent completed versions of the scales, reporting on their child's health-related quality of life, are available for toddlers (aged 2–4), young children (aged 5–7), children (aged 8–12), and adolescents (aged 13–18). Parallel, developmentally appropriate, child self-report versions of the scales are available for young children, children, and adolescents. Parent-report infant scales and self-report young-adult and adult scales are also available. The PedsQL 4.0 is available in many languages including Spanish, French, German, Italian, Hebrew, Portuguese, and Russian.

The psychometric properties of the PedsQL 4.0 are generally good. Adequate internal consistency has been demonstrated for the scales with most researchers reporting coefficient alphas greater than.70. As evidence of validity, scores on the scales have been shown to correlate with other measures of health-related quality of life and to functional indices of health such as the number of days the child was ill, the number of school days missed by the child, the number of work days missed by the parent, and objective measures of disease severity. The generic core scales have also been found to distinguish between children with chronic health conditions and those who are healthy.

Further information regarding the PedsQL™ 4.0 including access to the measures can be obtained at www.pedsql.org.

Cross-References

▶ Quality of Life
▶ Quality of Life: Measurement

References and Readings

Palermo, T. M., Long, A. C., Lewandowski, A., Drotar, D., Quittner, A. L., & Walker, L. S. (2008). Evidence-based assessment of health-related quality of life and functional impairment in pediatric psychology. *Journal of Pediatric Psychology, 33*, 983–996.

Varni, J. W., Limbers, C. A., & Burwinkle, T. M. (2007). Impaired health-related quality of life in children and adolescents with chronic conditions: a comparative analysis of 10 disease clusters and 33 disease categories/severities utilizing the PedsQL 4.0 Generic Core Scales. *Health and quality of life outcomes, 5*, 43. http://www.hqlo.com/content/5/1/43

Varni, J. W., Seid, M., & Kurtin, P. S. (2001). PedsQL™4.0: Reliability and validity of the Pediatric Quality of Life Inventory™ Version 4.0 generic core scales in healthy and patient populations. *Medical Care, 39*, 800–812.

PedsQL 4.0

▶ Pediatric Quality of Life Inventory (PedsQL)

Peer Coaches

► Promotoras

Peer Health Educators

► Promotoras

Peer Health Promoters

► Promotoras

Penetrance

Rong Jiang
Department of Psychiatry and Behavioral Sciences, Duke University, Durham, NC, USA

Definition

Penetrance in genetics is the proportion of individuals carrying a particular gene variant (allele or genotype) that also possess an associated trait (phenotype). If penetrance of a disease allele is 100%, then all individuals carrying that allele will have the associated disease. Penetrance only considers whether individuals express the trait or not. This differs from expressivity, which characterizes qualitatively or quantitatively the extent of phenotypic variation given a particular genotype. Penetrance is age related (Bessett et al., 1998) and is affected by environmental and behavioral factors such as diet and smoking. It is also modified by other genes and epigenetic regulation.

Cross-References

► Allele
► Epigenetics
► Genotype
► Phenotype

References and Readings

Bessett, J. H., Forbes, S. A., Pannett, A. A. J., Lloyd, S. E., Christie, P. T., Wooding, C., et al. (1998). Characterization of mutations in patients with multiple endocrine neoplasia type 1. *American Journal of Human Genetics, 62*(2), 232–244.

Pepper

► Capsaicin

Peptic Ulcer

► Gastric Ulcers and Stress

Peptide

► Leptin

Perceived Behavioral Control

► Perceived Control

Perceived Benefits

Yvonne Leung
Department of Psychosocial Oncology and Palliative Care, Princess Margaret Hospital, University Health Network/ University of Toronto, Toronto, ON, Canada

Synonyms

Benefit finding; Flourishing; Positive by-products; Positive changes; Positive meaning; Posttraumatic growth; Stress-related growth; Thriving

Definition

Perceived benefit refers to the perception of the positive consequences that are caused by a specific action. In behavioral medicine, the term perceived benefit is frequently used to explain an individual's motives of performing a behavior and adopting an intervention or treatment. Researchers and theorists attempt to measure positive perceptions because they believe that a behavior is driven by an individual's cognition in terms of acceptability, motives, and attitudes toward such behavior, especially if positive.

In psychology, five models may explain the performance of health behavior related to the construct of perceived benefit. First, the Health Belief Model (Becker, 1974) describes that the perceived benefit is one of the four major predictors of health-related behavior. Second, the Transtheoretical Model (Prochaska & DiClemente, 1986) posits that the progress of change depends upon the decisional balance weighting between perceived benefits and barriers. Third, the Protection Motivation Theory (Rogers, 1983) puts forward that the intention to protect oneself depends upon four cognitions among which is the perceived efficacy (inlcuding benefits) of the recommended preventive behavior. Finally, the Theory of Reasoned Action (Fishbein & Ajzen, 1975) and its extension the Theory of Planned Behavior (Ajzen, 1985) both suggest that a person's behavior is driven by the persons' attitude about the behavior, which consists of beliefs about the consequences of performing the behavior multiplied by his or her valuation of these consequences.

In trauma literature, perceived benefits essentially mean the perceptions of positive psychological changes as a result of coping with a trauma or a highly stressful event (McMillen & Fisher, 1998). For example, after struggling with a highly stressful event, an individual may experience increases in personal strength, relatedness to others, and one's appreciation of life. In particular, McMiller and Fisher have developed the Perceived Benefit Scales (PBS) to assess several commonly reported positive by-products of adversity. On the PBS, respondents rate 30 positive by-product items on how similar they were to their own experiences by using a 5-point scale. The PBS has eight subscales: increased self-efficacy, increased faith in people, increased compassion, increased spirituality, increased community closeness, increased family closeness, lifestyle changes, and material gain. This concept is a form of cognitive coping strategy often associated with improved outcomes including participation in cancer screening tests.

Cross-References

- Benefit Finding
- Coping Strategies
- Positive Psychology
- Posttraumatic Growth

References and Readings

Ajzen, I. (1985). From intentions to actions: A theory of planned behavior. In J. Kuhl & J. Beckmann (Eds.), *Action control: From cognition to behavior*. Berlin/Heidelberg/New York: Springer.

Ajzen, I., & Fishbein, M. (1980). *Understanding attitudes and predicting social behavior*. Englewood Cliffs: Prentice-Hall.

Becker, M. H. (Eds). (1974). The health belief model and personal health behavior. *Health Education Monographs*, *2*, 324–473.

Fishbein, M., & Ajzen, I. (1975). *Belief, attitude, intention, and behavior: An introduction to theory and research*. Reading: Addison-Wesley.

McMillen, J. C., & Fisher, R. H. (1998). The perceived benefit scales: Measuring perceived positive life changes after negative events. *Social Work Research, 22*, 173–186.

Prochaska, J. O., & DiClemente, C. C. (1986). Toward a comprehensive model of change. In W. R. Miller & N. Heather (Eds.), *Treating addictive behaviors: Processes of change* (pp. 3–27). New York: Plenum Press.

Rogers, R. W. (1983). Cognitive and physiological processes in fear appeals and attitude change: A revised theory of protection motivation. In J. Cacioppo & R. Petty (Eds.), *Social psychophysiology*. New York: Guilford Press.

Perceived Control

Chris Zehr[1] and Peter A. Hall[2]
[1]Department of Health Studies and Gerontology, University of Waterloo, Waterloo, ON, Canada
[2]Faculty of Applied Health Sciences, University of Waterloo, Waterloo, ON, Canada

Synonyms

Perceived behavioral control

Definition

Perceived behavioral control is the extent to which an individual perceives that they are in control of a particular behavior (Ajzen, 2002).

Description

Perceived behavioral control (PBC) was included in the Theory of Planned Behavior (TPB; Ajzen, 1991) in order to predict/explain behaviors that are not entirely under the volitional control of the individual. According to the TPB, PBC is determined by beliefs regarding factors that may act to facilitate or inhibit successful behavioral performance (Ajzen, 1991; Conner & Armitage, 1998). For example, a belief that exercising after work is associated with many barriers (i.e., cold weather, icy sidewalks, limited schedule) may lead to low perceived behavioral control over exercise, in turn leading to less frequent exercise during winter months. However, a belief that there are few barriers to exercising (i.e., favorable weather, few other time commitments) may result in greater perceived behavioral control over exercise, which in turn may lead to more frequent exercising during summer months. Importantly, control beliefs may concern factors external to the individual (e.g., weather conditions), or internal (e.g., innate ability; Conner & Armitage, 1998).

In the context of the TPB, PBC is thought to influence behavioral performance in two ways. First, PBC affects behavioral performance indirectly by influencing behavioral intentions to perform a particular behavior (Armitage & Connor, 2001). Those who perceive that they are in greater control of a given behavior may have stronger intentions to act compared to those who perceive less control over the behavior. That is, the influence of PBC on behavior is mediated through intentions.

PBC can also affect behavioral performance directly (Armitage & Connor, 2001). Given that perceptions of behavioral control may reflect actual behavioral control, PBC may influence behavioral performance as an immediate antecedent under conditions where there is little volitional control (Ajzen, 1991). For example, if an individual does not have access to a fitness facility or equipment, low PBC for engaging in resistance training would accurately reflect the individual's actual (low) control over the behavior.

Cross-References

▶ Theory of Planned Behavior

References and Readings

Ajzen, I. (1991). The theory of planned behavior. *Organizational Behavior and Human Decision Processes, 50*(2), 179–211.

Ajzen, I. (2002). Perceived behavioral control, self-efficacy, locus of control, and the theory of planned behavior. *Journal of Applied Social Psychology, 32*(4), 665–683.

Armitage, C. J., & Connor, M. (2001). Efficacy of the theory of planned behavior: A meta-analytic review. *British Journal of Social Psychology, 40*(4), 471–499.

Conner, M., & Armitage, C. J. (1998). Extending the theory of planned behavior: A review and avenues for further research. *Journal of Applied Social Psychology, 28*(15), 1429–1464. Special issue: Expectancy-value models of attitude and behavior.

Godin, G., & Kok, G. (1996). The theory of planned behaviour: A review of its application to health-related behaviors. *American Journal of Health Promotion, 11*(2), 87–98.

Perceived Risk

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

This term refers to an individual's subjective evaluation of his or her risk of an illness or an adverse outcome, often in relation to performing a certain risky behavior. This term maps onto the Health Belief Model (Rosenstock, 1966), which tries to model why people use health services or adhere to medically advocated healthy behaviors. Perceived risk, for example, can be in relation to having a myocardial infarction due to smoking or having skin cancer due to sun exposure or having an accident due to risk taking on the road. Relevant to perceived risk, Weinstein (1982) coined the terms "unrealistic optimism" and "unrealistic pessimism," where people are asked to estimate their risk of having a disease or an adverse outcome, compared to people of their age and sex. Answers are rated on a Likert scale ranging, for example, from −5 (far below others' risk) through 0 (same as others' risk) to +5 (far above others' risk). Levels of perceived risk could be related to prior exposure to a condition, one's knowledge of such a condition, exposure to one of a condition's risk factors, and to personality aspects. For example, in a recent review of 53 studies on risk perception among people at high risk for cancer, Tilburt et al. (2011) found that family cancer history, previous tests and treatments, younger age, believing in cancer's preventability and severity, monitoring coping style, distress, and the ability to process numbers all correlated with cancer risk perceptions. Concerning prediction, many studies have shown that perceived risk is related to various behavioral and health outcomes. For example, Mann, Allegrante, Natarajan, Halm, & Charlson (2007) found that level of perceived risk was one of several predictors of adherence to prescribed statins, which are used to treat hypercholesterolemia and prevent cardiac events. Knowing one's levels of perceived risk can help predicting his or her adherence to an advocated health behavior or to stopping an unhealthy behavior. Furthermore, perceived risk, if unrealistic, can be a target of brief cognitive restructuring, in the service of healthier behaviors, disease prevention, and treatment.

References and Readings

Mann, D. M., Allegrante, J. P., Natarajan, S., Halm, E. A., & Charlson, M. (2007). Predictors of adherence to statins for primary prevention. *Cardiovascular Drugs and Therapy, 21*, 311–316.
Rosenstock, I. M. (1966). Why people use health services. *The Milbank Memorial Fund Quarterly, 44*, 94–124.
Tilburt, J. C., James, K. M., Sinicrope, P. S., Eton, D. T., Costello, B. A., Carey, J., et al. (2011). Factors influencing cancer risk perception in high risk populations: A systematic review. *Hereditary Cancer in Clinical Practice, 19*, 2.
Weinstein, N. D. (1982). Unrealistic optimism about susceptibility to health problems. *Journal of Behavioral Medicine, 5*, 441–460.

Perceived Stress

Anna C. Phillips
Sport & Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Stress

Definition

Perceived stress is the feelings or thoughts that an individual has about how much stress they are under at a given point in time or over a given time period.

Perceived stress incorporates feelings about the uncontrollability and unpredictability of one's life, how often one has to deal with irritating hassles, how much change is occurring in one's life, and confidence in one's ability to

deal with problems or difficulties. It is not measuring the types or frequencies of stressful events which have happened to a person, but rather how an individual feels about the general stressfulness of their life and their ability to handle such stress. Individuals may suffer similar negative life events but appraise the impact or severity of these to different extents as a result of factors such as personality, coping resources, and support. In this way, perceived stress reflects the interaction between an individual and their environment which they appraise as threatening or overwhelming their resources in a way which will affect their well-being (Lazarus & Folkman, 1984). Perceived stress is commonly measured as the frequency of such feelings via a questionnaire such as the Perceived Stress Scale (Cohen, Kamarck, & Mermelstein, 1983). Perceived stress measures are often used to examine relationships between stress and health within behavioral medicine research.

Cross-References

- ▶ Life Events
- ▶ Negative Thoughts
- ▶ Stress

References and Readings

Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior, 24*, 385–396.

Lazarus, R. S., & Folkman, S. (1984). *Stress, coping and adaptation*. New York: Springer.

Perceived Stress Scale (PSS)

Sherilynn F. Chan and Annette M. La Greca
Department of Psychology, University of Miami, Coral Gables, FL, USA

Definition

The Perceived Stress Scale (PSS) is a 14-item self-report measure designed to assess "the degree to which situations in one's life are appraised as stressful" (Cohen, Kamarck, & Mermelstein, 1983, p. 385). Specifically, items are designed to measure the extent to which one's life is perceived as "unpredictable, uncontrollable, and overloading" (Cohen et al., 1983, p. 387). The measure was intended for use with community samples of adolescents or adults with an educational level of junior high school or more. Sample items include the following: "In the last month. . .how often have you been upset because of something that happened unexpectedly?," ". . .how often have you felt that you were unable to control the important things in your life?," and ". . .how often have you felt confident about your ability to handle your personal problems?" (Cohen et al., 1983). Half of the questions are positively stated and reverse coded. Each item is rated on a 5-point scale (0 = Never, 1 = Almost Never, 2 = Sometimes, 3 = Fairly Often, 4 = Very Often) and summed to create a total score. Ten-item (PSS-10) and four-item (PSS-4) versions of this measure have also been developed (Cohen & Williamson, 1988; Cohen et al., 1983).

The PSS is one of the most widely used instruments used to measure stress perceptions; both the 14- and 10-item versions have good psychometric properties. In studies of adults, the PSS-14 has strong internal consistency ($\alpha = .84$ to .86) and good test-retest reliability ($r = .85$ over a 2-day period, $r = .55$ over a 6-week period; Cohen et al., 1983). In terms of concurrent validity, the PSS-14 is positively related to the number and perceived impact of life stressors ($r = .17$ to .35; Cohen et al., 1983). In terms of predictive validity, PSS scores predict depressive symptoms ($r = .65$ to .76), various health-related outcomes ($r = .52$ to .65), and social anxiety ($r = .37$ to .48) (Cohen et al., 1983). Factor analyses conducted with psychiatric inpatients revealed two factors: perceived distress and perceived coping (Hewitt, Flett, & Mosher, 1992; Martin, Kazarian, & Brieter, 1995).

Psychometric data also support the reliability and validity of the PSS-10 (Roberti, Harrington, & Storch, 2006), and an exploratory factor analysis revealed two factors: perceived helplessness and perceived self-efficacy (Roberti et al., 2006).

Overall, no significant gender differences have been found with either version of the PSS (Cohen et al., 1983; Roberti et al., 2006). The PSS-4 can be a useful measure when an abridged version is needed; however, its internal reliability ($\alpha = .72$) and test-retest reliability ($r = .55$) are lower than that for the longer versions (Cohen et al., 1983). The PSS-14 and PSS-10 have been translated into many different languages, including Hungarian, Turkish, Spanish, Portuguese, Japanese, and Thai, with good reliability and validity.

The PSS can be used to examine the role of appraised stress in physiological and behavioral disorders and can also be employed in research and clinical settings as an outcome variable (Cohen et al., 1983). Furthermore, it can be used as a screening device to identify individuals at risk for certain psychiatric disorders such as depression (Cohen et al., 1983) and may be a valuable tool for use within clinical settings to aid treatment planning and monitor treatment response (Roberti et al., 2006).

Cross-References

- ▶ Mental Stress
- ▶ Perceived Stress
- ▶ Perceptions of Stress
- ▶ Stress
- ▶ Stress Responses

References and Readings

Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior, 24*, 385–396.

Cohen, S., & Williamson, G. (1988). Perceived stress in a probability sample of the United States. In S. Spacapam & S. Oskamp (Eds.), The social psychology of health: Claremont Symposium on applied social psychology. Newbury Park, CA: Sage

Hewitt, P. L., Flett, G. L., & Mosher, S. W. (1992). The perceived stress scale: Factor structure and relation to depression symptoms in a psychiatric sample. *Journal of Psychopathology and Behavioral Assessment, 14*, 247–257.

Martin, R. A., Kazarian, S. S., & Brieter, H. J. (1995). Perceived stress, life events, dysfunctional attitudes, and depression in adolescent psychiatric inpatients. *Journal of Psychopathology and Behavioral Assessment, 17*, 81–95.

Roberti, J., Harrington, L., & Storch, E. (2006). Further psychometric support for the 10-item version of the perceived stress scale. *Journal of College Counseling, 9*(2), 135–147.

Perception of Internal Noise (False)

▶ Tinnitus and Cognitive Behavior Therapy

Perceptions of Stress

Kristen Salomon[1] and Mardís Karlsdóttir[2]
[1]Department of Psychology, University of South Florida College of Arts & Sciences, Tampa, FL, USA
[2]Department of Psychology, The University of Iceland School of Health Sciences, Reykjavík, Iceland

Synonyms

Stress appraisals

Definition

The construals or appraisals of an event that result in the experience of stress. Major theoretical definitions of stress emphasize perception as an important component responsible for the experience of stress. According to Lazarus and Folkman (1984), events are perceived as stressful if they are perceived as (1) relevant to one's well-being and (2) having the potential for harm or loss. Primary appraisals of demand, difficulty, and/or uncertainty when weighed against secondary appraisals of coping resources and abilities may result in further perceptions of stress as a challenge to be met and overcome (resources outweigh demands) or as a threat to be endured (demands outweigh resources). These resultant perceptions can

influence the psychological and physiological responses to the stressor (Tomaka, Blascovich, Kelsey, & Leitten, 1993). Perceptions of a stressful event's duration (chronic vs. acute), severity, controllability, and predictability can also influence responses to the stressor. According to Hobfoll (1989), stress results from the perceived potential loss of resources. As a result, perceptions of current resources and the potential to gain resources are involved in perceiving an event as stressful.

Cross-References

- ▶ Mental Stress
- ▶ Stress
- ▶ Stress Responses

References and Readings

Hobfoll, S. E. (1989). Conservation of resources: A new attempt at conceptualizing stress. *American Psychologist, 44*, 513–524.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal and coping*. New York: Springer.

Tomaka, J., Blascovich, J., Kelsey, R. M., & Leitten, C. L. (1993). Subjective, physiological, and behavioral effects of threat and challenge appraisals. *Journal of Personality and Social Psychology, 65*, 248–260.

Performance Anxiety

- ▶ Anxiety

Peripheral Arterial Disease (PAD)/ Vascular Disease

Leah Rosenberg
Department of Medicine, School of Medicine, Duke University, Durham, NC, USA

Synonyms

Complications of atherosclerosis; Intermittent claudication; Rest pain

Definition

Peripheral arterial disease is the mismatch of blood flow supply and demand in the distal arteries.

Description

Peripheral arterial disease (PAD) is a common manifestation of atherosclerosis and is often a complication of hypertension and/or diabetes. It is estimated that PAD affects more than eight million Americans. PAD, or the accumulation of atherosclerotic plaques leading to narrowing in noncardiac vasculature, can affect renal arteries, carotid arteries, or any other branch vessels from the aorta like the subclavian artery or iliacs. When patients with PAD become symptomatic, there is a mismatch between the metabolic supply and demand of a tissue. When an upper or lower extremity is involved, PAD may starve the affected muscle of oxygenated blood flow and causes discomfort or pain, usually exacerbated by increased activity of the affected limb. In the lower extremity, this mismatch generally presents as intermittent claudication, commonly referred to as "walking pain," where patients develop lower extremity discomfort while ambulating. While there are anatomical entrapment syndromes, deep venous thromboses, and other neurological entities that must be ruled out, PAD is a significant cause of morbidity and mortality (American Heart Association, 2011).

PAD may present before arteriosclerotic disease of the great vessels, or heart becomes clinically relevant. Signs of lower extremity PAD on physical exam include shiny, tight, and hairless skin on the lower leg. Other signs of PAD include ischemic nonhealing leg ulcers and gangrene. The limb temperature often feels cool to the touch and has decreased sensation on examination. Most patients do not perceive the rest pain of intermittent claudication. In fact, only approximately 10% of patients with measurable PAD present to physicians complaining of the typical activity-related symptoms of PAD. Early signs of PAD include erectile dysfunction, leg cramps, or muscle fatigue that exceeds the expected effects of normal exertion.

The ankle-brachial index (ABI) is an excellent screening test for PAD of the distal extremity, although direct arteriography is the gold standard. Measuring a patient's ABI is a relatively low-cost, noninvasive diagnostic test that uses the systolic blood pressure readings of the brachial, posterior tibial, and dorsalis pedis arteries. Each lower extremity is examined separately. The formula involves by dividing the maximal ankle pressure in each lower extremity by the higher of the two brachial artery pressures.

The symptoms of PAD may be modifiable through lipid management and behavioral change. While not life threatening, moderate-to-severe PAD and claudication can have a serious impact on a patient's quality of life. Treatment for PAD may be behavioral (i.e., physical activity, smoking cessation), medication-based (statins and antiplatelet agents), or interventional (angioplasty, stent implantation, or bypass surgery).

References and Readings

Statistical Fact Sheet. Peripheral Arterial Disease. American Heart Association. http://www.americanheart.org. Accessed October 9, 2011.

Perseverative Cognition

J. F. Brosschot[1], Bart Verkuil[1] and Julian F. Thayer[2]
[1]Clinical, Health and Neuro Psychology, Leiden University, Leiden, Netherlands
[2]Department of Psychology, The Ohio State University, Columbus, OH, USA

Synonyms

Intrusive thoughts; Repetitive thinking; Rumination; Worry

Definition

Perseverative cognition is defined as "the repeated or chronic activation of the cognitive representation of one or more psychological stressors" (Brosschot, Gerin, & Thayer, 2006). Stressful events, or stressors, can make people "linger on" mentally. Humans, unlike animals, can make mental representations of stressors, long before and long after these events occur or are believed to occur. This continued cognitive representation of stressful events, before or after their occurrence, and even regardless of their actual occurrence, is called perseverative cognition. It can take the form of worry, rumination, angry brooding, etc., but also, for example, as mind wandering about negative topics. Perseverative cognition appears to play a causal or sustaining role in several major psychopathologies (anxiety disorders, depression, post-traumatic disorder) (Watkins, 2008) – indeed, worry is the hallmark of general anxiety disorder – as well as in somatic disease, including subjective bodily complaints as well as cardiovascular disease (Verkuil, Brosschot, Gebhardt, & Thayer, 2010).

During perseverative cognition, physiological activity can be increased. The "perseverative cognition hypothesis" (Brosschot et al., 2006) holds that the health damage due to stress is actually caused ("mediated") by perseverative cognition, because the latter prolongs the physiological responses to stressors. It has been suggested that perseverative cognition may partly be unconscious, while it still has physiological effects (Brosschot, Verkuil, & Thayer, 2010). This possibility, or the more general possibility of unconscious stress having health impacts, has the potential to open a new area in stress research.

There are several ways to reduce perseverative cognition. The most direct way is "postponing" or "scheduling" worrying or rumination, that is, limiting it to small daily time periods to obtain control over it. More indirect interventions are mediation mindfulness and computer-based attentional training techniques (Verkuil et al., 2010).

Cross-References

▶ Intrusive Thoughts
▶ Worry

References and Readings

Brosschot, J. F., Gerin, W., & Thayer, J. F. (2006). The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *Journal of Psychosomatic Research, 60*, 113–124.

Brosschot, J. F., Verkuil, B., & Thayer, J. F. (2010). Conscious and unconscious perseverative cognition: Is a large part of prolonged physiological activity due to unconscious stress? *Journal of Psychosomatic Research, 69*(4), 407–416.

Verkuil, B., Brosschot, J. F., Gebhardt, W., & Thayer, J. F. (2010). When worries make you sick: A review of perseverative cognition, the default stress response and somatic health. *Journal of Experimental Psychopathology, 1*(1), 87–118.

Watkins, E. R. (2008). Constructive and unconstructive repetitive though. *Psychological Bulletin, 134*(2), 163–206.

Persistent Pain

► Chronic Pain Patients

Personal Growth

► Resilience

Personal Health Record

► Electronic Health Record

Personality

Matthew Cribbet and Paula Williams
Department of Psychology, University of Utah, Salt Lake City, UT, USA

Synonyms

Disposition; Individual Difference Factors; Traits

Definition

Although definitions may vary across the theoretical and methodological approaches, the term *personality* generally refers to stable patterns in how people think, act, and feel that make them unique.

Description

The study of personality has been guided by two major themes: (a) the study of individual dimensions along which people differ (i.e., nomothetic approaches) and (b) the study of individuals as unique and integrated people (i.e., idiographic approaches). Diverse theoretical and methodological approaches that have guided research in these two major domains have also contributed to controversies that have waxed and waned throughout the history of this discipline. In addition, research focused on explicating the relative role of the person versus the situation, as well as biology versus environment has been central to this area of psychology; however, in recent years, the focus on such false dichotomies has yielded to more integrated theoretical perspectives on personality. Emerging personality research ranges from the study of genetics and biological systems to the influence of culture on personality development and expression. An overview of the prominent theoretical approaches to the study of personality is provided below, including psychoanalytic, trait, social-cognitive, and interpersonal approaches.

Psychoanalytic Approaches to the Study of Personality

Although less prominent in current personality research, it is important to describe the psychoanalytic perspective on the development and manifestation of personality. Specifically, psychoanalytic approaches assert that stable personality patterns develop in childhood. According to this perspective, early childhood experiences are important, because they shape how individuals relate to others as adults. As individuals develop into mature adults, mental

processes not only guide interpersonal interactions, but may also operate in parallel so that people can feel conflicted about the same person or situation. According to this perspective and start the sentence with Personality develops as individuals learn to relate to others by regulating tension associated with sexual and aggressive drives. Finally, psychoanalytic theory emphasizes the role of mental representations of the self, others, and relationships as a framework for how people interact with one another. Psychoanalytic approaches have emphasized that behavior arises from thoughts, feelings, and motives that are outside of awareness. This reliance on unconscious mental processes separates psychoanalytic approaches from other perspectives on personality development.

Early psychoanalytic writing placed motivation at the center of theories of personality. According to this perspective, human motives could be grouped into two broad classes: life instincts (that include self-preservation and sexual motives) and death instincts or aggressive motives. This dualistic approach to personality was criticized for being overly simplistic. Subsequent research efforts expanded upon these two instincts by creating a more extensive catalog of "needs" or motives. These attempts to reformulate the psychoanalytic concept of instincts and motives stimulated a great deal of empirical research. Despite stark criticism, research on unconscious motives on adult behavior has provided the foundation for several important developments in the study of personality. For example, empirical work on unconscious motives has stimulated the fields of cognitive information processing, including research on implicit psychological mechanisms that have made the study of the unconscious more scientifically acceptable.

An important criticism of the psychoanalytic approach to personality is that the operationalization of key theoretical constructs has proven difficult. Assessments of unconscious motives and thoughts through the traditional psychoanalytic methods of free association, the unpacking of defense mechanisms, the analysis of transference processes, and dream interpretation have generally failed to hold up to minimal standards of reliability and validity necessary for empirical investigation.

Among the many attempts to operationalize psychoanalytic constructs for empirical investigation of personality, the Thematic Apperception Test (TAT) is perhaps the most well known and widely used. The popularity of the TAT as a measure of unconscious motives greatly diminished due to early criticisms of low internal consistency, temporal reliability, and a lack of correlation with self-reported measures of motives.

Trait Approaches to the Study of Personality

Trait theories of personality have typically used three different strategies to study the number, nature, and organization of dimensions along which people differ. One trait approach uses statistical techniques, such as factor analysis, to identify underlying personality dimensions applicable to all people. Another approach is to construct typologies based on a priori theories that are applicable to subgroups of people. Finally, idiographic approaches to the study of personality reject the search for basic traits common to all people, and instead focus on patterns of behavior that are unique to an individual.

The *lexical hypothesis* has been central to trait approaches to personality. The *lexical hypothesis* states that most of the descriptors that distinguish one individual from another have become embedded in our natural language. If the *lexical hypothesis* is correct, the basic dimensions of personality can be discovered, because all important individual differences will be spoken and eventually encoded into trait descriptors. Early work guided by the *lexical hypothesis* provided some initial structure, but did not provide a framework for distinguishing, naming, and ordering individual differences in behavior and experience. Early taxonomic efforts paired down initial attempts to derive personality traits from large lists of terms, but were limited by data-analytic techniques that were not sophisticated enough to handle large and complex data sets. With statistical advancements, research focused on examining the factor structure of

personality descriptors in the lexicon grew considerably. Proponents of this approach debated about the number of factors sufficient to describe personality (with 16, 5, and 3 being the predominant models) and the applicability of applying a group of factors to all people. Critics of the trait approach argued that these individual difference factors lacked consistency across situations and were poor at actually predicting specific behavior.

Following a brief period of relative dormancy in the 1970s and 1980s, due in part to the above-stated criticisms, research on the trait structure of personality increased dramatically during the mid-1980s. By the early 1990s, many personality psychologists reached a general (though still not unanimous) consensus that the trait domains could be described most broadly by five orthogonal factors, or clusters of traits. This five-factor trait model has been typically measured with self-report questionnaires and includes the following factors: neuroticism – hostility, depression, and anxiety; extraversion – warm, active, and assertive; openness to experience – open to ideas, values, and fantasy; agreeableness – modest, straightforward, and altruistic; and conscientiousness–dutiful, self-disciplined, and ordered. Yet, some psychologists have maintained that three factors, not five, characterized as neuroticism, extraversion, and psychoticism – a dimension characterized by low agreeableness and low conscientiousness – account for a majority of the variance. Despite criticism regarding the underlying assumptions of factor-analytic techniques, the five-factor model approach to the study of personality has proven to be a useful model for predicting outcomes at the individual, interpersonal, and social levels of analysis.

Beyond factor-analytic approaches, some trait theorists propose that personality is based on bundles of trait-like characteristics that can be classified as types or typologies presumed to cover all people. Recent statistical advancements (e.g., multilevel modeling techniques) have greatly increased our understanding of individual differences by allowing for the examination of the trajectory and rate at which people change. These approaches tend to focus on limited subgroups by examining combinations of personality variables as well as other variables such as intelligence, conduct, and externalizing behavior. These statistical and methodological advances have allowed researchers to examine more variables and the ability to account for interactions among those variables when describing what constitutes personality. Whereas these approaches to the study of personality are innovative, they are not without flaw. Specifically, critics have noted that this approach dissembles people into component parts that results in "types" that account for a limited amount of the variance in whatever outcome is under investigation.

Finally, the idiographic approach to the study of personality rejects the search for underlying personality traits common to all people, and instead focuses on identifying central themes in an individual's life. The idiographic approach is also concerned with describing the patterning of traits within an individual and using that pattern to predict future behavior. Research advocating for an idiographic approach claims that factor-analytic approaches might not account for the full range of personality characteristics within a person.

Social-Cognitive Approaches to the Study of Personality

Personality psychology was greatly influenced by the cognitive revolution in psychology that occurred during the late 1950s and early 1960s. This early research rejected the construct of motivation and focused on the complexity of cognitive processes. Importantly, the cognitive revolution in psychology spurred interest in the importance of "the self," leading to the examination of constructs such as self-esteem, self-schema, self-monitoring, and self-regulation. Research on the self broadened the view of personality by considering not only conceptions of what constitutes "the self," but also the ways in which our self-concept reflects our perceptions of how others view and respond to us.

More broadly, the concept of self-identity has spawned research endeavors aimed at creating a

conceptual bridge between the self and the role that social variables such as gender, race, class, and nationality play in the development of personality. Recent theoretical models of self-identity emphasize a "life story" approach to the study of personality. According to this perspective, personality is constructed by an individual through self-defining life narratives. These life narratives are created with the intent of describing individuals as integrated, whole people.

During the late 1960s and early 1970s, critiques of personality psychology produced a major crisis within the field, and led to an in-depth examination of many of the fundamental theories and methods used by personality psychologists. This perspective placed emphasis on situations, claiming that the examination of broad dispositional personality variables was unnecessarily overemphasized.

This approach advocated the notion that it is difficult to demonstrate the consistency of individual difference factors from one situation to the next and that individual difference factors rarely predict specific behaviors. This assertion not only challenged the basic premise of personality psychology, but also generated a paradigmatic crisis, resulting in an ideological split between those who study individual difference factors and those who examine the effect of situations on people. Those psychologists who advocate for a situationist approach construe personality as an organized system of goals, motives, and expectancies that mediate psychological processes that occur across situations. Proponents of this approach maintain that this characterization of personality accounts for both stability within the person as well as adaptive behavior across situations.

The response to this critique was to improve measurement techniques and to conduct studies demonstrating the consistency of personality over time. Yet, other researchers responded by examining how moderator variables, such as gender, interact with situational factors and with traditional trait descriptors. Those who emphasize the importance of context or situation maintain that dispositional traits are manifest as affective and cognitive processes that become activated during a distinct situation. Over time, these situation-by-behavior profiles are thought to shape who we are as individuals, leading to "dispositional signatures" that distinguish us as unique individuals.

Interpersonal Approaches to the Study of Personality

From an interpersonal perspective, personality is considered to be expressed in interactions with other people. Interpersonal theories of personality development do not merely emphasize observable behavior between individuals. Instead, interpersonal theories extend beyond personality development to include personality structure, function, and even pathology. According this perspective, interpersonal interactions support the development and maintenance of personality as patterns of interpersonal interactions give rise to lasting concepts of the self and others.

There have been two distinct empirical traditions to describe interpersonal functioning – the individual differences approach and the dyadic approach. The individual differences approach focuses on the qualities of an individual that are assumed to give rise to behavior that is consistent over time. This perspective led to various formulations of a structural model of interpersonal traits, actions, and problems often referred to as the interpersonal circle or circumplex. Circumplex models of behavior are used to anchor descriptions of theoretical concepts. Circumplex models of personality maintain that individual differences can be described as combinations of the circle's two underlying dimensions of dominance/submission and warmth/hostility. Interpersonal qualities close to one another on the perimeter of the circle are conceptually and statistically similar, qualities 90° apart are conceptually independent, but related, whereas qualities located 180° apart on the circle are considered conceptual and statistical opposites. This model of interpersonal functioning is not typically tied to interactions with a specific person or context, but rather is most often used to describe qualities of an individual interacting with a generalized other person.

In contrast, the dyadic approach assumes that two people comprise a basic unit of analysis for understanding personality. Accordingly, the interpersonal learning of social behaviors and self-concept is based on a variety of interpersonal situations. Interpersonal learning occurs across situations when interactions with others shape, refine, and maintain lasting conceptions of the self and others in relation to the self. In addition, this perspective on personality emphasizes that interpersonal behavior does not occur at random; instead, reciprocal relational patterns between two or more people help to define an interpersonal field. Within this interpersonal field, behavior from one individual pulls for responses from another, creating a dynamic, transactional process that leads to a conceptualization of the self. Interpersonal theories also include aspects of other theories of personality, but uniquely contribute to personality psychology by combining structural models that describe behavior with an examination of interpersonal situations.

Emerging Approaches to the Study of Personality

In recent years, the study of personality has benefited from advances in molecular genetics and functional imaging techniques. Within the domain of molecular genetics, research has focused on identifying specific biological pathways that contribute to complex cognitive and emotional behaviors. Advancements in the field of behavioral genetics may increase our understanding of the biological underpinnings of how individual differences in personality emerge and how those individual differences confer risk or resilience for mental and physical health. Other approaches that apply molecular genetic techniques to the study of personality involve examining the association between a particular phenotype and a specific allele of a gene.

Functional neuroimaging techniques hold promise for understanding personality by examining brain activity among individuals with varying levels of individual difference factors. Emerging advances in neuroimaging techniques may also help to further refine our understanding of how people process emotional information, including social connections. Yet, examining a single variation in alleles or the functional contributions of one brain region will not hold much explanatory value for our understanding of individual differences in thought, behavior, and emotions. With advancing techniques, future research should involve the careful application of methods and concepts learned from decades of investigation in order to refine our understanding of personality.

Personality and Health: Implications for Behavioral Medicine

Theory and research examining the influence of personality on health and disease has been an influential force for the development of the fields of health psychology and behavioral medicine. Research in this domain has been concerned with understanding the effects of personality on both the development and trajectory of health and disease. Possible mechanisms linking personality and health include the psychophysiological effects of stress and the extent to which personality traits are related to specific behaviors that may either promote or compromise health. Of particular interest to behavioral medicine is research that examines how certain personality characteristics confer differential risk toward negative affective states and behavioral dysregulation that often accompanies the diagnosis of and adjustment to chronic illness. Evidence that personality is a powerful predictor of health and illness has not only contributed to the development of behavioral medicine and health psychology, but has also helped revitalize personality research by challenging the critique of personality traits as having limited predictive utility.

Cross-References

- ▶ Behavioral Medicine
- ▶ Character Traits
- ▶ Health Psychology
- ▶ Phenotype
- ▶ Trait Anger
- ▶ Trait Anxiety

References and Readings

Barenbaum, N. B., & Winter, D. G. (2009). History of modern personality theory and research. In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of personality: Theory and research* (pp. 3–28). New York: Guilford Press.

McCrae, R. R., & John, O. P. (1992). An introduction to the five-factor model and its applications. *Journal of Research in Personality, 60*, 175–215.

Pincus, A. L., & Ansell, E. B. (2003). Interpersonal theory of personality. In T. Millon & M. Lerner (Eds.), *Comprehensive handbook of psychology* (Personality and social psychology, Vol. 5, pp. 209–229). New York: Wiley.

Williams, P. G., Smith, T. W., & Cribbet, M. R. (2008). Personality and health: Current evidence, potential mechanisms, and future directions. In G. J. Boyle, G. Matthews, & D. H. Saklofske (Eds.), *Personality theory and assessment* (Vol. 1, pp. 635–658). Thousands Oaks: Sage.

Personality Hardiness

▶ Hardiness and Health

Pessimism

Ryan Garcia
University of Texas Southwestern Medical Center at Dallas, Dallas, TX, USA

Synonyms

Dispositional pessimism

Definition

Pessimism is a personality variable that reflects the generalized tendency for an individual to have negative expectations about the future. Its development emerged along with that of dispositional optimism from models of self-regulation and goal achievement. Originally, pessimism was construed to reflect low levels of optimism, but it has emerged as an independent construct as the field of research has developed and grown. It is associated with a coping style characterized by problem and emotion avoidance coping (Solberg Nes & Segerstrom, 2006). Research suggests a pessimistic orientation places one at increased risk for depression and anxiety. Pessimism has also been associated with several different adverse health outcomes across a variety of settings, ranging from HIV + populations to increased mortality rates in individuals with cancer. The following terms are related to pessimism: defensive pessimism, unrealistic pessimism, and self-handicapping; however, these terms are not identical to pessimism, and have different associations with other variables.

Cross-References

▶ Attribution Theory
▶ Avoidance
▶ Coping Styles
▶ Dispositional Optimism
▶ Explanatory Style
▶ Life Orientation Test (LOT)
▶ Negative Thoughts
▶ Optimism and Pessimism: Measurement
▶ Optimism, Pessimism, and Health
▶ Self-Regulation Model

References and Readings

Abela, J. R. Z., Auerbach, R. P., & Seligman, M. E. P. (2008). Dispositional pessimism across the lifespan. In K. S. Dobson & D. J. A. Dozois (Eds.), *Risk factors in depression* (pp. 195–220). San Diego: Academic Press.

Cantor, N., & Norem, J. K. (1989). Defensive pessimism and stress and coping. *Social Cognition, 7*, 92–112.

Carver, C. S., & Scheier, M. F. (2002). Optimism, pessimism, and self-regulation. In E. C. Chang (Ed.), *Optimism & pessimism: Implications for theory, research, and practice* (pp. 31–51). Washington, DC: American Psychological Association.

Norem, J. K. (2002). Defensive pessimism, optimism, and pessimism. In E. C. Chang (Ed.), *Optimism & pessimism: Implications for theory, research, and practice* (pp. 77–100). Washington, DC: American Psychological Association.

Scheier, M. F., & Carver, C. S. (1985). Optimism, coping, and health: Assessment and implications of

generalized outcome expectancies. *Health Psychology, 4*(3), 219–247.
Scheier, M. F., & Carver, C. S. (1987). Dispositional optimism and physical well-being: The influence of generalized outcome expectancies on health. *Journal of Personality, 55*(2), 169–210.
Scheier, M. F., & Carver, C. S. (1992). Effects of optimism on psychological and physical well-being: Theoretical overview and empirical update. *Cognitive Therapy and Research, 16*(2), 201–228.
Solberg Nes, L., & Segerstrom, S. C. (2006). Dispositional optimism and coping: A meta-analytic review. *Personality and Social Psychology Review, 10*(3), 235–251.

Pew Internet and American Life Project

Chad Barrett
Department of Psychology, University of Colorado Denver, Denver, CO, USA

Definition

The Pew Internet and American Life Project is part of the Pew Research Center and is a nonpartisan and nonprofit organization that conducts research in order to provide information on the issues, attitudes, and trends that influence America and the world. According to their website, The Pew Internet and American Life Project examines "the impact of the internet on families, communities, work and home, daily life, education, health care, and civic and political life" (Pew Internet and American Life Project website). Each year, The Pew Internet and American Life Project releases 15–20 reports of research that address "how Americans use the internet and how their online activities affect their lives" (Pew Internet and American Life Project website). Data collection methods typically involve nationwide random phone surveys, online surveys, and qualitative research. In addition, data collection efforts are often augmented by research conducted by government agencies, technology firms, academia, and by various other expert researchers. Pew Internet and American Life Project aims to provide authoritative reports while maintaining neutral positions on policy issues and abstaining from providing endorsements of specific technologies, industries, organizations, companies, and individuals.

Description

The website for the Pew Internet and American Life Project provides a brief summary of highlights from their research related to health and health care (Fox, 2011). Such highlights include the following findings from recent surveys. Seventy-eight percent of adults in the USA use the internet and 83% own a cell phone. Eighty percent of internet users, or 59% of all US adults, use the internet to search for health or medical information. Seventeen percent of cell phone owners, or 15% of all US adults, have used their cell phones to look up health or medical information. Fox notes that since young people, Latinos, and African Americans are more likely than other groups to access the internet through their cell phones, this finding is of particular interest for studies targeting trends in these groups. Internet users most commonly search for health and medical information related to specific diseases or conditions, treatments or procedures, and doctors or other health professionals.

Another line of research has been examining the trends in how the internet influences people's relationships with health and medical information and with each other. As summarized by Fox (2011), many people use the internet to search for health information from other people's personal health-related experiences or to connect with others with similar conditions. Thirty-four percent of internet users, 25% of adults in the USA, have read about another person's experience with health or medical related issues by visiting an online news group, a website, or a blog. Twenty-four percent of internet users, or 18% of adults in the USA, have read online reviews of particular drugs or medical treatments. Eighteen percent of internet users, or 13% of adults in the USA, have used the internet to

connect with other people who share similar health concerns. People with rare or chronic conditions are especially likely to attempt to connect with others through the internet. Twenty-seven percent of internet users, or 20% of adults in the USA, have used online applications to monitor their weight, diet, exercise routine, or some other health indicators or symptoms. Six percent of internet users, or 4% of adults in the USA, have visited one or more websites in order to post comments or questions concerning health or medical issues. Four percent of internet users, 3% of adults in the USA, have posted comments or discussions about their experiences with a particular drug or treatment.

In addition to such highlighted findings, the website of the Pew Internet and American Life Project provides links to relevant study reports. The Pew Internet and American Life Project collects a variety of data that could be relevant to the field of behavioral medicine. The Project's website provides access to various studies and raw data sets dating back to 2000.

Cross-References

- ▶ eHealth and Behavioral Intervention Technologies
- ▶ Health Care
- ▶ Health Care Access
- ▶ Health Care Utilization
- ▶ Home Health Care
- ▶ Internet-Based Interventions
- ▶ Internet-Based Studies
- ▶ Public Health
- ▶ Social Capital and Health
- ▶ Telehealth
- ▶ Telemedicine

References and Readings

Fox, S. (2011). *Pew internet: Health*. Retrieved November 25, 2011 from http://www.pewinternet.org/Commentary/2011/November/Pew-Internet-Health.aspx

Pew Internet and American Life Project website. (n.d.). Retrieved November 25, 2011 from http://pewinternet.org/About-Us.aspx.

PGD

▶ In Vitro Fertilization, Assisted Reproductive Technology

Pharmaceutical Industry: Research and Development

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Drug development; New drug development

Definition

Contemporary research and development within the pharmaceutical industry is best described employing a lifecycle perspective. Four components of this are: drug discovery and drug design; nonclinical development; clinical development; and postmarketing surveillance (Turner, 2010).

Description

Drug Discovery and Drug Design

Drug discovery can be thought of as the work done from the time of the identification of a therapeutic need in a particular disease area to the time the drug candidate deemed most likely to safely affect the desired therapeutic benefit is identified. This drug candidate may be a small molecule or a biological macromolecule such as a protein or nucleic acid. The traditional mode of drug discovery is a long iterative process in which each molecule more closely approximates the ideal drug candidate. The modern discipline of drug design is much quicker. Computer simulation modeling

examines the "docking" of the drug molecule with the drug receptor and identifies the most likely chemical structure that will best dock with the receptor. The science of molecular engineering is then used to create the molecule suggested by the computer simulation.

Nonclinical Development

Nonhuman animal research is currently necessary before regulatory permission will be given to test a new drug in humans. Since part of the overall nonhuman animal testing is done before the drug is first given to humans, the term "preclinical" has a certain appeal and is used by many authors in place of nonclinical. However, a significant amount of nonhuman animal testing is typically conducted after the first administration of the drug to humans. Some of the more lengthy, more complex, and more expensive nonhuman animal testing is typically not started until initial human testing reveals that the drug has a good safety profile in humans and therefore has a reasonable chance of being approved for marketing if it also proves to be effective in later clinical trials. In this entry, therefore, the term "nonclinical" has been adopted for research involving nonhuman animals.

While human pharmacological therapy is the ultimate goal, understanding nonclinical drug safety and efficacy is critical to subsequent rationally designed, ethical human trials. Nonclinical research gathers critical information concerning safety, drug dose, and route and frequency of administration. It involves in vitro, ex vivo, and in vivo testing. For example, when investigating the cardiac safety of noncardiac drugs (drugs for noncardiac indications are not supposed to influence the heart's activity, and if they do it is likely to be in a deleterious manner), the following progression of levels of testing occurs: subcellular (investigation of individual ion channels within cardiac cell muscles or cardiomyocytes); cellular; isolated cardiac tissue; isolated heart; anesthetized intact animal; and conscious animal.

Preapproval Clinical Development and Postmarketing Trials and Surveillance

Pharmaceutical clinical trials are often categorized into various phases, with any given trial being identified as belonging to one of them. These categories traditionally include Phase I, Phase II, Phase III, and Phase IV, described as follows:

- Phase I. Pharmacologically oriented studies that typically look for the best dose to employ. Comparison to other treatments is not typically built into the study design.
- Phase II. Trials that look for evidence of activity, efficacy, and safety at a fixed dose. Again, comparison to other treatments is not typically built into the study design.
- Phase III. Trials in which comparison with another treatment (e.g., placebo, an active control) is a fundamental component of the design. These trials are undertaken if Phase I and Phase II studies have provided preliminary evidence that the investigational drug is safe and effective.
- Phase IV. These are postmarketing trials, conducted once the drug has been approved and in therapeutic use. There are various sorts of Phase IV trials. Some can be quite similar in design and conduct to preapproval therapeutic confirmatory trials. Other kinds include open-label trials, when both investigators and subjects know what treatment subjects are receiving, and large simple trials.

However, while commonly employed, these designations are not always used consistently. Accordingly, two studies with the same aims may be classified into different phases, and two studies classified into the same phase may have different aims. This nomenclature, therefore, can be confusing, and alternate systems of categorization are arguably more informative. One such system is presented in Table 1. The four categories correspond closely to Phase I to Phase IV, respectfully, but are more descriptive.

Among the goals of clinical development are:

- Estimation of the investigational drug's safety and tolerance in healthy adults.

Pharmaceutical Industry: Research and Development, Table 1 Classifying clinical studies according to their objectives (Based on ICH E8: General considerations for clinical trials)

Objective of trials	Study examples
Human pharmacology • Assess tolerance • Describe or define pharmacokinetics (PK) and pharmacodynamics (PD) • Explore drug metabolism and drug interactions • Estimate (biological) activity	• Dose-tolerance studies • Single- and multiple-dose PK and/or PD studies • Drug interaction studies
Therapeutic exploratory • Explore use for the targeted indication • Estimate dosage for subsequent studies • Provide basis for confirmatory study design, endpoints, methodologies	• Earliest trials of relatively short duration in well-defined narrow populations with the disease or condition of clinical concern, using surrogate of pharmacological endpoints or clinical measures • Dose-response exploration studies
Therapeutic confirmatory • Demonstrate/confirm efficacy • Establish safety profile • Provide an adequate basis for assessing benefit/risk relationship to support licensing (market approval) • Establish dose-response relationship	• Adequate and well-controlled studies to establish efficacy • Randomized parallel dose-response studies • Clinical safety studies • Studies of mortality/morbidity outcomes • Large simple trials • Comparative studies
Therapeutic use • Refine understanding of benefit-risk relationship in general or special populations and/or environments • Identify less common adverse drug reactions • Refine dosing recommendations	• Comparative effectiveness studies • Studies of mortality/morbidity outcomes • Studies of additional endpoints • Large simple trials • Pharmacoeconomic studies

- Determination of a safe and effective dose range, safe dosing levels, and the preferred route of administration.
- Investigation of pharmacokinetics and pharmacodynamics following a single dose and a multiple-dose schedule.
- Establishment and validation of biochemical markers in accessible body fluids that may permit the assessment of the desired pharmacological activity.
- Identification of metabolic pathways.
- Evaluation of the drug's safety and efficacy in a relatively small group of subjects with the disease or condition of clinical concern (the targeted therapeutic indication).
- Selection and optimization of final formulations, doses, regimens, and efficacy endpoints for larger scale, multicenter studies. Efficacy endpoints should be able to be measured reliably and should quantitatively reflect clinically relevant changes in the disease or condition of clinical concern.
- Evaluation of the drug's comparative efficacy (measured against placebo or an active comparator) in larger scale, multicenter studies, and collection of additional safety data.

Cross-References

▶ Comparative Effectiveness Research
▶ Efficacy
▶ Metabolism
▶ Placebo and Placebo Effect

References and Readings

ICH E8. (1997). *General considerations for clinical trials*. Accessed April 09, 2011, from http://www.ich.org/fileadmin/Public_Web_Site/ICH_Products/Guidelines/Efficacy/E8/Step4/E8_Guideline.pdf

Turner, J. R. (2010). *New drug development: An introduction to clinical trials*. New York: Springer.

Pharmacological Challenge Tests

▶ Pharmacological Stress Tests

Pharmacological Stress Tests

Beate Ditzen[1], Urs M. Nater[2] and Christine Heim[3]
[1]Division of Clinical Psychology and Psychotherapy, Department of Psychology, University of Zurich, Binzmuhlestrasse, Zurich, Switzerland
[2]Department of Psychology, University of Marburg, Marburg, Germany
[3]Institute of Medical Psychology, Charité University Medicine Berlin, Berlin, Germany

Synonyms

HPA axis stimulation tests; Pharmacological challenge tests

Definition

The Hypothalamic-Pituitary-Adrenal (HPA) axis is the major neuroendocrine stress system in humans. Appropriate functioning of this highly dynamic multilevel system and its feedback mechanisms are assumed to modulate psycho-physiological adaptation to all major and minor challenges in life. In line with this, profound HPA axis alterations have been found in psychiatric disorders (most prominently affective disorders), in chronic medical disorders (e.g., cardiovascular disease), and in unexplained medical symptoms (e.g., chronic fatigue, Nater et al., 2008). Furthermore, it has been suggested that restoration of glucocorticoid receptor functioning and thereby improvement of HPA axis integrity might mediate treatment outcome in some disorders (such as depression) (Holsboer, 2000).

In order to assess the functional integrity of the HPA axis and its feedback mechanisms, standard pharmacological challenge tests have been developed. Targeting different levels of the axis, these tests meet two main goals: (a) to stimulate the HPA axis in order to assess its top-down reactivity, and (b) to stimulate negative feedback mechanisms of the HPA axis in order to test its feedback sensitivity.

Description

In general, HPA axis functioning can be assessed with repeated measures of its unstimulated effector steroids, that is, corticotropin-releasing hormone (CRH), adrenocorticotropic hormone (ACTH), and cortisol.

CRH is best measured in the cerebrospinal fluid (CSF). Plasma levels do not seem to reflect hypothalamic CRH secretion because (1) in addition to the CNS, many tissues in the periphery (such as the placenta) produce CRH and (2) the relatively high concentrations of CRH from the hypothalamic-hypophyseal portal venous blood are bound by CRH-binding protein until they reach the peripheral veins (Cunnah, Jessop, Besser, & Rees, 1987).

ACTH can be assessed in blood. Because the analysis of ACTH concentrations from blood plasma is relatively expensive, the number of repeated measures is usually limited. With a short plasma half-life and episodic secretion, ACTH levels in plasma have shown wide fluctuations and should be assessed in combination with repeated cortisol measures in order to increase reliability.

Cortisol can be measured in plasma (unbound and bound cortisol fraction), in saliva (unbound cortisol fraction), and in urine (unbound fraction).

Note that single measures of CRH, ACTH, or cortisol do not give a valid picture of HPA axis integrity, and more information is obtained by repeated testing.

Besides psychological challenge tests of HPA axis activity (Dickerson & Kemeny, 2004), highly standardized pharmacological challenge tests have also been developed. Among others, the administration of Insulin, Naloxone, Fenfluramine, Alprazolam, synthetic CRH, synthetic ACTH, Metyrapone, and Dexamethasone have been used to provoke changes in HPA axis activity. In the following, the most widely used HPA axis challenge tests will be briefly characterized.

Note that all challenge tests need to be employed under standardized conditions in a clinical setting.

CRH Stimulation Test

CRH stimulates ACTH secretion. Accordingly, administration of CRH is used to assess information about HPA axis dysregulation occurring down from the level of the pituitary (ACTH and cortisol).

Following one or two blood draws in order to assess ACTH and cortisol baseline levels, an IV bolus of 1 μg/kg of body weight CRH is administered. Repeated blood sampling at min 15, 30, 60, 90, and 120 will show a rapid rise and subsequent gradual decline in ACTH and cortisol following CRH administration.

In healthy subjects, the cortisol response in plasma after 30–60 min following injection is higher than 10 μg/dL (276 nmol/L). Usually, the test is well tolerated, with transient facial flushing in about 20% of the participants, occasional shortness of breath, or rare tachycardia, and hypotension.

ACTH Stimulation Test

With the ACTH stimulation test, the acute adrenal response to ACTH can be assessed. Before and following administration of 0.25 mg synthetic human α1-24-ACTH (tetracosactrin, cosyntropin, or "Synacthen") intramuscularly or intravenously, cortisol levels are measured repeatedly (e.g., baseline, 30, 45, and 60 min after injection). Due to ACTH administration, plasma cortisol rapidly increases within 30 min to at least 18–20 μg/dL (496–552 nmol/L), with peak responses at 30–60 min. Interestingly, substantially lower doses of ACTH were associated with the same endocrine response as the above-described high dosage. Consequently, the so-called low-dose ACTH test (1 μg) is gaining greater importance in endocrine research (Dickstein et al., 1991). Possible adverse side effects of this test include bradycardia, tachycardia, hypertension, peripheral edema, and rash. These side effects should disappear within a few hours after testing.

Dexamethasone Suppression Test

The synthetic corticosteroid Dexamethasone (Dex) binds to glucocorticoid receptors and thereby mimics the effects of cortisol. Consequently, researchers employ the Dexamethasone Suppression Test (DST) in order to assess HPA axis feedback sensitivity with an expected reduction in cortisol secretion following the administration of Dex.

In the standard DST procedure, 1 mg Dex is administered orally at 11:00 p.m. The following morning (8:00 a.m.), cortisol levels are determined in blood, saliva, or urine and may be again measured at 4:00 p.m. In healthy subjects, the standard DST will minimize cortisol secretion with plasma cortisol levels less than 2 μg/dL (50 nmol/L) at both measure time points. In depressed patients, the DST is thought to show non-suppression of cortisol due to *reduced* feedback sensitivity. However, studies on this test in depression have not shown high sensitivity and specificity (APA Task Force on Laboratory Tests in Psychiatry, 1987), with only about 40–60% of patients demonstrating a failure to suppress cortisol in response to the standard DST (Yehuda, 2006). In disorders characterized by *increased* HPA axis feedback sensitivity (such as Posttraumatic Stress Disorder, PTSD), the low-dose DST (0.5 mg or 0.25 mg) is preferably used. Following this test, normal cortisol suppression results in values around 5 μg/dL. The low-dose DST has been widely used in PTSD research, with PTSD subjects having been exposed to traumas (e.g., childhood abuse, combat, or Holocaust exposure) showing hyperresponsiveness (= lower post-DST cortisol) in this test (Yehuda, 2006). No adverse side effects have been reported for the DST.

Dex-CRH Test

In order to more precisely characterize underlying mechanisms of non-suppression in the Dex test, the combined Dex-CRH test has been developed, and improved sensitivity of this test compared to the Dex test has been shown in depression (Heuser, Yassouridis, & Holsboer, 1994). With the Dex-CRH test, the oral administration of Dexamethasone (1.5 mg) at 11:00 p.m. is combined with the intravenous administration of 100 μg CRH the following day in the afternoon (between 2:00 p.m. and 3:00 p.m.). Blood

samples will be repeatedly collected before CRH administration, and again at 15, 30, 60, 90, and 120 min after administration.

In healthy individuals, ACTH and cortisol will be suppressed prior to CRH administration. After CRH injection, ACTH and cortisol levels are first increasing and then decreasing (Carroll et al., 1981). Patients suffering from current major depression, but also from other psychiatric and medical conditions, show increased sensitivity, with markedly elevated ACTH and cortisol levels following the Dex-CRH test (Ising et al., 2005). Particularly in depression, repeated Dex-CRH testing has been discussed as a surrogate marker for drug efficacy (Ising et al., 2007). Adverse side effects following the combined Dex-CRH test are identical to those following CRH administration alone.

Metyrapone Test

Metyrapone is a method for assessing ACTH secretory reserve via the interruption of the negative feedback on the HPA axis. The drug inhibits P450c11 (11β-hydroxylase), the enzyme that catalyzes the final step in cortisol biosynthesis. The inhibition of cortisol secretion interrupts negative feedback of the HPA axis, which results in a compensatory increase in ACTH. This increase in ACTH secretion then stimulates biosynthesis in the cortisol precursor steroid (11-deoxycortisol) in plasma.

The overnight metyrapone test is used in order to test whether altered cortisol secretion is a function of increased/decreased ACTH drive from the pituitary or a result of adrenal alterations. A quantity of 30 mg/kg of metyrapone is administered orally, preferably at midnight. Plasma 11-deoxycortisol is then determined the following morning at 8 a.m. In healthy subjects, 11-deoxycortisol levels in plasma rise to more than 7 μg/dL (0.2 μmol/L) and plasma ACTH levels rise to greater than 100 pg/mL (22 pmol/L) the following morning. The test has been used in PTSD research (for a review see Yehuda, 2006) as well as in depression research (Young, Ribeiro, & Ye, 2007). Possible adverse side effects include gastrointestinal symptoms, headaches, dizziness, hypotension, and allergic skin reactions.

Summary and Outlook

The investigation of neuroendocrine alterations in psychiatric and medical disorders has largely improved our knowledge of the pathophysiology of these disorders. The next steps will be to further improve the sensitivity, specificity, and thereby validity of HPA axis challenge tests. Note that so far, reliable results can only be obtained with repeated application of challenge tests and repeated HPA axis assessment. Confounding factors, such as eating disorders, restrictive dieting, diabetes, gender, and alcohol consumption have been discussed in the literature with mixed results (e.g., APA Task Force on Laboratory Tests in Psychiatry, 1987; Ising et al., 2005; Young, Ribeiro, & Ye, 2007). This is particularly relevant, as oftentimes these factors might be substantially altered in psychiatric and medical conditions.

A variety of CNS processes can trigger or dampen HPA activation. Among others, these mechanisms may include alterations in levels vasopressin, serotonin, endorphins, oxytocin, neuropeptide Y, substance P, and cytokines. So far, on the level of HPA axis dynamics alone, we are not able to trace back specifically involved CNS mechanisms; in other words, the specificity of HPA axis challenge tests cannot exceed the specificity of the HPA axis itself. Future studies should combine imaging data, administration of centrally active substances (e.g., intranasal administration of vasopressin and oxytocin), in combination with HPA axis challenge tests, thus providing further insights into the specificity of the involved mechanisms in healthy as well as in pathological endocrine functioning.

Cross-References

- ▶ Adrenal Glands
- ▶ Hypothalamic-Pituitary-Adrenal Axis
- ▶ Pituitary-Adrenal Axis

References and Readings

APA Task Force on Laboratory Tests in Psychiatry. (1987). The dexamethasone suppression test: An

overview of its current status in psychiatry. The APA task force on laboratory tests in psychiatry. *American Journal of Psychiatry, 144*(10), 1253–1262.
Carroll, B. J., Feinberg, M., Greden, J. F., Tarika, J., Albala, A. A., Haskett, R. F., et al. (1981). A specific laboratory test for the diagnosis of melancholia. Standardization, validation, and clinical utility. *Archives of General Psychiatry, 38*(1), 15–22.
Cunnah, D., Jessop, D. S., Besser, G. M., & Rees, L. H. (1987). Measurement of circulating corticotrophin-releasing factor in man. *Journal of Endocrinology, 113*(1), 123–131.
Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130*(3), 355–391.
Dickstein, G., Shechner, C., Nicholson, W. E., Rosner, I., Shen-Orr, Z., Adawi, F., et al. (1991). Adrenocorticotropin stimulation test: Effects of basal cortisol level, time of day, and suggested new sensitive low dose test. *Journal of Clinical Endocrinology and Metabolism, 72*(4), 773–778.
Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., et al. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine, 14*(4), 245–258.
Heuser, I., Yassouridis, A., & Holsboer, F. (1994). The combined dexamethasone/CRH test: A refined laboratory test for psychiatric disorders. *Journal of Psychiatric Research, 28*(4), 341–356.
Holsboer, F. (2000). The corticosteroid receptor hypothesis of depression. *Neuropsychopharmacology, 23*(5), 477–501.
Ising, M., Horstmann, S., Kloiber, S., Lucae, S., Binder, E. B., Kern, N., et al. (2007). Combined dexamethasone/corticotropin releasing hormone test predicts treatment response in major depression – a potential biomarker? *Biological Psychiatry, 62*(1), 47–54.
Ising, M., Kunzel, H. E., Binder, E. B., Nickel, T., Modell, S., & Holsboer, F. (2005). The combined dexamethasone/CRH test as a potential surrogate marker in depression. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 29*(6), 1085–1093.
Nater, U. M., Maloney, E., Boneva, R. S., Gurbaxani, B. M., Lin, J. M., Jones, J. F., et al. (2008). Attenuated morning salivary cortisol concentrations in a population-based study of persons with chronic fatigue syndrome and well controls. *Journal of Clinical Endocrinology and Metabolism, 93*(3), 703–709.
Yehuda, R. (2006). Advances in understanding neuroendocrine alterations in PTSD and their therapeutic implications. *Annals of the New York Academy of Sciences, 1071*, 137–166.
Young, E. A., Ribeiro, S. C., & Ye, W. (2007). Sex differences in ACTH pulsatility following metyrapone blockade in patients with major depression. *Psychoneuroendocrinology, 32*(5), 503–507.

Pharmacotherapy for Depression

▶ Depression: Treatment

Phasic REM

▶ REM Sleep

Phenotype

Jeanette McCarthy
Community and Family Medicine, Duke University Medical Center, Durham, NC, USA

Definition

The term phenotype refers to an organism's outward appearance and characteristics. This contrasts with the individual's genotype, the set of alleles that an offspring inherits from both parents. In the behavioral sciences, including Behavioral Medicine, the fundamental issue of heredity is the extent to which differences in genotype account for differences in phenotype, i.e., observed differences among individuals (Plomin, DeFries, McClearn, & Rutter, 1997).

In contrast to single-gene disorders such as Huntington's disease and phenylketonuria (PKU), complex dimensions, disorders, and conditions of clinical concern in Behavioral Medicine are influenced by heredity, but not by one gene alone. Multiple genes are typically involved, and so too are multiple environmental influences, and phenotypes are often the result of the combined effects of both genotype and environmental factors.

Cross-References

▶ Allele
▶ Genotype

References and Readings

Plomin, R., DeFries, J. C., McClearn, G. E., & Rutter, M. (1997). *Behavioral genetics* (3rd ed.). New York: W.H. Freeman and Company.

Physical Ability/Disability

▶ Activities of Daily Living (ADL)

Physical Activity

▶ Benefits of Exercise
▶ Exercise

Physical Activity and Cancer

▶ Cancer and Physical Activity

Physical Activity and Health

Bernardine M. Pinto[1] and Georita Marie Frierson[2]
[1]Centers for Behavioral and Preventive Medicine, Brown University, Providence, RI, USA
[2]Department of Psychology, Southern Methodist University, Dallas, TX, USA

Synonyms

Assessment; Exercise

Definition

Physical activity (PA) is any body movement that leads to skeletal muscle contraction and noticeable increases in energy expenditure (US Department of Health and Human Services [USDHHS], 2008). Such activities can be walking, washing windows, or gardening.

Description

Introduction

There is strong evidence that there are greater physical, physiological, and possibly mental health benefits from a lifestyle that includes more occupational and leisure-time physical activity (PA) than a predominantly sedentary (inactive or underactive) lifestyle (USDHHS, 2008). These benefits include a risk reduction for type 2 diabetes, overweight/obesity, cardiovascular disease, stroke, high blood pressure, an adverse lipid profile, osteoporosis, sarcopenia, and loss of function and autonomy in older age. There is also a great interest in the benefits of PA for mitigating and possibly preventing cancer and its significant morbidity and mortality rates: the evidence is strongest for the prevention of colon and breast cancer. Regular PA can also help in weight loss (when combined with reduced calorie intake) and is associated with reduced depression and better cognitive functioning (among older adults) (USDHHS).

On the negative end, there is low risk of adverse events such as injuries, when generally healthy people engaged in moderate-intensity activity. However, when performing the same activity, people who are less fit are more likely to be injured than those who are fitter. The risk of cardiac events such as heart attacks during PA is rare. But there is a risk of such events when an individual suddenly becomes much more active than usual (e.g., shoveling snow). When both the benefits and risks of PA are considered, it is clear that the health benefits of PA far outweigh the risks of adverse events for a majority of people.

Given the overwhelming evidence for substantial benefits from PA, *physical inactivity* is a national public health problem. Recent data from the 2009 National Health Interview Survey (NHIS) indicates that 35% of US adults report engaging in regular leisure-time PA, 33% reported some leisure-time PA, and 33% report no participation in leisure-time PA (http://www.cdc.gov/nchs/fastats/exercise.htm.) NHIS data for 2005–2007 showed that 30.7% of US adults engaged in PA sufficient in frequency and duration to be classified as regular and 39.7% report

no leisure-time PA (Schoenborn & Adams, 2010). Men (61.9%) were more likely than women (58.9%) to engage in at least some leisure-time activity. The percentage of adults who engage in at least some leisure PA increased with education and with family income but decreased with increasing age. Married adults were more likely than those in other marital status groups to engage in at least some leisure-time PA. White adults (61.9%) and Asian adults (60.3%) were more likely than black adults (48.8%) to engage in at least some leisure-time PA. Finally, adults living in the US South (27.4%) were least likely to engage in regular leisure-time PA compared with adults living in any other region.

What Is Physical Activity?

Body movement for the purposes of health benefits can be defined by multiple constructs. Some of these constructs are similar but are still distinct. *PA* is any body movement that leads to skeletal muscle contraction and noticeable increases in energy expenditure (USDHHS, 2008). Such activities can be walking, washing windows, or gardening. To assess how much PA someone has to engage in for a specific health outcome, the term "dose response" is used (USDHHS). Dose response refers to the frequency, intensity, duration, and type of PA needed for a certain health outcome (i.e., fitness). Typically, PA is measured in kilocalorie (kcal), metabolic equivalent (MET), minutes, or MET-minutes per day or week (USDHHS). Such measurements occur through various assessment methods that will be addressed below.

A construct that can be seen as interchangeable with PA is exercise, but these terms are not identical. *Exercise*, a subset of PA, is planned, structured, and repetitive bodily movement (such as participating in an aerobics class) with the goal of improving or maintaining physical fitness (USDHHS, 2008). *Physical fitness* primarily consists of aerobic power or cardiorespiratory fitness measured by maximal and submaximal stress testing or a field test (USDHHS). PA is something an individual does which can help to achieve greater physical fitness. In addition to PA, various factors such as age, sex, health status, and genetics can also affect physical fitness. In *aerobic activity* or endurance activity, the body's large muscles move in a rhythmic manner for a sustained period of time as in brisk walking, biking, or swimming. *Resistance training* (or strength training) consists of repetitive movements geared toward greater skeletal muscle strength, power, endurance, and mass (USDHHS). *Flexibility training* refers to repetitive activities to improve the movement of joints through their full range of motion (USDHHS).

Assessment of Physical Activity

Reliable and valid measures of PA are essential to understand energy expenditure in various populations. There are three common ways to assess PA: (1) criterion methods, (2) objective measures, and (3) subjective/self-reports. The selection of any of these three methods can be based on feasibility, cost, the specific research or clinical setting, and type of population.

Criterion methods such as doubly labeled water and indirect calorimetry are the gold standards for measuring PA (Vanhees et al., 2005). Doubly labeled water (DWL) measures total energy expenditure and does not require individuals to log their daily activity because it uses the body's water to record metabolic rates. Thus, this technique is objective and requires little participant burden. Even with these advantages, various factors that lead to changes in energy expenditure such as PA, basal metabolic rate, and diet-induced energy expenditure cannot be teased apart from each other (Vanhees et al.). Another important criterion method is indirect calorimetry. Energy expenditure is measured by oxygen consumption and carbon dioxide production through collected respiratory gases or in a respiration chamber (Vanhees et al.). Both DWL and indirect calorimetry are expensive and have limited usability. They are less feasible in studies where participants have to monitor their daily PA in the community or home setting or over multiple assessments or over extended periods.

The objective measures of PA include fitness testing, accelerometers, and pedometers. As mentioned earlier, aerobic capacity is measured through fitness testing conducted on a treadmill,

stationary bike, or a field test. These tests yield levels of peak oxygen consumption (VO_2 peak) which is an index of cardiorespiratory capacity (Gelieber et al., 1997). These treadmill or bike tests must be overseen by a physician and are expensive but are used to assess fitness in clinical and research studies.

Accelerometers and pedometers are more commonly seen in the literature because they are relatively inexpensive ($10–$450 and above) and participants can be taught how to use them without extensive training. Accelerometers and pedometers are motion sensors but with different purposes. Accelerometers measure all body movement and physical activities and can yield the dose of PA (e.g., Fogelholm et al., 1998). Thus, accelerometers are significantly more expensive than pedometers due to their use of technology (Vanhees et al., 2005).

Pedometers are battery-operated step counters that are worn on the waist and measure steps when engaging in PA (Vanhees et al., 2005). They are efficient in monitoring steps but may produce inaccurate readings due to inadvertent body movements. Activities such as biking and other activities where the body torso is stationary are not suitable for pedometers as are water activities. The reliability and validity of pedometers have been addressed in the literature (e.g., Vanhees et al.). It is anticipated that the next generation of pedometers will yield information on the "dose" of PA (i.e., intensity, frequency, and duration).

The third category of assessments is the subjective, self-report measures which include diaries, interviews, and questionnaires (e.g., von Poppel, Chinapaw, Mokkink, van Mechelen & Terwee, 2010). These subjective methods are inexpensive and generally do not take significant time. The measures are commonly used when assessing PA of large numbers of individuals. As with all self-report measures, problems with recall, over-estimation, and interviewer skill limit the validity of these instruments.

Physical Activity Guidelines

There are various PA guidelines for public health benefits, weight loss, or weight management and for patients treated for diseases such as cancer (Schmitz et al., 2010; USDHHS, 2008). For the purposes of this chapter, PA guidelines for improving general health for adults will be discussed.

In the 1960s and 1970s, the PA literature provided information that many health benefits could be achieved through vigorous intensity or high levels of PA. Since then, a large research base led to the first public health and PA guidelines issued in 1995 by the American College of Sports Medicine (ACSM) and Centers for Disease Control and Prevention (CDC) (Pate et al., 1995). These guidelines recommended that "every US adult should accumulate 30 min or more of moderate-intensity PA on most, preferably all, days of the week." The guidelines were based on evidence that moderate-intensity PA accumulated in short bouts could lead to improved health outcomes.

Due to further advances in understanding the health benefits of PA, misunderstanding of the prior 1995 ACSM and CDC PA guidelines, and continued physical inactivity of many Americans, a new set of PA guidelines were issued in 2007 by the ACSM and American Heart Association (AHA) (Haskell et al., 2007). These 2007 guidelines were tailored for children, healthy adults between the ages of 18–65, and older adults over age 65. For healthy adults, the guidelines recommend moderate-intensity aerobic PA for at least 30 min on 5 days each week or vigorous-intensity aerobic activity for at least 20 min on 3 days each week (Haskell et al.). These guidelines also clarified that (a) 30 min of aerobic or endurance activity can be achieved in at least 10-min bouts, (b) resistance training should be performed at least twice a week, and (c) individuals who wish to engage in more activity to improve health or reduce risk of disease could surpass the minimum recommendations (Haskell et al.).

In 2008, the US Department of Human and Health Services (USDHHS) released PA guidelines for health benefits and recommended a minimum of 150 min of moderate-intensity PA, 75 min of vigorous-intensity PA, or 500–1,000 MET min of PA per week. A combination of moderate- and vigorous-intensity activity

could be used to achieve these recommendations. These guidelines did not provide an empirically supported dose response prescription that included frequency of PA (i.e., number of days a week) because of insufficient evidence. On the other hand, the guidelines did clarify that the upper limit of MET values for moderate activity is 5.9 and the lower limits of vigorous activity are 6 METs. In the past, 6 METs overlapped as the highest and lowest values for moderate and vigorous PA, respectively (USDHHS, 2008).

Finally, there are other PA guidelines such as those related to the number of steps per day needed to achieve health benefits. It is commonly known that 2,000 steps equal 1 mile. Current PA guidelines suggest that accumulating at least 10,000 steps and greater per day indicates that one is active, and accumulating at least 12,500 steps per day indicates that an individual is very active (e.g., Tudor-Locke & Bassett, 2004).

Theories and Interventions

To respond to the challenge of reducing sedentary behavior, numerous interventions have been developed and tested for specific patient populations (e.g., individuals with diabetes, cardiovascular disease, osteoarthritis, cancer), individuals with specific risk factors (high cholesterol, hypertension), and the general population at various phases of the lifespan (young children, school-aged, college-level, middle-aged, and older adults). The interventions have been offered at schools, work sites, and in communities. PA has also been targeted to reduce other risk behaviors (e.g., smoking, alcohol, and other drug addiction). More recently, PA has been emphasized as part of the initiatives to combat the obesity epidemic in the USA. The interventions have yielded varying degrees of success. The Task Force on Community Preventive Services (Kahn et al., 2002) concluded that there are six types of interventions that have been shown to increase PA and cardiorespiratory fitness: point-of-decision prompts, community-wide education, school physical education and community social support, individual health behavior, and enhanced access to places for PA combined with informational outreach activities. Interventions have been offered using various modalities (in-person, by telephone, web-based, and more recently, using mobile technology such as palmtop computers and mobile phones) with varying degrees of "reach" to modify sedentary behavior.

In developing interventions, there is growing interest in *community-based participatory research* especially to reach subgroups that are more challenging to reach but are characterized by sedentary lifestyles. This type of research addresses predictors of health at the community and individual levels and includes the community of interest in the whole research enterprise. Thus, the participants or community are commonly involved with all areas of research conceptualization, development, and data collection. Furthermore, culturally relevant strategies and theories and social marketing principles are integrated a priori in the study to enhance recruitment and retention efforts. Community-based participatory research is gaining popularity given the public health epidemic of obesity and physical inactivity in diverse populations (Yancey et al., 2004).

PA interventions have been based on theories of behavior change. One of the more commonly used theories is *Social Cognitive Theory* (SCT) (Bandura, 1986) which posits that behavior, environmental factors, and personal factors of the individual, such as cognitions, emotions, and physical characteristics, are mutually influential. Interventions based on SCT focus on the importance of individuals' ability to control their behavior and how changes in the individual or the environment can produce changes in behavior. Success in being able to initiate and maintain the behavior change is determined by an individual's ability to regulate his or her own behavior through personal strategies (e.g., setting PA goals, monitoring progress toward goals), as well as environmental approaches (e.g., using social support or environmental prompts).

The *Theory of Planned Behavior* (Tpb) (Ajzen, 1991) is another widely used theory that proposes that behavior is directly predicted by

intention, which in turn, is directly predicted by attitude, subjective norm, and perceived control. Perceived control is the belief that a behavior can be performed with ease or difficulty and it may directly predict the behavior, attitude is the personal evaluation of performing the behavior, and subjective norm is the perceived normative beliefs of relevant others regarding the behavior. Thus, according to the theory, individuals will intend, and be motivated to, perform a behavior such as PA when they view it favorably, believe that important others think they should be physically active, and believe that PA is under their control and can be carried out.

The *Transtheoretical Model* (TTM) of health behavior change (Prochaska & DiClemente, 1983) postulates that individuals move through a series of six stages of motivational readiness while making a behavior change (i.e., precontemplation, contemplation, preparation, action, maintenance, and termination), and this approach has been applied to PA (Marcus & Simkin, 1993). While progressing through these stages, the individual engages in ten different cognitive and behavioral processes of change that are important in the adoption and maintenance of a new behavior. For example, research suggests that cognitive processes of change (e.g., setting realistic goals) should be encouraged among those in precontemplation and contemplation, while behavioral processes (e.g., placing reminders to exercise at work or home) should be promoted among those in the more advanced stages of motivational readiness. TTM-based interventions attempt to tailor PA programs to a participant's motivational readiness to change and utilize the processes of change to encourage progression in motivational readiness for PA.

The *Self-Determination Theory* (SDT) posits that behaviors are regulated by motives that range on a continuum from highly controlled (extrinsically motivated) to fully autonomous (intrinsically motivated). Extrinsically motivated behaviors arise to avoid negative emotions, a threat or a demand. Extrinsic motivation is also involved when an individual performs a behavior that he or she feels is valuable (but not necessarily inherently enjoyable; Ryan & Deci, 2000). Intrinsically motivated behaviors are those that are done to provide the individual inherent enjoyment, satisfaction, or pleasure. It is thought that intrinsic motivation for behaviors such as PA leads to greater interest, more confidence, and longer persistence of the behavior.

The *Protection Motivation* Theory (Rogers, 1983) emphasizes threat and coping appraisal. Threat appraisal consists of perceived severity (estimated threat of disease) and perceived vulnerability (estimate of chance of developing the disease), and coping appraisal consists of response efficacy (expectancy that the recommended behavior, i.e., PA, can remove the threat) and self-efficacy (belief that one can carry out the recommended behavior successfully, i.e., adopt and maintain PA). Both threat and coping appraisal affect intention to be physically active and PA behavior.

Theories that focus on the individual (e.g., beliefs, attitudes) have led to the development of various interventions to promote PA. However, variables based on these theories do not explain more than a relatively small percentage of the variance in PA levels. *Ecological models* have become increasingly popular in acknowledging multiple levels of influence on PA: individual, social/cultural, organizational, community, physical environment, and policy. They are "macro" in the sense that they go beyond the individual-level choices and decisions to become active in an effort to reduce sedentary lifestyles (King, Stokols, Talen, Brassington & Killingsworth, 2002). Such models focus on the environmental variables, the related type of PA (e.g., transit vs. recreational), and the extent to which specific environmental conditions (e.g., built environment) can facilitate or constrain recreational activity, transit activities, or both. For example, availability of playgrounds, sidewalks, biking, or walking trails is likely to facilitate PA. Conversely, unsafe neighborhoods and lack of sidewalks are likely to hinder PA.

To achieve widespread adoption of physically active lifestyles, much needs to be done to determine what models will facilitate dissemination of PA interventions and which approaches will

help adapt interventions for culturally diverse subgroups. The costs and benefits of such interventions also require attention. Finally, sustaining PA over time will require change at the policy and legislative levels as has been the case with smoking cessation.

Conclusions

PA has an important role to play in the prevention and management of many chronic diseases. Although there has been improvement in the overall prevalence of regular PA in the USA, a large subgroup does not engage in regular PA. Efforts to promote PA have focused on individual-level factors and, to a lesser extent, factors at the community and population level. New technologies can help extend the reach of interventions. However, addressing the barriers to adopting and maintaining PA in the twenty-first century will require efforts at the individual, community, population, and policy levels.

References and Readings

Ajzen, I. (1991). The theory of planned behavior. *Organization Behavior and Human Decision Processes, 50*, 179–211.

Bandura, A. (1986). *Social foundations of thought and action: A social cognitive theory*. Englewood Cliffs, NJ: Prentice-Hall.

Fogelholm, M., Hilloskorpi, H., Laukkanen, R., Oja, P., Van Marken, L. W., & Westerterp, K. (1998). Assessment of energy expenditure in overweight women. *Medicine and Science in Sports and Exercise, 30*, 1191–1197.

Gelibeter, A., Maher, M. M., Gerace, L., Gutin, B., Heymsfield, S. B., & Hashim, S. A. (1997). Effects of strength of aerobic training on body composition, resting metabolic rate, and peak oxygen consumption in obese dieting subjects. *American Journal of Clinical Nutrition, 66*(3), 557–563.

Haskell, W. L., Lee, I.-M., Pate, R. R., Powell, K. E., Blair, S. N., Franklin, C. A., et al. (2007). Physical activity and public health: Updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Medicine and Science in Sports and Exercise, 39*(8), 1423–1434.

Kahn, E. B., Ramsey, L. T., Brownson, R. C., Heath, G. W., Howze, E. H., Powell, K. E., et al. (2002). The effectiveness of interventions to increase physical activity: A systematic review. *American Journal of Preventive Medicine, 22*(4S), 73–107.

King, A. C., Stokols, D., Talen, E., Brassington, G. S., & Killingsworth, R. (2002). Theoretical approaches to the promotion of physical activity. *American Journal of Preventive Medicine, 23*(2), 15–25.

Marcus, B. H., & Simkin, L. R. (1993). The stages of exercise behavior. *Journal of Sports Medicine and Physical Fitness, 33*(1), 83–88.

Pate, R. R., Pratt, R. M., Blair, S. N., Haskell, W. L., Macera, C. A., Bouchard, C., et al. (1995). Physical activity and public health: A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. *Journal of the American Medical Association, 273*, 402–407.

Prochaska, J. O., & DiClemente, C. C. (1983). Stages and processes of self-change of smoking: Toward an integrative model of change. *Journal of Consulting & Clinical Psychology, 51*(3), 390–395.

Rogers, R. W. (1983). Cognitive and physiological process in fear appeals and attitude change: A revised theory of protection motivation. In J. R. Cacioppo & R. E. Petty (Eds.), *Social psychology: A source book* (pp. 153–176). New York: Guildford Press.

Ryan, R. M., & Deci, E. L. (2000). Self-determination theory and the facilitation of intrinsic motivation, social development, and well-being. *American Psychologist, 55*(1), 68–78.

Schmitz, K. H., Courneya, K. S., Matthews, C. M., Demark-Wahnefried, W., Galvao, D. A., Pinto, B. M., et al. (2010). American College of Sports Medicine roundtable on exercise guidelines for cancer survivors. *Medicine & Science in Sports and Exercise, 42*, 258–266.

Schoenborn, C. A., & Adams, P. F. (2010). Health behaviors of adults: United States 2005–2007. National Center for Health Statistics. *Vital and Health Statistics, 10*(245), 1–132.

Tudor-Locke, C., & Bassett, D. R. (2004). How many steps/day are enough? Preliminary pedometer indices for public health. *Sports Medicine, 34*, 1–8.

U.S. Department of Health and Human Services. (2008). *Physical Activity Guidelines Advisory Committee report, 2008*. Washington, DC: U.S. Department of Health and Human Services.

Van Poppel, M. N., Chinapaw, M. J., Mokkink, L. B., van Mechelen, W., & Terwee, C. B. (2010). Physical activity questionnaires for adults: A systematic review of measurement properties. *Sports Medicine, 440*(7), 565–600.

Vanhees, L., Lefevre, J., Philippaerts, R., Martens, M., Huygens, W., Troosters, T., et al. (2005). How to assess physical activity? How to assess physical fitness? *European Journal of Cardiovascular Prevention and Rehabilitation, 12*, 102–114.

Yancey, A. K., Kumanyika, S. K., Ponce, N. A., McCarthy, W. J., Fielding, J. E., Leslie, J. P., et al. (2004). Population-based interventions engaging communities of color in healthy eating and activity living: A review. *Preventing Chronic Disease, 1*, A09.

Physical Activity Interventions

Rick LaCaille
Psychology Department, University of Minnesota Duluth, Duluth, MN, USA

Definition

Physical activity interventions primarily aim to encourage sedentary individuals or those at risk for chronic diseases to initiate and maintain healthy levels of activity. Secondary goals of interventions may include improved weight or disease management, enhanced psychological well-being and stress reduction, and better quality of life. There has been a recent concerted effort to examine changing multiple health behaviors (e.g., dietary, tobacco cessation) in combination with sedentary behaviors. Interventions are often evaluated in the context of a randomized clinical trial (efficacy trials) or quasi-experimental designs (effectiveness trials).

Description

Recently, the US government released its first formal set of recommendations on physical activity (PA), the *Physical Activity Guidelines for Americans* (United States Department of Health and Human Services [USDHHS], 2008), thereby establishing increasing PA as a significant health goal for the twenty-first century. In addition to making clear the health benefits of PA, these guidelines emphasize accumulating targeted doses of moderate to vigorous-intensity forms of PA across the week (rather than on a daily basis) as well as provide specific guidelines for particular groups of people (e.g., youth, adults, older adults, pregnant women, adults with disabilities). However, because of the many benefits of PA and relatively low rates of individuals who engage in routine leisure-time PA, public health professionals have for some time developed strategies and interventions to increase PA (Buckworth & Dishman, 2002). Typically, PA interventions target a goal of consistently achieving at least 30 min of moderate intensity activity on 5 or more days per week. However, some interventions have focused on increasing low-intensity PA, such as walking or stair usage, which may be engaged in more often and in various settings. Duration and intensity of PA interventions have varied greatly from brief, single contacts to multiple contacts extending over 2 years. Efforts to promote PA involvement have, in general, fallen into one or a combination of the following levels of intervening: Individual, group, and community context.

Individual level interventions attempt to promote the adoption and maintenance of active lifestyles by targeting attitudes, beliefs, and behaviors of particular individuals who are not meeting PA guidelines. The most common theoretical paradigms guiding these interventions include social learning theory/social cognitive theory, theory of planned behavior, health belief model, and transtheoretical model (e.g., Marcus & Forsyth, 2009). Interventions have employed several strategies and techniques either successively or concurrently, often in a home-based context, to affect behavioral initiation and/or maintenance of change. The following strategies have been commonly used in interventions: health education, health-risk appraisal, motivational interviewing, written exercise prescriptions, mastery experience/self-efficacy enhancements, goal setting, incentives, contingency contracts, self-monitoring, self-reinforcement, and stimulus control. Delivery of individual interventions has often been via face-to-face or telephone interactions and print materials, though text message/e-mail and web-based programs with expert system algorithms for interactive and tailored feedback have become increasingly popular and considered a viable mode that may be appealing to some individuals (Marcus et al., 2006; Vandelanotte, Spathonis, Eakin, & Owen, 2007).

In contrast to individual level programs, group-based interventions occur in the context of small group settings (e.g., structured classes) and utilize this social environment to encourage PA. Although group interventions often also make use of the theoretical paradigms and strategies discussed within individual approaches, the group structure (e.g., group norms) and processes (e.g., interactions

and communications) that facilitate group cohesion and support for changing sedentary behaviors are emphasized. Group-based specific strategies include developing a distinct group identity (along with group name/clothing), establishing shared goals, mentoring by more experienced members, and encouragement of "fitness friends" and additional support.

Community-level interventions emphasize prevention and risk reduction that affects large segments of a particular population or community. Such PA interventions may include a combination of site-based (e.g., schools, health care, work, church), mass media (e.g., print media campaigns), built environments (e.g., constructing multiuse paths and trails), or policy/legislative (e.g., time allotted for PA at worksite, insurance premium reductions for engaging in PA) strategies and target system changes rather than behavior change of individuals. Thus, a fundamental assumption of community-level PA interventions is that large numbers of individuals will be accessed that may not have been identified otherwise and that the systemic change will yield behavior change in many more individuals.

Numerous reviews and reviews of reviews of PA intervention studies have been conducted (e.g., Foster, Hillsdon, & Thorogood, 2005). Reviews focusing exclusively upon intervention modalities/settings and subpopulations are also available. Overall, the data have suggested that PA interventions (compared to no-intervention or minimal-intervention controls) have garnered modest gains (Muller-Riemenschneider, Reinhold, Nocon, & Willich, 2008), with behavior modification strategies yielding the largest effects. However, behavior modification approaches have been criticized for lacking sustained effects once the intervention is completed as well as potentially compromising more autonomous motivation to engage in PA. A recent meta-analytic review indicated that the strategy of self-monitoring accounted for the most behavioral change of intervention participants, whereas the delivery context (e.g., individual vs. group), number of sessions, target population, and setting were not significant in determining intervention effectiveness (Michie, Abraham, Whittington, McAteer, & Gupta, 2009). Data also suggest that interventions targeting lower intensity and more active leisure time (e.g., walking) appear more successful in affecting change in PA behavior than those advocating more vigorous intensities and structured activities (Williams, Matthews, Rutt, Napolitano, & Marcus, 2008). Although the findings are inconsistent on the effectiveness of worksite interventions for promoting PA, a recent review of randomized clinical trials indicated such interventions may achieve modest effects with promoting healthy weight among employees (Anderson et al., 2009). Worksite settings are thought to offer valuable opportunities for PA interventions because of the potential for high exposure and reach, with promising strategies that include an increased emphasis on social networks (e.g., "buddy system") and built environments (e.g., stairwell enhancements, bike racks) as well as communication change strategies (e.g., persuasive point-of-decision prompts for increased PA or stairwell use). Although environmental strategies that create or improve access to PA for leisure or transportation purposes appear promising, at present, solid empirical evidence for such interventions is lacking. Community-level interventions involving large-scale multicomponent strategies, as well as targeting a combination of health behaviors (e.g., dietary), have demonstrated some initial effectiveness for increasing the percentage of active people across a variety of communities and populations, provided materials and messages are culturally adapted to the needs of the particular community and/or populations (Zaza, Briss, & Harris, 2005). While school-based physical education programs also appear effective in increasing PA levels, generalizability outside of the school setting has been limited (Marcus et al., 2006; Zaza, Briss, & Harris, 2005). On balance, family-based PA intervention have yielded mixed results, whereas classroom-based health education and single-component mass media campaigns have not been found to be effective for increasing PA (Marcus et al.; Zaza, Briss, & Harris).

Unfortunately, maintenance of behavioral change beyond 3 months is not often reported in PA interventions. The data and reviews that are available suggest that determinants of PA initiation

and maintenance may be different. A recent review found that maintenance was more likely for PA interventions that involved more than six behavioral change strategies, occurred for more than 24 weeks, and included face-to-face contacts as well as follow-up prompts to reinforce intervention content (Fjeldsoe, Neuhaus, Winkler, & Eakin, 2011). Notably, assessment of long-term sustainability of PA improvements did not extend beyond 24 months post-intervention for any of the studies examined. Increased assessments of PA maintenance and intervention sustainability appear to be a much needed emphasis in future research. Although cost-effectiveness analyses are also underreported in the literature and in need of greater prioritization (Hagberg & Lindholm, 2006; Muller-Riemenschneider, Reinhold, & Willich, 2009), some data suggest that both behavioral and environmental focused PA interventions may be cost-effective with the latter appearing to have potentially greater cost-effectiveness. In a similar vein, determining the public health impact of PA interventions has become increasingly important and emphasized, with the RE-AIM (Reach, Efficacy, Adoption, Implementation, and Maintenance) framework (Glasgow et al., 2010) serving as a useful model for evaluating and comparing any given intervention across the various levels (i.e., individual to community). Finally, because poor fidelity and adherence to treatment protocols and theoretical frameworks has limited conclusions from the data, there has been a recent effort to systematically create a taxonomy of behavior change techniques to more clearly map intervention components reported in studies (Abraham & Michie, 2008).

Cross-References

- ▶ Benefits of Exercise
- ▶ Physical Activity and Health

References and Readings

Abraham, C., & Michie, S. (2008). A taxonomy of behavior change techniques used in interventions. *Health Psychology, 27*, 379–387.

Anderson, L. M., Quinn, T. A., Glanz, K., Ramirez, G., Kahwati, L. C., Johnson, D. B., Buchanan, L. R., Archer, W. R., Chattopadhyay, S., Kalra, G. P., & Katz, D. L. (2009). The effectiveness of worksite nutrition and physical activity interventions for controlling employee overweight and obesity: A systematic review. *American Journal of Preventive Medicine, 37*, 340–357.

Buckworth, J., & Dishman, R. K. (2002). *Exercise psychology*. Champaign, IL: Human Kinetics.

Fjeldsoe, B., Neuhaus, M., Winkler, E., & Eakin, E. (2011). Systematic review of maintenance of behavior change following physical activity and dietary interventions. *Health Psychology, 30*, 99–109.

Foster, C., Hillsdon, M., & Thorogood, M. (2005). Interventions for promoting physical activity. *Cochrane Database of Systematic Reviews, 25*(1), Art. No.: CD003180

Glasgow, R. E., Dzewaltowski, D. A., Estabrooks, P. A., Gaglio, B. A., King, D., & Klesges, L. (2010) *RE-AIM*. Retrieved June 22, 2011, from http://www.re-aim.org

Hagberg, L. A., & Lindholm, L. (2006). Cost-effectiveness of healthcare-based interventions aimed at improving physical activity. *Scandinavian Journal of Public Health, 34*, 641–653.

Marcus, B. H., & Forsyth, L. A. (2009). *Motivating people to be physically active* (2nd ed.). Champaign, IL: Human Kinetics.

Marcus, B. H., Williams, D. M., Dubbert, P. M., Sallis, J. F., King, A. C., Yancey, A. K., Franklin, B. A., Buchner, D., Daniels, S. R., & Claytor, R. P. (2006). Physical activity intervention studies: What we know and what we need to know. A scientific statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism (Subcommittee on Physical Activity); Council on Cardiovascular Disease in the Young; and the Interdisciplinary Working Group on Quality of Care and Outcomes Research. *Circulation, 114*, 2739–2752.

Michie, S., Abraham, C., Whittington, C., McAteer, J., & Gupta, S. (2009). Effective techniques in healthy eating and physical activity interventions: A meta-regression. *Health Psychology, 28*, 690–701.

Muller-Riemenschneider, F., Reinhold, T., Nocon, M., & Willich, S. (2008). Long-term effectiveness of interventions promoting physical activity: A systematic review. *Preventive Medicine, 47*, 354–368.

Muller-Riemenschneider, F., Reinhold, T., & Willich, S. N. (2009). Cost-effectiveness of interventions promoting physical activity. *British Journal of Sports Medicine, 43*, 70–76.

United States Department of Health and Human Services. (2008). *Physical activity guidelines for Americans*. Washington, DC: Author.

Vandelanotte, C., Spathonis, K. M., Eakin, E. G., & Owen, N. (2007). Website-delivered physical activity interventions: A review of the literature. *American Journal of Preventive Medicine, 33*, 54–64.

Williams, D. M., Matthews, C., Rutt, C., Napolitano, M. A., & Marcus, B. (2008). Interventions to increase walking behavior. *Medicine and Science in Sports, and Exercise, 40*, S567–S573.

Zaza, S., Briss, P. A., & Harris, K. W. (2005). *The guide to community preventive services: What works to promote health?* New York: Oxford University Press.

Physical Activity, Psychosocial Aspects, Benefits

Rick LaCaille
Psychology Department, University of Minnesota Duluth, Duluth, MN, USA

Definition

Numerous aspects of psychological well-being related to physical activity have been examined using epidemiological, cross-sectional, and experimental research strategies, including depression, anxiety, mood/affect, self-perception, cognitive performance, and quality of life. Moreover, these aspects have been considered in terms of acute bouts of exercise and chronic exercise/physical activity as well as potential underlying mechanisms.

Description

It is well accepted that physical activity (PA) has important and far-reaching benefits for enhanced physical health and reduced risk of premature mortality. However, notable psychological benefits have also been documented in the literature (Buckworth & Dishman, 2002). Recently, increased attention has been drawn to examining the impact of PA on serious mental illnesses (e.g., schizophrenia, substance abuse/dependence, eating disorders) and a call to consider integrating PA programs into mental health services as a means to improve psychological well-being and reduce risk for comorbid physical health problems (Holley, Crone, Tyson, & Lovell, 2011; Richardson et al., 2005). Although additional randomized clinical trials are needed to evaluate PA interventions for treating psychological disorders, some data exist supporting the psychosocial benefits in terms of alleviating both primary and secondary symptoms of psychological disorders, including reduced cravings and body dissatisfaction and improved mood and social functioning.

Depression

The most cited and striking evidence in support of the beneficial associations between psychosocial functioning and physical activity comes from studies examining depression. Prospective epidemiological studies have found depression to be associated with low levels of PA, whereas maintaining a moderately active lifestyle has been related to lower risk of developing depression (Biddle, Fox, & Boutcher, 2000). These data are suggestive of both a dose–response relationship and protective effect of PA. Both narrative reviews and meta-analyses of exercise interventions have reported moderate-to-large effects for individuals with clinical depression with the reduction in depression appearing for both aerobic and non-aerobic forms of exercise (Clark & Williams, 2011; Stathopoulou, Powers, Berry, Smits, & Otto, 2006). Importantly, the effect was more pronounced for individuals diagnosed with moderate-to-severe levels of depression than mild-to-moderate levels. These post-intervention effects have been demonstrated in older populations (>60 years) as well, though long-term intervention outcomes and follow-up appear to be lacking with most of the trials (Blake, Mo, Malik, & Thomas, 2009). Some data also point to PA yielding effects comparable to antidepressant medications. The effects for exercise may appear less immediate than medications, but exercise seems to offer a reduced risk for relapse with depression (Barbour, Edenfield, & Blumenthal, 2007). A recent meta-analysis of PA interventions and depressive symptoms (i.e., nonclinical depression) has reinforced the previous findings of somewhat smaller moderate effects with less depressive symptomatology for both men and women; however, the effect was strengthened when aerobic forms of exercise were combined

with non-aerobic exercise (i.e., resistance, flexibility) (Conn, 2010a). Supervised low-intensity PA interventions were shown to yield larger effect sizes than moderate- or high-intensity exercise, suggesting that increased aerobic fitness is not a primary mechanism responsible for antidepressive effects of PA.

Anxiety

Another central area of inquiry regarding the salutary effects of PA has involved investigating anxiety reductions in both clinical and nonclinical samples. Reviews of intervention studies using individuals with elevated levels of anxiety or diagnosed anxiety disorders have generally reported small-to-medium effect sizes with somewhat better effects demonstrated with aerobic forms of exercise and longer intervention programs (Biddle, Fox, & Boutcher, 2000). Some comparative studies have shown PA to provide similar results to pharmacological and other therapeutic techniques (e.g., relaxation) in anxiety reductions and related symptoms. However, similar to the findings with depressive disorders, the impact of PA on anxiety reduction generally appears to be more delayed but stable. A recent meta-analysis of anxiety outcomes of PA interventions with nonclinical samples found similar benefits of PA on reduced anxiety levels, though observed slightly more modest effects than seen with clinical samples (Conn, 2010b). Although no differential effect was found for aerobic versus non-aerobic forms of PA, greater anxiety reduction was observed with moderate-to-high intensity levels of exercise. In addition to chronic exercise (i.e., PA interventions), studies focusing on single bouts of exercise have examined changes in state anxiety with similar size effects. Larger reductions in anxiety appear for within-group than between-group comparisons and aerobic forms of exercise (rather than non-aerobic/resistance forms) appear most beneficial with reducing state anxiety. Single-session bouts of exercise have also been found to provide beneficial attenuated physiological reactivity and improved recovery from psychosocial stressors (Buckworth & Dishman, 2002).

Affective Response

Although some researchers have used mood and affect interchangeably, there has been considerable attention in the PA/exercise literature directed toward providing clarification of these constructs (e.g., Ekkekakis & Petruzzello, 2000). Overall, moods are emotion-related expressions (e.g., anxious mood, depressed mood) that are considered more complex and multifaceted, lasting only brief moments to days, and may develop without an identifiable event. While affect is also considered an emotion-related expression, it is thought to be more basic and vary along the orthogonal dimensions of valence (unpleasant/avoidance vs. pleasant/approach) and level of activation (calm vs. aroused). Thus, an affective response may entail unpleasant and low activation (e.g., fatigue, boredom), unpleasant and high activation (e.g., tension, distress), pleasant and low activation (e.g., relaxation, tranquility), and pleasant and high activation (e.g., vigor, excitement). A meta-analysis examining changes in pleasant high activation affect observed medium-sized effects shortly after a bout of aerobic exercise with larger effects noted for lower-intensity exercise (Reed & Ones, 2006). Other studies have also indicated that exercise may result in reports of increased vigor, relaxation, and tranquility and decreased tension, irritability, and fatigue (Biddle, Fox, & Boutcher, 2000). Additionally, high-intensity (or even moderate-intensity) exercise may result in increased negative/unpleasant affect, though there appears to be a great deal of variability in affective responses among moderate-intensity PA. More recent efforts to clarify the relationship between affect and exercise intensity have investigated affective responses that occur while physically active. This line of research has indicated that the ventilatory/lactate threshold (i.e., near maximal level of intensity and transition from aerobic to anaerobic metabolic supplementation) appears to be the point at which pleasant affective responses diminish while exercising and that postexercise affect may reflect a rebound from this decline or a continuation of pleasant affect if this level of intensity is not exceeded (Ekkekakis & Acevedo, 2006).

Cognitive variables (e.g., attentional focus, self-efficacy, attributions) appear to influence affective responses with intensities below or near the threshold; however, physiological/interoceptive variables (e.g., muscular or respiratory cues) come to play a more dominant role in influencing the affective response as the threshold is approached or exceeded. An important implication of this line of investigation lies in the potential to improve adherence to a PA program for novice exercisers by developing self-monitoring skills and cognitive strategies (e.g., attentional dissociation/association) while exercising and self-selecting intensity to maximize reinforcing positive affective responses.

Self-Perception

A related and equally important area, though considerably less studied, is the relationship between self-perception and PA. Self-perception is conceptualized as a multifaceted construct within which lie several related subjective attitudes and beliefs about one's self (e.g., self-esteem, self-concept) that may be organized in hierarchical levels (e.g., global self-esteem, physical self-esteem domain, situation-specific efficacy). Overall, cross-sectional studies have shown that engaging in PA appears weakly associated with higher global self-esteem, though a moderate relationship between better physical self-esteem and body image exists beginning in adolescence and extending into adulthood (Ekelund, Heian, & Hagen, 2005; Fox, 2000; Spence, McGannon, & Poon, 2005). Reviews of PA interventions reinforce this pattern of improved self-perceptions with the greatest improvements occurring for individuals with lower self-esteem. Some data also suggest that females may benefit more from participation in an exercise program than their male counterparts in terms of increased body image, physical self-esteem, and self-efficacy for exercise (Hausenblas & Fallon, 2006). Although both aerobic forms of PA and weight/resistance training seem beneficial, the combination of the two appears to offer greater improvements in body image and physical self-esteem.

Cognitive Performance

A growing body of evidence supports the beneficial effect of PA on cognitive functioning in both children and adults (Clark & Williams, 2011; Etnier, Nowell, Landers, & Sibley, 2006). Although cross-sectional design studies have suggested moderate-sized effects favoring fitter participants, most randomized exercise interventions indicate a more modest effect on improved cognitive processes. Studies with older adults have provided somewhat mixed results, with improved cognitive performance being most pronounced with simple reaction time-/speed-based tasks in some studies and executive-control tasks (e.g., planning, response inhibition, task choice/switching) showing greater benefits from PA in other studies. Some evidence indicates that regular participation in moderate PA during midlife may confer a reduced risk of later cognitive impairment/dementia (Colcombe & Kramer, 2003). With regard to children, benefits associated with PA have been observed in several cognitive domains (e.g., verbal abilities, attention and concentration, executive functioning) as well as improved academic performance. For both children and adults, such improved cognitive processing has been seen with acute bouts of exercise and longer term PA programs (Biddle, Fox, & Boutcher, 2000). Generally, acute bouts of exercise have demonstrated larger effects when lasting 30–60 min; PA programs exceeding 6 months have shown greater improvements than briefer interventions. Although the emphasis within the literature is usually on aerobic forms of PA, combining aerobic and resistive/weight training forms of exercise has demonstrated somewhat larger cognitive performance gains.

Quality of Life

A final area of consideration with regard to PA benefits involves the emerging concept of perceived quality of life, sometimes referred to as health-related quality of life. Quality of life is a multifaceted construct often encompassing physical (e.g., physical functioning, bodily pain), mental (e.g., vitality), and social components (e.g., role limitations, social functioning) or overall satisfaction with life and psychological

well-being. In terms of PA, the construct has been predominantly examined with older populations and those managing a chronic health diagnosis with exercise appearing to have a positive impact for several of these conditions (e.g., cancer, diabetes). A meta-analysis of older adults without clinical disorders found small-to-medium effects for PA on various aspects of quality of life/well-being, with aerobic forms of exercise and moderate intensity providing the greatest benefits (Netz, Wu, Becker, & Tenenbaum, 2005). Another more recent review examined the quality of life outcomes in the general adult population and reported consistently moderate-to-strong positive effects of PA in cross-sectional studies (Bize, Johnson, & Plotnikoff, 2007). However, few randomized clinical exercise trials have examined quality of life in healthy samples which limits the potential strength of the conclusions and generalizability to the general population.

Compulsive Physical Activity

Although the emphasis within the literature on psychosocial aspects of PA has been overwhelming on the beneficial effects, reports suggest potentially deleterious effects for some individuals. For instance, some investigations have documented compulsive/obligatory patterns of exercise engagement that may pose an increased risk for overuse injuries or reduced psychological well-being (e.g., increased self-criticism and body dissatisfaction despite improved fitness or some weight loss success) (Biddle, Fox, & Boutcher, 2000). The diagnostic validity of "exercise addiction" has been questioned by some investigators, and the incidence of such patterns is thought to be rare, though excessive PA/exercise has been more frequently reported as a secondary feature of some preexisting clinical disorders (e.g., body/muscle dysmorphia). Notably, some data exist supporting the use of PA/exercise as an adjunctive treatment for eating disorders with improvements in reduced drive for thinness and binge eating (Stathopoulou, Powers, Berry, Smits, & Otto, 2006).

Conclusions and Summary

Despite the large literature that exists on the psychosocial benefits of PA, a clear understanding of the underlying mechanisms is less evident. Numerous mechanisms have been proposed in the literature, including psychological/cognitive (e.g., sense of mastery, autonomy and personal control, distraction) and physiological/biochemical changes (e.g., thermogenic, cerebral adaptations, monoamine and endorphin levels) (Buckworth & Dishman, 2002). In summary, PA appears to have a sizeable antidepressant effect, with several other psychosocial benefits documented such as reduced anxiety, improved body image and physical self-esteem, enhanced cognitive performance, and perceptions of greater quality of life. A person's affect while being active, as well as following a bout of exercise, may fluctuate as a product of the level of intensity with higher levels potentially resulting in a diminished positive and increased negative affective response. Helping beginning exercisers self-identify and regulate this potential affect-intensity relationship may prove useful in maximizing the reinforcing positive affective responses and improving longer-term adherence to a healthier physically active lifestyle. Although PA may be associated with some reductions in psychological well-being for select individuals engaging in compulsive behavioral patterns, on balance, it appears that the benefits of PA exceed the risks of misuse.

Cross-References

- ▶ Benefits of Exercise
- ▶ Physical Activity and Health
- ▶ Physical Activity Interventions
- ▶ Stress, Exercise

References and Readings

Barbour, K. A., Edenfield, T. M., & Blumenthal, J. A. (2007). Exercise as a treatment for depression and other psychiatric disorders: A review. *Journal of Cardiopulmonary Rehabilitation and Prevention, 27*, 359–367.

Biddle, S. J. H., Fox, K. R., & Boutcher, S. H. (Eds.). (2000). *Physical activity and psychological well-being*. London: Routledge.

Bize, R., Johnson, J. A., & Plotnikoff, R. C. (2007). Physical activity level and health-related quality of life in the general adult population: A systematic review. *Preventive Medicine, 45*, 401–415.

Blake, H., Mo, P., Malik, S., & Thomas, S. (2009). How effective are physical activity interventions for alleviating depressive symptoms in older people? A systematic review. *Clinical Rehabilitation, 23*, 873–887.
Buckworth, J., & Dishman, R. K. (2002). *Exercise psychology*. Champaign, IL: Human Kinetics.
Clark, U. S., & Williams, D. (2011). Exercise and the brain. In R. A. Cohen & L. H. Sweet (Eds.), *Brain imaging in behavioral medicine and clinical neuroscience* (pp. 257–273). New York: Springer.
Colcombe, S., & Kramer, A. F. (2003). Fitness effects on the cognitive function of older adults: A meta-analytic study. *Psychological Science, 14*, 125–130.
Conn, V. S. (2010a). Depressive symptom outcomes of physical activity interventions: Meta-analysis findings. *Annals of Behavioral Medicine, 39*, 128–138.
Conn, V. S. (2010b). Anxiety outcomes after physical activity interventions: Meta-analysis findings. *Nursing Research, 59*, 224–231.
Ekelund, E., Heian, F., & Hagen, K. B. (2005). Can exercise improve self-esteem in children and young people? A systematic review of randomized clinical trials. *British Journal of Sports Medicine, 39*, 792–798.
Ekkekakis, P., & Acevedo, E. O. (2006). Affective responses to acute exercise: Toward a psychobiological dose–response model. In E. O. Acevedo & P. Ekkekakis (Eds.), *Psychobiology of physical activity* (pp. 91–109). Champaign, IL: Human Kinetics.
Ekkekakis, P., & Petruzzello, S. J. (2000). Analysis of the affect measurement conundrum in exercise psychology: I. Fundamental issues. *Psychology of Sport and Exercise, 1*, 71–88.
Etnier, J. L., Nowell, P. M., Landers, D. M., & Sibley, B. A. (2006). A meta-regression to examine the relationship between aerobic fitness and cognitive performance. *Brain Research Reviews, 52*, 119–130.
Fox, K. R. (2000). The effects of exercise on self-perceptions and self-esteem. In S. J. H. Biddle, K. R. Fox, & S. H. Boutcher (Eds.), *Physical activity and psychological well-being* (pp. 88–117). London: Routledge.
Hausenblas, H. A., & Fallon, E. A. (2006). Exercise and body image: A meta-analysis. *Psychology and Health, 21*, 33–47.
Holley, J., Crone, D., Tyson, P., & Lovell, G. (2011). The effects of physical activity on psychological wellbeing for those with schizophrenia: A systematic review. *British Journal of Clinical Psychology, 50*, 84–105.
Netz, Y., Wu, M. J., Becker, B. J., & Tenenbaum, G. (2005). Physical activity and psychological well-being in advanced age: A meta-analysis of intervention studies. *Psychology and Aging, 20*, 272–284.
Reed, J., & Ones, D. S. (2006). The effect of acute aerobic exercise on positive activated affect: A meta-analysis. *Psychology of Sport and Exercise, 7*, 477–514.
Richardson, C. R., Faulkner, G., McDevitt, J., Skrinar, G. S., Hutchinson, D. S., & Piette, J. D. (2005). Integrating physical activity into mental health services for persons with serious mental illness. *Psychiatric Services, 56*, 324–331.
Spence, J. C., McGannon, K. R., & Poon, P. (2005). The effect of exercise on global self-esteem: A quantitative review. *Journal of Sport & Exercise Psychology, 27*, 311–334.
Stathopoulou, G., Powers, M. B., Berry, A. C., Smits, J. A. J., & Otto, M. W. (2006). Exercise interventions for mental health: A quantitative and qualitative review. *Clinical Psychology: Science and Practice, 13*, 179–193.

Physical Capacity

▶ Physical Fitness

Physical Condition

▶ Physical Fitness

Physical Environment

▶ Built Environment

Physical Exam

▶ Physical Examination

Physical Examination

Margaret Hammersla
University of Maryland School of Nursing, Baltimore, MD, USA

Synonyms

Health assessment; Physical exam

Definition

Physical examination, assessment, is the systematic process of collecting data about a patient or client using the techniques of inspection, palpation, percussion, and auscultation to guide a clinician in the process of diagnosis of pathological states as well as developing a plan of care (Fennessey & Wittmann-Price, 2011). Physical assessment is an ongoing process that enables the clinician to continuously evaluate a patient's signs and symptoms, to monitor effectiveness of treatment, and to make adjustments in the plan of care as required (Zambas, 2010). This physical assessment is conducted in a systematic manner that is comfortable to both the patient and clinician; typically this is done using a head-to-toe approach.

Physical assessment is done for one of two reasons. The first reason is to conduct a complete physical exam of the entire body in order to screen the patient for potential health problems that have not yet manifested symptoms (Bickley & Szilagy, 2008) and monitor chronic health concerns. This exam is traditionally categorized based on body system (e.g., cardiovascular, respiratory, gastrointestinal). Each body system has its own set of unique advanced assessment procedures that allow the clinician to make a judgment about physical function based on what he or she sees, hears, and feels.

The second reason is to investigate a patient's chief complaint or follow-up on a current health problem such as hypertension or diabetes (Stern, Cifu, & Altkorn, 2009). For this more focused exam the clinician makes a determination of what body systems and exam components need to be conducted based on the differential diagnosis and/or the pathophysiology of the current health problem. This more focused exam typically utilizes more advanced techniques to obtain a deeper understanding of the physical changes that may be occurring due to disease process (Bickley & Szilagy, 2008).

Physical assessment, along with health history, is the first and most vital step in diagnosing and planning care for patients. A skilled clinician will be able to utilize findings from the physical exam to both support and rule out diagnoses.

Cross-References

- ▶ Clinical Settings
- ▶ Diabetes
- ▶ Hypertension
- ▶ Primary Care
- ▶ Primary Care Physicians

References and Readings

Bickley, L. S., & Szilagy, P. G. (2008). *Bates' guide to physical examination and history taking* (10th ed.). Philadelphia: Lippincott Williams and Wilkins.

Fennessey, A., & Wittmann-Price, R. A. (2011). Physical assessment: A continuing need for clarification. *Nursing Forum, 46*(1), 45–50.

Stern, S. D. C., Cifu, A. S., & Altkorn, D. (2009). *Symptom to diagnosis: An evidence based guide* (2nd ed.). New York: McGraw-Hill Medical.

Zambas, S. I. (2010). Purpose of the systematic physical assessment in everyday practice: Critique of a "sacred cow". *Journal of Nursing Education, 49*(6), 305–310.

Physical Fitness

Nerissa Campbell[1], Stefanie De Jesus[1] and Harry Prapavessis[2]
[1]Exercise and Health Psychology Laboratory, The University of Western Ontario, London, ON, Canada
[2]University of Western Ontario, London, ON, Canada

Synonyms

Functional health; Habitual performance; Physical capacity; Physical condition

Definition

Physical fitness is one's ability to execute daily activities with optimal performance, endurance, and strength with the management of disease, fatigue, and stress and reduced sedentary behavior.

Description

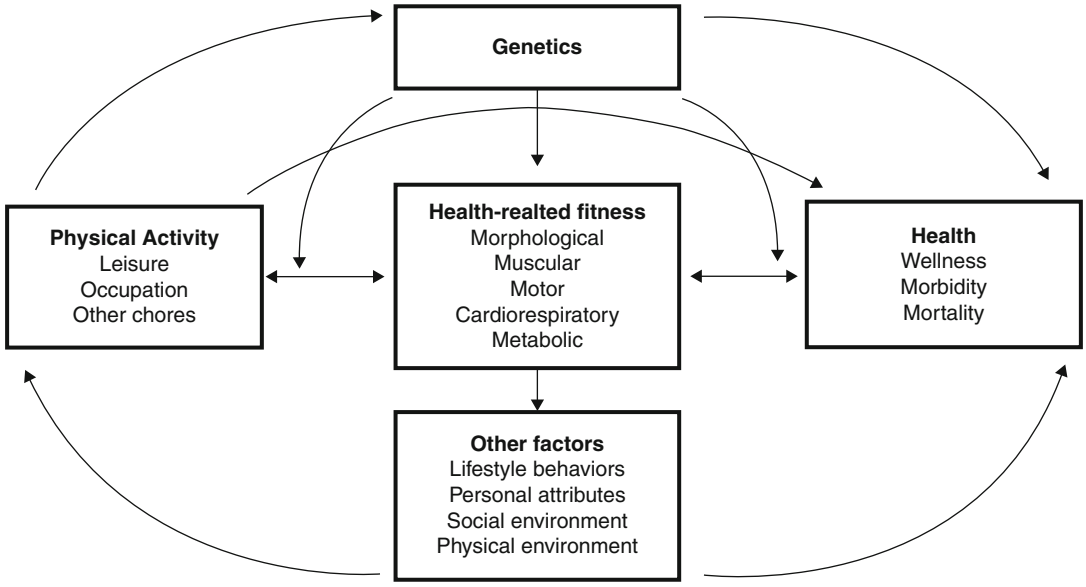
Physical fitness has multiple components and is conceptualized as either performance- or health-related. The specificity of performance-related fitness regarding one's athletic skill best relates to an individual's athletic performance. Conversely, health-related fitness is generalized to health status and is affected positively or negatively by one's habitual physical activity habits. Given the complexity of physical fitness and the epidemiological analysis taken presently, health-related fitness will be the focus of this discussion.

There are five major components of health-related fitness: morphological, muscular, motor, cardiorespiratory, and metabolic (see below), with muscular and cardiorespiratory fitness being the two primary facets assessed in research. As outlined in Fig. 1, a complex relationship exists among the five biological traits, physical activity level, and health outcome.

There are numerous methods to assess the different components of health-related physical fitness, ranging in feasibility and utility for a laboratory versus field-based testing. Morphology is typically assessed using body mass index (BMI, expressed as kg/m^2), a crude measure of body composition. More objective measures of body composition include skinfold technique, bioelectrical impedance, and dual-energy x-ray absorptiometry. Muscular strength can be evaluated by tensiometers, handgrip dynamometers, and strength gauges. Conversely, muscular endurance is measured by performing the maximum number of repetitions of common body movements, such as sit-ups or push-ups. Motor skills involve fine and gross motor skills and are most commonly assessed by a battery of tests that target balance, speed, control precision, reaction time, aim, and coordination. Cardiorespiratory fitness is the ability of the cardiovascular and respiratory systems to deliver oxygen to working muscles, in addition to the ability of those tissues to utilize that oxygen to produce energy. Peak VO_2, a reflection of cardiorespiratory fitness, is the highest rate of oxygen consumption by muscles during exercise and can be directly assessed by a maximal exercise test. Alternatively, peak VO_2 can be measured indirectly using a submaximal exercise test or timed distance run/walk protocol. Finally, to determine metabolic health, blood pressure and biochemical analyses of blood triglycerides and fasting plasma glucose are examined.

Natural differences exist in health-related physical fitness across the life span and by sex and ethnicity. The extent of the difference is dependent on the specific component of fitness. In general, level of physical fitness declines with age. Age-related decreases in peak VO_2 and muscular strength make even the simplest tasks physically demanding for the elderly compared to younger people. Sex differences in physical fitness are primarily attributed to differences in absolute muscle mass and morphology between males and females. Males generally tend to have higher levels of cardiorespiratory fitness and strength and decreased flexibility compared to females. Similarly, differences in cardiorespiratory fitness have been found between white and black individuals. On average, white males and females are found to have higher maximal VO_2 values compared to black males and females. It is important to keep in mind that depending on the population of interest, different approaches may be needed in order to appropriately assess physical fitness. For example, a maximal VO_2 exercise test may be appropriate for healthy adults or the fit elderly, whereas a different approach for assessing VO_2 in obese individuals or frail older persons may be required.

A multidisciplinary approach is necessary to achieve and maintain physical fitness. Specifically, meeting recommended physical activity and nutrition guidelines as well as acquiring



Physical Fitness, Fig. 1 Bouchard, Blair & Haskell (2006), p. 17

adequate rest are each important components to overall functional health. Not surprisingly, cardiovascular disease, diabetes, cancer, obesity, depression, osteoporosis, and premature death are associated with inadequate physical activity and hence poor physical fitness.

Health-Related Fitness Components and Traits

Morphological component	Body mass for height
	Body composition
	Subcutaneous fat distribution
	Abdominal visceral fat
	Bone density
	Flexibility
Cardiorespiratory component	Submaximal exercise capacity
	Maximal aerobic power
	Heart functions
	Lung functions
	Blood pressure
Muscular component	Power
	Strength
	Endurance
Motor component	Agility
	Balance
	Coordination
	Speed of movement
Metabolic component	Glucose tolerance
	Insulin sensitivity
	Lipid and lipoprotein metabolism
	Substrate oxidation characteristics

Bouchard, Blair, & Haskell (2006), p. 17

Cross-References

- Body Composition
- Body Mass Index
- Exercise Testing
- Handgrip Strength
- Maximal Exercise Stress Test
- Physical Activity and Health

References and Readings

Bouchard, C., Blair, S. N., & Haskell, W. L. (2006). *Physical activity and health*. Champaign, IL: Human Kinetic.

Bouchard, C., & Shephard, R. J. (1994). Physical activity, fitness and health: The model and key concepts. In C. Bouchard, R. J. Shephard, & T. Stephens (Eds.), *Physical activity, fitness and health, International Proceedings and concensus statement* (pp. 77–88). Champaign: Human Kinetics.

Caspersen, C. J., Powell, K. E., & Christenson, G. M. (1985). Physical activity, exercise, and physical fitness: Definitions and distinctions for health-related research. *Public Health Reports, 100*(2), 126–131.

Larson, L. A. (1974). *Fitness, health, and work capacity: International Standards for assessment*. New York: Macmillan.

Warburton, D. E. R., Nicol, C. W., & Bredin, S. S. D. (2006). Health benefits of physical activity: The evidence. *Canadian Medical Association Journal, 174*(6), 801–809.

Physical Fitness Testing

► Exercise Testing

Physical Fitness: Health-Related Fitness Components and Traits

► Physical Fitness

Physical Functioning

► Physical Therapy

Physical Health

► Therapy, Physical

Physical Illness

► Psychosocial Work Environment

Physical Inactivity

Tyler Clark
School of Psychology, The University of Sydney, Sydney, NSW, Australia

Definition

Physical inactivity is the failure to meet the minimum recommended physical activity guidelines (i.e., 30-min moderate-intensity exercise on at least 5, although preferably all, days of the week or 75-min vigorous-intensity exercise to be undertaken in no less than 20-min increments thrice a week). Physical inactivity is one of the World Health Organization's (WHO) 12 leading risks to health. Physical inactivity is widespread and associated with increases in all causes of mortality and is an independent risk factor for chronic diseases.

Physical inactivity differs from sedentary behavior (e.g., sitting and not moving); physical inactivity refers to not meeting the aforementioned guidelines.

Description

Prevalence

The American Heart Association estimates that 60% of the world population does not meet recommended physical activity guidelines (American Heart Association, 2001). The American Centre for Disease Control (CDC) estimates 25% of adults are not active at all.

Risk Factors

Physical inactivity increases with age. While physical activity typically peaks in early adolescence, it then begins to decline, regardless of gender (World Health Organization, 2011a). Other demographic risk factors include low income and less education.

Behavioral correlates of physical inactivity include a reduction in leisure-time physical activity and the inclusion of more sedentary occupational and domestic activities (Healey, 2007).

Other environmental correlates of physical activity include population overcrowding, increased levels of crime, high-density traffic, low air quality, and a lack of parks, sidewalks, and sports/recreation facilities (WHO, 2011a).

Health Risks

Physical inactivity increases all cause mortality (World Health Organization, 2011b). The WHO estimates as many as two million deaths worldwide as attributable to physical inactivity. It is also an independent risk factor for chronic diseases such as ischemic heart disease, stroke, type 2 diabetes, breast cancer, colon cancer, and depression. Physical inactivity is also a leading cause of falls and fall-related injuries, particularly in older populations.

Physical inactivity has indirect health burdens as well, which include pain, disability, anxiety, and increased suffering due to medical conditions. These indirect burdens often lead to a reduction in an individual's quality of life, as well as shorter life expectancy, less workforce participation, decreased bone and functional health, and weight gain (Taylor, 2009).

Cross-References

- ▶ Chronic Disease or Illness
- ▶ Lifestyle, Sedentary
- ▶ Physical Activity
- ▶ Quality of Life
- ▶ Risk Factors and Their Management
- ▶ Sedentary Behaviors

References and Readings

American Heart Association (2009). Physical inactivity. Available at americanheart.org. Accessed December 8, 2010.

Healey, J. (2007). *Physical activity*. Thirroul, N.S.W: Spinney Press.

Taylor, S. E. (2009). *Health psychology* (7th ed.). New York: McGraw Hill. International Edition.

World Health Organization (2011a). Global strategy on diet, physical activity and health. Available at http://www.who.int/dietphysicalactivity/pa/en/index.html. Accessed January 8, 2011.

World Health Organization (2011b). Physical activity. Available at: http://www.who.int/topics/physical_activity/en/. Accessed January 8, 2011.

Fletcher, G. F., Balady, G. J., Amsterdam, E. A., et al. (2001). Exercise standards for testing and training: a statement for healthcare professionals from the American Heart Association. *Circulation*, *104*:1694–1740.

Physical Therapy

Marilyn Moffat[1] and Catherine Sykes[2]
[1]Department of Physical Therapy, New York University, New York, NY, USA
[2]World Confederation for Physical Therapy, Victoria Charity Centre, London, UK

Synonyms

Kinesiotherapy; Physiotherapy; Therapy, Physical

Definition

Physical therapy is a health service concerned with identifying and maximizing quality of life and movement potential within the spheres of promotion, prevention, intervention/treatment, habilitation, and rehabilitation. The spheres of practice are aimed at physical, psychological, emotional, and social well-being of a patient/client or population group. Physical therapy involves the interaction between physical therapists and patients/clients, other health professionals, families, caregivers, and communities in a process where movement potential is assessed, goals are agreed upon, and interventions carried out using knowledge and skills unique to physical therapists (World Confederation for Physical Therapy [WCPT], 2011a, b).

Description

Physical therapy is the service provided only by, or under the direction and supervision of physical therapists, to people and populations to develop, maintain, and restore maximum movement and

functional ability throughout the life span. Physical therapist practice includes the provision of services in circumstances where movement and function are threatened by the process of aging or by injury, diseases, disorders, or other conditions of health. Functional movement is central to physical therapist practice since it is at the core of what it means to be healthy. Physical therapists are guided in their practice by the professional behaviors of accountability, altruism, compassion/caring, cultural competence, ethical behavior, integrity, personal/professional development, professional duty, and social responsibility and advocacy. Physical therapist education consists of university-based education leading to entry-level qualifications that range internationally from bachelors, to masters or doctorate entry qualifications.

Physical therapist practice follows a patient/client management model that includes examination/assessment, evaluation, diagnosis, prognosis, plan of care, intervention/treatment, and reexamination (World Confederation for Physical Therapy, 2011a, b).

Examination by the physical therapist involves history taking, screening of the patient's/client's systems (cardiovascular/pulmonary, musculoskeletal, neuromuscular, and integumentary) and the use of specific tests and measures, the results of which are evaluated within a process of evidence-based clinical reasoning to determine the facilitators and barriers to optimal human functioning. The tests and measures used by physical therapists include any of the following: aerobic capacity/endurance; anthropometric characteristics; arousal, attention, and cognition; assistive and adaptive devices; balance; circulation (arterial, venous, lymphatic); cranial and peripheral nerve integrity; environmental, home, and work access and barriers/facilitators; ergonomics and body mechanics; gait and locomotion; integumentary integrity; joint integrity and mobility; motor function (motor control and motor learning); muscle performance; neuromotor development and sensory integration; orthotic, protective, and supportive devices; pain; posture; prosthetic requirements; range of motion; reflex integrity; self-care and home management; sensory and proprioceptive integrity; ventilation and respiration/gas exchange; and work, education, community, and leisure integration or reintegration.

Based upon the examination results, physical therapists evaluate the findings from the examination (history, systems review, tests and measures, environmental facilitators/barriers) to make clinical judgments regarding patients/clients. Physical therapists formulate the diagnosis that results in the identification of existing or potential impairments, activity limitations, and participation restrictions and then determine patient/client prognoses and identify the most appropriate intervention/treatment strategies to optimize patient/client functioning. The plan of care is developed that is consistent with legal, ethical, and professional obligations and administrative policies and procedures of the practice environment. Specific interventions/treatments are determined with measurable outcomes and goals associated with the plan of care and with the involvement of the person and their care providers, both professional and personal. Plans may include referral to other agencies and service delivery providers.

Physical therapists provide, whenever possible, evidence-based physical therapy interventions to achieve patient/client goals and outcomes. These interventions/treatments encompass three major areas: (1) coordination, communication, and documentation; (2) patient/client-related instruction; and (3) procedural interventions.

The procedural interventions include:

1. Therapeutic exercise (including aerobic capacity/endurance conditioning or reconditioning; balance, coordination, and agility training; body mechanics and postural stabilization; flexibility exercises; gait and locomotion training; neuromotor development training; relaxation; and strength, power, and endurance training for head, neck, limb, pelvic-floor, trunk, and ventilatory muscles)
2. Functional training in self-care and home management (including activities of daily living training; barrier accommodations or modifications; device and equipment use and training; functional training programs;

instrumental activities of daily living training; and injury prevention or reduction)
3. Functional training in work, community and leisure integration or reintegration (including barrier accommodations or modifications; device and equipment use and training; functional training programs; instrumental activities of daily living training; injury prevention or reduction; and leisure and play activities and training)
4. Manual therapy techniques (including acupressure; manual lymphatic drainage; manual traction; massage; mobilization/manipulation; and passive range of motion)
5. Prescription, application, and, as appropriate, fabrication of devices and equipment (including adaptive, assistive, orthotic, prosthetic, protective, and supportive devices)
6. Airway clearance techniques (including breathing strategies; manual/mechanical techniques; and positioning)
7. Integumentary repair and protection techniques (including selective and nonselective wound debridement; dressings; oxygen therapy; and topical agents)
8. Electrotherapeutic modalities (including biofeedback; electrotherapeutic delivery of medications; and electrical stimulation)
9. Physical agents (including athermal agents; cryotherapy; hydrotherapy; light agents; sound agents; and thermotherapy) and mechanical modalities (including acupuncture, dry needling; compression therapies; gravity-assisted compression devices; mechanical motion devices; and traction devices)

Interventions/treatments are aimed at prevention of impairments, activity limitations, participation restrictions, and injury. Interventions provided by physical therapists also include prevention, health promotion, and fitness for individuals of all ages and for groups and communities.

Reexamination by physical therapists occurs throughout the episode of service delivery to evaluate the effectiveness of interventions/treatments and outcomes and to adjust the plan of care in response to findings. Outcomes monitoring is part of building the evidence base for modifying the patient/client plan, as well as for the research underpinning professional physical therapy practice.

Physical therapy is an essential part of the health and community/welfare services delivery systems. Professional education prepares physical therapists to practice independently of other health service delivery providers as autonomous practitioners and also within interdisciplinary rehabilitation/habilitation programs. Physical therapists may act as first contact practitioners, and patients/clients may seek direct services without referral from another health care professional. Physical therapists provide consultation within their expertise and determine when patients/clients need to be referred to an other professional provider.

The professional names of physical therapy or physiotherapy, the titles physical therapist or physiotherapist and appropriate abbreviations (e.g., PT, FT, physio) as such or in any translation, are the sole preserve of persons who hold qualifications approved by national professional associations. Members of the public wishing to access the services of a physical therapist are entitled to know that recognized qualifications are held and that professional behavior is governed by ethical codes (World Confederation for Physical Therapy, 2007).

Physical therapists, guided by their own code of ethical principles, are concerned with the following purposes:

- Promoting the health and well-being of individuals and the general public/society, emphasizing the importance of physical activity and exercise
- Preventing impairments, activity limitations, participatory restrictions, and disabilities in individuals at risk of altered movement behaviors due to health or medically related factors, socioeconomic stressors, environmental factors, and lifestyle factors
- Providing interventions/treatments to restore integrity of body systems essential to movement, maximize function and recovery, minimize disability, and enhance the quality of life and inclusion, independent living and workability in individuals and groups of individuals with altered movement behaviors resulting

from impairments, activity limitations, participatory restrictions, and associated environmental factors (disabilities)
- Modifying environmental, home, and work access and barriers to ensure full participation in one's normal and expected societal roles

Physical therapists contribute to the development of local, national, and international health policies and public health strategies. Physical therapists may have roles in management, administration, supervision of personnel, education, research, and consultation to businesses, schools, government agencies, other organizations, or individuals.

Physical therapy services may be provided to individuals or populations and in a wide range of service settings, including but not limited to community-based rehabilitation programs; community settings including primary health care centers; individual homes; field settings (including in response to disasters); educational and research centers; fitness centers; health clubs; gymnasia and spas; hospices; hospitals; nursing homes; occupational health centers; outpatient clinics; physical therapist private offices and clinics; prisons; public settings (e.g., shopping centers) for health screening and promotion; rehabilitation centers; residential homes; schools including preschools and special schools; senior citizen centers; sports centers/clubs; and workplaces/companies.

Cross-References

- ▶ Activities of Daily Living (ADL)
- ▶ Activity Level
- ▶ Aerobic Exercise
- ▶ Back Pain
- ▶ Cancer and Physical Activity
- ▶ Cardiac Rehabilitation
- ▶ Cardiovascular Disease Prevention
- ▶ Chronic Disease Management
- ▶ Exercise
- ▶ Exercise Testing
- ▶ Exercise, Benefits of
- ▶ Functional Versus Vocational Assessment
- ▶ Lifestyle, Active
- ▶ Lifestyle, Healthy
- ▶ Massage Therapy
- ▶ Physical Activity and Health
- ▶ Physical Activity Interventions
- ▶ Physical Fitness
- ▶ Physical Health

References and Readings

World Confederation for Physical Therapy. (2011a). *Policy statement: Description of physical therapy*. Accessed March 21, 2012, from http://www.wcpt.org/sites/wcpt.org/files/files/PS_Description_PT_Sept2011.pdf

World Confederation for Physical Therapy. (2011b). *Policy statement: Protection of title*. Accessed March 21, 2012, from http://www.wcpt.org/sites/wcpt.org/files/files/PS_Protection_Title_Sept2011.pdf

Physical Well-Being

- ▶ Happiness and Health

Physician-Assisted Suicide

- ▶ Euthanasia

Physiological Reactivity

Kristen Salomon
Department of Psychology, University of South Florida College of Arts & Sciences, Tampa, FL, USA

Synonyms

Stress reactivity; Stress responses

Definition

Physiological reactivity involves bodily changes in response to stressful stimuli or events.

The classic features of physiological reactivity are increases in sympathetic nervous system and hypothalamic-pituitary-adrenal axis (HPA) activity, often referred to as the "fight-or-flight" response (Cannon, 1932). These responses include increases in heart rate, blood pressure, cardiac contractility, and cortisol. Changes in parasympathetic nervous system activity, immune function (Cacioppo, 1994), and non-HPA endocrine function (Taylor et al., 2000) can also occur. For reactivity to serve as a meaningful metric, stress responses must be compared to an unstressed resting state, or baseline, to control for wide individual differences in resting levels (Jennings et al., 1992). Physiological reactivity is most often assessed in response to acute stressors on the order of minutes (Steptoe & Vögele, 1991).

Cross-References

- ▶ Mental Stress
- ▶ Perceptions of Stress
- ▶ Stress
- ▶ Stress Responses

References and Readings

Cacioppo, J. T. (1994). Social neuroscience: Autonomic, neuroendocrine, and immune responses to stress. *Psychophysiology, 31*, 113–128.

Cacioppo, J. T., Tassinary, L. G., & Berntson, G. G. (2007). *Handbook of psychophysiology*. New York: Cambridge University Press.

Cannon, W. B. (1932). *The wisdom of the body*. New York: Norton.

Jennings, J. R., Kamarck, T., Stewart, C., Eddy, M., & Johnson, O. (1992). Alternate cardiovascular baseline assessment techniques: Vanilla or resting baseline. *Psychophysiology, 29*, 742–750.

McEwen, B. S. (1998). Stress, adaptation, and disease: Allostasis and allostatic load. *Annals of the New York Academy of Sciences, 840*, 33–44.

Sapolsky, R. M. (1994). *Why zebras don't get ulcers*. New York: Holt.

Steptoe, A., & Vögele, C. (1991). Methodology of mental stress testing in cardiovascular research. *Circulation, 83*, II-14–II-24.

Stern, R. M., Ray, W. J., & Quigley, K. S. (2001). *Psychophysiological recording*. New York: Oxford University Press.

Taylor, S. E., Klein, L. C., Lewis, B. P., Gruenewald, T. L., Gurung, R. A., & Updegraff, J. A. (2000). Biobehavioral responses to stress in females: Tend-and-befriend, not fight-or-flight. *Psychological Review, 107*, 411–429.

Physiotherapy

- ▶ Physical Therapy

Pickering, Thomas G.

Eoin O'Brien
The Conway Institute, University College Dublin, Belfield, Dublin, Ireland

Biographical Information

Dr. Thomas George Pickering

Thomas George Pickering was born in the United Kingdom in 1940. He was educated at Bryanston School in Blandford, England, and went on to study medicine at Trinity College, Cambridge,

Editors' Note: Dr. Pickering passed away in 2009. The American Society of Hypertension has established the Thomas Pickering Memorial Lecture, the first of which was delivered at the Scientific Meeting in May 2012.

and the Middlesex Hospital Medical School, London, where he graduated in 1966.

Pickering's early postgraduate years were spent at the Middlesex and the Radcliffe Infirmary. He became a member of the Royal College of Physicians of London in 1968, becoming a fellow in 1980. He received a Ph.D. degree from Oxford University in 1970. In 1972, he went to New York to take up appointments as Associate Physician at the Rockefeller University Hospital and Assistant Professor at Cornell University. He spent 2 years as Assistant Professor at the Rockefeller University working with Neal Miller on biofeedback mechanisms. He was appointed Assistant Physician to the New York Hospital in 1974. He returned to theRadcliffe Infirmary in 1974 to work with Peter Sleight.

Pickering's earliest hypertension research at Oxford focused on baroreceptor function, the autonomic nervous system, and the emerging class of cardiovascular medications known as the adrenoreceptor blockers. Although he remained in Oxford from 1974 to 1976, the possibility of being able to work as both a practicing physician and a clinical investigator drew him back to New York City and Cornell University Medical College, where he spent more than 20 years in a productive career in cardiovascular behavioral medicine, clinical hypertension, and blood pressure measurement research. In 2000, he became Director of Behavioral Cardiovascular Health and the Hypertension Program at the Cardiovascular Institute of Mount Sinai Medical Center, and in 2003 he moved to Columbia University Medical College as Professor of Medicine and Director of the Behavioral Cardiovascular Health and Hypertension Program.

Major Accomplishments

Pickering practiced "translational research" long before the term became fashionable, translating his clinical observations in medical practice to research endeavors throughout his career. He made important observations on the relationship between renovascular disease and cardiovascular complications, and the impact of renal revascularization. He also observed that anxiety, perceived stress, job strain, and the medical care environment itself induced hypertension in some individuals who otherwise would not have been classified as hypertensive. He had a deep belief that psychosocial mechanisms played an important role in the pathogenesis of cardiovascular disorders.

Pickering's research interests also focused on new methods of blood pressure measurement, particularly the use of 24-h ambulatory monitoring and self monitoring. His identification of the importance of the circadian variability of blood pressure led to the study of the psychological influences of work and stress in hypertension and heart disease, a field in which he was regarded as the world authority. At a clinical level he studied the influence of sleep in hypertension and methods of improving adherence to medication in order to obtain better control of elevated blood pressure. Pickering also studied the application of non-pharmacological approaches to the management of hypertension, publishing prolifically in these areas. He published a total of almost 500 original research articles in a clear and concise manner. Several highly acknowledged experts acknowledge his leadership in coining the terms "white-coat hypertension" and "masked hypertension," conditions which he not only described, but did much to explain with well-designed studies. This work systematically investigated whether white-coat hypertension was benign, and whether masked hypertension enhanced risk.

Pickering served on many governmental and academic bodies including the American Society of Hypertension; the National Heart, Lung, and Blood Institute; the International Society of Hypertension; the American Heart Association; the US Cardiorenal Advisory Committee; the US Food and Drug Administration; and the Committee on Gulf War and Stress of the Institute of Medicine. As a senior editor of the Journal of Clinical Hypertension he wrote numerous editorials. In his later years he came to feel strongly that self-monitoring of blood pressure, as well as ambulatory blood pressure monitoring, should be covered by third-party insurance companies for patient hypertension care. In 2002, after 19 years

of lobbying, a scientific meeting of the Center for Medicare and Medicaid Services was established to develop a national policy for coverage of ambulatory blood pressure monitoring for patients with white-coat hypertension. At Pickering's suggestion, not only was evidence of the benefits of ambulatory monitoring presented at this meeting but the patients who had benefited gave testimony. The patients' stories had a substantial impact, and the meeting voted unanimously to approve national coverage for ambulatory monitoring.

Cross-References

- ▶ Adherence
- ▶ Hypertension

References and Readings

Devereux, R. B., Pickering, T. G., Harshfield, G. A., Kleinert, H. D., Denby, L., Clark, L., et al. (1983). Left ventricular hypertrophy in patients with hypertension: Importance of blood pressure response to regularly recurring stress. *Circulation, 68*, 470–476.

O'Brien, E. (2009). In memoriam. *Journal of Hypertension, 27*, 1715–1716.

O'Brien, E., & White, W. B. (2010). Thomas George Pickering, 1940–2009. Special Memorial Tribute Issue. *Blood Pressure Monitoring, 15*, 67–114.

Pickering, T. G. (1992). The ninth Sir George Pickering memorial lecture: Ambulatory monitoring and the definition of hypertension. *Journal of Hypertension, 10*, 401–409.

Pickering, T. G., Coats, A., Mallion, J. M., Mancia, G., & Verdecchia, P. (1999). Blood pressure monitoring: Task force V-white-coat hypertension. *Blood Pressure Monitoring, 4*, 333–341.

Pickering, T. G., Hall, J. E., Appel, L. J., Falkner, B. E., Graves, J., Hill, M. N., et al. (2005). Recommendations for blood pressure measurement in humans and experimental animals: Part 1-blood pressure measurement in humans: A statement for professionals from the Subcommittee of Professional and Public Education of the American Heart Association Council on High Blood Pressure Research. *Hypertension, 45*, 142–161.

Pickering, T. G., Harshfield, G. A., Kleinert, H. D., Blank, S., & Laragh, J. H. (1982). Blood pressure during normal daily activities, sleep, and exercise: Comparison of values in normal and hypertensive subjects. *Journal of the American Medical Association, 247*, 992–996.

Pickering, T. G., Herman, L., Devereux, R. B., Sotelo, J. E., James, G. D., Sos, T. A., et al. (1988). Recurrent pulmonary oedema in hypertension due to bilateral renal artery stenosis: Treatment by angioplasty or surgical revascularization. *Lancet, 2*, 551–552.

Pickering, T. G., James, G. D., Boddie, C., Harshfield, G. A., Blank, S., & Laragh, J. H. (1988). How common is white coat hypertension? *Journal of the American Medical Association, 259*, 225–228.

Pickering, T. G., Miller, N. H., Ogedegbe, G., Krakoff, L. R., Artinian, N. T., & Goff, D. (2008). Call to action on use and reimbursement for home blood pressure monitoring: Executive summary-a joint scientific statement from the American Heart Association, American Society of Hypertension, and Preventive Cardiovascular Nurses Association. *Hypertension, 26*, 2259–2267.

Pickering, T. G., Shimbo, D., & Haas, D. (2006). Ambulatory blood pressure monitoring. *The New England Journal of Medicine, 354*, 2368–2374.

Pickering, T. G., Sos, T. A., Vaughan, E. D., Case, D. B., Sealey, J. E., Harshfield, G. A., et al. (1984). Predictive value and changes in renin secretion in hypertensive patients with unilateral renovascular disease undergoing successful renal angioplasty. *The American Journal of Medicine, 76*, 398–404.

Pickering, T. G., & White, W. B. (2008). American Society of Hypertension position paper: Home and ambulatory blood pressure monitoring-when and how to use self (home) and ambulatory blood pressure monitoring. *Journal of Clinical Hypertension, 10*, 850–855.

White, W. B. (2009). In memoriam. Thomas G. Pickering 1940–2009. *Hypertension, 54*, 917–918.

Pitocin

- ▶ Oxytocin

Pituitary-Adrenal Axis

Jennifer Heaney
School of Sport and Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

Synonyms

Hypothalamic-pituitary-adrenal axis

Definition

The pituitary-adrenal axis comprises the pituitary and adrenal glands. The main interactions that take place between these two endocrine glands are as part of the hypothalamic-pituitary-adrenal axis (HPA axis). Corticotrophin-releasing hormone (CRH) stimulates the secretion of adrenocorticotropic hormone (ACTH) from the anterior pituitary; this in turn stimulates the release of cortisol from the adrenal cortex (Martin, Reichlin, & Brown, 1997). More detail on the pituitary-adrenal axis and its function in the HPA axis can be found in Widmaier, Raff, and Strang (2004), O'Riordan, Malan, and Gould (1988), and Greenspan and Forsham (1983).

References and Readings

Greenspan, F. S., & Forsham, P. H. (1983). *Basic and clinical endocrinology*. Los Altos, CA: Lange Medical Publications.

O'Riordan, F. L. H., Malan, P. G., & Gould, R. P. (1988). *Essentials of endocrinology* (2nd ed.). Oxford: Blackwell Scientific Publications.

Widmaier, E. P., Raff, H., & Strang, K. T. (2004). *Vander, Sherman, and Luciano's human physiology: The mechanism of body function*. New York: McGraw-Hill.

Martin, J. B., Reichlin, S., & Brown, G. M. (1997). *Clinical neuroendocrinology*. Philadelphia: F.A. Davis.

Placebo and Placebo Effect

Magne Arve Flaten[1] and Mustafa al'Absi[2]
[1]Department of Psychology, University of Tromsø, Tromsø, Norway
[2]University of Minnesota Medical School, University of Minnesota, 235 School of Medicine, Duluth, MN, USA

Synonyms

Conditioned response; Context effect; Expectancy effect

Definition

A placebo is any inert substance, procedure, apparatus, or similar, that alone has no effect in the body. The placebo effect is the psychological and/or physiological response to the placebo when it is administrated with a suggestion that the substance, procedure, apparatus, or similar will have an effect in the individual.

Description

Placebo comes from the Latin word "placere" which means "I shall please" and has been used in medicine to describe treatments that pleases the patient, but that has no specific effect on the symptom.

A placebo effect may occur when a substance, procedure, or other stimulus is administrated to a person, together with a suggestion that this will reduce or heal a symptom. The placebo effect may occur whether the substance or procedure is effective in reducing the symptom or not, as effective treatments may also induce placebo effects. Both the substance or procedure and the suggestion are necessary to produce a placebo effect. Administration of a pill without the suggestion, or the suggestion without the pill, will not generate a placebo effect. Thus, a placebo alone is not sufficient to generate a placebo effect, as it must be accompanied by a suggestion. The suggestion can be in the form of verbal information, e.g., "this capsule may contain a painkiller," that induces an expectation that the treatment will reduce the symptom. The expectation has been found to be correlated to the actual placebo response. Thus, it could be argued that the term "expectancy effect" should replace the vague and often misinterpreted term "placebo effect." Placebo effects may also be implicit and communicated through contextual factors. For example, a "pill" provided by a physician may have a stronger "placebo" effect than a pill provided by a nonmedical professional.

Placebo effects may also occur as a result of a conditioning process. For example, after the individual has been subject to effective treatment,

an association between treatment and its outcome may develop. The shape or color of tablets the patient has taken in the past to reduce pain or other problems may be associated with the drug effect, since the shape or color of the tablet (the conditioned stimulus) is reliably followed by reduction in the symptom (the unconditioned response). The features of the tablet can be associated with the effect of the drug in the central nervous system (the unconditioned stimulus) and come to elicit conditioned decreases in the symptom. Placebo effects induced by actual experience with the drug effect are stronger than placebo effects induced by verbal information alone (Flaten et al., 1999). For some symptoms, the conditioned stimulus exerts its effect by inducing an expectation, for other symptoms unconscious, automatic processes seem to be responsible for the placebo effect (Benedetti et al., 2003).

The placebo effect is observed as a reduction in a symptom in a group that receives placebo treatment with suggestion, compared to a natural history control group that receives no treatment and no suggestion. The natural history group controls for normal variations in the symptom due to normal healing processes or other changes that are not due to expectations of treatment effects. Response bias is serious problem, as subjects may feel obliged or may have a tendency to report on the symptom in accordance with the suggestion provided by the experimenter, without there being any improvement in the symptom, and studies must control for demand characteristics. Placebo analgesia, a reduction in pain due to expectations of having received a painkiller, is the most studied form of placebo effect. Pain is accompanied by changes in autonomic function, by well-defined changes in the event-related potential to painful stimulation, and by a cerebral response reliably involving the somatosensory cortex, the anterior cingulate cortex, and the insula. Placebo analgesia is accompanied by reduction in these correlates to the reported pain, indicating that the placebo effect is due to changes in the brain's response to the pain signal and not solely to a response bias, although this may contribute.

Multiple neurobiological pathways are thought to be involved in mediating effects of placebo, including those related to the endogenous opioid system and stress response systems. Placebo analgesia has been found to be reversible or partly reversible by the opioid antagonist naloxone. This is further evidence that the placebo effect is not due to a response bias, and indicates that expectations of pain relief activate the mid-brain descending pain inhibitory system. Wager et al. (2004) found that expectations of pain relief were associated with increased activity in the periaqueductal gray in the midbrain, a nucleus that controls pathways descending to the rostral ventral medulla and the dorsal horn. There, this pathway inhibits pain transmission, resulting in a reduced pain signal to the brain areas mentioned above, with a consequent reduction in pain sensation. Injections of opioids into the periaqueductal gray and the ventral medulla reduce pain, suggesting that endogenous opioids reduce pain via the same descending system. Eippert, Finsterbusch, Bingel, and Buchel (2009) furthermore showed that placebo analgesia involved dorsal horn activity, indicating that placebo analgesia is due to expectation of pain relief that in turn activates the descending endorphin-mediated pain inhibitory system.

In addition to pain, placebo effects are documented in, e.g., Parkinson's disease, depression, cardiac heart disease, sexual function, and airway resistance in asthmatic patients. This implies that there are several placebo effects with different underlying mechanisms. In Parkinson's disease, placebo treatment has been found to increase dopamine release in the basal ganglia, thereby improving motor function. Increased dopamine release has also been implicated in placebo analgesia. Most studies have found only a partial reversal of placebo analgesia by naloxone, whereas some studies have found no effect of naloxone on the placebo analgesic response. Thus, non-opioid mechanisms play a role in placebo analgesia. It has been hypothesized that placebo treatment leads to reduced anxiety or negative emotions, that could be a common factor across

placebo effects, and that different placebo treatments could activate additional disease-specific mechanisms.

Placebo effects can also be observed in the response to drugs of habitual use or abuse. Caffeine, e.g., increases arousal and decreases reaction time and fatigue, and habitual coffee drinkers who believe they receive caffeine but get a placebo, respond with caffeine-like reactions of increased alertness and faster reaction times, and increased dopamine release in areas associated with reinforcement. Subjects who believe they drink alcohol but receive placebo drinks report symptoms of intoxication and display deteriorated performance on cognitive and motor tasks. Likewise, subjects who believe they receive amphetamine but receive a placebo still report amphetamine-like effects.

Placebos are used as controls in randomized clinical trials, where the effect of the intervention is defined as the improvement over placebo. Since the participant does not know to which arm of the trial he or she has been randomized to, expectations of drug effects are the same in both arms, and expectations of receiving effective treatment are lower than in ordinary clinical practice or in experimental studies on the placebo effect. Thus, placebo effects are smaller or absent in clinical trials (Hróbjartsson & Gøtzscke, 2001). Drugs or other treatments may have noticeable subjective effects like drowsiness or nausea that may inform the participant that he or she has received active medication, thereby unblinding the trial for the participants in the active arm. To solve this problem active placebos are used, i.e., drugs that have similar subjective effects to the tested drug, but that have no effect on the symptom, instead of inactive placebos.

Cross-References

- ▶ Functional Magnetic Resonance Imaging (fMRI)
- ▶ Pain
- ▶ Randomized Controlled Trial

References and Readings

Benedetti, F., Pollo, A., Lopiano, L., Lanotte, M., Vighetti, S., & Rainero, I. (2003). Conscious expectation and unconscious conditioning in analgesic, motor, and hormonal placebo/nocebo responses. *Journal of Neuroscience, 23*(10), 4315–4323.

Eippert, F., Finsterbusch, J., Bingel, U., & Buchel, C. (2009). Direct evidence for spinal cord involvement in placebo analgesia. *Science, 326*(5951), 404–404.

Flaten, M. A., Simonsen, T., & Olsen, H. (1999). Drug-related information generates placebo and nocebo responses that modify the drug response. *Psychosomatic Medicine, 61*(2), 250–255.

Hróbjartsson, A., & Gøtzscke, P. C. (2001). Is the placebo powerless? *The New England Journal of Medicine, 344*(21), 1594–1602.

Wager, T. D., Rilling, J. K., Smith, E. E., Sokolik, A., Casey, K. L., Davidson, R. J., et al. (2004). Placebo induced changes in fMRIin the anticipation and experience of pain. *Science, 303*(5661), 1162–1167.

Plasma Lipid

- ▶ Lipid

Plasminogen Activator Inhibitor (PAI-1)

Jonathan Newman
Columbia University, New York, NY, USA

Definition

The rupture of an atherosclerotic plaque is a recognized key event in acute ischemic syndromes, such as myocardial infarctions. The intravascular thrombotic response to a ruptured plaque is a complex cascade of thrombogenic (clot-forming) and thrombolytic (clot-dissolving) mechanisms. A key component of the thrombotic cascade is plasminogen activator inhibitor type 1 (PAI-1). PAI-1 inhibits the activation of plasminogen by tissue plasminogen activator (tPA) and urokinase (uPA) and, hence, inhibits clot lysis.

PAI-1 is a single-chain glycoprotein composed of nearly 380 amino acids. It is a member of

the serine proteases family and is synthesized by vascular endothelium and smooth muscle cells in both normal and atherosclerotic arteries. By synthesizing molecules like PAI-1, arterial smooth muscle cells can prevent bleeding from small vascular injuries; congenital deficiencies of PAI-1 are a rare cause of abnormal bleeding.

Associations between PAI-1 and incident or recurrent coronary heart disease (CHD) have been demonstrated, but not definitively proven. While circadian variation in PAI-1 levels (highest in the morning) may correlate to circadian patterns of myocardial infarction (highest in the morning), the relationship of PAI-1 to CHD, independent of other prothrombotic risk factors for coronary heart disease, such as diabetes or insulin resistance, has not been clearly shown. Additionally, individuals with specific genetic variations leading to increased PAI-1 production have not been clearly shown to have an increased risk of CHD. Lastly, while important in constitutive pathways of fibrinolysis, it is not clearly known whether the increased PAI-1 expression seen following plaque rupture and thrombosis is a causal pathway or an effect of the inciting event.

References and Readings

Humphries, S. E., Panahloo, A., Montgomery, H. E., Green, F., & Yudkin, J. (1997). Gene-environment interaction in the determination of levels of haemostatic variables involved in thrombosis and fibrinolysis. *Thrombosis and Haemostasis, 78*(1), 457–461.

Juhan-Vague, I., Pyke, S. D., Alessi, M. C., Jespersen, J., Haverkate, F., & Thompson, S. G. (1996). Fibrinolytic factors and the risk of myocardial infarction or sudden death in patients with angina pectoris. ECAT Study Group. European Concerted Action on Thrombosis and Disabilities. *Circulation, 94*(9), 2057–2063.

Lee, M. H., Vosburgh, E., Anderson, K., & McDonagh, J. (1993). Deficiency of plasma plasminogen activator inhibitor 1 results in hyperfibrinolytic bleeding. *Blood, 81*(9), 2357–2362.

Platelet Plug

▶ Fibrinogen

Pleasant Affect

▶ Positive Affectivity

PMD

▶ Primary Care Physicians

Point of Care Testing

▶ Glucose Meters and Strips

Polymorphism

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

The term “polymorphism” refers to a locus with two or more alleles. The translation from Latin is “multiple forms.” It is therefore a difference in DNA sequence at a particular locus.

Cross-References

▶ Allele
▶ DNA
▶ Locus
▶ Single Nucleotide Polymorphism (SNP)

References and Readings

Britannica. (2009). *The Britannica guide to genetics* (Introduction by Steve Jones). Philadelphia: Running Press.

Polysomnogram

► Polysomnography

Polysomnography

Christopher Kline
Department of Psychiatry, School of Medicine, University of Pittsburgh, Pittsburgh, PA, USA

Synonyms

Polysomnogram; Sleep study

Definition

Polysomnography is the simultaneous recording of numerous physiological signals during attempted sleep, including activity of the brain, heart, eyes, and muscles. Polysomnography is considered the gold standard for the objective assessment of sleep and diagnosis of many clinical sleep disorders.

Description

Polysomnography (PSG), termed as such because of the multiple physiological signals that are recorded, has been employed for the characterization of sleep/wake status since the early 1900s. Measurement of the brain's electrical activity, or electroencephalography (EEG), is the primary physiological signal assessed during PSG. Concurrent measurement of eye movement (electrooculography, or EOG), submentalis muscle activity (electromyography, or EMG), and cardiac activity (electrocardiography, or ECG) are essential for the discrimination of specific stages of sleep. Besides the basic recording montage of EEG, EOG, EMG, and ECG, supplemental measures can be added to PSG for the assessment of respiratory and limb movement activity during sleep. See Fig. 1 for an example of a standard polysomnogram.

Polysomnography is most commonly performed in a sleep laboratory. However, due to digitization of sleep signals, portability of data collection units, and increased data storage capacities, full-scale polysomnography is now able to be performed in the home. Home-based PSG allows for a more ecological assessment of sleep, since patients often report altered sleep due to the artificial sleep laboratory environment (Edinger et al., 2001).

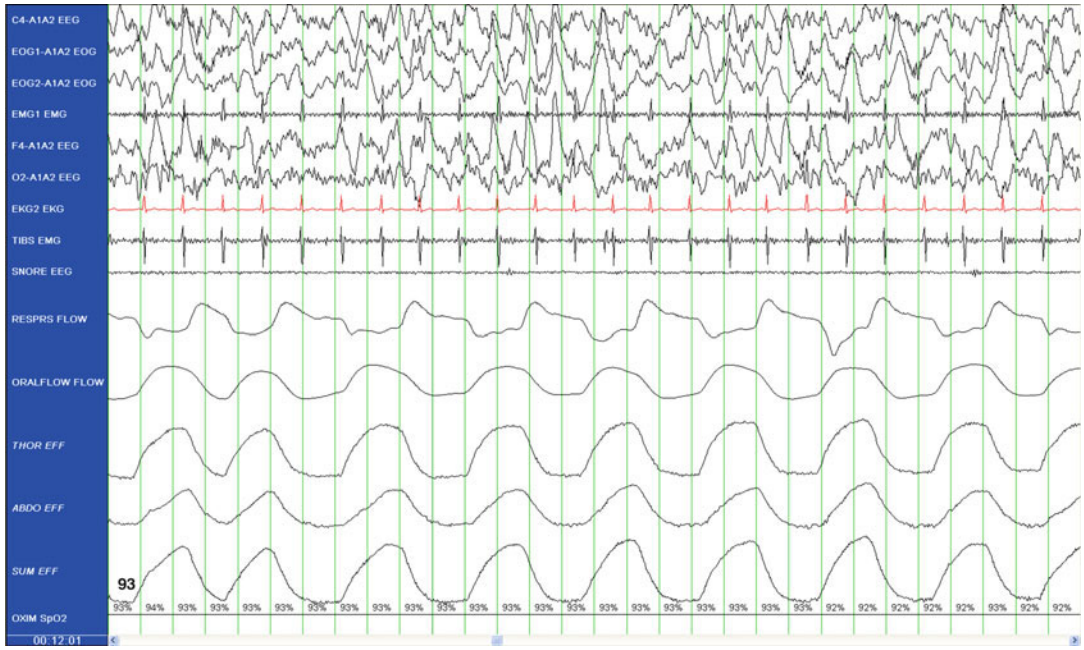
Indications

Polysomnography can be conducted for the objective assessment of sleep for any individual, regardless of whether a sleep disorder is suspected. However, PSG is indicated for the diagnosis of sleep-disordered breathing, narcolepsy, certain types of parasomnias (e.g., seizure disorders) and periodic limb movement disorder (Kushida et al., 2005).

Preparation

Prior to electrode placement, sites should be briefly cleaned with alcohol and an abrasive skin preparation to minimize impedance levels. Electrode cups are then filled with conducting paste and affixed to the proper site with medical tape or electrode paste backed by gauze. Alternatively, some sleep laboratories attach scalp electrodes with collodion glue. Electrode position for the electroencephalogram follows the International 10–20 system (Harner & Sannitt, 1974). A typical EEG montage for sleep includes bilateral electrodes in the occipital, central, and frontal regions, with bilateral mastoid process electrodes as reference electrodes (Iber, Ancoli-Israel, Chesson, & Quan, 2007).

For the electrooculogram, electrodes should be placed 1 cm lateral to and above the outer canthus of the right eye, and 1 cm lateral to and below the outer canthus of the left eye. For the electromyogram, electrodes are placed to assess the activity of the submentalis muscle of the chin; when limb movements need to be assessed, EMG activity of the anterior tibialis is measured with two electrodes on each leg.



Polysomnography, Fig. 1 A 30-s epoch of a standard clinical polysomnogram. Channels are listed on the left column. Electroencephalographic (C4-A1A2, F4-A1A2, O2-A1A2), electrooculographic (EOG1-A1A2, EOG2-A1A2), and electromyographic (EMG1) channels are shown here and are needed for the identification of sleep stages. Additional channels are included here for monitoring leg movements (TIBS; anterior tibialis EMG activity), cardiac activity (EKG2), snoring (SNORE), breathing patterns (RESPRS: nasal pressure; ORALFLOW: oronasal thermistor), respiratory effort (THOR EFF: thoracic effort; ABDO EFF: abdominal effort; SUM EFF: sum of thoracic and abdominal effort channels), and oxyhemoglobin saturation (OXIM SpO2)

A basic electrocardiogram lead, with torso electrodes corresponding to right arm and left leg placement, provides an assessment of cardiac activity. When sleep-disordered breathing is suspected, an oronasal thermal sensor and nasal cannula pressure transducer are used to detect airflow, respiratory effort is assessed with either esophageal manometry or (more commonly) inductance plethysmography belts around the thorax and abdomen, and measurement of oxyhemoglobin saturation is obtained with finger pulse oximetry. Sensors that measure snoring intensity and track body position through the night are commonly added to the recording montage.

Recording and Analysis

Following patient preparation, impedance checks and biocalibrations are performed to assure signal quality. Ideally, the electrode impedance for facial and scalp electrodes should not exceed 5 kΩ (Iber et al., 2007). Moreover, biocalibrations are a set of instructions delivered to the patient to verify signal quality. Recording system calibration is also undertaken before commencement of the sleep study.

With EEG, EOG, EMG, and ECG, changes in electrical potential are detected from electrodes at the skin surface, with the electrical potential at one site measured in relation to its referent electrode. Physiological signals are amplified, digitized, and then displayed for inspection and analysis. During digitization, signals are sampled at a specific rate (a minimum of 200 Hz is recommended for the recording of EOG, EEG, EMG, and ECG), with low- and high-frequency filter settings used to reduce signal artifact. For EOG, EEG, and EMG, signals are measured and displayed in microvolts, whereas ECG signals are displayed in millivolts.

Standardized scoring guidelines for sleep were first established in 1968 (Rechstschaffen & Kales, 1968) and updated in 2007 (Iber et al., 2007). Separate guidelines for scoring pediatric sleep were included in the 2007 update (Grigg-Damberger et al., 2007; Iber et al., 2007). Scoring of sleep stages occurs in 30-s epochs, with each epoch assigned a specific sleep stage that occupies the majority of the epoch.

Electroencephalographic activity is the primary characteristic that distinguishes sleep stages, with EOG and EMG being essential for the detection of rapid eye movement (REM) sleep. In adults, five distinct stages can be scored: wakefulness (W), stage 1 non-REM sleep (N1), stage 2 non-REM sleep (N2), stage 3 non-REM sleep (N3), and REM sleep (R). Stage W is characterized by a predominance of low-voltage, high-frequency EEG (most often alpha and beta frequency) and eye blinks, with elevated chin EMG activity. In stage N1 sleep, alpha EEG activity transitions to a slightly lower-frequency EEG (typically theta frequency), often accompanied by slow rolling eye movements, vertex sharp waves (sharply negative, transient waveforms distinguishable from background activity), and an attenuation of chin EMG from stage W. Stage N2 sleep is characterized by the presence of K complexes (high-amplitude biphasic waveforms typically $\geq$0.5 s in duration) or sleep spindles (rhythmic bursts of sigma-frequency EEG for $\geq$0.5 s) over a background of low-voltage, high-frequency EEG, along with a lack of EOG activity and further attenuation of chin EMG activity. Sleep is considered to be stage N3 sleep, also known as slow-wave sleep, when $\geq$20% of an epoch contains slow wave activity (delta EEG frequency with amplitude $>$75 μV). Finally, stage R sleep is characterized by low-amplitude, high-frequency EEG activity similar to wakefulness and N1 sleep, but with irregular rapid eye movements and minimal chin EMG activity (Iber et al., 2007).

The duration, percentage of total sleep time, and distribution of sleep stages across the night are typically retained for analysis. In addition, typical parameters measured from PSG-scored sleep include sleep onset latency (i.e., the length of time it takes to fall asleep), wakefulness after sleep onset (i.e., the amount of wakefulness that occurs after initially falling asleep), total sleep time (i.e., the total amount of sleep obtained), and sleep efficiency (i.e., the ratio of time spent asleep to the total duration of attempted sleep). In addition to these traditional measures of sleep, quantitative assessment of the sleep EEG is possible, in which the spectral content of the sleep EEG is decomposed to reveal the power of specific EEG frequency bands.

Additional guidelines are present when assessing respiratory events and periodic limb movements (Iber et al., 2007). An apnea is characterized by a $\geq$90% reduction in airflow for at least 10 s, whereas a hypopnea is defined by a $\geq$30% reduction in airflow for $\geq$10 s with a $\geq$4% decrease in oxyhemoglobin saturation. An apnea that is accompanied by inspiratory effort (as assessed by esophageal manometry or inductive plethysmography) is considered to be obstructive, whereas an apnea without inspiratory effort is classified as a central apnea. An apnea that is without inspiratory effort initially but resumes in the latter portion of the event is considered a mixed apnea. Significant leg movements during sleep are characterized by elevated tibialis anterior EMG activity that lasts for 0.5–10 s that is not immediately preceded or followed by a respiratory event.

Limitations

Despite its recognition as the gold standard for the objective assessment of sleep, there are many situations in which PSG might not be desirable and/or feasible. These include assessment of sleep across multiple nights, 24-h recording of sleep/wake activity, and assessment of a large cohort of patients. Under these conditions, the use of PSG may be excessive in cost, labor, or patient burden. Wrist actigraphy may be an acceptable alternative in these situations.

In addition, the "first night effect" is a well-documented phenomenon in which disturbed sleep is reported when assessed by PSG, presumably due to the discomfort and burden imposed by the numerous wires and electrodes and unfamiliar sleep environment (if conducted in the

laboratory). Although in some research studies this sleep disturbance has been shown to take multiple nights of PSG recording before resolution, most studies address the first night effect by conducting at least two consecutive nights of PSG recording and discarding the data from the first night in subsequent analyses.

Although considered an objective measure of sleep, the scoring of PSG is subject to human influence and therefore some degree of subjectivity. Although most sleep laboratories employ trained polysomnographic technicians to evaluate sleep studies, significant between-technician variation is possible. For that reason, quantitative EEG, an automated procedure in which the spectral content of the sleep EEG is analyzed across multiple frequency bandwidths, may provide unique information.

Cross-References

- ▶ Brain Wave
- ▶ Non-REM Sleep
- ▶ REM Sleep
- ▶ Sleep
- ▶ Sleep and Health
- ▶ Sleep Apnea
- ▶ Sleep Architecture

References and Readings

Butkov, N., & Lee-Chiong, T. (Eds.). (2007). *Fundamentals of sleep technology*. Philadelphia: Lippincott Williams & Wilkins.

Edinger, J. D., Glenn, D. M., Bastian, L. A., Marsh, G. R., Daile, D., Hope, T. V., et al. (2001). Sleep in the laboratory and sleep at home II: Comparisons of middle-aged insomnia sufferers and normal sleepers. *Sleep, 24*, 761–770.

Grigg-Damberger, M., Gozal, D., Marcus, C. L., Quan, S. F., Rosen, C. L., Chervin, R. D., et al. (2007). The visual scoring of sleep and arousal in infants and children. *Journal of Clinical Sleep Medicine, 3*, 201–240.

Harner, P. F., & Sannitt, T. (1974). *A review of the international ten-twenty system of electrode placement*. Quincy, MA: Grass Instrument Company.

Iber, C., Ancoli-Israel, S., Chesson, A., & Quan, S. F. (2007). *The AASM manual for the scoring of sleep and associated events: Rules, terminology and technical specifications* (1st ed.). Westchester, IL: American Academy of Sleep Medicine.

Keenan, S., & Hirshkowitz, M. (2011). Monitoring and staging human sleep. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5th ed., pp. 1602–1609). St. Louis, MO: Elsevier.

Kushida, C. A., Littner, M. R., Morgenthaler, T., Alessi, C. A., Bailey, D., Coleman, J., Jr., et al. (2005). Practice parameters for the indications for polysomnography and related procedures: An update for 2005. *Sleep, 28*, 499–521.

Rechtschaffen, A., & Kales, A. (Eds.). (1968). *A manual of standardized terminology, techniques and scoring system for sleep stages in Human subjects*. Los Angeles: UCLA Brain Information Service/Brain Research Institute.

Polyunsaturated Fats

▶ Fat, Dietary Intake

Polyunsaturated Fatty Acids

▶ Fat: Saturated, Unsaturated

Population Health

Chris Zehr[1] and Peter A. Hall[2]
[1]Department of Health Studies and Gerontology, University of Waterloo, Waterloo, ON, Canada
[2]Faculty of Applied Health Sciences, University of Waterloo, Waterloo, ON, Canada

Definition

Population health is a general approach to assessing and managing health at the level of the whole population. Reduction of health-care inequities, prevention of illness, and contextual improvement are all central objectives of the population health approach.

Description

Population health is an approach that aims to improve the health of entire population with an emphasis on understanding and decreasing health inequities among groups of people within the population (Hertzman, Frank, & Evans, 1994). This approach deviates from a traditional biomedical approach that focuses treatment at an individual level and instead targets group-level phenomena through the implementation of broad-based and widely diffusible health interventions (Jeffery, 1989; Rose, 1985). Rather than focusing on treating illnesses after they emerge, population health interventions are characterized by a strong emphasis on primary prevention, or preventing illness before it develops. As such, modifiable risk factors for illness are a common focus of population health intervention such as smoking, obesity, high blood pressure, physical inactivity, and unhealthy diet as well as preventable disease such as heart disease, stroke, certain cancers, and diabetes (Wanless, 2003).

A defining feature of population health is an emphasis on upstream determinants of illness rather than proximal causes (Hawe, 2007; Link & Phelan, 1995). For example, it has been established that cigarette smoking is associated with myriad negative health consequences. A population health approach would seek to elucidate the conditions and factors upstream that put individuals at greater risk of smoking (e.g., low education levels, low income levels, peer group influence). By identifying upstream determinants of illness, population health interventions are developed that can target these more distal factors.

The population health approach considers health status as a product of a diverse range of factors that include, but are not limited to, individual behavior and life-style; biological and genetic factors; early development; the physical, social, and economic environment; and the health-care system itself (Evans & Stoddart, 1990; Kindig & Stoddart, 2003). Rather than viewing these influences as distinct and individual causes of illness and/or health, these and any other health-determining factors are viewed to interact with each other to determine health.

Population health research has highlighted social conditions as a particularly important determinant of health (Link & Phelan, 1995; Marmot, 2003). Socioeconomic status (SES), a measure of income and social position, has received a significant amount of attention. A robust association exists that suggests that those relatively higher on the socioeconomic gradient tend to experience better health and a longer life expectancy than those lower on the gradient (Hertzman et al., 1994; Lynch, Smith, Kaplan, & House, 2000). Due to the emphasis on minimizing inequities in health, a central focus of population has been to determine the nature of the SES-longevity relationship and how to mitigate it.

Because population health research seeks to impact the greatest number of people possible, interventions based on population health research are often implemented through policy, but also include media campaigns that serve to educate and make individuals aware of certain health-protective behaviors while warning of health-risk behaviors (Hawe, 2007). Population health interventions can also be delivered as programming in settings such as schools, churches, and workplaces. Moreover, when considering the diverse array of determinants of health, population health interventions have the potential to span different sectors not traditionally associated with health intervention (i.e., public transportation, education, agriculture, urban planning) to address a particular health issue.

A criticism of the population health approach is that while biomedical approaches to health care provide tailored treatment for individuals with specific health conditions, population health interventions generally work in a broad stroke manner targeting groups of individuals for whom intervention may not be necessary or beneficial. For example, using data from the Framingham Heart Study, Rose (1985) calculated that even a 10 mmHg lowering of the blood pressure distribution of a population could result in 30% reduction in mortality attributed to blood pressure. Though this may be potentially beneficial for the overall health of a population, this may provide little personal benefit to each individual. This has been termed the "prevention paradox" (Rose).

Despite this, Rose (1985) notes that population strategies can be powerful in their influence. Given that population health approaches are concerned with affecting large groups of people, they have the capacity to change social norms so that a health-risk behavior becomes less socially acceptable, or alternatively, that a health-protective behavior becomes more accepted.

Cross-References

- ▶ Health Promotion
- ▶ Public Health

References and Readings

Evans, R. G., Barer, M. L., & Marmor, T. R. (Eds.). (1994). *Why are some people healthy and others are not? The determinants of health of populations*. Hawthorne: Aldine De Gruyter.

Evans, R. G., & Stoddart, G. L. (1990). Producing health, consuming health care. *Social Science & Medicine, 31*(12), 1347–1363.

Frankish, C. J., Green, L. W., Ratner, P. A., Chomik, T., & Larsen C. (1996). *Health impact assessment as a tool for population health promotion and public policy*. A Report Submitted to the Health Promotion Division of Health Canada. Institute of Health Promotion Research, University of British Columbia.

Glouberman, S., & Miller, J. (2003). Evolution of the determinants of health, health policy, and health information systems in Canada. *American Journal of Public Health, 93*(3), 388–392.

Hawe, P. (2007). What is population health? Retrieved from http://www.ucalgary.ca/PHIRC/pdf/Hawe_2007-PopHealth.pdf

Health, S. (1999). *A population health framework for Saskatchewan regional health authorities*. Regina: Saskatchewan Health.

Hertzman, C., Frank, J., & Evans, R. G. (1994). Heterogeneities in health status and the determinants of population health. In R. G. Evans, M. L. Barer, & T. R. Marmor (Eds.), *Why are some people healthy and others not?* (pp. 67–92). New York: Walter de Gruyter.

Jeffery, R. W. (1989). Risk behaviors and health: Contrasting individual and population perspectives. *American Psychologist, 44*(9), 1194–1202.

Kindig, D., & Stoddart, G. (2003). What is population health? *American Journal of Public Health, 93*(3), 380–383.

Link, B. G., & Phelan, J. (1995). Social conditions as fundamental causes of disease. *Journal of Health and Social Behavior, 35*, 80–94.

Lynch, J. W., Smith, G. D., Kaplan, G. A., & House, J. S. (2000). Income inequality and mortality: Importance to health of individual income, psychosocial environment, or material conditions. *British Medical Journal, 320*(7243), 1200–1204.

Marmot, M. G. (2003). Understanding social inequalities in health. *Perspectives in Biology and Medicine, 46*(3), S9–S23.

Public Health Agency of Canada (1996). *Towards a common understanding: Clarifying the core concepts of population health approach*. Discussion paper. Cat. No. H39-391/1996E ISBN 0-662-25122-9. Retrieved from: http://www.phac-aspc.gc.ca/ph-sp/docs/common-commune/index-eng.php

Rose, G. (1985). Sick individuals and sick populations. *International Journal of Epidemiology, 14*(1), 32–38.

Wanless, D. (2003). *Securing good health for the whole population: Population health trends*. London: HM Treasury.

Population Health Monitoring or Tracking

- ▶ Mental Health Surveillance

Population Stratification

Abanish Singh
Duke University Medical Center, Durham, NC, USA

Definition

Often an apparently homogenous population group contains subgroups that are genetically distinct. These subgroups may have allele frequency differences due to systematic ancestry differences. Such population structure is known as population stratification.

Description

The mixture of groups of individuals with different allele frequency, i.e., heterogeneous genetic backgrounds, undermines the reliability of

association testing results. The assumption of population homogeneity in association studies may not always be true, and violation of the assumption can result in statistical errors. Genetically distinct population subgroups, possibly resulted from interbreeding of two different population groups, can exhibit disequilibrium between pairs of unlinked loci which may create confounding or spurious associations. Therefore, it becomes important to find and quantify genetically distinct subgroups within a population group. Various techniques including principal component analysis have been successfully used to quantify population stratification.

Cross-References

- ▶ Allele
- ▶ Gene
- ▶ Genetics
- ▶ Genome-Wide Association Study (GWAS)

References and Readings

Cardon, L. R., & Palmer, L. J. (2003). Population stratification and spurious allelic association. *The Lancet, 361*, 598–604.

Engelhardt, B. E., & Stephens, M. (2010a). Analysis of population structure: A unifying framework and novel methods based on sparse factor analysis. *PLoS Genetics, 6*(9), 1–12. doi:10.1371/journal.pgen.1001117. e1001117.

Engelhardt, B. E., & Stephens, M. (2010b). Analysis of population structure: A unifying framework and novel methods based on sparse factor analysis. *PLoS Genetics, 6*, 1–12. e1001117.

Patterson, N., Price, A. L., & Reich, D. (2006). Population structure and eigenanalysis. *PLoS Genetics, 2*(12), 2074–2093. doi:10.1371/journal.pgen.0020190. e190.

Price, A. L., Patterson, N. J., Plenge, R. M., Weinblatt, M. E., Shadick, N. A., & David, R. (2006). Principal components analysis corrects for stratification in genome-wide association studies. *Nature Genetics, 38*(8), 904–909.

Tiwari, H. K., Barnholtz-Sloan, J., Wineinger, N., Padilla, M. A., Vaughan, L. K., & Allison, D. B. (2008). Review and evaluation of methods correcting for population stratification with a focus on underlying statistical principles. *Human Heredity, 66*, 67–86. doi:10.1159/000119107.

Wacholder, S., Rothman, N., & Caporas, N. (2000). Population stratification in epidemiologic studies of common genetic variants and cancer: quantification of bias. *Journal of the National Cancer Institute, 92*(14), 1151–1158. doi:10.1093/jnci/92.14.1151.

Population-Based Study

Roselind Lieb
Department of Psychology, Division of Clinical Psychology and Epidemiology, Basel, Switzerland

Definition

Population-based studies aim to answer research questions for defined populations. Answers should be generalizable to the whole population addressed in the study hypothesis, not only to the individuals included in the study. This point addresses the point of external validity of the findings. Therefore, the valid definition as well as the reliable and valid identification of populations in which research questions for specific populations can be studied is the most important issue in population-based studies.

Population-based studies may include a variety of study types. They may include case–control studies, cross-sectional studies, twin studies, or prospective and retrospective cohort studies. The important issue is the selection of the individuals that are included into the study – they should be representative of all individuals in the a priori defined specific population.

For example, in a population-based prospective cohort study, in which an association between a specific exposure and a specific outcome (i.e., the onset of a certain disease) is studied, all individuals sampled for the study should be representative for the addressed population. This means that the individuals under exposure and non-exposure should be identified within the same population. They should differ only on the exposition factor. Likewise, in a population-based case–control study, cases and controls should be also identified in the same population.

Otherwise, differences between cases and controls can be attributed to different population characteristics.

Cross-References

- ▶ Cohort Study
- ▶ Follow-Up Study

References and Readings

Rothman, K. J., Greenland, S., & Lash, T. L. (2008). *Modern epidemiology* (3rd ed.). Philadelphia: Lippincott Williams & Wilkins.

Susser, E., Schwartz, S., Morabia, A., & Bromet, E. J. (2006). *Psychiatric epidemiology*. Oxford: Oxford University Press.

Szklo, M. (1998). Poplation-based cohort studies. *Epidemiologic Reviews, 20*, 81–90.

Positive Affect

- ▶ Happiness and Health

Positive Affect Negative Affect Scale (PANAS)

Vincent Tran
University of Texas, Southwestern Medical Center, Dallas, TX, USA

Synonyms

Positive and negative affect schedule

Definition

The Positive and Negative Affect Schedule (PANAS) (Watson, Clark, & Tellegen, 1988) is one of the most widely used scales to measure mood or emotion. This brief scale is comprised of 20 items, with 10 items measuring positive affect (e.g., excited, inspired) and 10 items measuring negative affect (e.g., upset, afraid). Each item is rated on a five-point Likert Scale, ranging from $1 =$ *Very Slightly or Not at all* to $5 =$ *Extremely,* to measure the extent to which the affect has been experienced in a specified time frame. The PANAS was designed to measure affect in various contexts such as at the present moment, the past day, week, or year, or in general (on average). Thus, the scale can be used to measure state affect, dispositional or trait affect, emotional fluctuations throughout a specific period of time, or emotional responses to events.

The PANAS is based on a two-dimensional conceptual model of mood, where the full range of affective experiences are reflected along two broad dimensions of positive mood (i.e., extent to which one is experiencing a positive mood such as feelings of joy, interest, and enthusiasm) and negative mood (i.e., extent to which one is generally experiencing a negative mood such as feelings of nervousness, sadness, and irritation). Importantly, the PANAS was developed to provide a brief scale that measures positive and negative affect as separate and largely uncorrelated constructs, such that one can experience both positive and negative emotions simultaneously. Both the positive and negative affect scales have good internal consistency, with Chronbach's alpha $\geq .84$ for each scale across multiple time frames. The scales also demonstrate good convergent and discriminant validity. The two-factor structure of the PANAS has been examined extensively and appears to be robust across different populations and temporal instructions.

Other versions of the PANAS have been developed. The PANAS-X is an extended version of the PANAS that may be used when more discrete measures of specific affective experiences are necessary. The PANAS-X includes 60 items that measure not only the two higher order scales (positive affect and negative affect), but also specific affects (joviality, self-assurance, attentiveness, fear, hostility, guilt, sadness, shyness, fatigue, serenity, and surprise). The I-PANAS-SF (International-PANAS-Short Form)

contains 10 items to measure positive and negative affect, and was developed to reduce redundancy or eliminate ambiguous meanings of some of the original PANAS terms. The PANAS-C is a child version of the PANAS; emotion terms were altered and instructions were simplified for use in childhood populations. Finally, the PANAS has also been translated into other languages, such as Japanese and Spanish.

Cross-References

- ▶ Affect
- ▶ Emotions: Positive and Negative
- ▶ Mood

References and Readings

Eid, M., & Diener, E. (Eds.). (2006). *Handbook of multimethod measurement in psychology*. Washington, DC: American Psychological Association.

Joiner, T. E., Sandín, B., Chorot, P., Lostao, L., & Marquina, G. (1997). Development and factor analytic validation of the SPANAS among women in Spain: (More) cross-cultural convergence in the structure of mood. *Journal of Personality Assessment, 68*, 600–615.

Kaplan, R. M., & Saccuzzo, D. P. (2008). *Psychological testing: Principles, applications, and issues* (7th ed.). Belmont: Wadsworth.

Laurent, J., Catanzaro, S. J., et al. (1999). A measure of positive and negative affect for children: Scale development and preliminary validation. *Psychological Assessment, 11*(3), 326–338.

McDowell, I. (2006). *Measuring health: A guide to rating scales and questionnaires* (3rd ed.). New York: Oxford University Press.

Thompson, E. R. (2007). Development and validation of an internationally reliable short-form of the positive and negative affect schedule (PANAS). *Journal of Cross-Cultural Psychology, 38*(2), 227–242.

Watson, D., & Clark, L. A. (1994). *The PANAS-X: Manual for the positive and negative affect schedule-expanded form*. Iowa City: University of Iowa.

Watson, D., & Clark, L. A. (1997). The measurement and mismeasurement of mood: Recurrent and emergent issues. *Journal of Personality Assessment, 68*, 267–296.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*(6), 1063–1070.

Positive Affectivity

Katherine T. Fortenberry[1], Kate L. Jansen[2] and Molly S. Clark[2]

[1]Department of Family and Preventative Medicine, The University of Utah, Salt Lake City, UT, USA

[2]Department of Family Medicine, University of Mississippi Medical Center, Jackson, MS, USA

Synonyms

Pleasant affect; Positive emotion

Definition

Positive affect can be described as the experience of a set of emotions reflecting pleasurable engagement with the environment. Positive affect reflects neither a lack of negative affect, nor the opposite of negative affect, but is a separate, independent dimension of emotion (Watson & Tellegen, 1985). It may be exhibited as either a trait-like variable, typically referred to as *positive affectivity*, or as a state-like variable (Watson, 2002). Research on positive affectivity has focused on associations with beneficial coping mechanisms, increased cognitive flexibility, and certain health benefits and improved outcomes.

Description

Watson and Tellegen (1985) presented a two-factor model of mood and affect, in which high levels of positive affect reflect enthusiastic, active, and alert mood states. They contrast this to negative affect, which includes aversive mood states, such as anger, guilt, nervousness, and fear. They suggest that a lack of positive affect reflects sadness and lethargy, whereas a lack of negative affect reflects calmness and serenity. Alternately, positive affect has been conceptualized as including positive emotions that reflect both high and low levels of energy and activation, including

joy, interest, contentment, and love (Fredrickson, 1998). Positive affectivity (i.e., the trait-like disposition to experience positive affect) has been found to relate to the personality variable of extraversion (Lucas & Fujita, 2000), which is one broad factor in the Five Factor Model of personality. This trait is most frequently measured via the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). Lack of positive affect is considered central in the development and maintenance of depressive disorders (Watson et al., 1995); in fact, a highly effective treatment for depression utilizes behavioral activation to target anhedonia and induce positive experiences, which in turn increases positive affect (Jacobson et al., 1996).

Fredrickson's (1998) Broaden and Build Model of positive affect posits that positive emotion broadens individuals' awareness, encouraging creative, flexible, and exploratory thoughts and actions. She suggests that positive emotions serve an evolutionary function by expanding physical, intellectual, and social resources, enhancing overall well-being and health. Empirical research finds that individuals with induced positive, but not negative, affect experienced broader scope of attention and more a varied repertoire of potential action (Fredrickson & Branigan, 2005). Neuropsychological research has supported positive affect's beneficial influence on cognition. Specifically, positive affect enhances consolidation of long-term memory, working memory, and creative problem solving, potentially via an increase in brain dopamine levels (Ashby, Isen, & Turken, 1999).

The influence of positive affectivity on health and health processes has been extensively examined (see Pressman & Cohen, 2005). Positive affectivity is consistently and prospectively linked to lower reports of pain, fewer symptoms, and better self-rated heath. Positive affectivity is also associated with reduced morbidity from illness, including reduced risk of stroke incidence (Ostir, Markides, Peek, & Goodwin, 2001), and reduced risk of infection in healthy adults (Cohen, Doyle, Turner, Alper, & Skoner, 2003); however, less consistent evidence has been found in mortality and survival studies. Positive affectivity is thought to influence health either through a main effect model, in which positive affect directly affects physiological processes and/or coping behavior, or through a stress-buffering model, in which positive affectivity influences health by ameliorating potentially harmful influences of stressful life events on health.

The recent increase in research on positive affectivity can be associated with the rise of interest in Positive Psychology, which examines the science of positive human functioning (see Seligman & Csikszentmihalyi, 2000).

Cross-References

- ▶ Emotions: Positive and Negative
- ▶ Negative Affectivity
- ▶ Positive Affect Negative Affect Scale (PANAS)
- ▶ Positive Psychology

References and Readings

Ashby, F. G., Isen, A. M., & Turken, A. U. (1999). A neuropsychological theory of positive affect and its influence on cognition. *Psychological Review, 106*, 529–550.

Cohen, S., Doyle, W. J., Turner, R. B., Alper, C. M., & Skoner, D. P. (2003). Emotional style and susceptibility to the common cold. *Psychosomatic Medicine, 65*, 652–657.

Fredrickson, B. L. (1998). What good are positive emotions? *Review of General Psychology, 2*, 300–319.

Fredrickson, B. L., & Branigan, C. (2005). Positive emotions broaden the scope of attention and thought-action repertoires. *Cognition and Emotion, 19*, 313–332.

Jacobson, N. S., Dobson, K. S., Truax, P. A., Addis, M. E., Koerner, K., Gollan, J. K., et al. (1996). A component analysis of cognitive-behavioral treatment for depression. *Journal of Consulting and Clinical Psychology, 64*, 295–304.

Lucas, R. E., & Fujita, F. (2000). Factors influencing the relation between extraversion and pleasant affect. *Journal of Personality and Social Psychology, 79*, 1039–1056.

Ostir, G. V., Markides, K. S., Peek, K., & Goodwin, J. S. (2001). The associations of emotional well-being and the incidence of stroke in older adults. *Psychosomatic Medicine, 63*, 210–215.

Pressman, S. D., & Cohen, S. (2005). Does positive affect influence health? *Psychological Bulletin, 131*, 925–971.

Seligman, M. E. P., & Csikszentmihalyi, M. (2000). Positive psychology: An introduction. *American Psychologist, 55*, 5–14.
Watson, D. (2002). Positive affectivity: The disposition to experience pleasurable emotional states. In C. R. Snyder (Ed.), *Handbook of Positive Psychology* (pp. 106–119). New York, NY: Oxford.
Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS Scales. *Journal of Personality and Social Psychology, 54*, 1063–1070.
Watson, D., & Tellegen, A. (1985). Toward a consensual structure of mood. *Psychological Bulletin, 98*, 219–235.
Watson, D., Weber, K., Assenheimer, J. S., Clark, L. A., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: I. Evaluating the convergent and discriminant validity of anxiety and depression symptom scales. *Journal of Abnormal Psychology, 104*, 1–14.

Positive and Negative Affect

▶ Emotions: Positive and Negative

Positive and Negative Affect Schedule

▶ Positive Affect Negative Affect Scale (PANAS)

Positive By-Products

▶ Perceived Benefits

Positive Changes

▶ Perceived Benefits

Positive Emotion

▶ Positive Affectivity
▶ Happiness and Health

Positive Emotions

▶ Well-Being: Physical, Psychological, Social

Positive Meaning

▶ Perceived Benefits

Positive Psychology

Lisa G. Aspinwall[1] and Watcharaporn Pengchit[2]
[1]Department of Psychology, The University of Utah, Salt Lake City, UT, USA
[2]Faculty of Psychology, Chulalongkorn University, Bangkok, Thailand

Definition

Positive psychology, the scientific study of positive phenomena from the neurobiology of positive emotions to application in the clinic and in everyday life, encompasses multiple efforts to understand and promote well-being and health (Aspinwall & Staudinger, 2003; Aspinwall & Tedeschi, 2010; Lopez & Snyder, 2009; Ryff & Singer, 1998; Seligman & Csikszentmihalyi, 2000). Key elements include (a) the identification of human strengths (qualities and processes that allow people to navigate adversity, pursue their goals, and make the most of life) and (b) empirical research directed toward understanding the diverse conditions that create and sustain such strengths. These processes have been investigated in a wide variety of domains, including education, social development, close relationships, aging, work, and health.

Description

Core Concerns of Positive Psychology and Health

Positive psychology is an active and growing field, with thousands of published articles in the

last decade, two major handbooks, a new *Encyclopedia of Positive Psychology*, a specialized journal (the *Journal of Positive Psychology*), and several edited volumes, journal special issues, and international conferences devoted to this topic. Within this voluminous literature, there are three main areas that examine the relationship of positive phenomena to physical health: (a) psychological adaptation to illness, (b) the impact of positive phenomena, such as positive mood, optimism, and hope, on physical health, and (c) interventions that use insights from this research to improve well-being in general and among people managing serious illness.

Understanding Psychological Adaptation to Illness

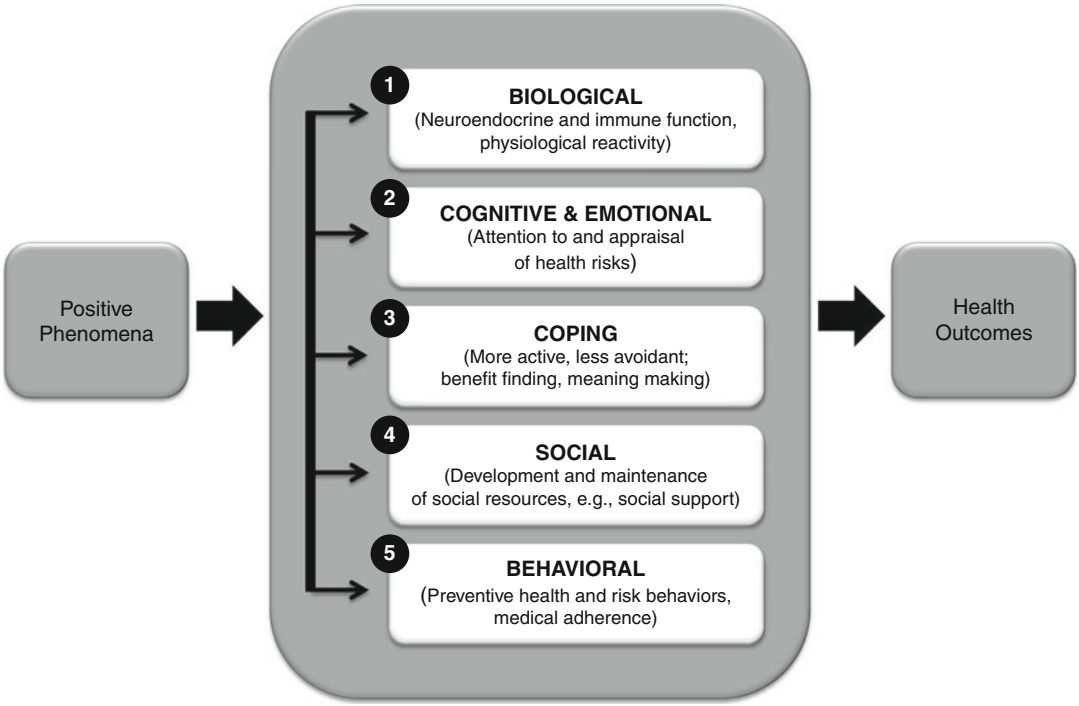
Multiple lines of research have examined the impact of negative life events, including serious illness, on people's beliefs about themselves, the benevolence of others, personal control, and views of the future. In general, research suggests that people who have experienced serious illness or other forms of adversity frequently report *both* positive and negative life changes. Frequently reported positive changes include a better sense of one's values and priorities, stronger social relationships, and an enhanced appreciation for life. Negative changes include fears for the future and feelings of personal vulnerability. Such changes are understood by many researchers to be the product of active efforts to manage and derive meaning and/or benefit from one's circumstances (Folkman, 2011; Taylor, 1983). Exciting developments in this line of research have related finding meaning and/or benefit to subsequent physical health outcomes, such as immune function (Park, Lechner, Antoni, & Stanton, 2009; Taylor, Kemeny, Reed, Bower, & Gruenewald, 2000).

As exciting as these findings are, it is important to understand their boundary conditions. In particular, not all events produce benefits or growth or do so for all people. Some particularly challenging events (severe interpersonal stressors, wartime experiences) seem to defy meaning-making coping. Further, large-scale longitudinal panel studies of life satisfaction following long-term unemployment and divorce suggest that people may not, on average, fully adjust to these events. Finally, there appear to be multiple "normal" responses to adversity. A recent advance in this area – made possible by rigorous prospective longitudinal studies of bereavement (Bonanno, 2004) – is the recognition that there may be multiple trajectories of mental health outcomes following adversity, with many respondents reporting stable good or even improving outcomes and a subset of respondents reporting either enduring preexisting distress or new elevations in distress. These different patterns of adjustment over time highlight the need to understand the antecedents and processes that account for them. The finding that there is considerable variation in response to adversity also suggests that global positive outcomes (e.g., "Cancer was the best thing that ever happened to me") may be true for some people, but may grossly misrepresent the experiences of others.

Understanding the Relation of Positive Phenomena to Physical Health

Although most research in positive psychology focuses on psychological well-being, there is increasing interest in the relation of such concepts as optimism, hope, positive affect, gratitude, benefit finding, and growth to physical health. Several recent meta-analyses suggest that positive phenomena show a reliable prospective beneficial relationship to multiple health outcomes, including longevity, in both healthy and ill samples (Diener & Chan, 2011; Howell, Kern, & Lyubomirsky, 2007; Pressman & Cohen, 2005; Rasmussen, Scheier, & Greenhouse, 2009). Importantly, this relationship is (a) not explained by the detrimental effects of either negative emotions or pessimistic expectations and (b) comparable in magnitude to that of these more widely studied negative phenomena. At present, the findings seem to be stronger for healthy samples than among people managing illness, and the strength of the findings varies by disease, with stronger and more encouraging findings for cardiovascular disease and HIV than for cancer mortality.

Researchers have examined multiple complementary pathways through which positive phenomena are related to health outcomes



Positive Psychology, Fig. 1 Five mutually interactive and complementary sets of pathways through which positive phenomena, such as positive mood and optimism, may influence human health

(Aspinwall & Tedeschi, 2010). Five sets of pathways are shown in Fig. 1. The first set of pathways involves direct relations to neuroendocrine and immune functions implicated in the etiology and progression of disease, as well as physiological reactivity to laboratory stressors. A second and third set of pathways involve the relation of positive phenomena to multiple aspects of attending to, appraising, and coping with health-risk information and the demands of serious illness, including the benefit-finding and meaning-making processes described earlier. A fourth set of pathways involves the relationship between positive phenomena and the development and maintenance of supportive social ties, especially during times of illness or other adversity. The fifth pathway represents the relationship of positive phenomena to the practice of preventive health behaviors, such as good nutrition, exercise, and sleep; the decreased practice of risk behaviors, such as substance abuse; and improved medical adherence.

Recent findings from the Women's Health Initiative illustrate the multiple pathways through which positive phenomena may influence health outcomes. In an 8-year prospective study of coronary heart disease in a sample of more than 97,000 US women, ages 50–79 (Tindle et al., 2009), dispositional optimism was found to prospectively predict decreased risk for coronary heart disease and all-cause mortality in the overall sample. Optimism also predicted lower cancer-related mortality for African-American women, but not Caucasian women. Compared to pessimists, optimists were less likely to have diabetes, hypertension, high cholesterol, or depressive symptoms; they were less likely to smoke, be sedentary, or be overweight; and they reported higher education and income, greater employment and health insurance, and greater religious attendance. However, the real kicker is that controlling for *all* of the above factors, optimism still predicted better health outcomes. These findings suggest that there is much that

remains to be understood about the relationship of positive beliefs to important health outcomes and that the multiple pathways outlined in Fig. 1 and described in the following sections may be implicated in such findings.

1. *Biological pathways*. At present, more is known about the prospective relationship of positive phenomena to diverse health outcomes than about the specific pathways through which these outcomes are realized. Most studies have focused on cardiovascular, neuroendocrine, and immune function. Both experimental laboratory studies and field experiments link the induction of positive self-beliefs to reduce cardiovascular and neuroendocrine reactivity to evaluative stressors and to more rapid recovery from induced negative states among healthy young adults. Recent reviews link optimism to improved immune function, both among healthy people managing naturalistic stressors such as law school entry and among people managing HIV infection. These lines of research have also identified some potential short-term physiological costs associated either with optimists' active coping efforts or with the relatively intense positive affect inductions used to study physiological outcomes in laboratory settings (Pressman & Cohen, 2005). However, these authors also noted that these costs were not seen in naturalistic ambulatory studies where positive affect was associated with health-protective responses. Many researchers have attributed the health-protective findings of optimism and positive mood to reductions in perceived stress, highlighting the importance of such appraisals in understanding how positive phenomena are related to health outcomes.
2. *Attention to and appraisal of threatening health information*. In contrast to the idea that being happy or optimistic reduces awareness of negative realities (e.g., seeing one's world through "rose-colored glasses"), several experiments have demonstrated that induced positive affect or positive self-beliefs (e.g., through self-affirmation or success manipulations) promote constructive attention to negative information about health risks and personal weaknesses when the information is relevant to the self and said to be useful (Aspinwall & Tedeschi, 2010). Similar findings have been obtained for dispositional optimism. Interestingly, this greater attention to negative information does not appear to be in the service of downplaying its negativity or relevance to the self, but instead appears to be strongly responsive to its potential value in warding off or managing future negative events. The ability to adaptively confront, manage, and remember such information may have beneficial links to both coping and health behavior.
3. *Coping with serious illness*. A large literature suggests that positive affect and optimism promote constructive methods of coping with adversity, involving planning, information-seeking, suppression of competing activities, and seeking both instrumental and emotional social support (Aspinwall & Tedeschi, 2010). Notably, positive beliefs are consistently inversely associated with deleterious forms of avoidant coping, such as denial, mental and behavioral disengagement, and substance use. Researchers have argued that the tendency toward active, engaged forms of coping with adversity shown in pathway #3 and the ability to attend to negative information shown in pathway #2 may work together to promote a more informed understanding of actual or potential stressors. The term "upward spiral" has been used to characterize such processes.
4. *Development and maintenance of social resources*. Recent meta-analytic evidence suggests that positive affect plays a major role in the maintenance of satisfying social relationships (Lyubomirsky, King, & Diener, 2005). Specifically, people who report frequent positive affect also report a greater number of – and more satisfying – social relationships. Similarly, college students who reported greater optimism on arrival to college reported both greater initial friendship network size and greater increases in perceived social support over the course of their first semester, and

these social ties buffered distress at the end of the semester. Existing evidence suggests that there may be more at work in such findings than the idea that it is pleasant to be around people in a good mood. People in a good mood seem better able to understand the goals and priorities of their interaction partners, to express more gratitude, and to be more likely to help others than people in a neutral mood. These particular processes suggest that an important direction for future research and intervention might be to examine these processes among people managing illnesses and other demands such as caregiving that are known to tax social resources over time.

5. *Health behaviors and medical adherence*. An emerging literature suggests that positive beliefs and states are robust prospective predictors of better health behavior, including diet, exercise, and sleep, and lower practice of risk behaviors, such as substance abuse (Aspinwall & Tedeschi, 2010). These findings have been obtained both in healthy community samples, including large samples of older adults, and among people managing serious illnesses, such as HIV infection. In contrast, prospective studies link negative beliefs, such as pessimism and fatalism, to the practice of a wide range of health-risk behaviors, including substance abuse and high-risk sexual behavior. Understanding how positive beliefs like hope and optimism are related to the practice of inherently forward-looking behaviors, like preventive health behavior, remains an important research question. Researchers are also starting to examine how positive phenomena may be related to behavior change and medical adherence. Although it is well known that negative affect and interpersonal stressors contribute to relapse from behavior change efforts in multiple domains, the question of whether and how positive phenomena may have facilitative effects needs further investigation. With respect to medical adherence, randomized controlled trials of interventions in hospital settings demonstrate that increasing patients' optimism about the success of cardiac rehabilitation improves outcomes like angina symptoms and return to work.

Summary and Remaining Conceptual Questions. This section outlined five sets of pathways through which positive feelings and beliefs may influence physical health. These multiple pathways are complementary, rather than competing, and are likely to work in concert (Aspinwall & Tedeschi, 2010). Specifically, the ability to maintain attention to negative information about health risks may work in tandem with more active forms of coping that are more likely to elicit information about one's situation and support for managing it. Similarly, finding benefits and growth in one's experience and maintaining supportive social ties may each be related to subsequent health behaviors in important ways. Research that examines the interplay of these different pathways will likely yield not only a more complex account of the ways in which positive phenomena may be linked to human health but also an understanding of some of the cumulative benefits that may result.

Interventions to Promote Health and Well-Being

Interventions derived from the principles and pathways reviewed here have been employed to promote well-being in healthy samples, as well as psychosocial adjustment to chronic illness (Folkman, 2011; Park et al., 2009). For example, a burgeoning literature on the kinds of experiences that make people happy and the processes that contribute to hedonic adaptation both to positive and negative events have been usefully employed in interventions to improve daily positive affect, primarily in healthy samples. For people living with chronic illnesses like diabetes, various interventions ranging from writing about one's positive experiences to multiple-component programs have proved effective in enhancing positive affect, at least in laboratory settings. Experimental interventions to increase hope and gratitude have also been shown to improve reported pain tolerance and physical symptoms, respectively. Finally, although they were not conducted under the rubric of positive psychology, psychosocial interventions to provide social support and reduce stress among

people managing serious illness suggest that such interventions reliably reduce pain and anxiety and improve quality of life (Aspinwall & Tedeschi, 2010; Park et al., 2009). Understanding how the various pathways suggested here may be implicated in such effects – and used to enhance them – among people managing serious illness remains an important goal for future research.

Controversies with Respect to Both Theory and Application

The study of how positive phenomena – especially positive thinking – are related to health has generated both academic and cultural controversies (Aspinwall & Staudinger, 2003; Aspinwall & Tedeschi, 2010; Becker & Marecek, 2008; Lazarus, 2003). Researchers have questioned whether the focus on positive or optimal human functioning is new or sufficiently distinctive or developed to merit a special name as a new field; whether separating positive phenomena from negative phenomena is practicable, desirable, or meaningful (e.g., coping with adversity may inherently involve both); and whether the field focuses too much on the individual as the unit of analysis at the neglect of important social, cultural, economic, structural, and environmental determinants of health and well-being. With respect to positive psychology and health, critics have questioned (a) whether the field is too narrowly focused on individual positive feelings and self-beliefs as the primary outcomes of optimal human functioning; (b) whether the promotion of positive thinking among those managing serious illness may be trivial, distracting, and ultimately useless; and (c) whether positive thinking might actually be actively harmful if it impairs attention to negative aspects of experience or contributes to a culture of blame for individual misfortune.

Several critics (as well as the present authors) have traced the history of these ideas to the New Thought movement and subsequent popular efforts to promote health, well-being, and financial success through positive thinking (e.g., *The Power of Positive Thinking, The Secret*; see Aspinwall & Tedeschi, 2010, for discussion). With respect to the application of positive psychology to understanding health and illness, a primary concern – reflected more in critiques of the popular self-help literature on positive thinking than in scientific discourse on these issues – is the potential misuse of findings suggesting that positive emotions and beliefs may predict physical health and recovery from illness. Specifically, the demonstration that a positive attitude (e.g., optimism or hope) is linked to better health outcomes for some people in some situations may create, or be used to promote, unrealistic expectations about the ability to cure disease through positive thinking. Similarly, the demonstration that some people with serious illness report benefits and growth can be misconstrued to suggest that all people should do so, all of the time and regardless of circumstance. These pressures to maintain uniformly positive thoughts and feelings and to ascribe deteriorating health to failures of positive attitude have been described as "saccharine terrorism" and the "tyranny of optimism." While researchers have in general been quite careful not to mandate positive feelings among people dealing with adversity (and in fact have documented the presence of both positive and negative life changes and multiple trajectories of adjustment), various popular treatments have promoted the notion that an exclusive focus on positive thoughts and feelings will cure illness. Some critics aptly note that this approach is particularly prevalent – and pernicious – in popular treatments of cancer survivorship (Aspinwall & Tedeschi, 2010).

Conclusion

As scientific research on the relationship of positive phenomena to health proceeds, rigorous process-oriented research should dispel the notion that there is some kind of magic bullet to be found in positive thinking. Continued examination of the multiple biological, cognitive, emotional, social, and behavioral pathways linking positive phenomena to health is essential. Further, researchers interested in a balanced understanding of the link between positive phenomena and health outcomes should continue to design studies that are sensitive to both potential benefits and liabilities of

particular kinds of positive (and negative) thoughts and feelings in different situations (Aspinwall & Staudinger, 2003; Aspinwall & Tedeschi, 2010). Indeed, what the study of health and illness has to offer positive psychology is the opportunity to examine the multiple ways in which positive and negative phenomena may go hand in hand as people manage serious illness and its treatment.

Cross-References

- ▶ Adherence
- ▶ Aging
- ▶ Behavior Change
- ▶ Benefit Finding
- ▶ Bereavement
- ▶ Cancer Survivorship
- ▶ Cardiac Rehabilitation
- ▶ Cardiovascular Disease
- ▶ Coping
- ▶ Coronary Heart Disease
- ▶ Denial
- ▶ Diabetes
- ▶ Exercise
- ▶ Fatalism
- ▶ Hypertension
- ▶ Immune Function
- ▶ Longevity
- ▶ Meta-Analysis
- ▶ Mortality
- ▶ Nutrition
- ▶ Optimism
- ▶ Overweight
- ▶ Perceived Benefits
- ▶ Pessimism
- ▶ Physiological Reactivity
- ▶ Sexual Risk Behavior
- ▶ Sleep
- ▶ Stressor
- ▶ Substance Abuse
- ▶ Well-Being

References and Readings

Aspinwall, L. G., & Staudinger, U. M. (Eds.). (2003). *A psychology of human strengths: Fundamental questions and future directions for a positive psychology*. Washington, DC: APA Books.

Aspinwall, L. G., & Tedeschi, R. G. (2010). The value of positive psychology for health psychology: Progress and pitfalls in examining the relation of positive phenomena to health. *Annals of Behavioral Medicine, 39*, 4–15.

Becker, D., & Marecek, J. (2008). Dreaming the American dream: Individualism and positive psychology. *Social and Personality Psychology Compass, 2*, 1767–1780.

Bonanno, G. A. (2004). Loss, trauma, and human resilience: Have we underestimated the human capacity to thrive after extremely aversive events? *American Psychologist, 59*, 20–28.

Diener, E., & Chan, M. Y. (2011). Happy people live longer: Subjective well-being contributes to health and longevity. *Applied Psychology: Health and Well-being, 3*(1), 1–43.

Folkman, S. (Ed.). (2011). *The Oxford handbook of stress, health, and coping*. New York: Oxford University Press.

Howell, R. T., Kern, M. L., & Lyubomirsky, S. (2007). Health benefits: Meta-analytically determining the impact of well-being on objective health outcomes. *Health Psychology Review, 1*, 1–54.

Lazarus, R. S. (2003). Does the positive psychology movement have legs? *Psychological Inquiry, 14*, 93–109.

Lopez, S. J., & Snyder, C. R. (Eds.). (2009). *Oxford handbook of positive psychology* (2nd ed.). New York: Oxford University Press.

Lyubomirsky, S., King, L., & Diener, E. (2005). The benefits of frequent positive affect: Does happiness lead to success? *Psychological Bulletin, 131*, 803–855.

Park, C. L., Lechner, S. C., Antoni, M. H., & Stanton, A. L. (Eds.). (2009). *Medical illness and positive life change*. Washington, DC: American Psychological Association.

Pressman, S. D., & Cohen, S. (2005). Does positive affect influence health? *Psychological Bulletin, 131*, 925–971.

Rasmussen, H. N., Scheier, M. F., & Greenhouse, J. B. (2009). Optimism and physical health: A meta-analytic review. *Annals of Behavioral Medicine, 37*, 239–256.

Ryff, C. D., & Singer, B. (1998). The contours of positive human health. *Psychological Inquiry, 9*, 1–28.

Seligman, M. E. P., & Csikszentmihalyi, M. (2000). Positive psychology: An introduction. *American Psychologist, 55*, 1–54.

Taylor, S. E. (1983). Adjustment to threatening events: A theory of cognitive adaptation. *American Psychologist, 38*, 1161–1173.

Taylor, S. E., Kemeny, M. E., Reed, G. M., Bower, J. E., & Gruenewald, T. L. (2000). Psychological resources, positive illusions, and health. *American Psychologist, 55*, 99–109.

Tindle, H. A., Chang, Y.-F., Kuller, L. H., Manson, J. E., Robinson, J. G., et al. (2009). Optimism, cynical hostility, and incident coronary heart disease and mortality in the women's health initiative. *Circulation, 120*, 656–662.

Positron Emission Tomography (PET)

▶ Brain, Imaging
▶ Neuroimaging

Posterior Hypothalamic Area

▶ Hypothalamus

Postpartum Blues

▶ Postpartum Depression

Postpartum Depression

Michele L. Okun
Sleep Medicine Institute and Department of Psychiatry, School of Medicine, University of Pittsburgh, Pittsburgh, PA, USA

Synonyms

Major depressive disorder; Postpartum blues

Definition

Postpartum depression is moderate to severe depression in a woman after she has given birth. It may occur soon after delivery or up to a year later. Most of the time, it occurs within the first 3 months after delivery.

Description

Major depressive disorder with a postpartum onset (PPD) is a prevalent and serious disorder. Postpartum major depression (PPMD) is moderate to severe depression in a woman after she has given birth, clinically resembling major depression as described in DSM-IV. Feelings of anxiety, irritation, tearfulness, and restlessness are common in the week or two after pregnancy. These feelings are often called the postpartum blues or "baby blues." These symptoms almost remit without the need for treatment. Postpartum depression may occur when the baby blues do not fade away or when signs of depression start 1 or more months after childbirth. Up to 20% of women will have an initial major depressive episode within the first 3 month postpartum (Gavin et al., 2005), with the risk of suffering recurrent postpartum major depression (PPMD) at about 25% (Wisner, Perel, Peindl, & Hanusa, 2004). Women are at the highest risk during their lifetimes for depressive episodes during the childbearing years (O'Hara, Zekoski, Philipps, & Wright, 1990).

PPMD is considered a serious public health concern (Gaynes et al., 2005; Wisner, Chambers, & Sit, 2006). The maternal role, which is vital to the infant's safety, survival, and well-being (Logsdon, Wisner, & Pinto-Foltz, 2006), can be compromised by PPMD. Children of mothers with PPMD are at an increased risk of impaired mental and motor development, poor self-regulation, and behavior problems (Moehler, Brunner, Wiebel, Reck, & Resch, 2006). Postpartum depression and its consequences can persist from months to years after childbirth, with lingering limitations in physical and psychological functioning after recovery from depressive episodes (Burt & Stein, 2002; Marcus & Heringhausen, 2009; McCarter-Spaulding & Horowitz, 2007).

A myriad of factors contribute to the etiology of both incident and recurrent PPMD (Beck, 2001; Bloch, Daly, & Rubinow, 2003; Gaynes et al., 2005). Among the established risk factors, previous episodes of depression, family history of depression, and depressive symptomatology during pregnancy (O'Hara, Schlechte, Lewis, & Varner, 1991; O'Hara, Schlechte, Lewis, & Wright, 1991) are the strongest predictors for both incident and recurrent episodes. Demographic variables, including marital status, race, age, and socioeconomic status, have also been

implicated as risk factors (Beck, 2001; Ross, Campbell, Dennis, & Blackmore, 2006). Unfortunately, these recognized risk factors have proven inadequate at predicting which women will have a recurrent episode. Other risk factors that have been identified include the following: (1) age below 20 years; (2) currently abusing alcohol, taking illegal substances, or smoking (these also cause serious medical health risks for the baby); (3) having an unplanned pregnancy, or have mixed feelings about the pregnancy; (4) a stressful event during the pregnancy or delivery, including personal illness, death or illness of a loved one, a difficult or emergency delivery, premature delivery, or illness or birth defect in the baby; (5) little support from family, friends, or the significant other.

Disturbed sleep during late pregnancy represents another potential risk factor for PPMD. Several investigators report that disturbed sleep is a prodromal symptom of both first onset and recurrent depressive symptoms and/or episodes outside of the postpartum (Ford & Kamerow, 1989) as well as during the postpartum (Coble et al., 1994; Wolfson, Crowley, Anwer, & Bassett, 2003). Recently, Okun and colleagues showed that poor sleep quality in late pregnancy (Okun, Hanusa, Hall, & Wisner, 2009) as well as in the first 8 weeks postpartum (Okun et al., 2011) significantly contributed to recurrent PPMD. Wolfson and colleagues noted that women reporting more sleep disturbances in late pregnancy are more likely to have clinically significant depressive symptomatology at 2–4 weeks postpartum than those with few sleep disturbances (Wolfson et al., 2003). Coble and colleagues found that women with a history of depression had more sleep disturbances throughout pregnancy and into the postpartum than women without a history of depression (Coble et al., 1994). Taken together, these findings suggest that sleep disturbances in late pregnancy increase vulnerability to PPMD, particularly in women who are susceptible, such as those with a history of PPMD (Wisner, Parry, & Piontek, 2002).

There are two identified biological systems attributed to have an effect on the risk of developing PPMD. First are endocrine factors. Hormones such as progesterone, estradiol, prolactin, and cortisol peak during the last few weeks of pregnancy, followed by a drastic drop in their levels following delivery and into the early postpartum period (Abou-Saleh, Ghubash, Karim, Krymski, & Bhai, 1998). Dramatic drops in hormone concentrations, especially progesterone and the estrogens, likely contribute to postpartum depression (Abou-Saleh et al., 1998; Bloch et al., 2003); however, their specific role is unclear. Administration of estrogens to postpartum women appears to reduce depressive symptoms (Dennis, 2004). However, the dramatic reduction in concentrations of gonadal steroids after delivery does not lead to PPMD in all women (Bloch et al., 2003).

The second pathway involves alterations in the cytokine milieu (Bloch et al., 2003; Maes et al., 2000). The "cytokine hypothesis of depression" states that both the etiology and pathophysiology of depression are linked to dysregulation of inflammatory cytokines (Maes, 1994). Puerperal women may be particularly vulnerable because inflammatory cytokines increase significantly during the last trimester of pregnancy in preparation for delivery (Romero et al., 2006). Women who report increased depressive symptoms in the postpartum have corresponding higher levels of pro-inflammatory cytokines (Maes et al., 2000).

PPMD is a significant health concern. While there is no single test to diagnose PPMD, it is imperative for a woman to be evaluated if there is any indication that she has signs of depression or risk for depression. This will buttress not only her own mental and physical health, but the health of her baby.

Cross-References

- ▶ Antidepressant Medications
- ▶ Women's Health

References and Readings

Abou-Saleh, M. T., Ghubash, R., Karim, L., Krymski, M., & Bhai, I. (1998). Hormonal aspects of postpartum depression. *Psychoneuroendocrinology, 23*, 465–475.

Beck, C. T. (2001). Predictors of postpartum depression: An update. *Nursing Research, 50*, 275–285.

Bloch, M., Daly, R. C., & Rubinow, D. R. (2003). Endocrine factors in the etiology of postpartum depression. *Comprehensive Psychiatry, 44*, 234–246.
Burt, V. K., & Stein, K. (2002). Epidemiology of depression throughout the female life cycle. *The Journal of Clinical Psychiatry, 63*(Suppl 7), 9–15.
Coble, P. A., Reynolds, C. F., Kupfer, D. J., Houck, P. R., Day, N. L., & Giles, D. E. (1994). Childbearing in women with and without a history of affective disorder. II. Electroencephalographic sleep. *Comprehensive Psychiatry, 35*, 215–224.
Dennis, C. L. (2004). Preventing postpartum depression part I: a review of biological interventions. *Canadian Journal of Psychiatry*, *49*(7), 467–475.
Ford, D. E., & Kamerow, D. B. (1989). Epidemiologic study of sleep disturbances and psychiatric disorders. An opportunity for prevention? *Journal of the American Medical Association, 262*, 1479–1484.
Gavin, N. I., Gaynes, B. N., Lohr, K. N., Meltzer-Brody, S., Gartehner, G., & Swinson, T. (2005). Perinatal depression: a systematic review of prevalence and incidence. *Obstetrics and Gynecology*, *106*(5 pt1), 1071–1083.
Gaynes, B. N., Gavin, N., Meltzer-Brody, S., et al. (2005). Perinatal depression: Prevalence, screening accuracy, and screening outcomes. *Evidence Report/Technology Assessment, Summ*, 1–8.
Logsdon, M. C., Wisner, K. L., & Pinto-Foltz, M. D. (2006). The impact of postpartum depression on mothering. *Journal of Obstetric, Gynecologic, and Neonatal Nursing, 35*, 652–658.
Maes, M. (1994). Cytokines in major depression. *Biological Psychiatry*, *36*(7), 498–499.
Maes, M., Lin, A. H., Ombelet, W., et al. (2000). Immune activation in the early puerperium is related to postpartum anxiety and depressive symptoms. *Psychoneuroendocrinology, 25*, 121–137.
Marcus, S. M., & Heringhausen, J. E. (2009). Depression in childbearing women: When depression complicates pregnancy. *Primary Care, 36*, 151–165.
McCarter-Spaulding, D., & Horowitz, J. A. (2007). How does postpartum depression affect breastfeeding? *The American Journal of Maternal/Child Nursing, 32*, 10–17.
Moehler, E., Brunner, R., Wiebel, A., Reck, C., & Resch, F. (2006). Maternal depressive symptoms in the postnatal period are associated with long-term impairment of mother-child bonding. *Archives of women's Mental Health*, *9*(5), 273–278.
O'Hara, M. W., Schlechte, J. A., Lewis, D. A., & Varner, M. W. (1991). Controlled prospective study of postpartum mood disorders: Psychological, environmental, and hormonal variables. *Journal of Abnormal Psychology, 100*, 63–73.
O'Hara, M. W., Schlechte, J. A., Lewis, D. A., & Wright, E. J. (1991). Prospective study of postpartum blues: Biologic and psychosocial factors. *Archives of General Psychiatry, 48*, 801–806.
O'Hara, M. W., Zekoski, E. M., Philipps, L. H., & Wright, E. J. (1990). Controlled prospective study of postpartum mood disorders: Comparison of childbearing and nonchildbearing women. *Journal of Abnormal Psychology, 99*, 3–15.
Okun, M. L., Hanusa, B. H., Hall, M., & Wisner, K. L. (2009). Sleep complaints in late pregnancy and the recurrence of postpartum depression. *Behavioral Sleep Medicine, 7*, 106–117.
Okun, M. L., Luther, J., Prather, A. A., Perel, J. M., Wisniewski, S., & Wisner, K. L. (2011). Changes in sleep quality, but not hormones predict time to postpartum depression recurrence. *Journal of Affective Disorders, 130*, 378–384.
Romero, R., Espinoza, J., Goncalves, L. F., Kusanovic, J. P., Friel, L. A., & Nien, J. K. (2006). Inflammation in preterm and term labour and delivery. *Seminars in Fetal and Neonatal Medicine*, *11*(5), 317–326.
Ross, L. E., Campbell, V. L., Dennis, C. L., & Blackmore, E. R. (2006). Demographic characteristics of participants in studies of risk factors, prevention, and treatment of postpartum depression. *Canadian Journal of Psychiatry, 51*, 704–710.
Wisner, K. L., Perel, J. M., Peindl, K. S., & Hanusa, B. H. (2004). Timing of depression recurrence in the first year after birth. *Journal of Affective Disorders*, *78*(3), 249–252.
Wisner, K. L., Chambers, C., & Sit, D. K. (2006). Postpartum depression: A major public health problem. *Journal of the American Medical Association, 296*, 2616–2618.
Wisner, K. L., Parry, B. L., & Piontek, C. M. (2002). Clinical practice. Postpartum depression. *The New England Journal of Medicine, 347*, 194–199.
Wolfson, A. R., Crowley, S. J., Anwer, U., & Bassett, J. L. (2003). Changes in sleep patterns and depressive symptoms in first-time mothers: Last trimester to 1-year postpartum. *Behavioral Sleep Medicine, 1*, 54–67.

Posttraumatic Growth

Vincent Tran
University of Texas, Southwestern Medical Center, Dallas, TX, USA

Synonyms

Adversarial growth; Benefit finding; PTG; Stress-related growth; Transformational coping

Definition

Posttraumatic growth is the experience of positive change after a traumatic or negative life

event. It is theorized to be the positive or adaptive outcome of a meaning-making process in which individuals are forced into a reevaluation process of their worldviews after experiencing a negative or life-changing event. Through this reevaluation process, some individuals may develop a more coherent understanding of themselves and the world. Common examples of growth after trauma include changes in life values, improved relationships with family and/or friends, growth in spiritual beliefs, and increased personal strength, empathy, or patience.

Description

The concept of growth from adversity is an ancient concept based in many religions and philosophical systems. However, it has been a formal focus of investigation in psychology only in the past few decades, coinciding with the development of psychometrically sound measures and the rise of positive psychology. The fields of health psychology and behavioral medicine, in particular, have latched onto the notion that one may experience positive change in response to adversity, including the adversity of coping with serious health threats and illnesses. For example, research on posttraumatic growth has been carried out in chronic illness populations such as patients with heart disease, cancer, rheumatoid arthritis, HIV/AIDS, and multiple sclerosis. It has also been studied in the context of veterans of war, victims of violence, bereavement, and family members/caregivers of those experiencing a negative life event.

Posttraumatic growth is conceptually very similar to benefit finding and stress-related growth, and these terms are often used interchangeably in the research literature. Multiple measures have been developed, which have facilitated the rigorous study of posttraumatic growth and related constructs. These scales include: benefit finding (Mohr et al., 1999; Tomich & Helgeson, 2004); Stress-Related Growth Scale (Park et al., 1996); and the Posttraumatic Growth Inventory (Tedeschi & Calhoun, 1996). Posttraumatic growth is differentiated from concepts such as resilience and hardiness, in which one may cope or adjust well in response to stress rather than experience positive transformation and an *improvement* in functioning, quality of life, or worldview. Posttraumatic growth is also conceptually differentiated from optimism because it reflects a change in experience rather than a dispositional trait, although the two constructs have been shown to be correlated.

Posttraumatic growth has been associated with adaptive coping processes including heightened problem-focused coping, social-support seeking, acceptance and positive reinterpretation, optimism, religion, cognitive processing, and positive affect. It has often been associated with better illness adjustment and health outcomes, less depression, and more positive well-being in chronic illness populations. Although the prevalence of posttraumatic growth varies widely from study to study, numerous studies suggest a majority of research participants endorse some type of growth from a negative or traumatic event.

Although research suggests that posttraumatic growth is associated with improved functioning and quality of life, there have been conflicting reports. Some studies report nonsignificant or even negative correlations between posttraumatic growth and well-being. These inconsistencies have driven researchers to more rigorously test proposed theories of posttraumatic growth and better clarify the processes by which posttraumatic growth occurs. More meticulous study has led to investigation of non-transformational change, mediators, moderators, and study of curvilinear associations between posttraumatic growth and well-being. Further elaboration on the theory of posttraumatic growth has led to studies on whether posttraumatic growth is the outcome of a series of life changes that have occurred or whether it is a cognitive process of reevaluation and understanding that unfolds over time. Researchers are also investigating whether posttraumatic growth reflects genuine changes in one's life (veridical growth) or whether it reflects perceptions of change or growth (non-veridical growth). Some have suggested that non-veridical growth may even reflect a maladaptive, defensive denial process,

although others claim that perceptions of growth that do not reflect true life changes may promote well-being in response to a traumatic event.

Cross-References

- ▶ Benefit Finding
- ▶ Coping
- ▶ Defensiveness
- ▶ Hardiness
- ▶ Optimism
- ▶ Positive Psychology
- ▶ Perceived Benefits
- ▶ Resilience

References and Readings

Affleck, G., & Tennen, H. (1996). Construing benefits from adversity: Adaptational significance and dispositional underpinnings. *Journal of Personality, 64*(4), 899–922.

Calhoun, L. G., & Tedeschi, R. G. (Eds.). (2006). *Handbook of posttraumatic growth: Research & practice*. Mahwah: Lawrence Erlbaum.

Helgeson, V. S., Reynolds, K. A., & Tomich, P. L. (2006). A meta-analytic review of benefit finding and growth. *Journal of Consulting and Clinical Psychology, 74*(5), 797–816.

Joseph, S., & Linley, P. A. (Eds.). (2008). *Trauma, recovery, and growth: Positive psychological perspectives on posttraumatic stress*. Hoboken: Wiley.

Linley, P. A., & Joseph, S. (2004). Positive change following trauma and adversity: A review. *Journal of Traumatic Stress, 17*(1), 11–21.

Lopez, S. J., & Snyder, C. R. (Eds.). (2009). *Oxford handbook of positive psychology* (2nd ed.). New York: Oxford University Press.

Mohr, D. C., Dick, L. P., Russo, D., Pinn, J., Boudewyn, A. C., Likosky, W., et al. (1999). The psychosocial impact of multiple sclerosis: Exploring the patient's perspective. *Health Psychology*, *18*(4), 376–382.

Park, C. L., Cohen, L. H., & Murch, R. L. (1996). Assessment and prediction of stress-related growth. *Journal of Personality*, *64*, 71–105.

Park, C. L., & Helgeson, V. S. (2006). Growth following highly stressful life events: Current status and future directions. *Journal of Consulting and Clinical Psychology, 74*(5), 791–796.

Park, C. L., Lechner, S. C., Antoni, M. H., & Stanton, A. L. (Eds.). (2009). *Medical illness and positive life change: Can crisis lead to personal transformation?* Washington, DC: American Psychological Association.

Tedeschi, R. G., & Calhoun, L. G. (1996). The Posttraumatic Growth Inventory: Measuring the Positive Legacy of Trauma. *Journal of Traumatic Stress*, *9* (3), 455–471.

Tedeschi, R. G., & Calhoun, L. G. (2004). Target article: Posttraumatic growth: Conceptual foundations and empirical evidence. *Psychological Inquiry, 15*(1), 1–18.

Tedeschi, R. G., Park, C. L., & Calhoun, L. G. (Eds.). (1998). *Posttraumatic growth: Positive changes in the aftermath of crisis*. Mahwah: Lawrence Erlbaum.

Tomich, P. L., & Helgeson, V. S. (2004). Is finding something good in the bad always good? Benefit finding among women with breast cancer. *Health Psychology, 23*, 16–23.

Posttraumatic Stress Disorder

Cortney J. Taylor, Whitney M. Herge and Annette M. La Greca
Department of Psychology, University of Miami, Coral Gables, FL, USA

Definition

By age 16 years, approximately two thirds of youth report having experienced at least one traumatic event (Costello, Erkanli, Fairbank, & Angold, 2002; Copeland, Keeler, Angold, & Costello, 2007). This is distressing in that exposure to a traumatic event can lead to the development of posttraumatic stress disorder (PTSD), which is a debilitating condition that is associated with several poor mental and physical health outcomes. As such, it is imperative to understand the development and course of PTSD, including its prevalence, comorbid conditions, assessment, treatment, and prognosis.

Diagnostic Criteria

PTSD is an anxiety disorder. Criteria for diagnosis include (a) exposure to a traumatic event that resulted in actual or threatened death or serious injury, or a threat to the physical integrity of the self or others, and also that the person's response involved intense fear, helplessness, or horror (American Psychiatric Association [APA], 2000, DSM-IV-TR, p. 467).

Further, an individual must meet criteria for three symptom clusters, each with more than 1 month duration: (b) one symptom of *reexperiencing* (e.g., recurrent distressing dreams of the event), (c) three or more symptoms of *avoidance* of stimuli associated with the trauma and *numbing* (e.g., efforts to avoid thoughts, feelings, or conversations associated with the trauma), and (d) two symptoms of increased *arousal* (e.g., difficulty falling or staying asleep). These symptoms must cause clinically significant distress or impaired functioning in the individual and cannot be accounted for by another disorder.

Proposed changes for DSM-V involve further explanation and expansion of the criteria. For example, criterion (a) may be expanded to include one or more of (1) experiencing the event, (2) witnessing the event, (3) learning the event occurred to someone close, or (4) experiencing repeated or extreme exposure to aversive details of the event. Also, the remaining criteria may be expanded to include the following: (b) one or more symptoms of *intrusion*, (c) one or more symptoms of *avoidance* of stimuli, (d) three or more *negative alterations in cognitions or mood* associated with the traumatic event, and (e) three or more symptoms of *alterations in arousal or reactivity* associated with the traumatic event (APA, 2010).

Further, there are differences in symptom presentation between children and adults who experience PTSD, which may be considered in DSM-V (APA, 2010). For example, within category B regarding intrusion, the DSM-V notes that children may have repetitive play reflecting the traumatic event, or trauma-specific reenactment during play, whereas adults may have recurrent, involuntary, or intrusive memories of the event. The DSM-V also specifies a different number of symptoms required to meet criteria for children than for adults. For example, adults are required to exhibit three or more symptoms, while children are only required to have two or more symptoms for both criterion D which are negative alterations in cognition and mood and criterion E alterations in arousal and reactivity, respectively.

Prevalence

Prevalence rates for PTSD vary greatly depending on the severity and type of traumatic event, as well as demographic factors. Prevalence rates of PTSD in youth range from 0.5% in typical community samples to 90% among sexually abused children (La Greca et al., 2012); the lifetime prevalence rate in adults is approximately 8% (APA, 2000, DSM-IV-TR, p. 466). Also, rates of PTSD are typically highest with events that involve violence, such as sexual abuse, terrorism, war, or the violent death of a loved one (Copeland et al., 2007). Women are twice as likely as men to evidence PTSD at some point in their lives (Foa, Keane, Friedman, & Cohen, 2008). The role of ethnicity is still unclear and evidence is mixed; some studies support an ethnic difference in rates of PTSD, while others suggest there are moderating factors such as differences in levels of exposure or socioeconomic status (Hamblen, 2007). Individuals from lower SES backgrounds have higher rates of PTSD than those with higher SES, and this may be due to the greater exposure to domestic violence or community violence among low SES individuals, which is associated with higher levels of PTSD.

Course of Disorder Over Time

Individuals who experience PTSD symptoms less than 1 month from the traumatic event may meet criteria for a diagnosis of acute stress disorder; if symptoms persist for more than 1 month, then the diagnosis is changed to PTSD. Individuals who present with PTSD for less than 3 months duration are considered to have the PTSD; if symptoms persist for more than 3 months, the diagnosis is *chronic* PTSD. Child physical or sexual abuse is often associated with a more chronic course of PTSD from childhood through adulthood. Finally, individuals who initially present with subsyndromal PTSD, but meet the full criteria at 6 months or more posttrauma, are considered to have *delayed-onset* PTSD. Some resilient individuals present with only a few symptoms of traumatic stress that never reach clinical thresholds and remit over time.

Common Comorbidities

PTSD is frequently comorbid with several other psychological disorders, including grief/bereavement, depression, anxiety, substance use and abuse, and health problems (Bonanno et al., 2010). Further, in children, PTSD is also commonly associated with behavioral concerns, such as acting out and disruptive behaviors (Cohen, Berliner, & Mannarino, 2010).

It is unclear whether PTSD plays a casual role in the development of additional comorbid conditions, or whether preexisting psychological conditions predispose a person to develop PTSD following a trauma (Perrin, Smith, & Yule, 2000). It is also possible that a broader underlying factor leads to the development of both PTSD and other conditions, which would account for the high rate of comorbidity (Perrin et al., 2000). Additional research is necessary to better understand the directionality of these comorbid relationships, which would also advance the conceptualization and treatment of PTSD.

Measures

There are several measures used in the assessment and diagnosis of PTSD in children and adults. The type of measure chosen depends on many factors, including the availability of a trained clinician, the individual's time and presenting condition(s), and the cost of the measure.

Clinician-administered interviews are the most thorough tools available and include (1) the Structured Clinical Interview for DSM-IV Axis I Disorders, used with adolescents and adults (SCID-I; First, Spitzer, Gibbon, & Williams, 1996); (2) the Kiddie-Schedule for Affective Disorders and Schizophrenia, used with children and adolescents (K-SADS-PL; Kaufman et al., 1997); and (3) the Diagnostic Interview for Children and Adolescents, used with children and adolescents (DICA-IV; Reich, Welner, & Herjanic, & MHS Staff, 1997). Each of these interviews includes a PTSD-specific module for assessing the presence of a traumatic event and any resulting symptoms required for diagnosis (APA, 2000). Although clinician-administered interviews are the most thorough diagnostic tool, they are also the most time consuming as well as the most expensive option.

Self-report measures of PTSD include the PTSD Checklist (PCL; Weathers et al., 1993) for adults, the Posttraumatic Stress Disorder Reaction Index (Steinberg, Brymer, Decker, & Pynoos, 2004), and the Trauma Symptom Checklist for Children (Briere, 1996). To assess a person's subjective response to trauma and/or stressful events more broadly, the Impact of Events Scale – Revised (IES-R; Weiss & Marmar, 1996) is often used with adolescents and adults. Although these measures are easy to administer and do not require a trained clinician, these measures provide significantly less detail and may require follow-up inquiry.

Treatment and Prognosis

Several treatment options are available for individuals with PTSD. Trauma-focused cognitive behavioral therapy (TFCBT) for children and adults and eye movement desensitization and reprocessing (EMDR) with adults have been indicated as effective treatments for individuals with PTSD (Foa et al., 2008; Bisson & Andrew, 2007). Another treatment option, sometimes utilized in conjunction with psychotherapy, is medication. Specifically, SSRIs and SNRIs are seen as first-line medication treatments for PTSD (Foa, et al., 2008).

Typically, PTSD symptoms decline drastically over the first-year posttrauma (Bonanno et al., 2010), while any further declines are more gradual (e.g., La Greca et al., 2010; Shaw et al., 1996). The prognosis for individuals with PTSD varies greatly depending on whether the individual has any comorbid conditions, their level of social support, and whether they have received treatment. Individuals with comorbid conditions, poor social support, and/or do not seek treatment will have a poorer prognosis than those who do not have these concerns. For individuals who do receive treatment, some evidence a decrease in symptoms after only four sessions of CBT, while others may require a longer course of treatment (Foa et al., 2008).

Cross-References

► Stress

References and Readings

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders, Text Revision* (4th ed.). Washington, DC: Author.

American Psychiatric Association. (2010). *DSM-5 development*. Downloaded from http://www.dsm5.org/ProposedRevisions/Pages/proposedrevision.aspx?rid=165

Bisson, J., & Andrew, M. (2007). Psychological treatment of post-traumatic stress disorder (PTSD). *Cochrane Database of Systematic Reviews*, *2007*(3), CD003388. doi:10.1002/14651858.CD003388.pub3.

Bonanno, G. A., Brewin, C. R., Kaniasty, K., & La Greca, A. M. (2010). Weighing the costs of disaster: Consequences, risks, and resilience in individuals, families, and communities. *Psychological Science in the Public Interest, 11*(1), 1–49.

Briere, J. (1996). *Trauma Symptom Checklist for Children (TSCC), professional manual*. Odessa, FL: Psychological Assessment Resources.

Cohen, J. A., Berliner, L., & Mannarino, A. (2010). Trauma focused CBT for children with co-occurring trauma and behavior problems. *Child Abuse & Neglect, 34*, 215–224.

Copeland, W. E., Keeler, G., Angold, A., & Costello, E. J. (2007). Traumatic events and posttraumatic stress in childhood. *Archives of General Psychiatry, 64*, 577–584.

Costello, E. J., Erkanli, A., Fairbank, J. A., & Angold, A. (2002). The prevalence of potentially traumatic events in childhood and adolescence. *Journal of Traumatic Stress, 15*, 99–112.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. (1996). *Structured clinical interview for the DSM-IV axis I disorders*. Washington, DC: American Psychiatric Association.

Foa, E. B., Keane, T. M., Friedman, M. J., & Cohen, J. (Eds.). (2008). *Effective treatments for PTSD: Practice guidelines from the International Society for Traumatic Stress Studies* (2nd ed.). New York: Guilford Press.

Hamblen, J. (2007). *PTSD in children and adolescents*. Downloaded from http://www.ncptsd.va.gov/facts/specific/fs_children.html.

Kaufman, J., Birmaher, B., Brent, D., Rao, U., Flynn, C., Moreci, P., et al. (1997). Schedule for affective disorders and schizophrenia for school-age children-present and lifetime version (K-SADS-PL): Initial reliability and validity data. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*, 980–988.

La Greca, A. M., Silverman, W., Lai, B., & Jaccard, J. (2010). Hurricane-related exposure experiences and stressors, other life events, and social support: Concurrent and prospective impact on children's persistent posttraumatic stress. *Journal of Consulting and Clinical Psychology, 78*(6). doi:10.1037/a0020775.

La Greca, A. M., Taylor, C. J., & Herge, W. (2012). Traumatic stress disorders in children and adolescents. In G. Beck & D. Sloan (Eds.), *Oxford Handbook of traumatic stress disorders*. Oxford: Oxford University Press.

Perrin, S., Smith, P., & Yule, W. (2000). Practitioner review: The assessment and treatment of post-traumatic stress disorder in children and adolescents. *Journal of Child Psychology and Psychiatry, 41*(3), 277–289.

Reich, W., Welner, Z., Herjanic, B., & Staff, M. H. S. (1997). *Diagnostic Interview for Children and Adolescents-IV (DICAIV): Software manual for the child/adolescent and parent versions*. Toronto: Multi-Health Systems.

Shaw, J. A., Applegate, B., & Schorr, C. (1996). Twenty-one-month follow-up study of school-age children exposed to Hurricane Andrew. *Journal of the American Academy of Child and Adolescent Psychiatry, 35*, 359–364.

Steinberg, A. M., Brymer, M. J., Decker, K. B., & Pynoos, R. S. (2004). The University of California at Los Angeles post-traumatic stress disorder reaction index. *Current Psychiatry Reports, 6*(2), 96–100.

Weathers, F., Litz, B., Herman, D., Huska, J., & Keane, T. (1993, October). *The PTSD Checklist (PCL): Reliability, validity, and diagnostic utility*. Paper presented at the Annual Convention of the International Society for Traumatic Stress Studies, San Antonio, TX. APA, 2000.

Weiss, D. S., & Marmar, C. R. (1996). The impact of event scale – revised. In J. Wilson & T. M. Keane (Eds.), *Assessing psychological trauma and PTSD* (pp. 399–411). New York: Guilford Press.

Potential Years of Life Lost (PYLL)

► Life Years Lost

Power Spectral Analysis

► Quantitative EEG Including the Five Common Bandwidths (Delta, Theta, Alpha, Sigma, and Beta)

Practice Guideline

► Clinical Practice Guidelines

Praise

► Prayer

Prayer

Donna C. Burdzy and Kenneth Pargament
Department of Psychology, Bowling Green State University, Bowling Green, OH, USA

Synonyms

Meditation; Praise; Supplication; Thanksgiving; Worship

Definition

Prayer in all of its variations can be defined by two fundamental principles: (1) prayer is a form of communication and (2) the exchange of communication takes place between the self and the transcendent, immanent, and numinous forces that represent human notions of the sacred. Defining prayer in this way broadens William James' classic conceptualization of prayer as "every kind of inward communion or conversation with the power recognized as divine" to include not only that which comes from God but everything that is imbued with the power of sacredness.

Description

Throughout history, humankind has manifested a yearning to communicate with the sacred through prayer. Expressed in vastly different cultures and religious traditions, prayer constitutes a universal phenomenon that plays a crucial role in humanity's religious experience. In fact, for many individuals, prayer is their primary religious practice.

Prayer, in the theocentric Judaic, Christian, and Islamic religious traditions, represents a way to express thanks to God, participate in God's will, and move closer to God. From a theocentric perspective, prayer is, simply put, communication with God. Prayer, however, is not limited to God-centered religions; it also features prominently in the practices of nontheistic religious traditions such as Zen and Theravada Buddhism, Jainism, and Taoism as well as animistic and pantheistic belief systems such as Shinto and the indigenous religions that feature the worship of nontheistic spiritual entities. Secular individuals without religious affiliations or theistic beliefs also pray regularly in a variety of ways.

Types of Prayer

Scholars have devised a number of classification schemes in an attempt to impose some order on the extraordinarily diverse modes of prayer expression. Traditional descriptive typologies have focused on the reasons people pray as well as on the content of prayers. Gill (1994) classified prayers according to their intent: petition, invocation, thanksgiving, dedication, supplication, intercession, confession, penitence, and benediction prayers. Heiler (1932) proposed a rich typology to capture the "astonishing multiplicity" of the forms of prayer: primitive; ritual; Hellenistic; philosophical; personal; mystical; prophetic; the prayers of great religious personalities; the prayers of great men, poets, and artists; prayer in public worship; and prayer as a law of duty and good works.

More recently, researchers have developed typologies by asking people what they pray about, when they pray, why they pray, and to whom or what they pray. Ladd and Spilka (2002) proposed a threefold scheme which distinguishes among recipients of prayer. Inward prayer is directed toward one's inner self, outward prayer represents a connection with another human, and upward prayer signifies sacred communication with the divine. Poloma and Pendleton (1991) classified prayer in terms of four concise categories. Petitionary prayer involves requests to fulfill one's own spiritual or material needs or those of others by asking for guidance, forgiveness, and physical well-being. Meditative prayer involves thinking about the

divine, communicating with the sacred, or merely being in the presence of the sacred. Ritual prayer involves silent or verbal recitation of specific religious texts or mantras from memory or by reading scripture. Colloquial prayer, which takes the form of a conversation between individuals and their God or divine figure, may blend aspects of petitionary and meditative prayer. Poloma and Gallup (1991) found that while Americans most widely practice colloquial prayer, meditative prayer was associated with a closer relationship with God and higher levels of well-being.

Most prayer typologies have been created out of research involving individuals from predominantly Western cultures in which prayer is typically experienced in the context of organized, theistic religion. However, Banzinger, Janssen and Scheepers (2008) found that the prayer experiences of individuals from the Netherlands, a highly secularized society, also could be categorized using a typology similar to Poloma and Pendleton's scheme. While Banzinger et al. argued for the creation of a secular prayer type, their findings support the notion that some types of prayer experiences may be common to both secular and nonsecular individuals.

Prayer and Well-Being

The way that people communicate with the sacred can influence their physical and psychological health. Studies have linked prayer to better medical outcomes among patients dealing with cardiovascular disease, cancer, migraines, chronic pain post surgical recovery, and HIV.

Research studies have also shown that people who pray more frequently report a greater sense of well-being. More specifically, prayer has been associated with higher levels of overall mental health, lower levels of depression and anxiety, higher levels of self-esteem, and more positive mood among individuals with post-traumatic stress disorder ("PTSD"). How well prayer works may depend at least in part on how people perceive God. Bradshaw, Ellison and Flannelly (2008) found that frequency of prayer was positively correlated with higher rates of psychopathology among individuals who perceived God as remote or not loving. In contrast, among individuals who viewed God as close and loving, more prayer was tied to lower psychopathology. Other factors may also affect the relationship between prayer and well-being, including the content and intent of prayers, an individual's level of spiritual maturity, and the availability of additional coping resources.

Although studies have consistently demonstrated significant correlations between prayer and well-being, researchers have not yet identified the underlying psychological mechanisms that account for this relationship. Prayer may affect well-being indirectly by strengthening the relationships between individuals, God, and their faith community which may buffer the negative impact of stressors on health and well-being. Prayer may also manifest its effects through other psychological factors, such as increasing feelings of gratitude. Researchers have shown that greater frequency of prayer is associated with increased feelings of gratitude which are, in turn, correlated with lower rates of depression as well as higher levels of optimism and fewer negative health symptoms.

Prayer for one's own health and for the health of others represent the two most frequently used alternative health treatments in the United States. The proportion of Americans who pray to alleviate health problems increased from 43% in 2002 to 49% in 2007. This number may grow even further as the number of individuals who live with chronic physical or psychological illnesses rises. Many individuals who pray have also expressed a desire to integrate prayer practices into their medical treatment. In one study, 79% of critical care providers were asked by patients or their families to pray for them. In another survey, 48% of American patients indicated that they would like their doctor to pray for them. Over 90% of these patients felt that their doctor's prayers on their behalf had enhanced their health and aided in their recovery.

Some clinicians have begun to respond to the promising research findings and to the desires of many of their patients by considering how to incorporate prayer into specific treatments. Seventy-three percent of critical care nurses surveyed by Tracy et al. (2005) prayed while treating clients,

and 81% of these nurses had recommended the use of prayer to their patients. Pargament, Smith, Koenig, and Perez (1998) suggested that positive forms of religious coping such as seeking spiritual support through prayer could be successfully integrated into psychotherapy. The appropriateness of integrating spiritual practices such as prayer into treatment, however, remains a highly divisive topic within the health-care community.

Cross-References

- ▶ Meditation
- ▶ Religion
- ▶ Religious Ritual
- ▶ Spirituality

References and Readings

Bänzinger, S., Janssen, J., & Scheepers, P. (2008). Praying in a secularized society: An empirical study of praying practices and varieties. *The International Journal for the Psychology of Religion, 18*, 256–265.

Bradshaw, M., Ellison, C. G., & Flannelly, K. J. (2008). Prayer, god imagery, and symptoms of psychopathology. *Journal for the Scientific Study of Religion, 47*(4), 644–659.

Fincham, F. D., Beach, S. R. H., Lambert, N., Stillman, T., & Braithwaite, S. R. (2008). Spiritual behaviors and relationship satisfaction: A critical analysis of the role of prayer. *Journal of Social and Clinical Psychology, 27*(4), 362–388.

Gill, S. D. (1994). Prayer. In M. Eliade (Ed.), *The encyclopedia of religion* (2nd ed., Vol. 11). New York: Macmillan Reference.

Heiler, F. (1932). *Prayer: A study in the history and psychology of religion*. London: Oxford University Press.

Krause, N. (2009). Lifetime trauma, prayer and psychological distress in later life. *International Journal for the Psychology of Religion, 19*(1), 55–72.

Ladd, K. L., & Spilka, B. (2002). Inward, outward, upward: Cognitive aspects of prayer. *Journal for the Scientific Study of Religion, 41*(3), 475–484.

Levin, J. (1996). How prayer heals: A theoretical model. *Alternative Therapies in Health & Medicine, 2*(1), 66–73.

McCullough, M. E. (1995). Prayer and health: Conceptual issues, research review, and research agenda. *Journal of Psychology and Theology, 23*(1), 15–29.

Pargament, K. I. (1997). *The psychology of religion and coping: Theory, research, practice*. New York: The Guilford Press.

Pargament, K. I., Smith, B. W., Koenig, H. G., & Perez, L. (1998). Patterns of positive and negative religious coping with major life stressors. *Journal for the Scientific Study of Religion, 37*(4), 710–724.

Poloma, M. M., & Gallup, G. H., Jr. (1991). *Varieties of prayer: A survey report*. Philadelphia: Trinity Press International.

Poloma, M. M., & Pendleton, B. F. (1991). The effects of prayer and prayer experiences on general wellbeing. *Journal of Psychology and Theology, 19*, 71–83.

Tracy, M. F., Lindquist, R., Savik, K., Watanuki, S., Sendelbach, S., Kreitzer, M. J., et al. (2005). Use of complementary and alternative therapies: A national survey of critical care nurses. *American Journal of Critical Care, 14*, 404–414.

Prediabetes

- ▶ Hyperinsulinemia
- ▶ Impaired Glucose Tolerance

Pregnancy

- ▶ Coffee Drinking, Effects of Caffeine
- ▶ Gestation

Pregnancy Complications

- ▶ Pregnancy Outcomes: Psychosocial Aspect

Pregnancy Outcomes: Psychosocial Aspect

Michele L. Okun
Sleep Medicine Institute and Department of Psychiatry, School of Medicine, University of Pittsburgh, Pittsburgh, PA, USA

Synonyms

Depression; Pregnancy complications

Definition

For most women, pregnancy is viewed as a natural and joyful event. In more than 80% of pregnancies, the delivery process is unremarkable, with no physiological or psychological complications. Indeed, the most notable changes associated with pregnancy often occur in social and partner relations, as well as changes to one's lifestyle. For instance, sleep deprivation and disturbance are the most frequently reported pregnancy-related disturbances. Pregnancy-related sleep disturbances, in turn, impact numerous facets of life including mood, cognition, social functioning, and memory (Harding, 1975).

For a small percentage of women, pregnancy and delivery are complicated by a variety of adverse outcomes. Preeclampsia, intrauterine growth restriction (IUGR), or preterm delivery can significantly affect the psychological health of women. Psychosocial aspects of pregnancy outcomes range along a continuum and are multidimensional. Psychosocial functioning in association with pregnancy complications may be a function of coping strategies, social support, and the overall emotional health of the mother and father. Sociodemographic factors, including parental age, socioeconomic status, and relationship quality also influence parental response to pregnancy complications. In the wake of adverse outcomes, parents may feel guilt, depression, anger, resentment, or withdrawal. Parents of infants transferred to the neonatal intensive care unit (NICU) may experience additional psychological burden, sometimes altering relationship dynamics (Zager, 2009) or mother-child bonding. Links between psychosocial factors and adverse pregnancy outcomes are currently underrecognized by health-care providers, which suggest that systematic prospective research is needed to more convincingly establish the significance of psychosocial factors and their potential for prevention.

Cross-References

▶ Psychosocial Factors

References and Readings

Harding, M. E. (1975). Maternity. In M. E. Harding (Ed.), *The Way of All Women* (pp. 160–170). New York: Harper and Row.

Lederman, R., & Weis, K. (2009). *Psychosocial Adaptation to Pregnancy: Seven Dimensions of Maternal Role Development* (3rd ed.). Dordrecht: Springer.

Zager, R (2009). *Glob. libr. women's med.*, (ISSN: 1756-2228); doi:10.3843/GLOWM.1015

Pregnancy Spacing

▶ Family Planning

Prehypertension

Jonathan Newman
Columbia University, New York, NY, USA

Description

In 2003, the seventh report of the Joint National Committee guidelines (JNC 7) proposed a classification for normal blood pressure (BP) and prehypertension based on the average of two more properly measured readings:

Normal blood pressure: systolic $<$ 120 mmHg and diastolic $<$ 80 mmHg

Prehypertension: systolic 120–139 mmHg or diastolic 80–89 mmHg

Compared to individuals with normal BP, prehypertensive individuals have a greater number of traditional cardiovascular disease (CVD) risk factors and have a greater risk of developing CVD independent of other CVD risk factors than individuals with BP $<$ 120/80. Prehypertensive individuals also have a greater risk of developing hypertension than normotensive individuals. Therefore, prehypertension can be conceptualized as an intermediate phenotype at elevated risk of developing traditional risk factors for CVD (such as hypertension) and at independent risk of developing CVD itself.

However, the ideal surveillance and management strategies for patients with prehypertension have not been well defined. Due to the associations between prehypertension and the development of overt hypertension and CVD, in 2007, the United States Preventive Services Task Force (USPSTF) recommended yearly BP screening for prehypertensive individuals and every other year screening for those with normotension. It remains unclear, however, how much of the excess CVD risk associated with prehypertension is due to the BP itself and how much is related to associated CVD risk factors: some analyses have shown that the excess CVD risk associated with prehypertension is attenuated after controlling for concomitant CVD risk factors. A more concrete problem with the diagnosis of prehypertension, however, may be the number of people affected: a recent analysis of the US population found that 39% of adults were normotensive, 31% prehypertensive, and 29% hypertensive. This suggests only a minority of Americans have normal blood pressure and raises the question of whether prehypertension should be defined as a "disease" state.

While there is limited evidence to suggest that treatment of prehypertension may prevent the development of hypertension, JNC 7 recommends careful follow-up for the development of hypertension or signs of end-organ damage (e.g., renal dysfunction or left-ventricular hypertrophy). Without the presence of CVD or other CVD risk factors (such as diabetes), prehypertension is generally treated with nonpharmacologic therapies such as weight loss, sodium restriction, dietary modification, and exercise.

References and Readings

Greenlund, K. J., Croft, J. B., & Mensah, G. A. (2004). Prevalence of heart disease and stroke risk factors in persons with prehypertension in the United States, 1999–2000. *Archives of Internal Medicine, 164*(19), 2113–2118.

Julius, S., Nesbitt, S. D., Egan, B. M., Weber, M. A., Michelson, E. L., Kaciroti, N., Black, H. R., Grimm, R. H., Jr., Messerli, F. H., Oparil, S., & Schork, M. A. (2006). Trial of Preventing Hypertension (TROPHY) Study Investigators. Feasibility of treating prehypertension with an angiotensin-receptor blocker. *The New England Journal of Medicine, 354*(16), 1685–1697.

U.S. Preventive Services Task Force. (2007). Screening for high blood pressure: U.S. Preventive Services Task Force reaffirmation recommendation statement. *Annals of Internal Medicine, 147*(11), 783–786.

Zhang, Y., Lee, E. T., Devereux, R. B., Yeh, J., Best, L. G., Fabsitz, R. R., & Howard, B. V. (2006). Prehypertension, diabetes, and cardiovascular disease risk in a population-based sample: The Strong Heart Study. *Hypertension, 47*(3), 410–414.

Preimplantation Genetic Diagnosis

► In Vitro Fertilization, Assisted Reproductive Technology

Prejudice

► Stigma

Premenstrual Headache

► Migraine Headache

Pressure

► Stress

Prevalence

Linda Carroll
Department of Public Health Sciences, University of Alberta, Edmonton, AB, Canada

Synonyms

Prevalence number; Prevalence rate

Definition

Prevalence is a measure of frequency of an illness, disease, or health conditions. Unlike incidence, which reflects new occurrences or changes in health states, prevalence is concerned with already existing health conditions, regardless of whether that health condition is of recent onset or is long-standing. Thus, prevalence of a particular condition refers to the proportion of the population which has that condition at a specified time. It is usually presented as x cases per 1,000 (or 10,000 or 100,000) people in the population. There are two types of prevalence: point prevalence (the type of prevalence most commonly reported) and period prevalence. Consider the example of the common cold. The "point prevalence" of the common cold in New York City means the proportion of people in New York City with a cold at a given point in time, i.e., the number of New York City residents with a cold on a specific day divided by the total number of New York City residents on that day. It is like a "prevalence snapshot." A 1-month "period prevalence" means the proportion of people in New York City who have had a cold at any point within the past month. It is analogous to time-lapse photography, reflecting the health state that has existed over a set period of time. A variation is lifetime prevalence or the proportion of people who have had the condition at any point during their lifetime.

Prevalence is usually measured in surveys or cross-sectional studies and reflects the burden of disease, rather than risk (incidence) of disease, which must be measured in longitudinal studies. Because prevalence is a measure of existing health conditions, it is affected by both incidence of that health state (the rate at which new cases develop) and the duration of that health state. A health condition might have a short duration because it is rapidly fatal, for example, rabies or Ebola, or because recovery is rapid, such as the case in the "24-h flu." Prevalence of a particular health condition can increase because there is an increase in incidence (e.g., during an influenza epidemic) or because people with that health condition are living longer with that condition (e.g., treatments may have extended the lives of those who suffer from that health condition). Alternatively, more effective treatments may shorten the recovery time.

Because prevalence is affected by recovery or death, it is an unreliable estimate of disease risk. This can be illustrated in the following example. The Framingham Heart Study reported equal prevalence of coronary heart disease in men and women. This could lead to the mistaken conclusion that men and women are at equal risk of developing coronary heart disease. However, the follow-up studies demonstrated that men were at greater risk of developing coronary heart disease and also had a higher fatality rate; while women were less likely to develop heart disease, but when they did, it was also less likely to be fatal. Thus, the prevalence in men and women was roughly equal because a lower risk was coupled with a longer duration of disease in women.

References and Readings

Oleckno, W. A. (2002). *Essential epidemiology: Principles and applications*. Long Grove, IL: Waveland Press.

Rothman, K. J., Greenland, S., & Lash, T. L. (2008). *Modern epidemiology* (3rd ed.). Philadelphia: Lippincott Williams & Wilkins.

Szklo, M., & Nieto, F. J. (2007). *Epidemiology: Beyond the basics* (2nd ed.). Sudbury, MA: James and Bartlett.

Prevalence Number

► Prevalence

Prevalence Rate

► Prevalence

Prevention: Primary, Secondary, Tertiary

Linda C. Baumann and Alyssa Karel
School of Nursing, University of Wisconsin-Madison, Madison, WI, USA

Synonyms

Levels of prevention

Definition

The natural history of disease is the course from onset to resolution (Last, 2000). The goal of epidemiology is to identify and understand causal factors of disease, disability, and injury so that effective interventions can be implemented to prevent the occurrence of adverse processes before they begin or progress (Stanhope & Lancaster, 2008). The definitions used in public health distinguish between primary prevention, secondary prevention, and tertiary prevention (Commission on Chronic Illness, 1957).

Description

The term "primary prevention" refers to intervention measures to prevent the occurrence (incidence) of new disease, disability, or injury (Leavell & Clark, 1965). This intervention must be implemented prepathogenesis and directed at individuals or groups at risk. Primary prevention efforts include health promotion and specific protection and are generally aimed at populations, not individuals (see Fig. 1). The application of primary prevention extends beyond medical problems and includes the prevention of other concerns that impact health and well-being, such as violence to environmental degradation. Education and public policy are major strategies for primary prevention.

Two other levels of prevention are termed secondary and tertiary prevention. Secondary prevention is a set of measures used for early detection and prompt intervention to control a problem or disease (prevalence) and minimize the consequences. Secondary prevention encompasses interventions that increase the probability that a person with a condition will have it diagnosed at a stage that treatment is likely to result in cure or reduction in the severity of a condition. Health screening is a major strategy of secondary prevention. Tertiary prevention focuses on the reduction of further complications of an existing disease, disability, or injury, through treatment and rehabilitation.

A landmark report published by the Institute of Medicine entitled: *Reducing Risks for Mental Disorders* (IOM, 1994) evaluated the body of research on the prevention of mental disorders. This report offered new definitions of prevention and provided recommendations on federal policies and programs. Levels of prevention across the natural disease history (Fig. 1) were defined as "prevention, treatment, and rehabilitation." Prevention, according to the IOM report (1994), is similar to the concept of primary prevention and refers to interventions to delay or avoid the initial onset of a disorder. Further, prevention has three types: universal, selective, and indicated, to reduce new cases. Universal efforts are directed to the entire population; selective prevention is for those at significant risk of a disorder due to biological, social, or psychological risk factors; and indicated prevention is for those with a mild disorder that has the potential to become more severe if not addressed in a timely manner.

Treatment refers to the identification of individuals with a disorder and providing treatment for those disorders, which includes interventions to reduce the likelihood of future co-occurring disorders. Maintenance refers to interventions that are oriented to reduce relapse and recurrence and to provide rehabilitation. Maintenance incorporates what public health defines as some forms of secondary and all forms of tertiary prevention.

THE NATURAL HISTORY OF ANY DISEASE OF MAN				
Interrelations of Agent, Host, and Environmental Factors Production of STIMULUS	Reaction of the HOST to the STIMULUS Early pathogenesis	Discemible Early Lesions	Advanced Disease	Convalescence
Prepathogenesis period	**Period of Pathogenesis**			

HEALTH PROMOTION	SPECIFIC PROTECTION	EARLY DIAGNOSIS and PROMPT TREATMENT	DISABILITY LIMITATION	REHABILITATION
Health education Good standard of nutrition adjusted to developmental phases of life Attention to personality development Provision of adequate housing, recreation and agreeable working conditions Marriage counseling and sex education Genetics Periodic selective examinations	Use of specific immunizations Attention to personal hygiene Use of environmental sanitation Protection against occupational hazards Protection from accidents Use of specific nutrients Protection from carcinogens Avoidance of allergens	Case-finding measures, individual and mass Screening surveys Selective examinations Objectives: To cure and prevent disease processes To prevent the spread of communicable diseases To prevent complications and sequelae To shorten period of disability	Adequate treatment to arrest the disease process and to prevent further complications and sequelae Provision of facilities to limit disability and to prevent death	Provision of hospital and community facilities for retraining and education for maximum use of remaining capacities Education of the public and industry to utilize the rehabilitated As full employment as possible Selective placement Work therapy in hospitals Use of sheltered colony
Primary Prevention		**Secondary Prevention**		**Tertiary Prevention**
LEVELS of APPLICATION of PREVENTIVE MEASURES				

Prevention: Primary, Secondary, Tertiary, Fig. 1 Levels of application of preventive measures in the natural history of disease

The concepts of risk and protective factors, risk reduction, and enhancement of protective factors (also referred to as fostering resilience) are central to most evidence-based prevention programs. Risk factors are those characteristics, variables, or hazards, that if present for a given individual, make it more likely that this individual, rather than someone selected at random from the general population, will develop a disorder. Protective factors improve a person's response to an environmental hazard resulting in an adaptive outcome.

The Agency for Health Research and Quality (AHRQ) provides ongoing administrative, research, technical, and dissemination support to the US Preventive Services Task Force (USPSTF). http://www.USPreventiveServicesTaskForce.org. The USPSTF is an independent panel of non-federal experts in prevention and evidence-based medicine, and is composed of an interdisciplinary mix of primary care providers (physicians, nurses, and health behavior specialists). The USPSTF conducts scientific evidence reviews of a broad range of clinical preventive health care services (such as screening, counseling, and preventive medications) and develops recommendations for primary care clinicians and health systems. These recommendations are published in the form of "Recommendation Statements."

References and Readings

Commission on Chronic Illness. (1957). *Chronic illness in the United States* (Vol. 1). Cambridge, MA: Harvard University Press.

Institute of Medicine. (1994). *Reducing risks for mental disorders*. Washington, DC: National Academy Press.

Last, J. M. (2000). *A dictionary of epidemiology* (4th ed.). New York: Oxford University Press.

Leavell, H. R., & Clark, E. G. (1965). *Preventive medicine for the doctor in his community* (3rd ed.). New York: McGraw-Hill.

Stanhope, M. & Lancaster, J. (2008). *Public health nursing: Population-centered health care in the community* (7th ed.). St. Louis, MO: Mosby Elsevier.

Preventive Care

Shannon Idzik
University of Maryland School of Nursing and the University of Maryland Medical Center Emergency Department, Baltimore, MD, USA

Definition

Preventive care is the branch of health care that strives to prevent mental and physical illnesses. Many members of the health care team must partner together to achieve proper preventive care. Preventive care is divided into three levels of care: primary prevention, secondary prevention, and tertiary prevention.

Primary preventive care is the prevention of disease in a susceptible population through health promotion, education, and protective efforts. Ensuring adequate nutrition, advising patients about skin protection from ultraviolet radiation, educating about seat belt use, promoting safe home and work environments, prescribing oral fluoride supplementation in children with fluoride-deficient water, and administrating immunizations are all examples of primary preventive care.

Secondary preventive care is the prevention of disease through screening and early detection. Early recognition of disease through health care screening allows treatment to occur early in the course of the disease and may decrease complications. Examples of screening procedures that lead to the prevention of disease include fecal occult blood testing for detecting colon cancer, Pap smear for detecting early cervical cancer, routine mammography for early breast cancer, blood pressure and blood cholesterol measurement, oral examinations for early dental caries, and use of screening tools for depression.

Tertiary preventive care is the prevention of disease progression and disease sequelae after a chronic or irreversible disease diagnosis has been made. Limiting disability and promoting rehabilitation is important in tertiary prevention. Examples of tertiary prevention efforts include prescribing anticlotting agents such as aspirin in

patients who have cardiovascular disease, physical rehabilitation in patients who have suffered a stroke, and endodontic therapy in patients with severe dental decay.

Cross-References

► Prevention: Primary, Secondary, Tertiary

References and Readings

Partnership for Prevention. (2007). *Preventive care: A national profile on use, disparities, and health benefits*. Retrieved October 20, 2011 from http://www.prevent.org/Publications-and-Resources.aspx

Patient Protection and Affordable Care Act (PPACA), Pub. L. No. 111–148, 124 Stat. 119 (2010). Retrieved October 20, 2011 from http://www.gpo.gov/fdsys/pkg/PLAW-111publ148/pdf/PLAW-111publ148.pdf

U.S. Preventive Services Task Force. (2011). Retrieved October 20, 2011 from http://www.uspreventiveservicestaskforce.org/recommendations.htm

Preventive Medicine Research Institute (Ornish)

Manjunath Harlapur[1] and Daichi Shimbo[2]
[1]Center of Behavioral Cardiovascular Health, Division of General Medicine, Columbia University, New York, NY, USA
[2]Center for Behavioral Cardiovascular Health, Columbia University, New York, NY, USA

Synonyms

Dean Ornish

Definition

Preventive Medicine Research Institute (PMRI) is a nonprofit research institute located in Sausalito, California, and founded by Dr. Dean Ornish. The mission of PMRI is to conduct scientific research examining the effects of diet and lifestyle choices on health outcomes.

Description

PMRI is a nonprofit research institute that is involved with scientific research studying the effects of diet and lifestyle choices on health and diseases. PMRI was founded by Dr. Dean Ornish, a leading researcher and physician advocating this type of prevention. The mission of PMRI is to conduct scientific research examining the effects of diet and lifestyle choices on health outcomes. In addition to research, another emphasis of PMRI is to educate health professionals and the lay public about the importance of preventive medicine and the benefits of lifestyle changes including diet, exercise, and stress management.

Cross-References

► Coronary Artery Disease

References and Readings

http://www.pmri.org/

Previous Smokers

► Ex-Smokers

Pride

► Self-Esteem

Primary Care

Shannon Idzik
University of Maryland School of Nursing and the University of Maryland Medical Center Emergency Department, Baltimore, MD, USA

Definition

Primary care is a component of integrated health care in which comprehensive and accessible care

is provided to a defined population. It is not disease- or organ-specific, but rather examines a person's overall state of health and well-being. Primary care is often the first point of contact into a health system for persons with a health concern including those with acute and chronic physical, mental, and social health issues. Primary care is a longitudinal and continuous approach to health maintenance including health promotion, disease prevention, health education, and counseling and includes diagnosis, treatment, and management of acute and chronic conditions. Primary care focuses on the provision of primary, secondary, and tertiary prevention measures, such as screenings, immunizations, and prevention of disease progression or sequelae. In primary care, the patient is seen as a partner in their health and health decisions. The primary care provider partners with the patient to coordinate other health services which includes a collaboration with and referral to other members of the health care team. Continuity in primary care is essential to develop and establish a patient-provider relationship. Primary care is not setting-specific and can be provided across a continuum of health settings such as the patient's private residence, provider office, hospital, or long-term care facility.

Cross-References

- ▶ Clinical Settings
- ▶ Family Practice/Medicine
- ▶ Primary Care
- ▶ Primary Care Physicians
- ▶ Primary Care Provider

References and Readings

American Academy of Family Physicians. (2011). *Primary care*. Retrieved from http://www.aafp.org/online/en/home/policy/policies/p/primarycare.html#Parsys0002. Accessed October 20, 2011.

Patient Protection and Affordable Care Act (PPACA), Pub. L. No. 111–148, 124 Stat. 119 (2010). Retrieved from http://www.gpo.gov/fdsys/pkg/PLAW-111publ148/pdf/PLAW-111publ148.pdf. Accessed October 20, 2011.

Primary Care Physicians

Steven Gambert
Department of Medicine, School of Medicine, University of Maryland, Baltimore, MD, USA

Synonyms

Family physician; General internist; PCP; PMD; Primary care provider; Primary medical doctor

Definition

A primary care physician is the physician who provides primary care, the physician selected to be the first doctor contacted for any medical condition. The physician acts as the patient's "gatekeeper," providing ongoing medical care, preventive services, medical counseling, and referrals to specialists as needed. Examples of physicians who may be considered to be primary care physicians include family medicine physicians, internal medicine physicians, OB/GYN physicians, pediatricians, and at times emergency medicine physicians. The number of primary care physicians has been declining in recent years with more physicians seeking careers as subspecialists or pursuing specialty care with financial rewards and increasing demands on time being major reasons for this change (Bodenheimer, 2006).

Cross-References

- ▶ Primary Care Providers

References and Readings

Bodenheimer, T. (2006). Primary care – will it survive? *New England Journal of Medicine, 355*(9), 861–864.

Primary Care Provider

- ▶ Primary Care Physicians
- ▶ Primary Care Providers

Primary Care Providers

Shannon Idzik
University of Maryland School of Nursing and the University of Maryland Medical Center Emergency Department, Baltimore, MD, USA

Synonyms

Family physician; General internist; Primary care provider; Primary medical doctor

Definition

Primary care provider is a generalist clinician who provides integrated accessible health care to a defined population. Nurse practitioners, physicians, and physicians' assistants who provide primary care are specially trained to provide primary care services. The primary care provider develops a sustained relationship with the patient and oversees all aspects of the patient's health. The primary care provider partners with the patient to coordinate other health services which includes a collaboration with and referral to other members of the health care team. Primary care providers are advocates for the patient throughout the entire health care system.

Cross-References

- ▶ Primary Care
- ▶ Primary Care Physicians
- ▶ Primary Care Provider

References and Readings

American Academy of Family Physicians. (2011). *Primary care*. Retrieved October 20, 2011, from http://www.aafp.org/online/en/home/policy/policies/p/primarycare.html#Parsys0002

Patient Protection and Affordable Care Act (PPACA), Pub. L. No. 111–148, 124 Stat. 119 (2010). Retrieved October 20, 2011, from http://www.gpo.gov/fdsys/pkg/PLAW-111publ148/pdf/PLAW-111publ148.pdf

Primary Medical Doctor

- ▶ Primary Care Physicians
- ▶ Primary Care Providers

Primary Raynaud's Phenomenon

- ▶ Raynaud's Disease and Stress
- ▶ Raynaud's Disease: Behavioral Treatment

Principle of Equipoise

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Clinical equipoise; Equipoise

Definition

Clinical equipoise exists when all of the available evidence about a new intervention/treatment does not show that it is more beneficial than an alternative and, equally, does not show that it is less beneficial than the alternative. For example, to be able to conduct a clinical trial that involves administering an investigational treatment that may confer therapeutic benefit to subjects for whom such benefit is desirable to some individuals, and to administer a control intervention treatment that is not capable of conferring therapeutic benefit to others, there cannot be any evidence that suggests that the investigational intervention shows greater efficacy than the control treatment or that it leads to greater side effects than the control treatment.

When individuals agree to participate in a clinical study, they do so with the understanding that all of the treatments are assumed to be of equal value. By the end of the trial, there may be compelling evidence that the investigational intervention is acceptably safe and statistically significantly more effective than the control intervention, but the study must be started with a good faith belief that the two treatments are of equal merit.

Treating subjects in clinical studies (trials) in an ethical manner is of paramount importance. Clinical equipoise is a cornerstone of such ethical conduct. Other fundamental ethical principles include respect for persons, beneficence, and justice (see Turner, 2010).

Derenzo and Moss (2006) commented as follows:

> Each study component has an ethical aspect. The ethical aspects of a clinical trial cannot be separated from the scientific objectives. Segregation of ethical issues from the full range of study design components demonstrates a flaw in understanding the fundamental nature of research involving human subjects. Compartmentalization of ethical issues is inconsistent with a well-run trial. Ethical and scientific considerations are intertwined.

Cross-References

- ▶ Clinical Trial
- ▶ Randomized Clinical Trial

References and Readings

Derenzo, E., & Moss, J. (2006). *Writing clinical research protocols: Ethical considerations*. San Diego: Elsevier.

Turner, J. R. (2010). *New drug development: An introduction to clinical trials*. New York: Springer.

Privacy

- ▶ Confidentiality

Probability

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

In situations where certainty is not possible, it can be helpful to assess how likely it is that something will occur. Quantification of this likelihood is particularly helpful in statistical analysis. The concept of probability is used in everyday language, but more loosely than in statistics. The statement "I'll probably be there on Saturday" involves a probabilistic statement, but there is no precise degree of quantification. If you know the individual making this statement, past experience may lead you to an informed judgment concerning the relative meaning of probably, but this is still a subjective judgment, not a quantitative statement.

In statistics, a probability is a numerical quantity between zero (represented here as 0.00) and one (1.00) that expresses the likely occurrence of a future event. Past events cannot be associated with a probability of occurrence, since it is known in absolute terms whether they occurred or not. A probability of zero denotes that the event will not (cannot) occur. A probability of one denotes certainty that the event will occur. Any numerical value between zero and one expresses a relative likelihood of an event occurring. Additionally, the decimal expression of a probability value can be multiplied by 100 to create a percentage statement of likelihood. A probability of 0.50 would thus be expressed as a 50% chance that an event would occur. Similarly, and more relevantly to inferential hypothesis testing, probabilities of 0.05 and 0.01 would be expressed as a 5% chance and a 1% chance, respectively, that an event would occur.

Cross-References

- ▶ Hypothesis Testing
- ▶ Null Hypothesis
- ▶ Statistics

Problem Drinking

- ▶ Binge Drinking

Problem Solving

Seth A. Margolis[1], Patricia Osborne[1] and Jeffrey S. Gonzalez[1,2]
[1]Clinical Psychology, Health Emphasis, Ferkauf Graduate School of Psychology, Yeshiva University, Bronx, NY, USA
[2]Diabetes Research Center, Albert Einstein College of Medicine, Yeshiva University, Bronx, NY, USA

Synonyms

Problem-solving skills training (PSST); Problem-solving therapy – primary care (PST-PC); Problem-solving therapy – SO (PST-SO); Social problem-solving therapy (SPST)

Definition

Problem-solving therapy (PST) is a brief, empirically supported, cognitive-behavioral intervention aimed at training clients to identify, evaluate, and resolve everyday problems through the methodical application of problem-solving skills. In addition to teaching specific coping skills, PST emphasizes the importance of maintaining a positive *problem-solving orientation* and a rational *problem-solving style* (D'Zurilla & Nezu, 2010).

An individual's *problem-solving orientation* encompasses how one perceives problems, to what/whom they attribute these problems, how they appraise problematic situations, and the degree to which they view their problems as under their control. A major goal of PST is to help clients view problems as solvable challenges instead of insurmountable impasses.

A person's *problem-solving style* addresses the characteristic way in which he or she attempts to manage problems in living. PST advocates a rational approach. This form of problem solving is taught through the use of four essential skills: defining the problem in precise and objective terms (i.e., problem definition and formulation), brainstorming potential solutions (i.e., generation of alternatives), weighing the pros and cons of each alternative and creating a plan of action (i.e., decision making), and implementing the most appropriate solution and evaluating the outcome (i.e., implementation and verification). In combination, these skills are meant to help individuals manage their problems in an organized and systematic manner.

Problem solving serves a critical function in the successful management of chronic medical conditions. PST has, therefore, been adapted for individuals affected by cancer, obesity, diabetes, cardiovascular disease, traumatic brain injury, stroke, as well as older adults, caregivers, and to promote treatment adherence.

PST has been successfully delivered in individual, group, and family formats by psychologists, psychiatrists, social workers, nurses, and graduate-level trainees. Most forms of PST are manual based and all make use of homework assignments. PST therapists tend to balance directive and collaborative treatment styles.

PST interventions have been implemented in primary care settings (Catalan et al., 1991; Mynors-Wallis & Gath, 1997; Oxman, Hegel, Hull, & Dietrich, 2008; Areán et al., 2010), via telephone and in the home (Grant, Elliott, Weaver, Bartolucci, & Ginger, 2002), within community clinics and online (Wade, Wolfe, Brown, & Pestian, 2005; Wade, Walz, Carey, & William, 2008).

Even though PST has been provided as both a stand-alone treatment and as a component of multifaceted interventions, more research is

needed to adequately compare the two. Although some studies have addressed PST's applicability to minority groups (Sahler et al., 2005) and different age ranges (Areán et al., 2010), supplementary research is needed in these areas. Finally, given the evidence that PST can be a successful intervention for behavioral medicine settings, it would behoove future investigators to attempt to extend findings to other chronic health conditions.

Cross-References

- ▶ Cognitive Behavioral Therapy (CBT)
- ▶ Stroop Color-Word Test

References and Readings

Allen, S. M., Shah, A. C., Nezu, A. M., Ciambrone, D., Hogan, J., & Mor, V. (2002). A problem-solving approach to stress reduction among younger women with breast carcinoma: A randomized controlled trial. *Cancer, 94*(12), 3089–3100.

Arean, P. A., Perri, M. G., Nezu, A. M., Schein, R. L., Christopher, F., & Joseph, T. X. (1993). Comparative effectiveness of social problem-solving therapy and reminiscence therapy as treatments for depression in older adults. *Journal of Consulting and Clinical Psychology, 61*(6), 1003–1010.

Areán, P. A., Raue, P., Mackin, R. S., Kanellopoulos, D., McCulloch, C., & Alexopoulos, G. S. (2010). Problem-solving therapy and supportive therapy in older adults with major depression and executive dysfunction. *American Journal of Psychiatry, 167*, 1391–1398.

Catalan, J., Gath, D. H., Anastasiades, P., Bond, S. A., Day, A., & Hall, L. (1991). Evaluation of a brief psychological treatment for emotional disorders in primary care. *Psychological Medicine, 21*(4), 1012–1018.

Chang, E. C., D'Zurilla, T. J., & Sanna, L. J. (Eds.). (2004). *Social problem solving: Theory, research, and training*. Washington, DC: American Psychological Association.

D'Zurilla, T. J., & Nezu, A. M. (2010). Problem-solving therapy. In K. S. Dobson (Ed.), *Handbook of cognitive-behavioral therapies* (3rd ed., pp. 197–225). New York: Guilford Press.

Grant, J. S., Elliott, T. R., Weaver, M., Bartolucci, A. A., & Ginger, J. N. (2002). Telephone intervention with family caregivers of stroke survivors after rehabilitation. *Stroke, 33*(8), 2060–2065.

Hegel, M. T., Barrett, J. E., & Oxman, T. E. (2000). Training therapists in problem-solving treatment of depressive disorders in primary care: Lessons learned from the "treatment effectiveness project". *Families, Systems, & Health, 18*, 423–435.

Houts, P. S., Nezu, A. M., Nezu, C. M., & Bucher, J. A. (1996). The prepared family caregiver: A problem-solving approach to family caregiver education. *Patient Education and Counseling, 27*, 63–73.

Mynors-Wallis, L., & Gath, D. (1997). Predictors of treatment outcome for major depression in primary care. *Psychological Medicine, 27*(3), 731–736.

Nezu, A. M., Nezu, C. M., & D'Zurilla, T. J. (2010). Problem-solving therapy. In N. Kazantzis, M. S. Reinecke, & A. Freeman (Eds.), *Cognitive and behavioral theories in practice* (pp. 76–114). New York: Guilford Press.

Nezu, A. M., Nezu, C. M., Felgoise, S. H., McClure, K. S., & Hots, P. S. (2003). Project genesis: Assessing the efficacy of problem-solving therapy for distressed adult cancer patients. *Journal of Consulting and Clinical Psychology, 71*(6), 1036–1048.

Oxman, T. E., Hegel, M. T., Hull, J. G., & Dietrich, A. J. (2008). Problem-solving treatment and coping styles in primary care for minor depression. *Journal of Consulting and Clinical Psychology, 76*(6), 933–943.

Perri, M. G., Nezu, A. M., McKelvey, W. F., Shermer, R. L., Renjilian, D. A., & Viegener, B. J. (2001). Individual versus group therapy for obesity: Effects of matching participants to their treatment preferences. *Journal of Consulting and Clinical Psychology, 69*(4), 722–726.

Sahler, O. J., Fairclough, D. L., Phipps, S., Mulhern, R. K., Dolgin, M. J., Noll, R. B., et al. (2005). Using problem-solving skills training to reduce negative affectivity in mothers of children with newly diagnosed cancer: Report of a multisite randomized trial. *Journal of Consulting and Clinical Psychology, 73*(2), 272–283.

Wade, S. L., Walz, N. C., Carey, J. C., & William, K. M. (2008). Preliminary efficacy of a web-based family problem-solving treatment program for adolescents with traumatic brain injury. *Journal of Head Trauma Rehabilitation, 23*(6), 369–377.

Wade, S. L., Wolfe, C., Brown, T. M., & Pestian, J. P. (2005). Putting the pieces together: Preliminary efficacy of a web-based family intervention for children with traumatic brain injury. *Journal of Pediatric Psychology, 30*(5), 437–442.

Problem-Focused Coping

Linda Carroll
Department of Public Health Sciences, University of Alberta, Edmonton, AB, Canada

Synonyms

Active coping

Definition

Coping refers to the intentional efforts we engage in to minimize the physical, psychological, or social harm of an event or situation. There are many different frameworks for understanding coping and many different ways of classifying coping strategies, but one such classification is problem-focused coping vs. emotion-focused coping. Problem-focused coping is that kind of coping aimed at resolving the stressful situation or event or altering the source of the stress. Coping strategies that can be considered to be problem-focused include (but are not limited to) taking control of the stress (e.g., problem solving or removing the source of the stress), seeking information or assistance in handling the situation, and removing oneself from the stressful situation.

Problem-focused coping is distinguished from emotion-focused coping, which is aimed at managing the emotions associated with the situation, rather than changing the situation itself. For example, when anxious about an upcoming exam, use of problem-focused coping strategies might involve checking with the teacher about material one is unsure of, or increasing the time spent studying, or even deciding not to take the exam (although removing oneself from the stressor might have other negative consequences in this particular example). In contrast, emotion-focused coping strategies might involve self-talk to increase one's confidence in one's test taking ability or using relaxation techniques to decrease fear and anxiety. Problem-focused coping works best when the source of the stress is potentially under an individual's control; however, when the source of the stress is beyond the individual's control, such strategies are not usually helpful. Examples are dealing with bereavement. In situations like this, problem-focused coping is less likely to be helpful than emotion-focused coping, for example, processing one's feelings or releasing one's feelings. Problem- and emotion-focused coping are not mutually exclusive, and individuals frequently use both problem- and emotion-focused coping strategies to deal with stress. For example, when feeling threatened, initial use of emotion-focused coping to gain control over the fear can facilitate the subsequent use of problem-focused coping. Problem- and emotion-focused coping are the two subscales that comprise the Ways of Coping Checklist.

Cross-References

- ▶ Active Coping
- ▶ Coping

References and Readings

Field, T., McCabe, P. M., & Schneiderman, N. (1985). *Stress and coping*. Hillsdale, NJ: Erlbaum.

Lazarus, R. S. (1999). *Stress and emotion: A new synthesis*. New York: Springer.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal and coping*. New York: Springer.

Moos, R. H. (1986). *Coping with life crises: An integrated approach*. New York: Plenum Press.

Zeidner, M., & Endler, N. S. (1996). *Handbook of coping: Theory, research, applications*. New York: Wiley.

Problem-Solving Skills Training (PSST)

- ▶ Problem Solving

Problem-Solving Therapy – Primary Care (PST-PC)

- ▶ Problem Solving

Problem-Solving Therapy – SO (PST-SO)

- ▶ Problem Solving

Productivity

► Job Performance

Progress

► Aging

Promotoras

Amelie Ramirez[1] and Barbara Turner[2]
[1]Department of Epidemiology & Biostatistics, The University of Texas Health Science Center at San Antonio, San Antonio, TX, USA
[2]The University of Texas Health Science Center at San Antonio, San Antonio, TX, USA

Synonyms

Community health advisors; Community health representatives; Community health workers (CHW); Health navigators; Lay health advisors; Lay health advocates; Outreach educators; Peer coaches; Peer health educators; Peer health promoters

Definition

Promotoras (or *promotoras de salud*) are female community health workers who provide a variety of services in their role as liaison between underserved Hispanics and traditional health care services. Their male counterparts are *promotores*, although it is less common to have men serve in this role. These terms translate to "promoters" of health, in this case among Hispanics who have historically experienced challenges accessing health care services and deficiencies in outcomes of care. These individuals are either volunteers or employees of the local health care system or other entities administering community interventions. They typically live in the community or neighborhood that they serve and thus can better communicate and relate to their clients because of their shared community experiences.

Description

Essentially, the *promotora* concept is a form of community-based peer support: nonprofessionals helping others with various health needs. These needs include: culturally appropriate education, informal advice and counsel, social support and encouragement, interpretation and translation, advocacy for health needs, disease management and prevention, health care system navigation, and community resource guidance. Depending upon the community health priorities and the specific goals and objectives of particular programs, *promotoras* may provide one or a combination of these services.

Historically, *promotoras* are part of a long tradition of lay health workers who serve critical health care and disease prevention roles in cultures around the world. For example, in eighteenth century Russia, *feldshers* began providing medical assistance in urban hospitals and the army. In China, during the 1960s, farmworkers were trained as "barefoot doctors" to give first aid, immunizations, and health education in rural areas. In Haiti, village health workers known as *accompagnateurs* attended individuals suffering from tuberculosis and HIV/AIDS. In Africa, health care workers provide the main form of health care delivery and especially have served as the linchpin for HIV prevention and treatment. Origins of the *promotoras* can also be found in Latin American countries, where laymen and laywomen were trained by church groups and other organizations to give community health assistance.

Efforts to establish formal community health worker programs in this country can be traced back over a half-century. Such outreach was subsequently included in the federal Migrant Health Act of 1962 and the Economic Opportunity Act of 1964, mandating utilization of

community health aides in many neighborhoods and migrant worker camps (Hill, Bone, & Butz, 1996). The Affordable Care Act of 2010 provided a more recent endorsement of the concept, specifying community health workers as an integral component of the nation's health care workforce (Affordable Care Act of 2010). With the rise in health care costs in recent times and increased awareness of the scope and cost of health disparities in this country, the peer-support approach has gained increasing support as a viable and potentially cost-effective means of helping fill gaps in the health care system. However, the evidence so far is conflicting and comes from less than optimally designed studies.

In the USA today, the nomenclature for peer health promotion personnel is diverse, including: community health workers (CHW), community health advisors, lay health advisors, lay health advocates, peer coaches, outreach educators, health navigators, peer health promoters, peer health educators, community health representatives, and, in Hispanic communities, *promotoras*. Often, some of these terms, including CHW and *promotoras*, are used interchangeably or in combination.

Surveys have found that these community health workers operate in all 50 states (U.S. Department of Health and Human Services Health Resources and Services Administration, 2007). Programs utilizing *promotoras* have flourished especially in states with large Hispanic populations such as California and along the USA-Mexico border. In 2009, Hispanics represented 16% of the US population (U.S. Census Bureau News, 2010) and this proportion is expected to rise dramatically in the coming decades. With the growing number of Hispanics, the peer-assistance concept has been incorporated into health programs and interventions across the country. This model has been evaluated in large cities, mid-size and smaller communities, as well as agricultural areas (working with farmworkers and their families), with favorable results in many studies.

Promotora involvement has been found to be useful in a broad range of needs, such as management of diabetes and other health conditions prevalent among Hispanics, screening for cancer and other diseases, and access to health care overall. They also can focus on addressing specific needs such as prenatal care, or healthy lifestyle behaviors in general (e.g., proper diet and exercise). In addition to working with diabetes and cancer patients, survivors, and at-risk individuals, *promotoras*/CHW have been involved in interventions targeting cardiovascular disease, HIV/AIDS, high blood pressure, asthma, mental illness, and other diseases.

Firsthand knowledge of the local Hispanic community and the personal and institutional barriers that residents of the community face in attaining adequate health care uniquely prepares *promotoras* for their liaison role. As Spanish speakers and residents of the neighborhoods they serve, *promotoras* offer assistance to reduce or remove linguistic and cultural barriers for segments of the population that have historically been difficult to reach for local health care agencies and services. At the same time, *promotoras* can provide assistance to professional health care personnel by educating providers and their staff about sociocultural factors that influence the health knowledge, beliefs, and attitudes, as well as the values and behaviors, of their Hispanic patients.

Among the strengths of the *promotora*/CHW models employed in various community health programs is the broad diversity of services peer health workers have been able to deliver. For example, as liaisons between health providers and underserved Hispanics, *promotoras* sometimes serve as interpreters as well as health system navigators, assisting in identification of benefits that clients are eligible to receive, and helping them complete necessary applications and forms. As case managers, *promotoras* typically facilitate contacts of health care providers with community members by maintaining accurate contact information and a record of interactions. As community organizers, *promotoras* have been called upon to motivate and encourage members of the community to participate actively in efforts to improve neighborhood living conditions. And as health educators, *promotoras* distribute and explain print and

other informative materials aimed at promoting screening, preventing disease, discouraging smoking, managing chronic diseases, and other health promotion purposes. In addition, interventions have utilized *promotoras* in various other roles, including as group presentation leaders; role models; and guides to community social, transportation, childcare, and other services and resources.

Promotoras are typically respected and trusted members of the community they serve. Due to their familiarity with their community and neighborhood, *promotora*s can move freely within the community and engage with various sectors. They provide their services in convenient locations, including homes, schools, churches, clinics, hospitals, community centers, job sites, and other locales. In many programs, they also participate in community events, such as health fairs.

In the past, *promotoras* and other community health workers have typically been trained on the job. With the increased utilization of peer-support models over the years, concerns about the quality of this training and the need for more standard certification of these workers have been increasingly raised. However, as of 2010, only a handful of states had taken steps to address these issues. In 1999, Texas was the first state to legislate voluntary training and certification for *promotoras* and community health workers (Nichols, Berrios, & Samar, 2005). In Minnesota, select community colleges offer a standardized curriculum for community health worker certification, with completion required for a worker to be eligible as a Medicaid provider (Rosenthal et al., 2010). In numerous states, certificates are being awarded, typically by community colleges and other programs, for completion of a course of community health worker studies. Proponents of required certification or credentialing note potential benefits, including offering assurance that workers possess basic competencies and promoting perceived legitimacy within health/human services fields. Others, however, contend that requiring certification may have a detrimental effect on the number of peer health workers from poor, minority neighborhoods and communities that most need to be served (Family Strengthening Policy Center, 2006).

From the substantial experience with *promotora*/CHW program implementation and research, the evidence is growing that peer support offers unique benefits to improve health care delivery and outcomes. This is particularly relevant, given rapidly expanding lower-socio economic status (SES) racial/ethnic segments of the population (particularly Hispanics), growing numbers of patients with chronic diseases, and spiraling health care costs. Systematic reviews of the benefit of peer support currently find that the evidence lacks sufficient rigor. Consequently, much more research needs to be conducted to define roles and scope of activities, determine effectiveness, develop consistent and adequate training, provide fair compensation, and, via certification and/or similar means, define a universally accepted and respected niche for *promotoras* among the nation's health care workforce.

Cross-References

- Health Disparities
- Health Education
- Health Literacy

References and Readings

Balcazar, H. G., Byrd, T. L., Ortiz, M., Tondapu, S. R., & Chavez, M. (2009). A randomized community intervention to improve hypertension control among Mexican Americans: Using the *promotoras de salud* community outreach model. *Journal of Health Care for the Poor and Underserved, 20*(4), 1079–1094.

Boothroyd, R. I., & Fisher, E. B. (2010). Peers for progress: Promoting peer support for health around the world. *Family Practice, 27*(Suppl. 1), i62–i68.

Centers for Disease Control and Prevention (2011). Community health workers/*Promotores de Salud*: Critical connections in communities. Accessed online from http://www.cdc.gov/diabetes/projects/comm.htm

Doull, M., O'Connor, A. M., Welch, V., Tugwell, P., Wells, G. A. (2008). Peer support strategies for improving the health and well-being of individuals with chronic diseases (Protocol). Copyright © 2008 The Cochrane Collaboration. Published by JohnWiley & Sons, Ltd.

Family Strengthening Policy Center. (2006). *Community health workers: Closing gaps in families' health resources* (Policy Brief No. 14). Accessed online from http://www.nydic.org/fspc/practice/documents/Brief14.pdf

Hill, M. N., Bone, L. R., & Butz, A. M. (1996). Enhancing the role of community health workers in research. *Image, 28*, 221–226.

Kumar, P. (2007). Providing the providers – Remedying Africa's shortage of health care workers. *The New England Journal of Medicine, 356*, 2564–2567.

Nichols, D. C., Berrios, C., & Samar, H. (2005). Texas' community health workforce: from state health promotion policy to community-level practice. *Preventing Chronic Disease, 2*(Special Issue), A13. Published online 2005 October 15.

O'Brien, M. J., Halbert, C. H., Bixby, R., Pimentel, S., & Shea, J. A. (2010). Community health worker intervention to decrease cervical cancer disparities in Hispanic women. *Journal of General Internal Medicine, 25*(11), 1186–1192. Epub 2010 July 7.

Patient Protection and Affordable Care Act, PL111-148, § 5101, 5102, 5313, 5403, and 3509 (2010).

Rosenthal, E. L., Brownstein, J. N., Rush, C. H., Hirsch, G. R., Willaert, A. M., Scott, J. R., et al. (2010). Community health workers: Part of the solution. *Health Affairs, 29*(7), 1338–1342.

U.S. Census Bureau News, U.S. Department of Commerce. (2010, July 15). Facts for Features CB10-FF.17. Accessed online from http://www.census.gov/newsroom/releases/pdf/cb10ff-17_hispanic.pdf

U.S. Department of Health and Human Services Health Resources and Services Administration. (2007, March). Community health workers national workforce study. Accessed online from http://bhpr.hrsa.gov/healthworkforce/chw/default.htm#preface

Vargas, R. B., & Cunningham, W. E. (2006). Evolving trends in medical care-coordination for patients with HIV and AIDS. *Current HIV/AIDS Reports, 3*(4), 149–153.

Viswanathan, M., Kraschnewski, J., Nishikawa, B., Morgan, L. C., Thieda, P., Honeycutt, A., et al. (2009). *Outcomes of community health worker interventions* (Evidence Report/Technology Assessment No. 181, Prepared by the RTI International-University of North Carolina Evidence-based Practice Center under Contract No. 290 2007 10056 I, AHRQ Publication No. 09-E014). Rockville, MD: Agency for Healthcare Research and Quality.

Prophylactic Use

► Condom Use

Prospective Cohort Study

► Cohort Study

Prospective Study

► Follow-up Study

Prostate

Marc A. Kowalkouski[1], Heather Honoré Goltz[1,2], Stacey L. Hart[3] and David Latini[4]
[1]HSR&D Center of Excellence, Michael E. DeBakey VA Medical Center (MEDVAMC 152), Houston, TX, USA
[2]Department of Social Sciences, University of Houston-Downtown, Houston, TX, USA
[3]Department of Psychology, Ryerson University, Toronto, ON, Canada
[4]Scott Department of Urology, Baylor College of Medicine, Houston, TX, USA

Synonyms

Prostate gland

Definition

Prostate Anatomy

The prostate is a walnut-sized gland that functions in the male reproductive system. It is positioned in front of the rectum and directly below the bladder, which stores urine. The prostate also encircles the proximal urethra, the canal which carries urine from the bladder and through the penis.

Three distinct zones of glandular tissue make up the prostate: the peripheral zone, central zone, and transition zone. Additionally, there is an area of fibromuscular tissue on the anterior surface. Each anatomic zone is uniquely affected by different disease processes. The majority of

prostate cancers develop in the peripheral zone, the largest zone by volume, while benign prostatic hyperplasia originates in the transition zone.

Description

Prostate Function

The prostate produces the thick, milky-white alkaline fluid that forms part of semen. The fluid provides nourishment to sperm and, along with fluid from the bulbourethral (Cowper's) glands, helps to neutralize the acidity of the urine residue in the male urethra and of the female vaginal canal, increasing the life span of sperm. During ejaculation, contraction of smooth muscles moves the fluid from the prostate into the urethral tract where it mixes with the sperm produced by the testicles and additional fluid from the bulbourethral glands and seminal vesicles. The resulting mixture, semen, passes from the urethra and out through the penis.

Common Disorders of the Prostate

There are three common conditions of the prostate: prostatitis, benign prostatic hyperplasia, and prostate cancer. Generally, older men are more susceptible to prostate disease. However, prostatitis can affect men at any age. Prostatitis is a benign infection of prostatic tissue, usually caused by bacteria. Inflammation can result in urine retention in the bladder, resulting in bladder distention (i.e., enlargement) and exposing the bladder to additional risk for infection. Additionally, prostatitis can trigger several urinary problems (e.g., pain or burning upon urination, urgency, and trouble voiding). Benign prostatic hyperplasia, or BPH, is another common prostatic condition caused by the noncancerous enlargement of the prostate gland in aging men. As the prostate enlarges, it compresses the urethra and irritates the bladder. Obstruction of the urethra, as well as gradually diminishing bladder function, results in the symptoms of BPH including dribbling after urination or a need to urinate often, especially at night. Some men also experience urinary incontinence, the involuntary discharge of urine. BPH symptoms can severely affect a man's, as well as his partner's, quality of life and can be further compounded by psychological factors (e.g., depression and anxiety) associated with BPH symptoms. Prostate cancer is the most common malignancy and the second most common cause of cancer death among men in the United States. Response to treatment is best when the disease is caught early. However, prostate cancer is generally asymptomatic when the disease is localized to the prostate. Screening for prostate cancer includes serum prostate-specific antigen (PSA) testing and digital rectal examination. There are many treatment options available to men diagnosed with prostate cancer (e.g., active surveillance, radiotherapy, and surgery). However, there is currently no consensus regarding the optimal treatment.

Cross-References

▶ Prostate-Specific Antigen (PSA)

References and Readings

Gacci, M., Bartoletti, R., Figlioli, S., Sarti, E., Eisner, B., Boddi, V., et al. (2003). Urinary symptoms, quality of life and sexual function in patients with benign prostatic hypertrophy before and after prostatectomy: A prospective study. *British Journal of Urology International, 91*, 196.

Mitropoulos, D., Anastasiou, I., Giannopoulou, C., Nikolopoulos, P., Alamanis, Z., & Dimopoulos, C. (2002). Symptomatic benign prostatic hyperplasia: Impact on partners' quality of life. *European Urology, 41*, 240–245.

Ramakrishnan, K., & Salinas, R. C. (2010). Prostatitis: Acute and chronic. *Primary Care, 37*, 547–563, vii–ix.

Tanagho, E. A., & McAninch, J. W. (Eds.). (2008). *Smith's general urology* (17th ed.). New York: McGraw-Hill.

Wein, A. J., Coyne, K. S., Tubaro, A., Sexton, C. C., Kopp, Z. S., & Aiyer, L. P. (2009). The impact of lower urinary tract symptoms on male sexual health: EpiLUTS. *British Journal of Urology International, 103*(Suppl. 3), 33–41.

Prostate Gland

▶ Prostate

Prostatectomy

► Radical Prostatectomy, Psychological Impact

Prostate-Specific Antigen (PSA)

Marc A. Kowalkouski[1], Heather Honoré Goltz[1,2], Stacey L. Hart[3] and David Latini[4]
[1]HSR&D Center of Excellence, Michael E. DeBakey VA Medical Center (MEDVAMC 152), Houston, TX, USA
[2]Department of Social Sciences, University of Houston-Downtown, Houston, TX, USA
[3]Department of Psychology, Ryerson University, Toronto, ON, Canada
[4]Scott Department of Urology, Baylor College of Medicine, Houston, TX, USA

Synonyms

Kallikrein-3; P-30 antigen; Semenogelase; Seminin

Definition

Prostate-specific antigen (PSA) is a protein produced by cells of the prostate gland. PSA functions in male fertility, and most of it gets expelled from the body in semen. However, low levels circulate in the blood. Elevated serum PSA levels often indicate the presence of prostate cancer and other prostate disorders.

Description

Measuring PSA

The PSA screening test measures the total level of PSA circulating in the blood serum. PSA levels above 4 ng per milliliter are considered above normal. However, total PSA does not offer a definitive diagnosis for cancer or other prostate disease. Therefore, additional measures of PSA have been integrated to depict a more comprehensive profile of prostate disease characteristics. For example, PSA velocity measures the rate of increase in PSA over time. Generally, larger PSA velocity is associated with prostate cancer. Free PSA is the percentage of circulating PSA that is not bound to other proteins. On average, men with a low percentage of free PSA are more likely to have cancer. PSA density is the PSA level divided by prostate volume. The likelihood of cancer is increased when PSA density is high.

PSA Testing and Current Guidelines

PSA testing has become increasingly popular because it allows detection of prostate cancer at early stages, before palpable detection during digital rectal examination. However, PSA testing also presents a high rate of false-positive and false-negative results, which has generated substantial controversy with regard to whether or not PSA screening is effective at reducing cancer deaths. Two large, randomized clinical trials are currently evaluating the efficacy of PSA screening. Preliminary findings indicate modest reductions in cancer deaths in the screening group but also note over-detection and potential overtreatment of clinically insignificant cancers in the same group. Accounting for these complex issues, the American Cancer Society (ACS) has revised its recommendations regarding routine PSA screening. The revised guidelines now recommend that doctors initiate comprehensive discussions with patients about their options for screening and that men use decision-making tools to make an informed choice about testing. The ACS suggests these guidelines are appropriate for patients over age 50 who are in good health with greater than 10 years of life expectancy. For men at higher risk (e.g., African American men and men with a positive family history), ACS guidelines suggest that discussions about screening begin earlier. The ACS does not endorse screening for men with several comorbid conditions or for men with less than 10 years of life expectancy. In response to the preliminary findings from the previously mentioned studies, the American Urological Association (AUA) also revised its screening recommendations, which

suggest extending screening to younger men. The AUA advises that PSA screening should be offered to well-informed men aged 40 years or older who have a life expectancy of at least 10 years. Additionally, AUA guidelines emphasize a shift away from using a single PSA threshold to determine whether to proceed to additional diagnostics. Instead, the combination of many factors (e.g., PSA profile over time, family history, race, age) should be considered.

References and Readings

American Cancer Society. (2010). *Cancer facts and figures 2010*. Atlanta, GA: Author.

American Urological Association. (2009). *Prostate-specific antigen best practice statement: 2009 Update*. Washington, DC: American Urological Association Education and Research.

Andriole, G. L., Grubb, R. L., Buys, S. S., Crawford, E. D., Chia, D., Church, T. R., et al. (2009). Mortality results from a randomized prostate-cancer screening trial. *The New England Journal of Medicine, 360*, 1310–1319.

Carroll, P. R., Whitson, J. M., & Cooperberg, M. R. (2010). Serum prostate-specific antigen for the early detection of prostate cancer: Always, never, or only sometimes? *Journal of Clinical Oncology, 28*, 1–3.

Jacobsen, S. J., Katusic, S. K., Bergstralh, E. J., Oesterling, J. E., Ohrt, D., Klee, G. G., et al. (1995). Incidence of prostate cancer diagnosis in the eras before and after serum prostate-specific antigen testing. *Journal of the American Medical Association, 274*, 1445–1449.

Schroder, F. H., Hugosson, J., Roobol, M. J., Tammela, T. L., Ciatto, S., Nelen, V., et al. (2009). Screening and prostate-cancer mortality in a randomized European study. *The New England Journal of Medicine, 360*, 1320–1328.

Tanagho, E. A., & McAninch, J. W. (Eds.). (2008). *Smith's general urology* (17th ed.). New York: McGraw-Hill.

Vickers, A. J., Savage, C., O'Brien, M. F., & Lilja, H. (2008). Systematic review of pretreatment prostate-specific antigen velocity and doubling time as predictors for prostate cancer. *Journal of Clinical Oncology, 27*, 398–403.

Prostatic Adenocarcinoma

► Cancer, Prostate

Protected Sex

► Condom Use

Protection of Human Subjects

Marianne Shaughnessy
School of Nursing, University of Maryland, Baltimore, MD, USA

Synonyms

Human subjects protections

Definition

The organized oversight of research to ensure the rights and well-being of participants.

Description

The protection of human subjects in clinical research evolved in response to conditions surrounding medical research that were deemed unacceptable in the early to mid-twentieth century. In December 1946, an American military tribunal opened criminal proceedings against 23 German physicians who conducted medical experiments on thousands of concentration camp prisoners without their consent. Most of the subjects of these experiments died or were permanently disabled as a result. The Nuremberg Code of 1948 resulted, stating that "voluntary consent of a human subject is absolutely essential" and that "the benefits of research must outweigh the risks" (NIH). In 1964, the World Medical Association published recommendations for research involving human subjects, and the Declaration of Helsinki governs international research ethics and defines rules for "research combined with clinical care" and

"non-therapeutic research." This declaration was revised in 1975, 1983, 1989, and 1996 and remains the basis for Good Clinical practices used today (WMA).

In the United States, the Tuskeegee Syphilis Study (1932–1972) described the natural history of syphilis in 600 low-income African American males, 400 of whom were infected. Over 40 years, researchers provided medical examinations, but infected subjects were never told of their disease nor offered treatment, even after a known cure became available in 1947. Many of the subjects died from syphilis-related causes during the study. The study was stopped in 1973 by the U.S. Department of Health, Education and Welfare after its existence was publicized (CDC). The publicity generated by the Nuremberg trials, the Tuskeegee study, and other studies created a mistrust of medical researchers and a demand for a standards and guidelines for the ethical conduct of research. On July 12, 1974, the National Research Act (Pub. L. 93–348) was signed into law, creating the National Commission for the Protection of Human Subjects of Biomedical and Behavioral Research. This commission generated the Belmont Report in 1976, a summary of the ethical principles identified by the Commission that should be used to guide human subjects research. These principles include (1) respect for persons, (2) beneficence, and (3) justice. The application of these general principles is further described in specific guidance for obtaining informed consent, complete assessment of risks, and benefits and selection of research subjects.

Medical research is conducted in a variety of academic and corporate settings, but all human subjects research studies should be evaluated by an Institutional Review Board or similar research oversight committee prior to initiation. This is a requirement for all federally funded research in the United States. These oversight boards are responsible for the review of each proposed study to ensure that the principles of the Belmont Report are applied and that the rights of potential and actual participants are respected. The Association for the Accreditation of Human Research Protection Programs (AAHRPP) reviews the processes established by these research oversight bodies and grants accreditation for such programs that meet general requirements and demonstrate processes geared toward continual improvement of research and the protection of human subjects.

Cross-References

▶ Research Participation, Risks and Benefits of

References and Readings

Association for the Accreditation of Human Research Protection Programs. Retrieved May 18, 2011 from http://www.aahrpp.org

Center for Information and Study on Clinical Research Participation (CISCRP). Retrieved May 18, 2011 from http://www.ciscrp.org/

Centers for Disease Control and Prevention (CDC). Retrieved May 18, 2011 from http://www.cdc.gov/tuskegee/timeline.htm

NIH Office of Human Subjects Research. Retrieved May 18, 2011 from http://ohsr.od.nih.gov/guidelines/nuremberg.html

The Belmont report. Retrieved May 18, 2011 from http://ohsr.od.nih.gov/guidelines/belmont.html

The Hastings Center. Retrieved May 18, 2011 from http://www.thehastingscenter.org

World Medical Association (WMA). Retrieved May 18, 2011 from http://www.wma.net/en/30publications/10policies/b3/

Protective Factors

▶ Cardiovascular Risk Factors

Protein Methylation

▶ Methylation

Proteomics

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

Proteomics is the study of all of a genome's putative proteins and involves the systematic analysis of proteins to determine their identity, quantity, and function (Soloviev, Barry, & Terrett, 2004).

Description

The Human Genome Project and related work have focused considerable attention on sequencing aspects of genomic research. However, as Holmes, Ramkissoon, & Giddings (2005) noted, "The eventual goal of these projects is actually to determine how the genome builds life through proteins. DNA has been the focus of attention because the tools for studying it are more advanced and because it is at the heart of the cell, carrying all the information – the blueprint – for life. However, a blueprint without a builder is not very useful, and the proteins are the primary builders within the cell."

It is of interest to characterize the complement of expressed proteins from a single genome. Monitoring the expression and properties of a large number of proteins provides important information about the physiological state of a cell and, by extension, an organism. Cells can express very large numbers of different proteins, and the "expression profile" (the number of proteins expressed and the expression level of each of them) can vary in different cell types. Given that each cell contains all genomic material and hence information, this differential expression of proteins explains why cells perform different functions (Soloviev et al., 2004).

The 20,000–25,000 genes in the human genome actually generate many more than the commensurate number of proteins, with estimates as high as one million appearing in the literature (Augen, 2005). This huge number results from the observation that multiple, distinct proteins can result from a single gene. Consider the following steps in the journey from the genome to the proteome (Holmes et al., 2005):

- DNA replication results in many gene forms.
- Ribonucleic acid (RNA) transcription leads to pre-messenger RNA.
- RNA maturation results in mature messenger RNA.
- Protein translation results in an immature protein.
- Protein maturation results in a mature protein in the proteome (posttranslational modifications are possible here).

The tremendous diversity of proteins in the proteome is facilitated by multiple possible means of protein expression. At each stage of the multistep process just described, alternate mechanisms produce variants of the "standard" protein, resulting in a proteome that is far greater than the genome that generates it. Posttranslational modifications play a considerable part in this creation of diversity. These modifications include, for example, the process of glycosylation in which proteins are glycosylated and hence become glycoproteins, which act as receptors and enzymes.

Nobel Laureate James Watson commented as follows with regard to the fields of proteomics and transcriptomics (Watson, 2004): "In the wake of the Human Genome Project, two new postgenomic fields have duly emerged, both of them burdened with unimaginative names incorporating the '-omic' of their ancestor: proteomics and transcriptomics. Proteomics is the study of the proteins encoded by genes. Transcriptomics is devoted to determining where and when genes are expressed – that is, which genes are transcriptionally active in a given cell."

Cross-References

▶ DNA
▶ Genomics
▶ Human Genome Project

References and Readings

Augen, J. (2005). *Bioinformatics in the post-genomic era: Genome, transcriptome, proteome, and information-based medicine*. Boston: Addison-Wesley.

Holmes, M. R., Ramkissoon, K. R., & Giddings, M. C. (2005). Proteomics and protein identification. In A. D. Baxevanis & B. F. F. Ouellette (Eds.), *Bioinformatics: A practical guide to the analysis of genes and proteins* (3rd ed.). Hoboken, NJ: Wiley-Interscience.

Soloviev, M., Barry, R., & Terrett, J. (2004). Chip-based proteomics technology. In R. Rapley & S. Harbron (Eds.), *Molecular analysis and genome discovery*. Hoboken, NJ: Wiley.

Watson, J. D. (2004). *DNA: The secret of life*. New York: Alfred A Knopf.

Proxy

► Surrogate Decision Making

Prozac®

► Selective Serotonin Reuptake Inhibitors (SSRIs)

Psychiatric Diagnosis

Toru Okuyama
Division of Psycho-oncology and Palliative Care, Nagoya City University Hospital, Nagoya, Aichi, Japan

Synonyms

Diagnostic criteria; Psychiatric disorder

Definition

Psychiatric diagnosis defines a psychiatric disorder which causes subjective distress and disability and can be conceptualized based on symptomatology, epidemiology, and pathophysiology. The primary purpose of the psychiatric diagnosis is to distinguish a certain condition from non-disease conditions or other disease conditions and to make health providers communicate better with each other by using shared concepts and languages. Psychiatric diagnosis facilitates researches by maintaining the internal validity of each psychiatric disease and is important for examining the external validity of findings when applying research evidences into individual patients.

Description

The top two most important and frequently used psychiatric diagnoses classification systems are the International Classification of Disease (ICD) by World Health Organization (WHO) and the Diagnostic and Statistical Manual of Mental Disorders (DSM) edited by the American Psychiatric Association.

The ICD is the international standard diagnostic classification of physical and psychiatric diseases and other health conditions, defined by WHO. It has been developed for the purpose of international use since the beginning, and WHO Member States compile national mortality and morbidity statistics based on this diagnostic system. The tenth revision was published in 1992. The classification of mental and behavioral disorders is included in Chap. F. ICD-10 consists of Clinical Descriptions and Diagnostic Guidelines and Diagnostic Criteria for Research. The former provides clinical descriptions detailing the principal signs and symptoms of each disorder. The latter is intended to help those researching specific disorders to maximize the homogeneity of study groups.

The latest version of DSM is the fourth edition text revision published in 2000 (DSM-IV-TR), in which several important features are included. First, it adopts the descriptive approach: the diagnoses criteria are defined based on the symptomatology, rather than the underlying causes. Secondly, clearly defined diagnostic criteria are provided for each specific disorder. These criteria include lists of features that must be present for diagnoses to be made. Thirdly, DSM-IV-TR is

a multidimensional evaluation system (Axis I: clinical disorders and other conditions that may be a focus of clinical attention, Axis II: personality disorders and mental retardation, Axis III: any physical disorder or general medical condition, Axis IV: the psychosocial and environmental problems that contribute significantly to the development or exacerbation of the current disorder, Axis V: a global assessment of functioning). The codes and terms used in the DSM-IV are designed to correspond with the codes used in the ICD-10.

It is also important how to apply these diagnostic criteria into each actual patient, in order to maximize the reliability of psychiatric diagnosis. Structured interviews were developed for this purpose. In structured interviews, the procedures for interviews including how to ask about the presence or absence of certain symptom and how to categorize patients' responses are strictly defined so that high inter-rater reliability of the diagnosis can be achieved. Since currently there are no gold standard objective diagnostic tests for psychiatric diagnosis, structured interviews are considered to be gold standard.

A clinical diagnosis includes the whole process of diagnostic formulation in addition to a psychiatric diagnosis based on the diagnostic criteria. A diagnostic formulation is an attempt to understand each patient comprehensively and individually by describing what has been influencing the feeling and behavior of the patient and what relationship might exist between the patient's life situation/background and the psychiatric illness.

Cross-References

▶ Diagnostic Interview Schedule

References and Readings

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders (Text revision)* (4th ed.). Washington, DC: Author.

World Health Organization. (1992). *ICD-10: The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines*. Geneva: Author.

Zimmerman, M., & Spitzer, R. L. (2009). Classification in psychiatry. In B. J. Sadock, V. A. Sadock, & P. Ruiz (Eds.), *Comprehensive textbook of psychiatry* (pp. 1003–1052). Philadelphia: Lippincott Williams & Wilkins.

Psychiatric Disorder

▶ Psychiatric Diagnosis
▶ Psychological Disorder
▶ Psychiatric Illness

Psychiatric Illness

Maxine Holmqvist
Clinical Health Psychology, University of Manitoba, Winnipeg, MB, Canada

Synonyms

Mental illness; Psychiatric disorder; Psychological disorder

Definition

A condition or syndrome with distinctive cognitive, affective, and/or behavioral symptoms, arising from underlying psychobiological dysfunction and causing significant distress, impairment, or an increased risk of death, pain, disability, or an important loss of freedom (American Psychiatric Association [APA], 2000).

Description

Psychiatry is a branch of medicine that focuses on the diagnosis and treatment of mental illness. It did not emerge as an independent discipline until the late 1800s; however, the roots of psychiatric assessment and treatment extend back to the ancient

Greeks and Egyptians (for a comprehensive overview of the history of psychiatry, see Wallace & Gach, 2008). Psychiatric illnesses are currently diagnosed with reference to either the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV; APA, 2000) or the International Classification of Disease, 10th revision (ICD-10; for more information on these systems, see *Psychological Disorders*, this volume). Psychiatric diagnoses are descriptive, and with few exceptions, they convey very little about etiology. McHugh and Slavney (1998) identify different perspectives that psychiatrists use to conceptualize cases and plan interventions. Disease reasoning presupposes that a patients' condition is the result of changes or defects in brain functioning. In contrast to this, behavioral reasoning presupposes that a patient's condition is the result of dysfunctional behavior. These two perspectives suggest different interventions; where the presumed cause is pathology of the brain, it follows that the appropriate treatment would likely involve medical intervention (e.g., pharmaceutical drugs, surgery, transcranial magnetic stimulation), while a behavioral disorder would respond best to behavioral treatment (e.g., attention to antecedents and consequences, behavior modification, exposure therapy). The use of the word "illness" in the term "psychiatric illness" implies that disease reasoning is being used; an illness is something a patient *has* rather than something that they are *doing* (McHugh & Slavney, 1998). Therefore, this term may be more commonly used to refer to psychoses and disorders with known biological pathophysiology (e.g., Huntington's disease) and less commonly used to refer to conditions where symptoms are primarily behavioral or appear more voluntary (e.g., gambling, anorexia nervosa).

Regardless of their presumed origin, psychiatric illnesses such as depression and post-traumatic stress disorder are risk factors for the onset of complex medical conditions like coronary heart disease. Furthermore, psychiatric illnesses may develop as a consequence of disease processes, and can be significant independent predictors of poor prognosis in several conditions, including heart disease and end-stage renal disease.

Cross-References

- ▶ Psychiatric Diagnosis
- ▶ Psychiatric Disorder
- ▶ Psychological Disorder

References and Readings

American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders, Fourth Edition, Text Revision*. Washington, DC: American Psychiatric Association. See also http://www.psych.org/mainmenu/research/dsmiv.aspx

Lippi, G., Montagnana, M., Favaloro, E. J., & Franchini, M. (2009). Mental depression and cardiovascular disease: A multifaceted, bidirectional association. *Seminars in Thrombosis and Hemostasis, 35*, 325–336.

McHugh, P. R., & Slavney, P. R. (1998). *The perspectives of psychiatry*. Baltimore: Johns Hopkins University Press.

Wallace, E. R., & Gach, J. (Eds.). (2008). *History of psychiatry and medical psychology: With an epilogue on psychiatry and the mind-body relation*. New York: Springer.

World Health Organization. (2007). *International Statistical Classification of Diseases and Related Health Problems, 10th Revision, Version for 2007*. Retrieved January 1, 2011 from http://apps.who.int/classifications/apps/icd/icd10online/

Psychiatric Surgery

▶ Psychosurgery

Psychoeducation

Shin-ichi Suzuki[1] and Asuka Tanoue[2]
[1]Faculty of Human Sciences, Graduate School of Human sciences, Waseda University, Tokorozawa-shi, Saitama, Japan
[2]Advanced Research Center for Human Science, Waseda University, Tokorozawa, Saitama, Japan

Definition

Psychoeducation is a method of providing patients/clients and their families a theoretical and practical

approach to understanding and coping with the consequences of their psychological disorders/problems or physical illnesses/responses.

The main goals of psychoeducation are the enhancement of adherence; improvement of illness management or stress control skills, such as early recognition of episode recurrence and development of strategies for effective coping with symptoms; improvement of social and occupational functions; and quality of life.

The role of psychoeducation encompasses not only imparting knowledge and information regarding treatment/psychological support through media such as leaflets or information web sites or feedback to individuals based on test results, but it is also characterized by active cooperation such as intervention exercises with patients and their families.

Psychoeducation may be conducted in a group including individuals with similar problems (e.g., chronic diseases such as diabetes, HIV/AIDS, and PTSD) and individual therapy sessions. Group therapy is also expected to foster support among patients/clients. Thus, the psychoeducational approach has a lot in common with general psychosocial support for schizophrenia, mood disorders, eating disorders, and drug addiction, but it can also be applied to further the field of education.

Particularly, in the context of a medical setting, the therapists will try to explain the normal reactions of symptoms related to each psychological disorder or physical illness at the beginning. This will prevent any misinformation from being circulated among the patients and expand their understanding of the intervention, and it will lead to an improvement in the interactions of patients/clients in the treatment. Evidence from systematic reviews has reinforced the effect of these treatments stating that psychoeducational interventions for unipolar depression are effective, can prevent the worsening of depression, and be used as a preventive instrument (e.g., Cuijpers, 1997).

As mentioned above, psychoeducation is a treatment that is routinely practiced in a number of fields, and it will continue to be considered essential in terms of the treatment and prevention of relapses.

Cross-References

▶ Health Education

References and Readings

Cuijpers, P. (1997). Bibliotherapy in unipolar depression: A meta-analysis. *Journal of Behavioral Therapy and Experimental Psychiatry, 28*, 139–147.

Psychological and Social Conditions People Experience in the Workplace

▶ Psychosocial Work Environment

Psychological and Social Effects

▶ Psychosocial Impact

Psychological Disorder

Maxine Holmqvist
Clinical Health Psychology, University of Manitoba, Winnipeg, MB, Canada

Synonyms

Behavioral disorder; Emotional disorder; Mental disorder; Psychiatric disorder; Psychiatric illness

Definition

A distinctive pattern of cognitive, and/or behavioral symptoms in an individual, arising from underlying psychobiological dysfunction and causing significant distress, impairment, or an increased risk of death, pain, disability, or an important loss of freedom (American Psychiatric Association, 2000). Psychological disorders do not include culturally sanctioned responses to life events (e.g., feeling sad after a significant loss).

Description

The term "psychological disorder" is often used interchangeably with similar terms, including "mental disorder" and "psychiatric illness." It may be a preferable term to "mental disorder," which evokes a mind/body dualism that is inconsistent with modern theories that emphasize the biopsychosocial origins of disease (Fulford, Thornton, & Graham 2006). The term "psychological disorder" is broad, encompassing both disorders that manifest primarily with dysfunctional behaviors (e.g., anorexia nervosa, alcohol dependence) and disorders that manifest primarily with involuntary symptoms (e.g., schizophrenia, depression; McHugh & Slavney, 1998). In contrast, the term "psychiatric illness" more typically refers to the latter category, assuming that these disorders are more rooted in brain dysfunction (see *Psychiatric Illness*, this volume). To date, genetic studies have not supported this distinction (e.g., Bienvenu, Davydow, & Kendler 2011).

Historical Background

Psychological disorders are cultural constructs, created through a variety of social processes, including debate, voting, and expert consensus (Raskin & Lewandowski, 2000). Classifying psychological symptoms into disorders allows service providers to communicate with one another and for evidence-based knowledge to be accumulated and shared. It can enhance treatment planning and is critical for health care management (e.g., allowing governments to monitor the incidence and prevalence of various conditions in the population and allocate resources appropriately). While modern classification systems are empirically informed, current diagnostic categories are heavily influenced by historical forces (Kendler, 2009). Today, the two most common classification systems for psychological disorders are the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV), and the International Statistical Classification of Diseases and Related Health Problems, 10th revision (ICD-10). Both the DSM-IV and the ICD-10 are categorical systems that enable the user to record the presence or absence of a condition or disease according to certain standardized rules; in contrast to this, dimensional systems of classification are quantitative and are more likely to utilize psychometric measures and statistical procedures. In addition to more closely mirroring theoretical models of psychological pathology that characterize symptoms on a continuum with normal functioning (see "▶ Psychological Pathology", this volume), these systems can be helpful in providing additional information that is likely to be important clinically. The importance of dimensional assessment to treatment planning and monitoring in particular has also been highlighted, and several proposed dimensional assessments are being evaluated for feasibility during the upcoming DSM-V field trials (Stein et al. 2010).

The Diagnostic and Statistical Manual of Mental Disorders (DSM)

The DSM is published by the American Psychiatric Association (APA). Its aim is to provide common language and standard criteria for the classification of mental disorders, and it is the primary system used in North America. There have been five revisions since the DSM was first published in 1952 (APA, 2000). The current edition is the 4th edition, text revision (DSM-IV-TR); the fifth edition (DSM-V) is due to be published in May 2013 (American Psychiatric Association, 2011). The DSM describes how qualified individuals can assign diagnoses based on predetermined criteria. Diagnoses are recorded using a multiaxial system, with different disorder subtypes coded on different axes. While efforts have been made to keep the diagnostic codes in the DSM consistent with the ICD, this has not always been possible due to the differing revision cycles. Primary goals for the DSM-V include improving diagnostic validity and reliability and enhancing clinical utility.

International Statistical Classification of Diseases and Related Health Problems (ICD)

The ICD originated in the 1850s, when medical statisticians identified the need for a standard nomenclature to record cause of death and other

important health statistics. Originally published as the International List of Causes of Death by the International Statistical Institute in 1893, responsibility for the ICD was taken over by the World Health Organization (WHO) when it was created in 1948 (World Health Organization, 2007). Currently, the ICD is the international standard diagnostic classification system for epidemiological reporting and research among WHO member states (e.g., monitoring the incidence and prevalence of diseases in a population, recording national mortality and morbidity statistics); it also aims to provide useful information for health management and clinical purposes. The scope of the ICD has increased with each revision and update. In the early 1960s, a series of meetings were held with the aim of improving the diagnosis and classification of mental health disorders. The current edition, the ICD-10, was completed in 2007 and contains a comprehensive listing of "mental and behavioral disorders," which are coded on axis V (F00-F99).

As with psychiatric illnesses, psychological disorders can often predict the risk of developing complex medical conditions (e.g. heart disease); the reverse is also true. Furthermore, psychological disorders have prognostic value in some illnesses, including cancer, heart disease and renal failure.

Cross-References

- ▶ Mental Illness
- ▶ Psychiatric Disorder
- ▶ Psychiatric Illness
- ▶ Psychological Pathology

References and Readings

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders, Fourth Edition, Text revision*. Washington, DC: American Psychiatric Association. See also http://www.psych.org/mainmenu/research/dsmiv.aspx

American Psychiatric Association. (2011). DSM-5 Development Page. Retrieved from http://www.dsm5.org/Pages/Default.aspx

Bienvenu, O. J., Davydow, D. S., & Kendler, K. S. (2011). Psychiatric 'diseases' versus behavioral disorders and degree of genetic influence. *Psychological Medicine, 41*, 33–40.

Fulford, K. W. M., Thornton, T., & Graham, G. (2006). *Oxford textbook of philosophy and psychiatry*. Oxford: Oxford University Press.

Kendler, K. S. (2009). An historical framework for psychiatric nosology. *Psychological Medicine, 39*, 1935–1941.

Ladwig, K. H., Baumert, J., Marten-Mittag, B., Kolb, C., Zrenner, B., & Schmitt, C. (2008). Posttraumatic stress symptoms and predicted mortality in patients with implantable cardioverter-defibrillators: results from the prospective living with an implanted cardioverter-defibrillator study. *Archives of General Psychiatry, 65*, 1324–1330.

McHugh, P. R., & Slavney, P. R. (1998). *The Perspectives of Psychiatry*, 2nd ed. Baltimore: The Johns Hopkins University Press.

Raskin, J. D., & Lewandowski, A. M. (2000). The construction of disorder as human enterprise. In R. A. Neimeyer & J. D. Raskin (Eds.), *Constructions of disorder: Meaning-making frameworks for psychotherapy* (pp. 15–40). Washington, DC: American Psychological Association.

Stein, D. J., Phillips, K. A., Bolton, D., Fulford, K. W. M., Sadler, J. Z., & Kendler, K. S. (2010). What is a mental/psychiatric disorder? From DSM-IV to DSM-V. *Psychological Medicine, 40*, 1759–1765.

World Health Organization. (2007). *International statistical classification of diseases and related health problems, 10th Revision, version for 2007*. Retrieved January 1, 2011 from http://apps.who.int/classifications/apps/icd/icd10online/

Psychological Factors

▶ Psychosocial Factors

Psychological Factors and Health

Jane Upton
School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Psychological variables; Psychosocial factors

Definition

Thoughts, feelings, and attitudes that influence behavior

Description

Behavioral medicine incorporates not only the effect of individual's actions on their health but also how psychological factors affect the physical body. This is a progression from traditional biomedicine, which conceptualizes the mind and the body as separate entities. Behavioral medicine has started to break down this artificial boundary, illuminating the close relationship between mind and body, and therefore the role that psychological factors play in disease.

A key psychological factor is stress, which is known to affect many systems of the body. This summary will focus on the immune system, a system once thought to be independent of psychological factors. Research investigating the effect of stress on vaccinations generally indicates that chronic psychological stress impacts on the immune response. In a study conducted by Cohen and colleagues, 394 healthy individuals completed a questionnaire to assess degree of psychological stress and were then given nasal drops containing a respiratory virus (Cohen, Tyrrell, & Smith, 1991). The rates of respiratory infection increased in a dose-response manner with increases in psychological stress. This relationship was not altered when the researchers controlled for variables that might affect this relationship (e.g., the infectious status of subjects in close vicinity to each other). The level of psychological stress reported by the individuals prior to exposure to the virus was therefore directly related to the level of infection. This relationship has been consistently duplicated; for example, Phillips and colleagues found that the stress of bereavement in the year prior to influenza vaccination was associated with a poorer antibody response (Phillips et al., 2006).

However, it is too simplistic to conclude that stress suppresses the immune system. Different types of psychological stress have different effects on the immune system. It is therefore important to differentiate between acute, short-term stress and long-term chronic stress. It has generally been found that long-term stress suppresses the immune system (Bauer et al., 2000), whereas acute stress potentiates it (Herbert et al., 1994).

It is important to note though that the effect of psychologically stressful events varies between individuals based on how they evaluate the event and their coping strategies (Lazarus & Folkman, 1984). Pettingale and colleagues investigated the effect of coping strategy on recovery from cancer (Pettingale, Morris, Greer, & Haybittle, 1985). Fifty-seven women who had recently undergone mastectomy were interviewed 4 months after their operation. They were categorized in to four groups dependent on coping strategy: stoic acceptance, denial, fighting spirit, and helplessness/hopelessness. They found that coping strategy was related to 10-year disease-free survival; those women who adopted denial or fighting spirit coping strategies tended to survive longer.

The level of chronic stress and coping styles may interact to affect the immune system. Stowell and colleagues found that under conditions of high chronic stress, people who had active coping styles had higher proliferation of leukocytes to stimulation by mitogens than people who had avoidance coping mechanisms (Stowell, Kiecolt-glaser, & Glaser, 2001). However, they also found that when experiencing low levels of chronic stress, people that used avoidance mechanisms to cope had higher proliferation levels of leukocytes than those that used more active mechanisms to cope. The relationships between certain coping methods and immune function may therefore depend on perceived stress levels.

Immune response may also be affected by personality traits such as neuroticism (Phillips, Carroll, Burns, & Drayson, 2005), internalization in adolescents (Morag, Morag, Reichenberg, Lerer, & Yirmiya, 1999), and trait negative affect (Marsland, Cohen, Rabin, & Manuck, 2001). More widely, it has been suggested that a constellation of personality traits such as high levels of anxiety, neuroticism, depression, anger, and hostility may be linked with a range of

diseases (Friedman & Booth-Kewley, 1987). However, prospective studies are required to confirm that these traits contribute to the etiology and progression of disease.

Schnurr and colleagues investigated the effect of posttraumatic stress disorder (PTSD) in 605 veterans of World War II and the Korean conflict (Schnurr & Avion, 2000). They controlled for age, smoking, alcohol use, and body weight at study entry. They found that PTSD symptoms were associated with increased onset of arterial lower gastrointestinal dermatologic and musculoskeletal disorders. The authors state that it is premature to draw firm conclusions from their study about the relationship of PTSD to these disorders. However, their findings were very similar to those reported by Boscarino and colleagues in their study of Vietnam War veterans (Boscarino, 1997)

Less dramatically, it has also been found that daily hassles affect the physical body. In a study of 48 undergraduate students, levels of daily hassles correlated with the General Health Questionnaire somatic symptoms scale (Sheffield, McVey, & Carroll, 1996). Daily hassles have also been linked with fluctuations in blood pressure. Steptoe and colleagues (1996) recorded the blood pressure (BP) hourly from 49 male firefighters on work and nonwork days (Steptoe, Roy, & Evans, 1996). They found that systolic BP readings accompanied by feelings of anger and stress were significantly greater than those without negative moods in both work and nonwork settings. They concluded that the raised systolic BP during working hours observed was affected both by physical activity and concurrent mood and that stress and anger were particularly influential.

Cross-References

- ▶ Psychosocial Predictors
- ▶ Psychosocial Variables

References and Readings

Bauer, E., Vedhara, K., Perks, P., Wilcock, G. K., Lightman, S. L., Shanks, N., et al. (2000). Chronic stress in caregivers of dementia patients is associated with reduced lymphocyte sensitivity to glucocorticoids. *Journal of Neuroimmunology, 103*, 84–92. doi:10.1016/S0165-5728(99)00228-3.

Boscarino, J. (1997). Diseases among men 20 years after exposure to severe stress: Implications for clinical research and medical care. *Psychosomatic Medicine, 59*(6), 605–614.

Cohen, S., Tyrrell, D., & Smith, A. (1991). Psychological stress and susceptibility to the common cold. *The New England Journal of Medicine, 325*, 606–612.

Friedman, H., & Booth-Kewley, S. (1987). The "disease-prone personality": A meta-analytic view of the construct. *The American Psychologist, 42*, 539–555. doi:10.1037/0003-066X.42.6.539.

Herbert, T., Cohen, S., Marsland, A. L., Bachen, E., Rabin, B., Muldoon, M., et al. (1994). Cardiovascular reactivity and the course of immune response to an acute psychological stressor. *Psychosomatic Medicine, 56*(4), 337–344.

Lazarus, R., & Folkman, S. (1984). *Stress, appraisal and coping*. New York: Springer.

Marsland, A. L., Cohen, S., Rabin, B., & Manuck, S. (2001). Associations between stress, trait negative affect, acute immune reactivity, and antibody response to hepatitis b injection in healthy young adults. *Health Psychology, 20*, 4–11. doi:10.1037/0278-6133.20.1.4.

Morag, M., Morag, A., Reichenberg, M., Lerer, B., & Yirmiya, R. (1999). Psychological variables as predictors of rubella antibody titers and fatigue – A prospective double blind study. *Journal of Psychiatric Research, 33*, 389–395.

Pettingale, K., Morris, T., Greer, S., & Haybittle, J. (1985). Mental attitudes to cancer; an additional prognostic factor. *Lancet, 1*(8431), 750.

Phillips, A. C., Carroll, D., Burns, V. E., & Drayson, M. (2005). Neuroticism, cortisol reactivity, and antibody response to vaccination. *Journal of Personality, 42*, 232–238. doi:10.1111/j.1469-8986.2005.00281.x.

Phillips, A. C., Carroll, D., Burns, V. E., Ring, C., Macleod, J., Drayson, M., et al. (2006). Bereavement and marriage are associated with antibody response to inXuenza vaccination in the elderly. *Immunity, 20*, 279–289. doi:10.1016/j.bbi.2005.08.003.

Schnurr, P. P., & Avion, S. (2000). Physician-diagnosed medical disorders in relation to PTSD symptoms in older male military veterans. *Health Psychology, 19*(1), 91–97. doi:10.1037//0278-6133.19.1.91.

Sheffield, D., McVey, C., & Carroll, D. (1996). Daily events and somatic symptoms: Evidence of a lagged relationship. *The British Journal of Medical Psychology, 69*, 267–269.

Steptoe, A., Roy, M., & Evans, O. (1996). Psychosocial influences on ambulatory blood pressure over working and non-working days. *Journal of Psychophysiology, 10*(3), 218–227.

Stowell, J. R., Kiecolt-glaser, J. K., & Glaser, R. (2001). Perceived stress and cellular immunity: When coping counts. *Journal of Behavioral Medicine, 24*(4), 323–339.

Psychological Pathology

Maxine Holmqvist
Clinical Health Psychology, University of Manitoba, Winnipeg, MB, Canada

Synonyms

Abnormal psychology; Psychopathology

Definition

The scientific study of psychological disorders and their causes.

Description

Psychological pathology is the study of the causes, components, course, and consequences of psychological disorders. These are characterized by *abnormality* and *dysfunction*.

Abnormality

Psychological disorders are defined by diagnostic criteria, like those outlined in the Diagnostic and Statistical Manual of Mental Disorders (DSM; American Psychiatric Association [APA], 2000) or the International Statistical Classification of Diseases and Related Health Conditions (ICD; World Health Organization, 2007). These criteria are composed of "marker symptoms," or thoughts, feelings, and/or behaviors identified as abnormal for a variety of reasons (e.g., because they cause distress, disadvantage, or disability or are highly inflexible or irrational; Stein et al., 2010). Symptoms can be understood as qualitatively different from normal or as extreme variants of common traits. Some symptoms are abnormal because they deviate significantly from a statistical mean. The notion of deviance from the mean is the underlying rationale for many commonly used psychometric tests, but is perhaps most clearly illustrated using the concept of intelligence. It is assumed that intelligence is normally distributed in the population; thus, individuals whose scores on a standardized test of intelligence fall below a specific cutoff may be diagnosed with mental retardation. It is important to note that even in this circumstance, meeting this cutoff is not sufficient for a diagnosis; there must be additional evidence of dysfunction (e.g., deficits in self-care and academic performance; APA, 2000). Thus, a trait or behavior is not necessarily "pathological" simply because it is highly unusual – high intelligence may be equally rare, but is not usually debilitating. Similarly, some characteristics that are presumed to be dysfunctional may be quite common (e.g., depressive thoughts, binge drinking). Importantly, abnormality can only be defined in reference to a given population; thus, the boundaries between normal and abnormal will shift over time and across cultures.

Dysfunction

Symptoms that cause significant impairment in important life domains may also be considered "pathological." Accordingly, dysfunction is often assessed with reference to the consequences of the symptom or disorder. Increasingly, psychological research is providing evidence of dysfunction by showing that certain psychological traits or behavioral patterns (e.g., perfectionism, type "A" personality, avoidance of feared stimuli) are reliably associated with undesirable physiological, social, and occupational outcomes (e.g., procrastination, chronic hypertension, strengthening of a phobic response). Dysfunction is also sometimes assessed in evolutionary terms, with symptoms referred to as "maladaptive," suggesting that they deviate from functioning that would have led to survival and reproductive success in the past.

Cross-References

▶ Psychiatric Disorder
▶ Psychiatric Illness
▶ Psychological Disorder

References and Readings

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders, text revision* (4th ed.). Washington, DC: Author.

Stein, D. J., Phillips, K. A., Bolton, D., Fulford, K. W. M., Sadler, J. Z., & Kendler, K. S. (2010). What is a mental/psychiatric disorder? from DSM-IV to DSM-V. *Psychological Medicine, 40*, 1759–1765.

World Health Organization. (2007). *International statistical classification of diseases and related health problems, 10th revision, version for 2007*. Retrieved January 1, 2011 from http://apps.who.int/classifications/apps/icd/icd10online/

Psychological Predictors

▶ Psychosocial Predictors

Psychological Researcher

▶ Psychologist

Psychological Science

Peter A. Hall
Faculty of Applied Health Sciences, University of Waterloo, Waterloo, ON, Canada

Synonyms

Scientific psychology

Definition

The term *psychological science* refers to the accumulated body of psychological knowledge (i.e., pertaining to brain, behavior, social, or mental processes) that has been generated through the systematic application of the scientific method. The term *psychological science* may also refer to the process of conducting psychological research through the use of the scientific method.

The scientific approach to studying social, mental, and behavioral phenomena has existed for the full history of the field of psychology. Though some have questioned the applicability of scientific methods to researching mental phenomena for at least as long as its existence, psychological science has always been at the core of psychology as a field, and scientific rigor has been an aspiration even within the applied subdisciplines of psychology, including clinical and health psychology. Psychology as a discipline has more strict adherence to the scientific method than most social sciences, and so the nature of accumulated knowledge within psychology in the first century of its existence would be largely considered scientific in nature.

Commitment to the scientific method in psychological research is traceable back to William James (1842–1910) in North America and Wilhelm Wundt (1832–1920) in Europe, though its roots likely extend even earlier than these two individuals. Notably, both James and Wundt were trained initially as physicians, highlighting the long-standing interconnectedness of psychological science and health science from the time that psychology emerged as a legitimate area of scientific inquiry. Psychological knowledge aims to be scientifically based by following basic scientific criteria of empiricism, replicability of a method, and the testing of generalizable hypotheses and models which eventually explain psychological phenomena.

In 1989, the inaugural issue of *Psychological Science* was published by the Association for Psychological Science (formerly named the American Psychological Society). This flagship journal was intended to be a showcase for leading psychological research conducted with rigorous adherence to the scientific method. The prominence of *Psychological Science* has grown steadily from its inception to present, and it is currently among the highest ranking empirical journals in the field of psychology (Association for Psychological Science, 2012).

References and Readings

Association for Psychological Science. (2012). *Psychological Science (journal home page)*. http://www.psychologicalscience.org/index.php/publications/journals/psychological_science

Psychological Scientist

▶ Psychologist

Psychological Stress

Shin-ichi Suzuki[1] and Daisuke Ito[2]
[1]Faculty of Human Sciences, Graduate School of Human Sciences, Waseda University, Tokorozawa-shi, Saitama, Japan
[2]Health Service Center, Kanazawa University, Kanazawa, Ishikawa, Japan

Synonyms

Distress; Strain; Stress; Stressor

Definition

H. Selye (1936) defined stress as "non-specific responses that be resulted from a variety of different kinds of stimuli." However, Selye's stress theory has only focused on physiological stress, and psychological factors have not been considered. Research on life stress examined the relationship between diseases and life events. Many studies were conducted for clarifying the psychological factors related to stress, and the results revealed that psychological factors play a significant role in the occurrence of physiological and psychological stress responses. Lazarus and Folkman (1984) proposed that stress occurs when people perceived that the demands from external situations were beyond their coping capacity. Today, the definition "stress is the process of interaction from resolution requests from the environment (known as the *transactional* model)" is widely accepted.

From the perspective of psychological stress research, the ambiguous elements related to stress have distinguished two aspects of stress. One is called "stressors," which cause stress (e.g., interpersonal problem, hard work, noise, and trauma). Another is called "stress responses," which are nonspecific physical and mental changes induced by stressors (e.g., frustration, depression, anxiety, and stomachache). Psychological stress responses that caused by various daily experiences are emotional, cognitive, and behavioral changes; their degrees have also become main factors affecting physical and mental health. However, as mentioned in the definition of Lazarus and Folkman (1984), psychological stressors are related to one's cognition and coping process rather than induced stress responses directly. Therefore, an effective approach for reducing psychological stress responses should include not only the removal of stressors but also enhancing the cognitive and behavioral coping capability.

Cross-References

▶ Cognitive Appraisal
▶ Coping
▶ Mental Stress
▶ Stress, Emotional
▶ Stress Responses
▶ Stressor

References and Readings

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.
Selye, H. (1936). A syndrome produced by diverse nocuous agents. *Nature, 138*, 32.

Psychological Stress Task

▶ Stress Test

Psychological Stressor

► Stress Test

Psychological Testing

► Assessment

Psychological Thriving

► Resilience

Psychological Variables

► Psychological Factors and Health
► Psychosocial Variables

Psychologist

Vincent Tran
University of Texas, Southwestern Medical Center, Dallas,
TX, USA

Synonyms

Mental health professional; Psychological researcher; Psychological scientist

Definition

A psychologist is a professional who has earned a doctoral degree in psychology at a regionally accredited university or professional school. Some psychologists are primarily involved in conducting research and contributing to the scientific body of knowledge in psychology, while others use this scientific knowledge in applied settings. Psychologists work in several different work sectors, including clinical practice settings, academic and research settings, and consultation to apply psychological principles in private or public industries. Major settings in which psychologists work include universities and medical schools, private practice, clinics and counseling centers, industry and government, hospitals, and school districts. In recent decades, psychologists have increasingly focused their work on health-related research and with patients in medical settings. This is evidenced in the rapid growth of the membership in the Health Psychology Division of the American Psychological Association (Division 38), and in the fact that psychologists comprise 73% of the interdisciplinary membership of the Society of Behavioral Medicine.

Although psychologists often enjoy a variety of professional roles, some psychologists (e.g., clinical and counseling psychologists) are primarily involved in the practice of psychology. Applied psychologists focus on the identification, assessment, treatment, and/or consultation related to psychological issues impacting human behavior in applied settings. Interventions may include individual, group, or family psychotherapy, as well as consultation with community and private organizations. In order to regulate the practice of psychology and to ensure mental and behavioral health services are provided by qualified professionals, psychologists must obtain a professional license. Each state has its own requirements for licensure; in addition to the doctoral degree, states commonly require additional postdoctoral training and passing scores on national and state licensure exams.

Other psychologists primarily conduct basic or applied research and/or teach in academic or university settings. Psychological science uses statistical and empirical methods of measurement to understand mental processes and behavior, which can be at the social, cognitive, biological, and/or emotional level. Major specialty areas studied in psychology include clinical, counseling, educational, experimental, industrial/organizational, developmental, social, personality, physiological, and quantitative psychology. The American Psychological Association, an organization representing psychologists in the United States,

has 54 divisions and interest groups, demonstrating the range and variety of subdisciplines or specialty interest areas of psychologists. The Association for Psychological Science is an organization dedicated to promoting scientifically oriented psychologists and psychology as a scientific discipline.

Cross-References

- Health Psychology
- Medical Psychology
- Psychological Science

References and Readings

APA. (2011). *American Psychological Asssociation*. Retrieved December 13, 2011, from http://www.apa.org/

APA. (2011). *What is APA's definition of a psychologist*? American Psychological Asssociation. Retrieved January 11, 2011, from http://www.apa.org/support/about/apa

APS. (2011). *Association for Psychological Science*. Retrieved December 13, 2011, from http://www.psychologicalscience.org

Keefe, F., Portner, L., & Somers, T. (2010). *The SBM career trajectories survey: Findings and next steps*. Society of Behavioral Medicine. Retrieved December 13, 2011, from http://www.sbm.org/about/councils/education-training-and-career-development/career-trajectories

Smith, M. W., & Passer, R. E. (2001). *Psychology: Frontiers and applications*. New York: McGraw-Hill.

Stricker, G., Widiger, T. A., & Weiner, I. B. (Eds.). (2003). *Handbook of psychology: Clinical psychology (Vol. 8)*. Hoboken: Wiley.

Psychometric Properties

Annie T. Ginty
School of Sport and Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

Synonyms

Psychometrics

Definition

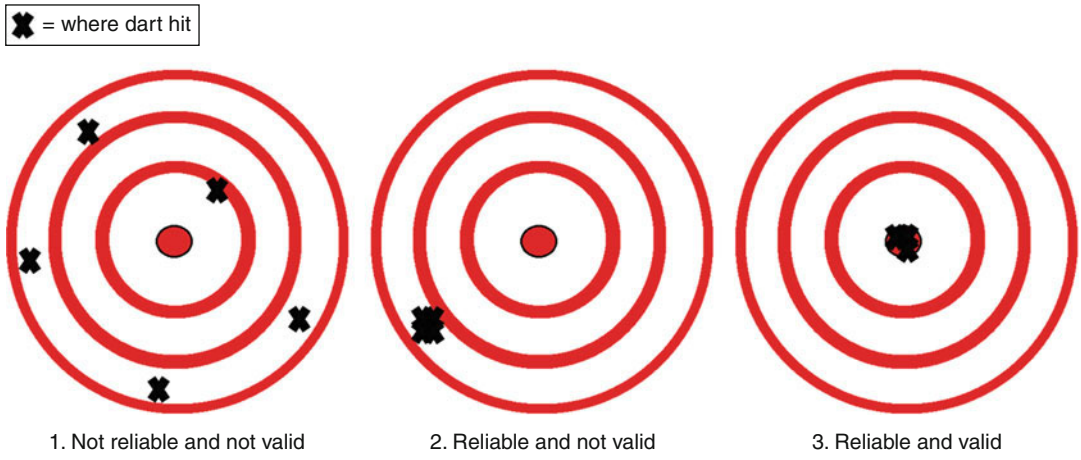
Psychometrics is the construction and validation of measurement instruments and assessing if these instruments are reliable and valid forms of measurement. In behavioral medicine, psychometrics is usually concerned with measuring individual's knowledge, ability, personality, and types of behaviors. Measurement usually takes place in the form of a questionnaire, and questionnaires must be evaluated extensively before being able to state that they have excellent psychometric properties, meaning a scale is both reliable and valid.

Description

A reliable scale consistently measures the same construct. This can occur across testing sessions, individuals, and settings. A valid measure measures what it says it is going to measure. If something is valid, it is always reliable. However, something can be reliable without being valid.

For example (see Fig. 1), if someone has five darts and is told to hit the bull's eye every time, their ideal aim is to be both reliable (consistent) and valid (accurate) with their throws. In part 1, the throws are neither reliable nor valid because their efforts are scattered across the board. In part 2, the throws were reliable, hitting the same spot every time. However, they did not hit the targeted spot of the bull's eye so they were not valid. Part 3 shows the perfect example of being both reliable and valid. The throws hit the spot they intended to hit and did so consistently. This same concept is applied to questionnaire measurements.

If a researcher is trying to create a new scale to measure depression, they want to make sure the scale reliably measures depression in someone with depression. For example, if the same individual filled out the questionnaire three times in the same day, they would produce the same score each time. The researcher also wants to make sure the scale is valid, which means that the scale is actually measuring depressive symptoms and not some other mood or behavior.



Psychometric Properties, Fig. 1 An example of reliability and validity

Cross-References

- ▶ Construct validity
- ▶ Validity

References and Readings

DeVellis, R. F. (2003). *Scale development: Theory and applications* (2nd ed.). London: Sage.
Grimm, G. L., & Yarnold, P. R. (2000). *Reading and understanding more multivariate statistics*. Washington, DC: American Psychological Association.

Psychometric Theory

- ▶ Psychometrics

Psychometrics

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Psychometric theory

Definition

Psychometric theory is a well-recognized approach for developing psychological measurement and standardized education tests (Kim-O & Embretson, 2010). It can be divided into two categories: classical test theory (CTT) and item response theory (IRT). CTT was pioneered a century ago by Spearman, while IRT developed in the 1950s and 1960s.

Kim-O and Embretson (2010) provided a thorough discussion of IRT and why it has become the preferred approach. In the CTT model, estimates of examinees' true test scores are often linear transformations of the raw test score, and are related to relevant normative populations by the transformation. Alternative test forms can be used to estimate true scores if the forms are parallel tests with the same expected true scores and error distributions. Psychometric indices for items in CTT are related to the properties of the test scores, particularly reliability and variance. In other words, item difficulty is defined as the proportion of persons passing or endorsing an item, while item discrimination is defined as the correlation of the item with the total test score.

The CTT approach, however, has several limitations. An examinee's true score depends on the difficulty level of a test, i.e., it is test-dependent: Scores will not be comparable between easy and hard tests. Second, the item characteristics

depend on the ability of examinees, i.e., they are sample-dependent. Item difficulty, for example, will vary substantially if the true score distributions vary between populations. Third, one of the key assumptions, that two true test scores and two error variances are identical in the two tests (the parallel test assumption), is never fully met in practice. Therefore, it becomes difficult to compare examinees who take different tests, and to contrast items whose characteristic indices are computed using different groups of examinees.

In contrast, in the IRT approach the examinee's true score is not test-dependent, the item parameters are not sample-dependent, and the parallel test assumption is not needed. See Kim-O and Embretson (2010) for further discussion of this approach.

Psychometric theory is used extensively in behavioral medicine research. As two examples, Kiernan, Moore, and Schoffman (2011) assessed the psychometric properties, initial levels, and predictive validity of a measure of perceived social support and sabotage from friends and family for healthy eating and physical activity. Second, Lo et al. (2011) introduced the Death and Dying Distress Scale (DADDS), a new, brief measure developed to assess death-related anxiety in advanced cancer and other palliative populations. Their paper described its preliminary psychometrics based on a sample of 33 patients with advanced or metastatic cancer. Additional examples are provided in the "References and Readings" section of this entry.

Cross-References

- ▶ Psychometric Properties
- ▶ Validity
- ▶ Variance

References and Readings

Kiernan, M., Moore, S. D., & Schoffman, D. E. (2011) Social support for healthy behaviors: Scale psychometrics and prediction of weight loss among women in a behavioral program. *Obesity (Silver Spring, MD)* Oct, 13th [Epub ahead of print].

Kim-O, M.-A., & Embretson, S. E. (2010). Item response theory and its application to measurement in behavioral medicine. In A. Steptoe (Ed.), *Handbook of behavioral medicine: methods and applications* (pp. 113–123). New York: Springer.

Kowalchuk Horn, K., Jennings, S., Richardson, G., et al. (2012). The patient-specific functional scale: Psychometrics, clinimetrics, and application as a clinical outcome measure. *The Journal of Orthopaedic and Sports Physical Therapy, 42*(1), 30–42.

Lo, C., Hales, S., Zimmermann, C., et al. (2011). Measuring death-related anxiety in advanced cancer: preliminary psychometrics of the death and dying distress scale. *Journal of Pediatric Hematology/Oncology, 33* (Suppl. 2), S140–S145.

Maddux, R. E., Lundh, L. G., & Bäckström, M. (2011). The Swedish depressive personality disorder inventory: Psychometrics and clinical correlates from a DSM-IV and proposed DSM-5 perspective. *Nordic Journal of Psychiatry*, Sep 22nd [epub ahead of print].

Robinson, D. W., Jr., Cormier, J. N., Zhao, N., et al. (2012). Health-related quality of life among patients with metastatic melanoma: Results from an international phase 2 multicenter study. *Melanoma Research, 22*(1), 54–62.

Psychoneuroendocrinology

Jutta M. Wolf[1] and Eve Saucier[2]
[1]Department of Psychology, Brandeis University, Waltham, MA, USA
[2]Brandeis University, Waltham, MA, USA

Synonyms

Behavioral endocrinology

Definition

Psychoneuroendocrinology (PNE) is an interdisciplinary field of research integrating psychology, endocrinology, and neuroscience to study the interactions between mind, brain, and hormonal function (Dantzer, 2010).

More specifically, PNE focuses on the way psychological factors influence neuroendocrine functions and, conversely, the way hormones influence higher brain functions. As such, PNE is not only interested in hormone synthesis, release, transport, breakdown, and feedback control but in

the interaction of hormones with their target tissues (e.g., the immune system), including the molecular mechanisms of their action.

PNE research is often concerned with health implications associated with even subtle chronic changes in hormonal patterns. While sex is one of the biggest factors influencing the body's neuroendocrine state, other factors of interest include health effects of age-related changes and changes observed in the context of diseases such as the metabolic syndrome or mood and anxiety disorders. By far, the most intensely studied area, however, is PNE of stress, which aims at describing and understanding neuroendocrine changes associated with acute as well as chronic physiological and especially psychological stress and how stress-related changes in turn impact behavior, cognition, affect, and health (for a review, see Dantzer, 2010).

Cross-References

- ▶ Behavioral Immunology
- ▶ Behavioral Medicine
- ▶ Psychoneuroimmunology
- ▶ Stress
- ▶ Sympathetic Nervous System (SNS)
- ▶ Sympatho-Adrenergic Stimulation

References and Readings

Dantzer, R. (2010). Psychoneuroendocrinology of stress. In F. K. George, M. Le Michel, & F. T. Richard (Eds.), *Encyclopedia of behavioral neuroscience* (pp. 126–131). Oxford: Academic Press.

Psychoneuroimmunology

Jutta M. Wolf and Kirsten Rene
Department of Psychology, Brandeis University, Waltham, MA, USA

Synonyms

Behavioral immunology

Definition

Psychoneuroimmunology (PNI) is the study of the functional relationships between central nervous system, behavior, and immune system.

These relationships have been documented to be multidirectional. For example, while behavior can influence immune processes through changes in nervous system signals, immune signals have been shown to alter the function of the central nervous system, thereby influencing behavior. Further, all systems exert regulatory control over each other, forming a complex communication network.

PNI research aims at describing this network and thus to contribute to the understanding of the behavioral and biological mechanisms underlying the links between psychosocial factors and health as well as disease development and progression. Psychosocial factors studied in PNI thereby range from negative psychological states such as depression and anxiety, to social support, interpersonal relationships, and personality factors. Disease-related processes investigated include cancer, susceptibility to infection, wound healing, HIV/AIDS, autoimmune diseases, and cardiovascular diseases. One important PNI branch focuses on how stress and stress-related neuroendocrine processes (see ▶ Psychoneuroendocrinology) affect health as well as disease development and progression.

As such, PNI is truly interdisciplinary, integrating not only knowledge from immunology, neuroscience, and psychology, but also from areas such as psychiatry, endocrinology, physiology, and pharmacology.

Cross-References

- ▶ Behavioral Immunology
- ▶ Behavioral Medicine
- ▶ Cytokines
- ▶ Immune Responses to Stress
- ▶ Inflammation
- ▶ Psychoneuroendocrinology
- ▶ Sickness Behavior
- ▶ Wound Healing

References and Readings

Ader, R. (2001). Psychoneuroimmunology. *Current Directions in Psychological Science, 10*(3), 94–98.
Cohen, S., & Herbert, T. B. (1996). Health psychology: Psychological factors and physical disease from the perspective of human psychoneuroimmunology. *Annual Review of Psychology, 47*, 113–142.
Maier, S. F., Watkins, L. R., & Fleshner, M. (1994). Psychoneuroimmunology: The interface between behavior, brain, and immunity. *American Psychologist, 49*(12), 1004–1017.
Schedlowski, M., & Tewes, U. (Eds.). (1996). *Psychoneuroimmunology: An interdisciplinary introduction*. New York/Boston/Dordrecht/London/Moscow: Kluwer Academic/Plenum.
Vedhara, K., & Irwin, M. (Eds.). (2007). *Human psychoneuroimmunology*. New York: Oxford University Press.

Psycho-oncology

► Cancer: Psychosocial Treatment

Psychopathology

► Psychological Pathology

Psychophysiologic Disorders

► Psychosomatic Disorder

Psychophysiologic Reactivity

Mark Hamer
Epidemiology and Public Health, Division of Population Health, University College London, London, UK

Synonyms

Stress response

Definition

Psychophysiologic reactivity refers to cardiovascular and biological responses to situations that are perceived as stressful, threatening, and/or physically harmful. Reactivity is defined as the response with respect to resting values. Some of the stressors that are commonly used in laboratory-based psychophysiological studies are designed to replicate real life, such as problem solving and public speaking tasks (Kamarck & Lovallo, 2003). It is, however, often advantageous to use novel stressors, such as the Stroop word-color conflict task and mirror tracing, in order to remove the potential confounding influences of education and work experience. Psychophysiologic stress testing allows individual differences in responses to standardized stress to be evaluated and related to psychosocial factors and health outcomes (Chida & Hamer, 2008). Behaviorally evoked psychophysiological responses are a relatively stable individual trait, consistent across time and stressor type. The magnitude or pattern of an individual's stress response is largely augmented by the immediate actions of the autonomic nervous system and delayed response of the hypothalamic-pituitary-adrenal axis, which releases various hormones (i.e., catecholamines, cortisol, etc.) into the circulation. These systems drive specific responses that include an increase in blood pressure and heart rate, changes in cardiac sympatho-vagal balance, skeletal muscle vasodilatation, the release of hemostatic and inflammatory markers, and activation of various immune cells. Although psychophysiologic reactivity is beneficial for maximizing performance, excessive and enduring responses are also relevant to health and well-being and are thought to contribute to underlying disease pathology.

Cross-References

► Blood Pressure Reactivity or Responses
► Heart Disease and Cardiovascular Reactivity
► Psychophysiologic Recovery
► Stroop Color-Word Test

References and Readings

Chida, Y., & Hamer, M. (2008). Chronic psychosocial factors and acute physiological responses to laboratory-induced stress in healthy populations: A quantitative review of 30 years of investigations. *Psychological Bulletin, 134*(6), 829–885.

Kamarck, T. W., & Lovallo, W. R. (2003). Cardiovascular reactivity to psychological challenge: Conceptual and measurement considerations. *Psychosomatic Medicine, 65*(1), 9–21.

Psychophysiologic Recovery

Mark Hamer
Epidemiology and Public Health, Division of Population Health, University College London, London, UK

Synonyms

Relaxation; Return to baseline; Rumination

Definition

Psychophysiologic recovery is defined as the rate at which a cardiovascular or biological variable returns to resting levels following a stressor. It is not uncommon to observe prolonged elevation in blood pressure following induction of mental stress, and this might last for up to an hour or so following the cessation of the stressor. This has also been observed in naturalistic settings, for example, in teachers, blood pressure has been shown to remain elevated throughout the evening following a stressful working day at school. A slower rate of psychophysiologic recovery has been linked to several risk factors and poorer health outcomes (Brosschot, 2010). One difficulty with isolating the predictive value of recovery is that those taking the longest time to return to baseline are likely to be those who showed the greatest reactivity. Nevertheless, recent evidence suggests that poor recovery and heightened reactivity are in fact independent predictors of health outcomes. The mechanisms underlying poorer psychophysiologic recovery are incompletely understood, although various psychological factors such as rumination and coping strategies have been implicated.

Cross-References

▶ Cardiovascular Recovery
▶ Psychophysiologic Reactivity

References and Readings

Brosschot, J. F. (2010). Markers of chronic stress: Prolonged physiological activation and (un)conscious perseverative cognition. *Neuroscience and Biobehavioral Reviews, 35*(1), 46–50.

Psychophysiological

Douglas Carroll
School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Definition

Psychophysiology is an interdisciplinary science concerned with the impact of psychological exposures and behavioral challenges on physiological systems. The adjective "psychophysiological" describes this impact. For example, the effect on blood pressure of exposure to a mental stress task would be described as a psychophysiological effect.

Cross-References

▶ Psychophysiology: Theory and Methods

Psychophysiology: Theory and Methods

William Lovallo
Department of Psychiatry and Behavioral Sciences, University of Oklahoma Health Sciences Center Veterans Affairs Medical Center, Oklahoma City, OK, USA

Synonyms

Psychophysiological

Definition

How does mental and emotional life tie in with the workings of the body? Psychophysiology is the branch of psychology that studies the behavior of the individual in a biological context. It is an attempt to chart the mutual interactions between psychological processes and the workings of the body, giving equal emphasis to both.

Description

A fundamental principle of psychophysiology is that thoughts and feelings cannot exist apart from the body. It follows that a full understanding of psychological processes depends on understanding the biological context from which they proceed. Due to its emphasis on integrating our understanding of mental and physiological processes, psychophysiology has contributed to research methods and theory building in behavioral medicine and to the neurosciences of cognition and emotion. Psychophysiology does this by providing a theoretical basis and a set of measurement methods that help to disentangle relationships between psychology and biology and between our thoughts and emotional experience in relation to good and poor health. From this perspective, psychophysiologists bring a physiological emphasis to the study of behavior and mental processes, and one expression of this emphasis is the contributions psychophysiology has made is to the understanding of emotions and their impact on good and poor health. Although it is accepted in psychophysiology that thoughts and feelings do not exist without the brain and the body, it is necessary to emphasize that the thoughts and feelings of interest are not equivalent to or directly reducible to these physiological processes.

The emphasis of psychophysiology, like that of behavioral medicine itself, is primarily on the whole person. However, it is necessary to measure the functions of specific systems, such as the cardiovascular system, endocrine system, or immune system in the course of psychophysiological investigations. This calls for a methodology that allows emotional experience to be studied simultaneously with physiological functioning in ways that are unobtrusive and minimally invasive. This ensures that the person being studied is behaving in a normal manner, as in everyday life, and is not reacting unduly to the apparatus or laboratory setting. Psychophysiological principles have been used to study responses to stress in the laboratory, responses to stressors in daily life, and individual differences in such responses.

Behavioral medicine is both a science and an approach to clinical practice. These two parts are concerned with the influence of behavioral factors on health and disease. Behavioral medicine holds that states of health can be influenced by overt behaviors, such as dietary habits, and by covert behaviors, such as emotional states and stress responses. This perspective leads behavioral medicine researchers to ask questions about the ways that emotional states and stress responses can affect health through their influence on physiology. The goal is to bring to light how our behaviors and our ways of perceiving and reacting to the world may affect our well-being for better or worse. Such research addresses questions in several major areas, including (1) how the body responds during positive and negative emotion states, (2) how a given person may differ from one time to the next in stress

reactivity, (3) the ways in which persons differ from one another in their stress responses, and (4) on the positive side, to establish the effects of behavior on good health and longevity. To carry out such research, behavioral medicine draws in part on the theory and methods developed in the field of psychophysiology.

In laboratory studies, persons are often exposed to stressors to determine how they react to such challenges both emotionally and physiologically. The results are thought to indicate how emotionally relevant events and behavioral stressors can affect physiology in daily life and therefore whether they may contribute to disease. As one example, a commonly used stressor is public speaking. This challenges the subject to make up a short speech and deliver it without notes and to do so in a fluent and convincing manner. Public speaking is stressful because most persons wish to avoid the embarrassment of doing poorly and to be seen as masterful and competent by observers in the laboratory. Using this method, the social world can be modeled in a small way in the laboratory, and the participant's disposition is invoked to produce a stress response. During public speaking, this process of social evaluation, along with the resulting fear and anxiety, produces substantial increases in heart rate and blood pressure and stress hormones, including catecholamines and cortisol. The person's mood states usually are assessed at rest before the task begins and again at the end using paper-and-pencil measures or brief interviews. Similarly, autonomic reactions are often measured at rest and during stress using automated blood pressure monitors and impedance cardiographs, and endocrine responses may be observed using saliva or blood sampling. In this manner, the person's psychological, cardiovascular, and endocrine reactions may be measured to provide a picture of how physiological reactions are set off by psychologically meaningful events.

This research strategy can then be extended to compare different kinds of people for potential differences in their physiological reactivity to stress. One common example is for the researcher to identify young, healthy individuals who have a family history of high blood pressure and also to find those with no such history. These family history groups can then be compared in the laboratory for differences their stress responses, perhaps using the public speaking stressor or some other method. This allows potential differences in stress reactivity to be assessed in relation to a family history of this prevalent cardiovascular disease. It is then possible to follow such persons for a period of years to establish which persons become hypertensive and which retain a normal blood pressure. Do persons from the family history group have a greater likelihood of becoming hypertensive in middle age? Are persons with greater reactions to stress more likely to become hypertensive, regardless of family history? Such studies therefore allow potential interactions between family history and stress reactivity to be studied. If persons with a family history of hypertension who are also highly reactive to social stress are much more likely to become hypertensive, then we would conclude that the family history created a biologically based risk factor that was enhanced by an elevated level of stress responsivity. In contrast, should risk of hypertension be increased equally by high reactivity in persons with and without a family history of hypertension, we would conclude that family history and reactivity tendencies contribute to hypertension risk in an additive manner.

Although the laboratory provides a well-controlled environment with an extensive range of measurement techniques, ambulatory methods have been used with increasing frequency outside the laboratory to document how challenges in persons' daily lives can affect cardiovascular, endocrine, or immune systems. Such methods measure the person's responses to naturalistic stressors, such as work stress, or challenges in the home, such as family conflict or the stress of caring for a chronically ill spouse. Such studies rely on small, lightweight monitors that can be worn comfortably as persons go about their daily routines. These monitors can make reliable measurements in a wide range of circumstances. Such systems are able to track heart rate, blood pressure, and physical activity. In addition, the person usually reports on their subjective state using brief paper diaries or personal digital assistants.

As in laboratory studies, this ambulatory method may be used to estimate the interaction of stress responses and disease risk. Persons with and without a family history of hypertension may be compared as they go about their daily lives. As in the laboratory, persons with the largest or most prolonged reactions to stress at home or at work are suspected of having greater risk of future disease, and again, they may be followed up for actual occurrence of hypertension in future years. Ambulatory systems currently in use include traditional Holter electrocardiographs, blood pressure monitors, and impedance cardiographs. The success of these systems has led several commercial companies to develop reliable products for research and clinical use.

Although some research focuses on family history, other work seeks to connect psychological dispositions, such as hopelessness, depression, or hostile style to disease risk. Studies using this strategy may compare highly hostile persons with nonhostile individuals with a specific hostility provoking interaction, such as harassing comments during work on a difficult task. By measuring physiological reactions to such specific challenges in persons with different psychological characteristics, a clearer picture may be developed of the psychological and physiological interplay that is suspected of contributing to disease.

While much of this research focuses on negative emotion states, stress responses, and risk of disease, there is a growing interest in positive emotional states and in studying persons who tend frequently to experience the positive emotions of joy and happiness. As in the above examples such persons can be selected for their emotion traits using a combination of self-report techniques and in laboratory tests of brain function. Persons high in typical positive affect can then be compared to those with less positive affective states in their resistance to the effects of stress and in their long-term states of health.

The research examples listed above all depend on testing persons while they are relaxed and resting, as well as when they are under stress or perhaps in a pleasurable mood. For these reasons, it is desirable to use measurement methods that do not cause discomfort or distress. Behavioral medicine research has therefore relied on methods of psychophysiological measurement that are noninvasive or minimally invasive and cause the volunteer no discomfort. The examples above focused on the cardiovascular system which can be studied using methods such as the electrocardiogram, blood pressure monitoring, and impedance cardiography to measure pumping action of the heart and constriction of the blood vessels, and, occasionally, fluid output to assess kidney function. Stress research often uses additional methods to track responses of the endocrine system, involving collection of urine, blood, or saliva for measurement of stress hormones and other substances associated with stress and pain responses. Still, other studies examine the immune system here using minimally invasive techniques in the collection of blood for later measurement of the numbers of immune system cells and their biological activity. Closely related to these physiological measurements is the need to classify persons as to personality and temperament characteristics to establish relationships between acute stress responses or chronic allostatic responses in the lab or in daily life. These considerations call for use of interviews or paper-and-pencil measures of personality and mood states. Finally, the application of such psychophysiological techniques calls for appropriate selection of tasks and ways to analyze the data.

Cross-References

- ▶ Ambulatory Blood Pressure
- ▶ Ambulatory Monitoring
- ▶ Blood Pressure
- ▶ Blood Pressure Reactivity or Responses
- ▶ Heart Rate
- ▶ Hypertension
- ▶ Psychophysiological
- ▶ Psychosocial Factors
- ▶ Psychosocial Predictors
- ▶ Psychosocial Variables
- ▶ Stress Test
- ▶ Systolic Blood Pressure (SBP)

References and Readings

Gerin, W. (2010). Laboratory stress testing methodology. In A. Steptoe (Ed.), *Handbook of behavioral medicine: Methods and applications* (pp. 633–648). New York: Springer.
Lovallo, W. R. (2005). *Stress and health*. Thousand Oaks: Sage.
Obrist, P. A. (1981). *Cardiovascular psychophysiology: A perspective*. New York: Plenum Press.
Steptoe, A., & Poole, L. (2010). Use of biological measures in behavioral medicine. In A. Steptoe (Ed.), *Handbook of behavioral medicine: Methods and applications* (pp. 619–632). New York: Springer.
Turner, J. R. (1984). *Cardiovascular reactivity and stress: Patterns of physiological response*. New York: Plenum Press.

Psychosocial Adaptation

► Psychosocial Adjustment

Psychosocial Adjustment

Fong Chan[1], Elizabeth da Silva Cardoso[2], Julie Chronister[3] and Emma Hiatt[4]
[1]Department of Rehabilitation Psychology and Special Education, University of Wisconsin-Madison, Madison, WI, USA
[2]Department of Educational Foundations & Counseling Programs, The City University of New York-Hunter College, New York, USA
[3]Department of Counseling, San Francisco State University, San Francisco, CA, USA
[4]Rehabilitaion Psychology and Special Education, University of Wisconsin-Madison, Madison, WI, USA

Synonyms

Psychosocial adaptation; Rehabilitation psychology; Response to disability

Definition

Chronic illness and disability profoundly impacts the lives of many individuals. For example, approximately one in five Americans has physical, sensory, psychiatric, or cognitive impairments that affect their daily activities. Psychosocial adaptation entails the integration of illness or disability into the individual's life, identity, self-concept, and body image. Psychosocial adaptation is defined as the process in which a person with a disability moves from a state of disablement to a state of enablement and is characterized by the transformation from negative to positive well-being (Livneh & Antonak, 2005). Observed across disability groups, psychosocial adaptation occurs as the individual moves toward a state of optimal person-environment congruence. The final stage of psychosocial adaptation, known as adjustment, represents maximum congruence between the individual's subjective experience and his or her external environment.

Description

Introduction

Psychosocial adjustment to chronic illness and disability (CID) is a long-term, dynamic process influenced by intrinsic and extrinsic variables within a specific context (Chan, Cardoso, & Chronister, 2009; Livneh & Antonak, 2005). There are several terms used to describe the adjustment to disability process. Specifically, *adjustment, adaptation*, and *acceptance* of disability are concepts commonly used to describe the process and outcome of coping with CID. *Adaptation* has been defined as the dynamic process a person with CID experiences in order to achieve the final state of optimal person-environment congruence known as *adjustment* (Smedema, Bakken-Gillen, & Dalton, 2009). The term *acceptance* was coined by Beatrice Wright (1983) who defined *disability acceptance* as an outcome in which the disability is incorporated as part of the individual's self-concept and is accepted as non-devaluing.

Today, *response to disability* is a widely accepted terminology considered to most accurately describe the adjustment process, because it fully conveys response to disability as a subjective experience that is not necessarily negative. Furthermore, response to disability involves a dynamic process as opposed to a one-time event (Smart, 2009). The response to disability process reaches beyond psychological aspects of adjustment to include a complex and dynamic interaction of a wide variety of psychological, societal, environmental, and personal factors. This entry will provide readers with a review of the prominent psychological, social, environmental, and personal factors that interact to influence the individual process.

Psychological Factors

Somatopsychology. Psychological aspects of CID have been most broadly explored in the context of *somatopsychology*, the study of the physique's influence on behavior and how that relationship is mediated by the effectiveness of the body as a tool for actions. The emphasis of somatopsychology is on the meaning of disability that is unique to the individual, as well as the value the disability holds for other individuals in a person's life (Smedema et al., 2009). The theory considers self-concept, body image, and coping to be psychological schemas and functions crucial to successful adaptation. Specifically, *self-concept* and *body image* represent mental schemas by which humans perceive and identify themselves. An individual with CID must alter one's body image and self-concept to incorporate the physical changes into one's daily life. Therefore, reorganization of these mental schemas is critical to successful adaptation (Livneh & Antonak, 2005).

Coping. *Coping* strategies also affect the response to disability process. Coping requires the individual draw on some personal or environmental resource to reduce the negative impact of a stressor (Chronister, Johnson, & Lin, 2009), including both adaptive and maladaptive strategies. In the context of disability adjustment, coping generally refers to cognitions, emotions, or behaviors that mediate the relationship between disability-related stressors (i.e., nature, type, duration, prognosis, perception, and severity) and the response to disability. Coping strategies associated with response to disability can be divided into three psychological categories: cognitive, behavioral, and affective (Smart, 2009). *Cognitive* response refers to how an individual chooses to think about or view the disability. *Behavioral* response refers to actions taken to manage the disability, including compliance to treatment recommendations, seeking social support, returning to work, and using self-advocacy strategies to manage the impact of stigma and prejudice. *Affective* response refers to how the individual feels about the disability and how he or she manages the emotions associated with the disability (Smart, 2009). A positive coping strategy within the context of disability may include having a realistic view of the disability and awareness of limitations without exaggerating them. Conversely, a negative coping strategy may involve substance use or self-blame. The coping strategies employed strongly influence the response to disability. For example, healthy coping strategies, such as seeking out social support or redefining life goals, may improve body image and quality of life while decreasing social isolation and feelings of helplessness.

Based upon the disability acceptance theory (Wright, 1983), Wright developed a cognitive restructuring framework known as the *coping* versus *succumbing* model. In this model, a person said to be *succumbing* to disability overemphasizes negative effects of impairment and neglects the challenge for change and meaningful adaptation. Conversely, a person said to be *coping* with disability is able to focus on personal assets and is oriented to activities that are within the individual's physical capabilities (Smedema et al., 2009). Wright (1983) proposed four primary value changes that reflect the *coping* perspective including (a) *Enlargement of the scope of values*, which requires the individual recognize that values beyond those presumed lost can be achieved despite limitations of CID; (b) *Subordination of the physique* occurs when

the individual changes the cognitive belief that physical is a true representation of one's worth, desirability, or competency; (c) *Containment of disability effects* occurs when the individual recognizes disability as limited to the impact of the actual impairment, rather than being globally debilitating; and (d) *Transformation from comparative status to asset values*, which occurs when the individual avoids comparing oneself to a nondisabled standard and focuses attention on his or her assets (Smart, 2009). The four primary value changes challenge each of the previously held beliefs, resulting in acceptance of disability (Wright, 1983). Conversely, signs of the *succumbing* perspective include (a) denial or acting as if the disability does not exist; (b) idolizing normal standards or applying old values to the new situation; (c) failure to modify previous aspirations within the context new situations; (d) eclipsing behavioral possibilities and limiting one's opportunity to learn new skills to address the new situations more effectively; and (e) overcompensation of perceived deficiencies in one area by exaggerated striving in another area (Wright, 1983).

Stage model. The response to disability process has also been conceptualized as a sequence of psychological stages, similar to those experienced during grief (Smedema et al., 2009). Stage models typically describe the process of adjustment as a linear series of psychological stages, requiring the completion of previous stages before the final stage of adjustment. Many stage models have been proposed to describe stages of adjustment. Livneh and Antonak (2005) concluded that the numerous stage models may be described within these five broad categories: (a) initial impact, which includes shock and anxiety; (b) defense mobilization, which includes bargaining and denial; (c) initial realization, which includes mourning, depression, and internalized anger; (d) retaliation, which includes externalized anger or aggression; and (e) reintegration or reorganization, which includes acknowledgment, acceptance, and final adjustment. The stage models provide a structure for understanding and predicting the course and outcome of an individual's response process (Smart, 2009). Nonetheless, the applicability of the stage theory to persons with CID has been criticized for the following three reasons: (a) "stages" are not universally experienced; (b) a state of final adjustment (e.g., resolution, acceptance, assimilation) is not always achieved; and (c) psychological recovery does not follow an orderly sequence of reaction phases (Livneh & Antonak, 2005). Finally, the existence of a universal, progressive, phase-like, orderly sequence of predetermined psychosocial reactions to disability has not been adequately supported by empirical research (Livneh & Antonak).

Ecological model. The most contemporary approach to conceptualizing and understanding the response to disability process is the *ecological* approach. Considered atheoretical, this approach incorporates components of somatopsychology and stage theory, while emphasizing the interaction of personal *and* contextual variables (social/environmental and personal factors) on psychosocial adaptation (Smedema et al., 2009). This approach also adopts a comprehensive and holistic approach to outcome measurement beyond that of acceptance or adjustment to CID, encompassing a person's overarching quality of life (Livneh & Antonak, 2005). As such, understanding response to disability beyond the psychological processes described above is critical in developing a full perspective of an individual's response to disability. Below is a review of key contextual variables important to consider in the response process.

Societal Factors

Societal definitions of disability. The manner in which society defines CID influences the adjustment to process. Definitions of disability help to identify the location of the problem and who is held responsible for the solution (Smart, 2009). Four of the most popular models of disability include the (a) biomedical model; (b) environmental model; (c) functional model; and (d) sociopolitical model. The biomedical model has the longest history. This model defines disability as pathology located within the individual and represents a deviation from the norm.

Therefore, treatment is focused solely on "fixing" the individual. The environmental model suggests that the individual's environment may cause, define, or exaggerate the disability. For example, if a person with paraplegia does not have a wheelchair, then the impairment is worsened. The functional model posits that the functions of the individual influence the definition of the disability. For example, an individual who is physically active would be much more affected by mobility impairments. Finally, the sociopolitical model, also known as the Minority Group Model or the Independent Living Model, proposes that disability is not a personal attribute, but caused by society, and thus society should bear the responsibility for dealing with disability (Smart, 2009). *Self-advocacy* is a critical component of the sociopolitical model of disability. Self-advocacy is rooted in the American ideals of autonomy and self-determination. In contrast to consequences of the medical model, including dependency, marginality, and social exclusion, self-advocacy refers to persons with disabilities taking control of their own lives, speaking up for themselves, being in control of their own resources, and having the right to make life decisions without undue influence or control from others.

The World Health Organization International Classification of Functioning, Disability and Health (WHO, 2001) is a contemporary, biopsychosocial approach to defining disability that considers the biomedical, environmental, functional, and social models in its explanation of disability. This model conceptualizes disability along five major domains: (a) body functions and structures; (b) activities; (c) participation; (d) personal factors; and (e) environmental factors (Chan, Gelman, Ditchman, Kim, & Chiu, 2009). The ICF recognizes disability as an interaction between all of these factors and cannot be defined apart from the individual's context.

Societal attitudes. Negative attitudes toward persons with disabilities are well documented in the literature (Chan, Livneh, Pruett, Wang, & Zheng, 2009). Negative attitudes or unfavorable evaluative statements related to a person, object, or event are considered *invisible barriers* that arise from the environment and impact the response process by limiting opportunities, access, and help-seeking behaviors, as well as reducing overall quality of life (Chan, Livneh et al., 2009). Related concepts include *prejudice, discrimination*, and *stigma*. *Prejudice* is a negative generalization toward a group of people and the assumption that an individual belonging to that group has the characteristics based on the generalization. For example, "all persons with CID are intellectually inferior." *Discrimination* is the action carried out based upon prejudice. For example, an employer who does not hire a person with CID because he or she believes persons with CID are "unsafe." Finally, *stigma* is a term that encompasses the problems associated with stereotyping, prejudice, and discrimination; it is the chain of events resulting from negative attitudes and beliefs, resulting in discrimination. Persons with disabilities often have limited access to work, housing, and other community resources because of stigmatizing attitudes that have led to discriminatory behavior (Chan, Livneh et al.).

Commonly cited sources of negative attitudes and stereotypical views regarding persons with CID include the *safety threat*, the *ambiguity of disability*, the *salience of the disability, spread or overgeneralization*, *moral accountability for the cause and management of the disability*, *inferred emotional consequences of the disability*, and the *fear of acquiring a disability* (Smart, 2009). Cook (1998) indicated that the general public also exhibits a "hierarchy of preferences" for specific groups of persons with CID; for example, people hold more favorable attitudes toward persons with physical disabilities than individuals with mental disabilities and persons tend to have more positive attitudes toward persons with intellectual disabilities than those with psychiatric disabilities.

Environmental Factors

A large majority of persons with CID live at or below the poverty level (Smart, 2009). Although most persons with CID report they want to work (Louis Harris Associate Inc. Polls, 1994), and despite protections afforded by the Americans with Disabilities Act, persons with CID have

greater difficulty finding or maintaining work, and unemployment and underemployment rates of persons with disabilities are high (Smart, 2009). This is due in part to prejudice and discrimination, worksite inaccessibility, and financial disincentives built into many disability benefits programs (e.g., SSI/SSDI). Other environmental factors that influence the response process include limited mobility and access to transportation, architectural barriers, frequency and duration of hospitalizations, lack of institutional support (medical services, educational programs and technological supports; political and religious groups), poor living conditions, limited availability of job opportunities, and inadequate accessibility of worksites (Livneh & Antonak, 2005).

Personal Factors

Age and developmental status. Age and developmental stage, such as those identified by Erikson (1968), interact with CID to influence the response process. For example, the process of adaptation differs significantly between individuals born with CID and those who acquire CID later in life. Developmentally speaking, for an *infant* with a congenital disability, the primary task of establishing trust in the world through the relationship with mother or primary caregiver may be compromised if the infant is hospitalized for long periods and is cared for by multiple health professionals (Smart, 2009), whereas during *early adulthood*, the tasks of establishing a family and beginning a career are important and may be impacted by CID. For *older adults*, the resulting impact of CID on independence and functioning is variable, ranging from minimal impact to substantial lifestyle change. While functional loss and disability may be a normal part of aging for some, social isolation, dependence, restricted activities and participation, and the loss of loved ones surrounding the individual can impact quality of life. Indeed, with disability, older adults face multiple life transitions in later years of life.

Gender. Gender is important to consider in the response process. Patterson, DeLaGarza, and Schaller (2005) suggest that men and women respond differently to various CIDs. For example, in the area of spinal cord injury, men experience greater difficulties related to sexual functioning than women, resulting in feelings of loss of "manhood" or "masculinity." With respect HIV/AIDS, women are more socially isolated than men because of cultural backgrounds commonly associated with females with HIV/AIDS, and because rates of transmission are lower and occur through a different mode. Furthermore, a woman with HIV/AIDS experiences issues related to pregnancy, including potential transmission from mother to infant. Women and men also respond differently following myocardial infarction, such that men return to work sooner and are more likely to participate in physical activity to cope with stressors related to the condition.

Culture. *Culture* involves the beliefs, customs, practices, social behaviors, and set of attitudes of a particular nation or group of people (Chronister & Johnson, 2009), and *worldview* is the framework of ideas and beliefs through which an individual interprets the world and interacts with it according to their philosophy, values, emotions, and ethics. Culture and worldview inform how disability is defined and experienced, and both aspects contribute to the response to disability process. For example, in contrast to the explanation of disability perpetuated by the medical model, some cultures perceive the origin of disability to be of the metaphysical-spiritual realm. For other cultures, disability is a condition that should not be altered, because it is considered to be predetermined by fate and not amenable to adaptive intervention. For example, the deaf culture rejects the notion that group members have a disability and identify as individuals with a different communication modality that live in a hearing world. Furthermore, the number of persons from culturally diverse backgrounds in the USA with CID is disproportionate, and these individuals are often at risk for experiencing multiple negative experiences or "double" discrimination and stigmatization, barriers that are likely to influence response to CID (Chronister & Johnson, 2009).

Disability. Disability characteristics are also important to consider in the adjustment process and vary significantly across and within

conditions. According to Smart (2009), there are ten disability factors that influence response to disability including time of onset (congenital, acquired), type of onset (insidious versus acute), course of disability (direction, pace of movement, degree of predictability), functions impaired (meaning of functioning, degree of intrusiveness, residual functioning and assistive technology), severity of the disability (number of disabilities experienced and areas of functioning affected, treatment necessary, and degree of stigma directed at the individual), visibility of the disability, degree of disfigurement, and prognosis (what is expected for the future).

Sexual orientation/identity. Sexual orientation is an important factor to consider in the response to disability process. Persons with CID that are identified with sexual orientations, such as lesbian, gay, bisexual, transgender, queer, questioning, intersex, and asexual (LGBTQQIA), are vulnerable to stereotypes, bigotry, abuse, bullying, and violence. They are subject to a complex array of prejudices by the mainstream population, including double prejudice for equal rights, higher rates of marginalization and discrimination, confusion in navigating identity development, and the prevailing view that persons with disability are asexual or unsuitable sexual partners (Miville, Romero, & Corpus, 2009).

Conclusion

Response to CID involves a complex and dynamic interaction of psychological, social, environmental, and personal variables. Somatopsychology and stage models provide a foundation for understanding the psychological processes of disability adjustment. The ecological approach builds upon this work to facilitate a broader conceptualization of psychosocial adjustment to address the importance of additional personal and contextual variables. Indeed, response to CID is a unique, personal experience that must be considered within the individual's context, including the sociocultural influences that contribute to how society defines and responds to CID. Contemporary definitions of disability conceptualize disability from a biopsychosocial perspective (i.e., WHO ICF model), recognizing body functioning and contextual factors as critical to disability determination and intervention planning. This framework, coupled with the ecological approach to adjustment to disability, replaces traditional approaches to yield a more holistic picture of adaptation.

Acknowledgments The contents of this entry were developed with support through the *Rehabilitation Research and Training Center on Effective Vocational Rehabilitation Service Delivery Practices* (EBP-VR-RRTC) established at both the University of Wisconsin-Madison and the University of Wisconsin-Stout under a grant from the Department of Education, National Institute on Disability and Rehabilitation Research (NIDRR) grant number PR# H133B100034. However, those contents do not necessarily represent the policy of the Department of Education, and endorsement by the Federal Government should not be assumed.

Cross-References

▶ Attitudes
▶ Chronic Disease or Illness
▶ Coping
▶ Depression
▶ Disability
▶ Health Disparities

References and Readings

Chan, F., Cardoso, E., & Chronister, J. A. (2009). *Understanding psychosocial adjustment to chronic illness and disability: A handbook for evidence-based practitioners in rehabilitation*. New York: Springer Publishing.

Chan, F., Gelman, J. S., Ditchman, N. M., Kim, J. H., & Chiu, C. Y. (2009). The World Health Organization ICF model as a conceptual framework of disability. In F. Chan, E. Cardoso, & J. Chronister (Eds.), *Psychosocial interventions for people with chronic illness and disability: A handbook for evidence-based rehabilitation health professionals*. New York: Springer.

Chan, F., Livneh, H., Pruett, S., Wang, C.-C., & Zheng, L. X. (2009). Societal attitudes towards disability: Concepts, measurements, and interventions. In F. Chan, E. Da Silva Cardoso, & J. A. Chronister (Eds.), *Understanding psychosocial adjustment to chronic illness and disability: A handbook for*

evidence-based practitioners in rehabilitation (pp. 333–367). New York: Springer.
Chronister, J., & Johnson, E. (2009). Multiculturalism and adjustment to disability. In F. Chan, E. Da Silva Cardoso, & J. A. Chronister (Eds.), *Understanding psychosocial adjustment to chronic illness and disability: A handbook for evidence-based practitioners in rehabilitation* (pp. 479–528). New York: Springer.
Chronister, J., Johnson, E., & Lin, C. P. (2009). In F. Chan, E. Da Silva Cardoso, F. Chan, E. Da Silva Cardoso, & J. A. Chronister (Eds.), *Understanding psychosocial adjustment to chronic illness and disability: A handbook for evidence-based practitioners in rehabilitation* (pp. 111–148). New York: Springer.
Cook, D. (1998). Psychosocial impact of disability. In R. M. Parker & E. M. Szymanski (Eds.), *Rehabilitation counseling: Basics and beyond* (3rd ed., pp. 303–326). Austin, TX: Pro-Ed.
Erikson, E. H. (1968). Identity: Youth and crisis. New York: Norton.
Livneh, H., & Antonak, R. F. (2005). Psychosocial aspects of chronic illness and disability. In F. Chan, M. J. Leahy, & J. Saunders (Eds.), *Case management for rehabilitation health professionals* (2nd ed., Vol. 2, pp. 3–43). Osage Beach, MO: Aspen Professional Services.
Louis Harris Associate Inc. Polls. (1994). *N.O.D.L. Harris survey of disabled Americans*. Washington, DC: National Organization of Disability.
Miville, M. L., Romero, L., & Corpus, M. J. (2009). Incorporating affirming, feminist and relational perspectives: The case of Juan. In M. E. Gallardo & B. W. McNeil (Eds.), *Intersections of multiple identities: A casebook of evidence-based practices with diverse populations* (pp. 175–201). New York: Routledge.
Patterson, J. B., DeLaGarza, D., & Schaller, J. (2005). Rehabilitation counseling practice: Considerations and interventions. In R. M. Parker, E. M. Szymanski, & J. B. Patterson (Eds.), *Rehabilitation counseling, basics and beyond* (4th ed., pp. 155–186). Austin, TX: ProEd.
Smart, J. (2009). *Disability, society, and the individual* (2nd ed.). Austin, TX: Pro-Ed.
Smedema, S. M., Bakken-Gillen, S. K., & Dalton, J. (2009). Psyhosocial adaptation to chronic illness and disability: Models and measurement. In F. Chan, E. Da Silva Cardoso, & J. A. Chronister (Eds.), *Understanding psychosocial adjustment to chronic illness and disability: A handbook for evidence-based practitioners in rehabilitation* (pp. 51–73). New York: Springer.
World Health Organization. (2001). *International classification of functioning, disability, and health: ICF*. Geneva: World Health Organization. Retrieved November 28, 2008, from http://www.who.int/classification/icf
Wright, B. A. (1983). *Physical disability: A physical approach* (2nd ed.). New York: Harper and Row.

Psychosocial Aspects

► Psychosocial Characteristics

Psychosocial Characteristics

Adriana Dias Barbosa Vizzotto[1], Alexandra Martini de Oliveira[2], Helio Elkis[3], Quirino Cordeiro[4] and Patrícia Cardoso Buchain[1]
[1]Occupational Therapist of the Occupational Therapy Service, Institute of Psychiatry, Hospital das Clínicas University of São Paulo Medical School, São Paulo, SP, Brazil
[2]Institute of Psychiatry – Hospital das Clínicas University of São Paulo Medical School, São Paulo, SP, Brazil
[3]Department and Institute of Psychiatry, University of São Paulo Medical School, São Paulo, SP, Brazil
[4]Department of Psychiatry and Psychological Medicine, Santa Casa Medical School, São Paulo, SP, Brazil

Synonyms

Psychosocial aspects; Psychosocial factors

Definition

Psychosocial characteristics is a term used to describe the influences of social factors on an individual's mental health and behavior.

Description

A psychosocial approach to human behavior involves the relation between intrapersonal psychological and environmental aspects. Psychosocial characteristics is commonly described as an individual's psychological development in relation to his/her social and cultural

environment. "Psychosocial" means "pertaining to the influence of social factors on an individual's mind or behavior, and to the interrelation of behavioral and social factors" (Oxford English Dictionary, 2012). Psychosocial factors, at least in the context of health research, can be defined as the mediation of the effects of social structural factors on individual health, conditioned and modified by the social structures contexts in which they exist (Martikainen, Bartley, & Lahelma, 2002). These statements raise the question of what the relevant broader social structural forces are, and how such forces might influence health through their effects on individual features.

Individual psychological and social aspects are related to individual's social conditions, mental and emotional health. For example, the main factors that influence children's mental health are the social and psychological environment (Halpen & Figueiras, 2004).

Environmental factors play an important role in the etiology of emotional problems in childhood. The cumulative risk effects from a "vulnerable environment," e.g., negligence, poverty, drug abuse, are more important in determining emotional problems in children than the presence of one single stressor, regardless of its magnitude. According to Barylnik (2003), the analysis of the characteristics of a juvenile delinquent's sample showed a high rate of psychiatric disorder and social phobia, alcoholism, organic brain dysfunctions, low intelligence quotients, and behavior problems.

The term "psychosocial" is widely used for determining an individual's health outcome. Psychological and social factors expressed as thoughts, expressive emotions, and behaviors are significant for human functioning and the occurrence of disease. Mentally healthy people tend to react in positive ways to negative situations, compared to emotional unstable people, who react negatively to similar situations. Hence, irrational thoughts may be a sign of bad psychosocial health. Therefore, for psychosocial instable people it is preferable to have special social bonds with and social support from other people. On the other hand, prejudice from others is often a result of poor psychosocial health that causes poor social relations.

Hence it is understood that health is better understood in terms of a combination of biological, psychological, and social factors rather than only in biological terms (Santrock, 2007). This is in contrast to the traditional and reductionist biomedical model of medicine which suggests that every disease process can be explained in terms of an underlying deviation from normal function such as a pathogen, genetic or developmental abnormality or injury (Engel, 1977).

The World Health Organization's (WHO, 2001) definition of health is "a state of complete physical mental and social well-being, and not merely the absence of disease and infirmity." This WHO definition has been criticized because cause and effect is mixed. The concept of "psychosocial health," in some cases, may combine traditional medical definitions of disease and infirmity with measures that reflect individual responses to disease and even in some cases indicators of the social context itself. However, such measures have merit in recognizing individuals' experiences and quality of life, meaning an important outcome of individual's psychosocial condition (Martikainen et al., 2002).

Cross-References

▶ Psychosocial Impact

References and Readings

Barylnik, J. (2003). Psychopathology, psychosocial characteristics, and family environment in juvenile delinquents. *The German Journal of Psychiatry, 6*, 30–32. http://www.gjpsy.uni-goettingen.de. ISSN 1433-1055.

Engel, G. L. (1977). The need for a new medical model: A challenge for biomedicine. *Science, 196*, 129–136.

Halpen, R., & Figueiras, A. C. M. (2004). Environmental influences on child mental health. *Journal of Pediatrics, 80*(2 Suppl), S104–S110.

Martikainen, P., Bartley, M., & Lahelma, E. (2002). Psychosocial determinants of health in social

epidemiology. *International Journal of Epidemiology, 31*, 1091–1093.
OED Online. (2012). Oxford University Press. Dictonary on line http://www.oed.com/. Retrieved 20120429.
Santrock, J. W. (2007). *A topical approach to human life-span development* (3rd ed.). St. Louis: McGraw-Hill.
World Health Organization (WHO). (2001). *The mental health report 2001: Mental health, new understanding hope*. Genebra: Author.

Psychosocial Factors

Jane Upton
School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Psychological factors; Psychosocial variables

Definition

Social factors include general factors at the level of human society concerned with social structure and social processes that impinge on the individual. Psychological factors include individual-level processes and meanings that influence mental states. Sometimes, these words are combined as "psychosocial." This is shorthand term for the combination of psychological and social, but it also implies that the effect of social processes are sometimes mediated through psychological understanding (Stansfeld & Rasul, 2007).

Description

The relationship between psychological factors and the physical body can be influenced by social factors, the effects of which are mediated through psychological understanding. Examples of psychosocial factors include social support, loneliness, marriage status, social disruption, bereavement, work environment, social status, and social integration. To illustrate that the role psychosocial factors can play in physical disease, this entry will focus on the relationship between social support and mortality.

In 1979, Berkman and Syme conducted a prospective study to investigate the relationship between social support and mortality (Berkman & Syme, 1979). The study included 6,928 adults from the general population of Alameda County, California. They recorded four sources of social contact: marriage, contacts with close friends and relatives, church membership, and informal and formal group associations; mortality was followed-up 9 years later. They found that respondents with each type of social tie had lower mortality rates than respondents lacking such connections. From these four variables, they then constructed a Social Network Index, which weighted intimate contacts more heavily than more superficial ones. Using this index, they found a consistent pattern of increased mortality rates with each decrease in social connection. Men who were the most isolated had an age-adjusted mortality rate 2.3 times higher than men with the most connections. This relationship was independent of self reported physical health status, year of death, socioeconomic status, and such health behaviors as smoking, alcohol ingestion, physical inactivity, obesity, and low utilization of preventive health services or health practices.

The effects of level of social support on mortality rates were investigated by Rosengren and colleages (Rosengren, Orth-Gomer, Wedel, & Wilhelmsen, 1993). They invited half of all men in Gothenburg who were born in 1933 (then 50 years old) to have a health examination and complete a measures of emotional support and social integration. Seventy-six percent responded. These men were then followed-up after 7 years, and mortality ascertained. Their data indicated that emotional support may attenuate the impact of adverse life events, possibly by strengthening the psychobiological resistance to stress.

Frasure-Smith and colleagues found in their study of 887 post-myocardial infarction (MI)

patients that depression, but not social support, was directly related to mortality (Frasure-Smith et al., 2000). However, very high levels of support protected patients from the negative prognostic consequences of depression. They found that three different measures of social support independently improved depression: higher scores on a measure of perceived social support, a greater number of close friends and relatives, and living with other people. They suggest that clinicians should ascertain patient's views of their social support in their assessments of post-MI depression.

Differences in health status between people with differing levels of social integration cannot simply be attributed to differences in health behaviors between the two groups. In two parallel studies, Cacioppo and colleagues investigated four mechanisms by which loneliness (a discrepancy between their desired and actual relationships) may negatively impact on morbidity and mortality (Cacioppo et al., 2002): (1) poorer health behaviors than nonlonely individuals, (2) altered cardiovascular activation, (3) elevated levels of hypothalamic pituitary adrenocortical activation, and (4) poorer sleep quality. Participants were 89 undergraduate students, and in a second study, 25 older adults (age range 53–78 years). They found that the health behaviors of lonely and nonlonely participants were similar. However, cardiovascular function differed between these groups in both the younger and older participants. They speculated that the differences they found in the hemodynamic function observed in the younger subjects may contribute to elevated blood pressure in lonely older adults. They also found that younger and older lonely adults suffered lower quality sleep, possibly leading to diminished health and well-being.

The plethora of research in this area, measuring different aspects of social relationships, led to uncertainty as to which aspect increased the risk of mortality. A recent meta-analytic review of 148 studies has revealed that stronger social relationships increased the likelihood of survival by 50% (Holt-Lunstad, Smith, & Layton, 2010). They found that complex measures of social integration were better predictors of mortality than binary indicators of residential status (e.g., living alone or with others). This is partly because living in a negative social relationship can increase risk of mortality. However, they also caution against assuming causation, due to the difficulty of conducting randomized controlled trials to investigate this topic. They conclude that the data they present makes a compelling case for social relationship factors to be viewed as an important risk factor alongside factors such as smoking, diet, and exercise.

Cross-References

- ▶ Acute Myocardial Infarction
- ▶ Bereavement
- ▶ Cardiovascular Risk Factors
- ▶ Daily Stress
- ▶ Loneliness
- ▶ Psychological Factors and Health
- ▶ Psychosocial Characteristics
- ▶ Social Support

References and Readings

Berkman, L., & Syme, S. (1979). Social networks, host resistance, and mortality: A nine-year follow-up study of alameda county residents. *American Journal of Epidemiology, 109*(2), 186–204.

Cacioppo, J., Hawkley, L., Crawford, L., Ernst, F., Burleson, M., Kowalewski, R., et al. (2002). Loneliness and health: Potential mechanisms. *Psychosomatic Medicine, 64*, 407–417.

Frasure-Smith, N., Lesperance, F., Gravel, G., Masson, A., Juneau, M., Talajic, M., et al. (2000). Social support, depression, and mortality during the first year after myocardial infarction. *Circulation, 101*, 1919–1924.

Holt-Lunstad, J., Smith, T., & Layton, J. (2010). Social relationships and mortality risk: A meta-analytic review. *PLoS Medicine, 7*(7). doi:10.1371/journal.pmed.1000316.

Rosengren, A., Orth-Gomer, K., Wedel, H., & Wilhelmsen, L. (1993). Stressful life events, social support, and mortality in men born in 1933. *British Medical Journal, 307*, 1102–1105.

Stansfeld, S., & Rasul, F. (2007). Psychosocial factors, depression and illness. In A. Steptoe (Ed.), *Depression and physical illness* (pp. 19–52). Cambridge: Cambridge University Press.

Psychosocial Factors and Traumatic Events

Shin-ichi Suzuki[1] and Yuko Takei[2]
[1]Faculty of Human Sciences, Graduate School of Human Sciences, Waseda University, Tokorozawa-shi, Saitama, Japan
[2]Faculty of Medicine, University of Miyazaki Hospital, Miyazaki-shi, Japan

Synonyms

Interpersonal relationships; Psychosocial stress; Stressful life event

Definition

Psychosocial factors are influences that affect a person psychologically or socially. There are multidimensional constructs encompassing several domains such as mood status (anxiety, depression, distress, and positive affect), cognitive behavioral responses (satisfaction, self-efficacy, self-esteem, and locus of control), and social factors (socioeconomic status, education, employment, religion, ethnicity, family, physical attributes, locality, relationships with others, changes in personal roles, and status).

Description

Psychosocial Factors in Everyday Life

Psychosocial factors and influences differ across individuals and may contribute to the development or aggravation of mental and physical disorders. Previous studies have indicated that depression, social isolation, and behavioral escape-avoidance coping were associated with the risk of mortality for cancer patients (Falagas et al., 2007) and patients with cardiac disease (Rozanski, Blumenthal, & Kaplan, 1999). A serious loss (bereavement, divorce, and disability), relationship problems, work stress, family crisis, financial setback, or any unwelcome life change can trigger depressive disorders (Meltzer, Gill, & Petticrew, 1995). Furthermore, those disorders may negatively impact some psychosocial factors. For example, depressive disorders substantially reduce a person's ability to work effectively and personal and family income and increase the probability of unemployment (Ormel et al., 1999).

Psychosocial Factors in Natural and Technological Disasters

Repeated disasters in Japan such as the Great Hanshin Earthquake and the Great East Japan Earthquake showed us the importance of these psychosocial factors in life events. Furthermore, the Great East Japan Earthquake caused not only death and destruction but also secondary disasters such as the Fukushima nuclear accident. The severity of these natural and technological disasters (e.g., the extent of death and destruction, the length of exposure, evacuation, proximity to the epicenter, and contradictory media reports about the health effects of radiation) are likely to have an adverse impact on victims' mental health (Bromet, Havenaar, & Guey, 2011).

In contrast, positive psychosocial factors in life events such as connectedness to others, the spirit of patience, politeness, and mutual aid may contribute to prevent an aggravation of the situation and change things for the better. In The Great Hanshin Earthquake and The Great East Japan Earthquake, Japanese people have handled their grief and loss and overcome numerous difficulties with mutual cooperation and the spirit of patience. These psychosocial factors provide us the energy to recover from severe life events.

Cross-References

▶ Cardiovascular Disease
▶ Depression: Symptoms
▶ Life Events
▶ Religion/Spirituality
▶ Self-esteem
▶ Socioeconomic Status (SES)

References and Readings

Bernard, L. B. (1988). *Health psychology: A psychosocial perspective*. Englewood Cliffs, NJ: Prentice Hall.
Bromet, E. J., Havenaar, M., & Guey, L. T. (2011). A 25 year retrospective review of the psychological consequences of the Chernobyl accident. *Clinical Oncology, 23*, 297–305.
Davison, L. M., Weiss, L., O'Keefe, M., & Baum, A. (1991). Acute stressors and chronic stress at Three Mile Island. *Journal of Traumatic Stress, 4*, 481–493.
Falagas, M. E., Zarkadoulia, E. A., Ioannidou, E. N., Peppas, G., Christodoulou, C., & Rafailidis, P. I. (2007). The effect of psychosocial factors on breast cancer outcome: A systematic review. *Breast Cancer Research, 9*(4), R44. doi: 10.1186/bcr1744.
Meltzer, H., Gill, B., & Petticrew, M. (1995). The prevalence of psychiatric morbidity among adults living in private households. *OPCS surveys of psychiatric morbidity, Report 1*. London: HMSO.
Ormel, J., Von Korff, M., Oldehinkel, T., Simon, G., Tiemens, B. G., & Ustrun, T. B. (1999). Onset of disability in depressed and non-depressed primary care patients. *Psychological Medicine, 29*, 847–853.
Rozanski, A., Blumenthal, J. A., & Kaplan, J. (1999). Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation, 99*, 2192–2217.

Psychosocial Impact

Alexandra Martini de Oliveira[1], Patrícia Cardoso Buchain[2], Adriana Dias Barbosa Vizzotto[2], Helio Elkis[3] and Quirino Cordeiro[4]
[1]Institute of Psychiatry – Hospital das Clínicas University of São Paulo Medical School, São Paulo, SP, Brazil
[2]Occupational Therapist of the Occupational Therapy Service, Institute of Psychiatry, Hospital das Clínicas University of São Paulo Medical School, São Paulo, SP, Brazil
[3]Department and Institute of Psychiatry, University of São Paulo Medical School, São Paulo, SP, Brazil
[4]Department of Psychiatry and Psychological Medicine, Santa Casa Medical School, São Paulo, SP, Brazil

Synonyms

Psychological and social effects

Definition

Psychosocial impact is defined as the effect caused by environmental and/or biological factors on individual's social and/or psychological aspects.

Several psychiatric disorders may affect psychological and social aspects of individual's lives. Examples are (a) obsessive-compulsive disorder (OCD), whereas these patients might present social marital disabilities, problems related to occupations and low income (Vikas, Avasthi, & Sharan, 2011), (b) people with cancer, who experienced negative psychological effect such as bad feelings and fears, as well as moderate to high levels of anxiety and psychological distress (Primo et al., 2000), (c) traumatic events such disasters, urban violence, and expose of terrorism may also impact on present psychosocial status (Eisenman et al., 2009). Natural disasters, like flooding, have been reported to cause a wide range of psychosocial impacts, leading the victims to present psychiatric symptoms (Paranjothy et al., 2011). For example, after the tsunami in Southern Thailand and Hurricane Katrina in the USA caused high levels of posttraumatic stress disorder (PTSD) among the victims. The prevalence of mental health disorders has been significantly higher among individuals who experienced floodwater in their houses compared to individuals who did not face this type of personal experience (Paranjothyet al., 2011). Individuals who are victims of these environmental phenomena might need more substantial and sometimes sustained intervention (de Zulueta, 2007).

Cross-References

▶ Psychosocial Characteristics

References and Readings

de Zulueta, C. F. (2007). Mass violence and mental health: Attachment and trauma. *International Review of Psychiatry, 19*, 221–233.

Eisenman, D. P., Glik, D., Ong, M., Zhou, Q., Tseng, C. H., Long, A., et al. (2009). Terrorism-related fear and avoidance behavior in a multiethnic urban population. *American Journal of Public Health, 99*, 168–174.

Paranjothy, S., Gallacher, J., Amlôt, R., Rubin, G. J., Page, L., Baxter, T., et al. (2011). Psychosocial impact of the summer 2007 floods in England. *BMC Public Health, 11*, 145.

Primo, K., Compas, B. E., Oppedisano, G., Howell, D. C., Epping-Jordan, J. E., & Krag, D. N. (2000). Intrusive thoughts and avoidance in breast cancer: Individual differences and associations with psychological distress. *Psychology and Health, 14*, 1141–1153.

Vikas, A., Avasthi, A., & Sharan, P. (2011). Psychosocial impact of obsessive-compulsive disorder on patients and their caregivers: A comparative study with depressive disorder. *The International Journal of Social Psychiatry, 57*, 45–56.

Psychosocial Implications

▶ Genetic Testing, Psychological Implications

Psychosocial Intervention

▶ Cancer: Psychosocial Treatment

Psychosocial Oncology

▶ Cancer: Psychosocial Treatment

Psychosocial Predictors

Joanna Long and Jennifer Cumming
School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Health behavior predictors; Psychological predictors

Definition

Psychosocial variables which act as predictors either of other psychosocial variables or behaviors, cognitions, risk, severity, mortality, or a number of other factors which may relate to behavioral medicine research, such as health outcomes.

Description

Psychosocial variables encompass both the social and psychological aspects of someone's life and cover a broad range of both positive and negative factors often measured in behavioral medicine research. Social factors include quality of life, health behaviors (alcohol consumption, smoking status, drug use), physical activity level, and socioeconomic status, whereas personal factors include depressive symptoms, perceived stress levels, anxiety, and mood (see ▶ Psychosocial Variables). Psychosocial variables often interrelate and can be used to predict behavioral and/or health outcomes. These variables also act as risk factors for mental health and chronic conditions, such as rheumatoid arthritis, HIV, gastrointestinal disorders, and Parkinson's disease, among many others. For these reasons, psychosocial predictors are important to assess when investigating cofactors of disease and targeting interventions within a population.

Within cross-sectional research, psychosocial predictors may correlate with other psychosocial variables or the behavior and health outcomes investigated. Examples of cross-sectional studies using psychosocial predictors within behavioral medicine research includes Ng and Jeffery (2003) who found an inverse relationship between perceived stress and exercise, and Blair and Church (2004) who investigated the link between physical activity status with obesity and health behaviors, such as smoking and diet. Although this design provides immediate research, it is limited to a particular time point for assessing relationships that may vary over time. Alternatively, longitudinal designs are used to study

psychosocial predictors in a group of individuals over a period of time, perhaps for many years. This may permit researchers to predict future behaviors, risk, and health outcomes in research. For example, Leserman et al. (1999) investigated the relationship between perceived stress and AIDS diagnosis in HIV patients over 5.5 years. Also, Whooley et al. (2008) investigated the relationship between depressive symptoms and health behaviors such as physical activity levels, smoking status, and alcohol consumption with risk of cardiovascular events over 5 years in patients with existing coronary heart disease. Taking assessments at multiple time points permits researchers to examine whether changes in psychosocial factors relate to long-term disease or condition changes. Compared to cross-sectional research, a longitudinal study also provides a more accurate idea of the direction of the predictor's relationship. However, cross-sectional and longitudinal studies cannot infer causation or show the direction of the relationship.

Cross-References

▶ Psychosocial Variables

References and Readings

Blair, S. N., & Church, T. S. (2004). The fitness, obesity, and health equation: Is physical activity the common denominator? *Journal of the American Medical Association, 292*(10), 1232–1234.

Leserman, J., Jackson, E. D., Petitto, J. M., Golden, R. N., Silva, S. G., Perkins, D. O., et al. (1999). Progression to AIDS: The effects of stress, depressive symptoms, and social support. *Psychosomatic Medicine, 61*, 397–406.

Ng, D. M., & Jeffery, R. W. (2003). Relationships between perceived stress and health behaviors in a sample of working adults. *Health Psychology, 22*(6), 638–642.

Whooley, M. A., de Jonge, P., Vittinghoff, E., Otte, C., Moos, R., Carney, R. M., et al. (2008). Depressive symptoms, health behaviors, and risk of cardiovascular events in patients with coronary heart disease. *Journal of the American Medical Association, 300*(20), 2379–2388.

Psychosocial Stress

▶ Psychosocial Factors and Traumatic Events
▶ Trier Social Stress Test

Psychosocial Traits

▶ Character Traits

Psychosocial Variables

Joanna Long and Jennifer Cumming
School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Health behavior variables; Psychological variables

Definition

Variables encompassing psychological and social factors.

Description

The term "psychosocial" has a broad meaning when considering health research and social epidemiology. It is formed from two words: psychological and social. Psychological factors can be positive, such as happiness, affect, and vitality, or negative, such as anxiety, perceived stress, and depressive symptoms. These can also be split to distinguish between trait and state aspects. Personality traits, depressive factors, well-being, quality of life, and the impact of

significant life events and trauma are less likely to fluctuate on a day to day basis (i.e., more trait-like or stable variables), whereas anxiety, perceived stress, mood, affect, happiness, and vitality are more unstable (i.e., more state-like). Furthermore, cognitive, behavioral, and affective facets within psychosocial factors can be identified. For example, someone may think about taking up smoking, and subsequently begin smoking, which in turn may lower perceived stress levels.

Social factors involve the relationship a person has with their environment, such as their age, sex, ethnicity, level and perception of social support, socioeconomic status, neighborhood factors, family history, and health behaviors. The environment can also promote or hinder whether individuals engage in positive or negative health behavior. For example, certain behavioral factors, such as the likelihood of exercise participation, smoking, alcohol intake, and drug abuse may be influenced by the physical environment. If an individual lives within a community that is safe and accessible, for example, they may be more likely to engage in high levels of outdoor physical activity, such as walking around the neighborhood.

Psychosocial variables therefore encompass a large range of factors relating to an individual's psychological state and social environment and potentially have either positive and negative consequences for health and behavioral outcomes (see ▶ Psychosocial Predictors). These variables are also important to consider when investigating either the risk, or progression, of an illness or disease. For example, high perceived stress levels, anxiety, and depression may accelerate progression of HIV or coronary heart disease (Barefoot et al., 1996; Leserman, 2008). Similarly, understanding of these variables allows researchers to examine developmental processes, such as healthy aging or the effects of a long-term intervention within the population.

There are two main ways of measuring psychosocial variables. Administering questionnaires is the most common method used in research. For example, the Perceived Stress Scale (PSS) (Cohen et al., 1983) assesses the degree to which situations in one's life are perceived as stressful, whereas the Centre for Epidemiological Studies Depression Scale (CES-D) (Kohout et al., 1993) measures current level of depressive symptomatology. The items making up the questionnaire are often summed together to create an overall score for the variable being measured (e.g., depression) and can be compared to norms generated for clinical and general populations. For example, the CES-D questionnaire has a range between 0 and 30, with a score of 10 or more indicating possible clinical depression. It is important that the questionnaires used are reliable, valid, and specific to the population which is being studied. Alternatively, researchers may choose to use structured or semi-structured interviews to assess psychosocial variables. A structured interview involves asking respondents a predetermined and limited number of questions about a specific topic, whereas a semi-structured interview is more flexible and allows new questions to be brought up or emerging topics to be explored. Interviews provide a qualitative aspect to the research, which is often used to complement the quantitative data provided by questionnaires when taking a mixed-methods approach.

Cross-References

▶ Psychosocial Factors
▶ Psychosocial Predictors

References and Readings

Barefoot, J. C., Helms, M. J., Mark, D. B., Blumenthal, J. A., Califf, R. M., Haney, T. L., O'Connor, C. M., Siegler, I. C., Williams, R. B. (1996). Depression and long term mortality risk in patients with coronary heart disease. *American Journal of Cardiology 78*, 613–617.

Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behaviour, 24*, 385–396.

Kohout, F. J., Berkman, L. F., Evans, D. A., & Cornoni-Huntley, J. (1993). Two shorter forms of the CES-D depression symptoms index. *Journal of Aging and Health, 5*, 179–193.

Leserman, J. (2008). Role of depression, stress and trauma in HIV disease progression. *Psychosomatic Medicine 70*, 539–545.

Psychosocial Work Environment

Karen Jacobs[1], Miranda Hellman[2], Jacqueline Markowitz[2] and Ellen Wuest[2]
[1]Occupational Therapy, College of Health and Rehabilitation Science, Sargent Collage, Boston University, Boston, MA, USA
[2]Boston University, Allston, MA, USA

Synonyms

Mental strain; Physical illness; Psychological and social conditions people experience in the workplace; Stress and occupational health

Definition

Psychosocial work environment pertains to interpersonal and social interactions that influence behavior and development in the workplace. Research has been conducted to determine the effects of the psychosocial work environment on stress levels and overall health. One study in particular found that low levels of support and control at work leads to increased rates of sickness absence (North, Syme, Feeney, Shipley, & Marmot, 1996). In other words, a positive and supportive psychosocial work environment is beneficial to employees in an occupational organization.

Cross-References

- ▶ Job Demand/Control/Strain
- ▶ Positive Affectivity
- ▶ Positive Psychology
- ▶ Psychological Factors
- ▶ Psychosocial Characteristics
- ▶ Psychosocial Impact

References and Readings

North, F. M., Syme, S. L., Feeney, A., Shipley, M., & Marmot, M. (1996). Psychosocial work-environment and sickness absence among British civil servants: The Whitehall II study. *American Journal of Public Health, 86*, 332–340.

Psychosomatic

Makiko Ito
Department of Stress Science and Psychosomatic Medicine, Graduate School of Medicine, The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Definition

Psychosomatic is defined as one involving or depending on both the mind and the body as mutually dependent entities.

The term has been used to refer to the following:

1. Physical disorders, those caused or aggravated by psychological factors and, less often, to mental disorders caused or aggravated by physical factors
2. The branch of medicine concerned with the mind-body relations
3. The field of study, one sometimes designated "psychosomatics," concerned with the relationship between mind and body

Description

It is said that the foundation for psychosomatic movement was laid 2,500 years ago in ancient Greece.

In the fifth century BC, Hippocratic principles emphasized what we consider to be some of the basic tenets of psychosomatic medicine: concern about the relationship between the physician and

the patient and about importance of the environment and of the adaptive factors in health and disease.

Francis Bacon advocated investigation of the mental faculties and of the interaction of body and mind by case studies and by study of the relationships between the individual and society. A passage written by Bacon in 1605 is the first explicit scientific statement about psychosomatic medicine in English.

Psychiatry and psychosomatic medicine owe an immense debt to Johann Reli, who was the first use the word psychiatry.

Another pioneer in German psychiatry was Johann Heinroth, who was the first to use the word psychosomatic in 1818. He insisted that the mind in health and disease was essential to the treatment of illness.

The significance of Freud's dynamic principles of psychological causality and of the unconscious is enhanced.

In the 1920s and 1930s, psychosomatic concepts were supported by two major advances in physiology as well as by psychoanalytic findings. Pavlov's discovery of the conditioned reflex furnished a tool for measuring emotional correlates of stress, and Cannon's work on adrenaline, the endocrine glands, and on the autonomic nervous system stimulated the development of research in psychophysiology.

Resurgent interest in psychosomatic medicine in the 1920s and the 1930s started with clinical work, initially case histories that described psychosomatic phenomena. Within a few years, Alexander and Dunbar proposed theories of psychosomatic illness.

In 1950s, Hans Selye advocated a concept that an external stressor had an influence on physical health, which is called "general adaptation syndrome." The theory of Selye contributed to mind and body medical advance greatly.

George L. Engel proposed that health is affected by a biological factor, a psychological factor, and the social support.

After that, including a famous Framingham study, a way of thinking that an unhealthy habit and action had an influence on health directly came to attract attention worldwide. Health and a complicated biological, psychological, and sociological health model about the disease came to be recognized in the medicine, and a field called the behavior medicine was born in this way. The current psychosomatic medicine regards this behavior medicine as the one of the important theoretical bases, but aims at the promotion of the medical care of all people while not only it but also psychology takes in various study.

Cross-References

▶ Psychosomatic Disorder

References and Readings

Kannel, W. B., & Eaker, E. D. (1986). Psychosocial and other features of coronary heart disease: Insight from Framingham study. *American Heart Journal, 112*(5), 1066–1073.

Lipowski, Z. J. (1984). What does the word "Psychosomatic" Really mean? A historical and semantic inquiry. *Psychosomatic Medicine, 46*(2), 153–171.

Schwab, J. J. (1985). Psychosomatic medicine: Its past and present. *Psychosomatics, 26*(7), 583–593.

Psychosomatic Diseases

▶ Psychosomatic Disorder

Psychosomatic Disorder

Tetusya Ando
Department of Psychosomatic Research, National Institute of Mental Health, National Center of Neurology and Psychiatry, Kodaira-shi, Tokyo, Japan

Synonyms

Psychophysiologic disorders; Psychosomatic diseases; Psychosomatic illness

Definition

1. Somatic diseases or disorders characterized by objective organic changes and/or functional changes that could be induced, progressed, aggravated, or exacerbated by psychological, social, and/or behavioral factors.
2. Physical illness or symptom believed to be caused by psychological factors.

Description

The term "psychosomatic" carries two connotations having an ancient tradition in Western thinking and medicine: psychogenesis of disease and holism. Psychogenesis is an etiologic hypothesis about the role of psychological factors in human disease. The core notion of holism is that mind and body is inseparable and mutually dependent aspects of man, and it implies a view of the human being as a whole.

The idea of psychogenesis resulted in the concept of psychosomatic disorder, a physical illness or symptoms believed to be caused by psychological factors. Notion of psychogenesis has been criticized because it is incompatible with current multifactorial view of diseases, and the term "psychosomatic disorder" is misleading since it implies a special class of disorders of psychogenic etiology and absence of psychosomatic interface in other diseases.

Holistic concept resulted in multifactorial model of illness called "biopsychosocial" model by Engel. In this model, illness is a result of interacting mechanisms at the cellular, tissue, organic, interpersonal, and environmental level. Biopsychosocial approach to illness and health covers the psychosomatic medicine, behavioral science, social science, neuroscience, stress physiology and epidemiology, psychoneuroendocrinology/immunology, psycho-oncology, and so on.

Psychosomatic medicine has focused on the study of the interaction of psychosocial and biological factors in health and disease. Psychosocial factors may induce, sustain, or modify the course of virtually all kind of diseases including infections and cancer, though their relative weight may vary from disease to disease and from patient to patient suffering from the same disease. Japanese Society of Psychosomatic Medicine defined psychosomatic disorders as somatic diseases "characterized by objective organic changes and/or functional changes that could be induced, progressed, aggravated, or exacerbated by psychological, social, and/or behavioral factors."

Psychosocial factors possibly affecting individual vulnerability, onset, course, and outcome of diseases are early and recent life events, chronic stress, personality variables, coping ability, social support, psychological state such as depression, anxiety, anger, hostility, irritability, psychological well-being, and abnormal illness behavior. Mechanisms through which psychosocial factors could influence health and disease are mediated by central and autonomic nervous systems, neuroendocrine systems, and immune systems. Psychosocial stressors may also affect health and disease through changes in health-related habits and behaviors, such as smoking, eating, drinking, exercise, and drug use. Psychological factors may also play role in adjustment to disease, attitude to medical care, doctor-patient relationship, adherence to treatment, impairment in social functioning, and quality of life of patients.

For example, high levels of stress, low levels of social support or social isolation, low socioeconomic status, personality factors, and negative emotions such as anger or hostility, depression, and anxiety are associated with increased cardiovascular disease morbidity and mortality. These psychosocial factors have been associated with enhanced sympathetic nervous system activation, impaired parasympathetic activity, increased circulating levels of catecholamines and corticosteroid, enhanced blood coagulation and fibrinolysis, endothelial dysfunction, coronary vasospasm, and various inflammatory markers. Lifestyle modification (e.g., smoking cessation, diet, exercise) improves cardiovascular health. Psychological interventions, such as relaxation, stress management, cognitive, and behavior therapies may improve psychological distress and psychological functioning in cardiovascular patients.

As a summary, the term "psychosomatic disorders" is misleading and discouraged to be used at least in the sense of psychogenic disease. Holistic concept, another major connotation of "psychosomatic," resulted in biopsychosocial model of illness. Irrespective of whether the term "psychosomatic disorder" is used or not, it is important to perform research and medical practice of a disease from a multifactorial perspective.

Cross-References

► Psychoneuroendocrinology

References and Readings

Engel, G. L. (1977). The need for a new medical model: A challenge for biomedicine. *Science, 196*, 129–136.
Fava, G. A., & Sonino, N. (2000). Psychosomatic medicine: Emerging trends and perspectives. *Psychotherapy and Psychosomatics, 69*, 184–197.
Lipowski, Z. J. (1986). Psychosomatic medicine: Past and present. *Canadian Journal of Psychiatry, 31*, 2–21.

Psychosomatic Illness

► Psychosomatic Disorder

Psychosomatic Medicine

► Health Psychology

Psychosurgery

Douglas Carroll
School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Psychiatric surgery

Definition

Psychosurgery is brain surgery conducted explicitly to amend aspects of human behavior. As such, it can be distinguished from neurosurgery, where the aim is to address some specific and identifiable brain pathology such as a tumor. Although there are noticeable gray areas such as brain surgery for intractable pain or to halt the spread of epileptic seizures from one brain hemisphere to the other, the above distinction is important in differentiating between the primarily behavioral aims of psychosurgery and the primary aims of treating physical pathology that characterize neurosurgery.

Description

The first psychosurgical operation was undertaken on November 12, 1935 by Egas Moniz at the Neurological Institute of the University of Lisbon, Portugal. Moniz's surgery was conducted on severely disturbed psychiatric patients who had proved resistant to other forms of treatment. He called his operation the prefrontal leucotomy; a wire garrote inserted via a cannula or "leucotome" was used to severe connections between the prefrontal cortex and more posterior parts of the brain. In 1949, Moniz was awarded the Nobel Prize in Medicine "for his discovery of the therapeutic value of prefrontal leucotomy in certain psychoses." Moniz's activities in the field of psychosurgery were curtailed, though, when he was shot by one of his psychosurgical patients; the bullet lodged in his spine and he retired, a hemiplegic, in 1944.

However, Moniz's initiative was enthusiastically transported to the USA and UK, where the operation was re-branded: the frontal lobotomy was born. As indicated, the target patient group were psychiatric patients suffering from severe and intransigent psychoses. The Second World War through up a great number of such patients; the late 1940s and early 1950s were to prove the heyday of the frontal lobotomy. It has been estimated that between 40,000 and 50,000 operations were conducted in the USA during

that period with a further 12,000 undertaken in the UK. From its outset, psychosurgery was subject to fierce controversy. Its opponents regarded it as a grievous, unjustified, and irreversible assault on the human personality. Its proponents, on the other hand, confidently testified that it was a valid and efficacious treatment for many seemingly intractable psychiatric disorders. Sober and objective assessment was difficult since the vast majority of psychosurgical data stem from uncontrolled observations where even the minimal attributes of good experimental design are absent: control groups, independent evaluation of treatment effects, lengthy follow-ups, etc. Accordingly, claims of efficacy based on data of such dubious scientific status can command little confidence.

By the late 1950s, psychosurgery was on the wane; the newly introduced major tranquilizers offered a seemingly easier and decidedly less contentious way of managing the behavior of severely disturbed psychiatric patients. Nevertheless, just as it seemed safe to go back into mental hospital, psychosurgery began to re-brand itself once more. By the late 1960s and early 1970s, psychosurgery was enjoying a modest renaissance. This time around, the operations were technically more sophisticated. The main differences, though, were the neural and behavioral targets. No longer was the prefrontal cortex the focus but rather structures that lay deep within the brain: components of the limbic system such as the amygdala, which is implicated in emotion and motivation. No longer was severe psychosis the sole or even main target; instead, the focus had shifted to vaguer and more diagnostically problematic behaviors such as aggression and hyperactivity. Deviance had replaced psychiatric disturbance. As before, the battle between opponents and proponents was enjoined, and as before, the available data did not afford anything like a proper scientific assessment. Again, its day in the sun was short-lived; psychosurgery had all but disappeared by the 1980s. This time, though, it was regulation and legislation that did the trick.

References and Readings

Valenstein, E. S. (1973). *Brain control: A critical examination of brain stimulation and psychosurgery*. New York: Wiley.

Psychotherapy for Depression

▶ Depression: Treatment

Psychotherapy for IBS

▶ Irritable Bowel Syndrome (IBS): Psychological Treatment

PTG

▶ Posttraumatic Growth

PTS

▶ Stress, Posttraumatic

Puberty

Adriana Carrillo and Carley Gomez-Meade
Department of Pediatrics, Miller School of Medicine, University of Miami, Miami, FL, USA

Synonyms

Menarche; Sexual maturation

Definition

Puberty is the transition from being sexually immature to sexual maturity and attainment of

reproductive capacity. Complex interactions of hypothalamic-pituitary hormones and neuroendocrine factors take place to initiate puberty. Recent research indicates kisspeptin and its receptor GPR54 to play a key part in the initiation of puberty. Kisspeptin and GPR54 increase at the onset of puberty. Kisspeptin neurons innervate and stimulate hypothalamic gonadotropin releasing hormone (GNRH) neurons. In addition, kisspeptin neurons express estrogen and androgen receptors which may be important for the onset and tempo of puberty. The onset of puberty is also marked by increasing pulse and frequency of GNRH from the hypothalamus. GNRH then stimulates the pituitary to secrete gonadotropins (luteinizing hormone (LH) and follicle stimulating hormone (FSH)) which directly stimulates the ovaries or testes to produce sex steroids. The increase of sex steroids induces the physical changes of puberty. The progressive physical changes during puberty are described by the Tanner staging system. Both boys and girls have five stages. The first Tanner stage (Tanner stage I) is prepubertal or sexual immaturity and the fifth Tanner stage describes a sexually mature adult. Tanner stage II marks the beginning of sexual maturation (Lifshitz, 2007; Oakley, Clifton, & Steiner, 2009).

Puberty begins earlier in girls than boys. The normal range for the onset of puberty in females is 8–13 years of age. The first sign of puberty in girls is breast development at an average age of 10.4 years in Caucasian girls. African American girls begin puberty earlier at a mean age of 9.5 years. Tanner stages follow progress through puberty with changing contour of the breast. The development of pubic hair is not under the same control as the ovaries. Adrenarche is a result of increased adrenal androgens and occurs before breast development in 10% of girls. Pubic hair is also described by Tanner stages. Puberty is also marked by an increase in percent body fat, maturation of the vaginal mucosa, and uterine and endometrial growth. Accelerated growth begins early in puberty for girls with peak growth velocity during Tanner stage II– III. The average age of menarche for Caucasian girls is 12.5 years and 12 years for African American girls. Menarche is associated with a deceleration in growth and typically occurs during Tanner stage IV. Menarche occurs approximately 2 years after Tanner stage II breast development in normally maturing girls. Early menstrual cycles may be anovulatory and irregular with subsequent ovulation and development of regular cycles. Adult contour breast and adult pubic hair distribution marks Tanner stage V and the completion of puberty.

The normal age range for the onset of puberty in boys is 9–14 years. The average age for the development of Tanner stage II for Caucasian males is 12 years and 11.2 years for African American males. The first sign of puberty in boys is increased testicular volume. Puberty progresses with continued testicular enlargement and penile enlargement as described by the Tanner stages. Mid-puberty axillary hair and androgen sensitive hair on the face, chest and back begins. Also in mid-puberty, males have peak linear growth, voice change, and acne. There is a progressive increase in total bone mineral content and lean body mass and a decline in body fat. Spermarche is attained at Tanner stage III. Peak growth velocity occurs during Tanner stage IV in males. Puberty is complete at Tanner stage V with adult genitalia and adult distribution of pubic hair.

Deviations from normal in the onset of puberty and progression through puberty may represent normal variation or pathological disease. Normal variations include premature adrenarche, gynecomastia during male puberty, and premature thelarche in females. Precocious puberty or delayed puberty may represent abnormal pubertal development. It is important to recognize abnormal variations in puberty that may require intervention.

Premature adrenarche is the early development of pubic hair, typically after 6 years of age for males and females. It is more common in females and is usually benign. Females with a history of premature adrenarche have an increased risk of polycystic ovarian syndrome and insulin resistance. A bone age radiograph can be used to determine effects of sex steroids on skeletal maturation. The bone age in

premature adrenarche is normal. In addition, gonadotropin hormones are normal and there is no breast development in females or testicular enlargement in males.

Gynecomastia is usually a self-limiting development of palpable breast tissue in 40% of males during puberty. It usually begins in early puberty and resolves within 2 years. It is typically minimal and does not require treatment. Some conditions are associated with excessive or prolonged gynecomastia including Klinefelter syndrome and other causes of decreased testosterone production. Other pathological causes of gynecomastia are some testicular tumors, liver failure, ketaconazole, spirinolactone, marijuana, and other medications.

In females, early benign breast development is called premature thelarche. It is usually present at birth and increases in size over the first 2 years of life. It may also occur around 6 years of age. It is nonprogressive and is not associated with accelerated growth, elevated pubertal hormones, or other changes associated with puberty. As with premature adrenarche, this is not associated with accelerated growth, normal bone age, or elevated pubertal hormones.

Precocious puberty is the abnormal development of secondary sexual characteristics before 7 years of age in Caucasian females, 6 years of age in African American females, and before 9 years of age in males. Precocious puberty in females is most commonly idiopathic premature activation of the hypothalamic-pituitary-gonadal axis, termed idiopathic central precocious puberty. Other causes of central precocious puberty include abnormalities of the central nervous system (CNS) such as brain tumors, intracranial irradiation, infections, or congenital malformations. CNS pathology is more common in males than in females. Outside of the CNS, estrogen or testosterone production may occur independent of gonadotropin secretion from the pituitary gland. In females, estrogen from ovarian cysts, ovarian tumors, or exogenous estrogen exposure may stimulate the onset of puberty. Rarely, genetic mutations can stimulate ovarian estrogen production. McCune Albright syndrome is due to an activating mutation of a signaling protein that results in café au lait spots, fibrous dysplasia, and precocious puberty in females. In males, androgen production stimulating penile growth and pubic hair can come from adrenal or testicular tumors. Congenital adrenal hyperplasia is due to a genetic mutation that leads to the overproduction of adrenal androgens that causes precocious puberty in males and virilization in females. Virilization in females includes clitoromegaly, hirsutism, and acne.

If puberty does not occur spontaneously prior to 14 years in males and 13 years in females it is considered delayed. In addition, puberty in males should be completed within 4.5 years after its onset. If a female has not menstruated by 16 years of age or 5 years after the onset of puberty it is termed primary amenorrhea. The most common reason for delayed puberty is constitutional delay of growth and puberty. Constitutional delay is characterized by a decline in growth velocity during early childhood followed by a normal growth velocity with a delayed bone age. Puberty occurs late and final height is normal. There is typically a family history of late puberty.

Pathologic causes of delayed puberty can be divided into those due to failure of the hypothalamus or pituitary to secrete gonadotropins and those due to failure of gonads to respond to gonadotropins. Delayed puberty with low gonadotropins can be secondary to chronic disease, genetic syndromes, gene mutations, or CNS pathology. Syndromes that are associated with low gonadotropins are Prader-Willi, Lawrence Moon, and Kallman syndromes. Anorexia, malnutrition, HIV, Crohn's disease and hemoglobinopathies are some of the chronic conditions that may cause delayed puberty. Endocrine disorders associated with delayed puberty are hypothyroidism and hyperprolactinemia. Tumors, radiation, infection, or congenital malformation can affect the production and secretion of gonadotropins.

Delayed puberty due to gonadal failure is most commonly due to sex chromosome abnormalities. Klinefelter syndrome males carry two X chromosomes and one Y chromosome. Klinefelter syndrome is characterized by testicular failure, disproportionate long limbs, poor

musculature, and gynecomastia. In females, Turner syndrome is due to loss of genetic material from one X chromosome. Females with turners have ovarian failure, short stature, and a spectrum of other dysmorphisms. Gonadal failure can also be due to gonadal dysgenesis or direct damage to the gonads such as trauma, medication, radiation or infection (Lifshitz, 2007; Sperling, 2008).

References and Readings

Lifshitz F (2007) Pediatric endocrinology, 5th edn. Informa Healthcare, New York
Oakley AE, Clifton DK, Steiner RA (2009) Kisspeptin signalling in the brain. Endocrine Reviews 30(6):713
Sperling M (2008) Pediatric endocrinology, 3rd edn. Suanders/Elsevier, Philadelphia, PA

Public Health

Marc D. Gellman
Behavioral Medicine Research Center,
Department of Psychology, University of Miami,
Miami, FL, USA

Definition

Public health refers to those activities by which a society attempts to increase life expectancy, decrease morbidity, and help improve health-related quality of life.

Description

There has often been a widespread misconception that public health is limited to "health care for low-income families." The Centers for Disease Control and Prevention created a list of the ten Great Public Health Achievements in the twentieth century that remind us of how far we have come, how we got here, and exactly what public health is: the active protection of a nation's health and safety, credible information to enhance health decisions, and partnerships with local minorities and organizations to promote good health. The choices of topics for this list were based on the opportunity for prevention and the impact on death, illness, and disability: they are not ranked by order of importance. The list includes the following: vaccination, motor-vehicle safety, safer workplaces, control of infectious diseases, decline in deaths from coronary heart disease (CHD) and stroke, safer and healthier foods, healthier mothers and babies, family planning, fluoridation of drinking water, and last but not least, the recognition of tobacco use as a health hazard.

During this period, the health and life expectancy of persons residing in the United States improved dramatically. Since 1900, the average lifespan of persons in the United States has lengthened by greater than 30 years, of which 25 years of this gain are attributable to advances in public health. The unprecedented increase in longevity seen during the first half of the twentieth century was also seen in other countries, primarily in economically advanced countries. The decrease in mortality rate was largely due to a decline in infectious diseases related to vaccination, decreased exposure to infection because of improved hygiene, improved nutrition, and the development of antibiotics to cope with bacterial infections. However, as infectious diseases declined as the leading cause of mortality in economically advanced countries, they were eclipsed by chronic diseases. By the middle of the twentieth century, CHD, cancer, and stroke accounted for more than 60% of the death rate in the United States.

Public health efforts to eradicate infectious diseases have been successful in an increase in longevity in economically developed and even many less developed countries. Similarly, improvements in healthy lifestyle have led to decreases in morbidity and increases in longevity in these countries during the second half of the twentieth century. At the same time, the HIV/AIDS pandemic in sub-Saharan Africa has led to an even steeper decline in life expectancy. The growing spread of HIV/AIDS across the Asian continent is of considerable concern.

At this point, early in the twenty-first century, the major causes of death in the United States

included (1) heart disease, (2) cancer, (3) stroke, (4) unintentional injuries, (5) chronic obstructive pulmonary disorder, (6) pneumonia and influenza, (7) diabetes, (8) suicide, (9) liver disease, (10) HIV/AIDS, and (11) homicide. Behavioral psychosocial, and sociocultural factors associated with lifestyle contribute to virtually all of these causes of mortality. Even in the case of infectious disease such as pneumonia, risk factors can be related to disruptions of natural pulmonary host mechanisms related to lifestyle factors such as smoking and alcohol abuse. Similarly, infection from HIV is primarily spread through high-risk sexual practices and the sharing of contaminated drug paraphernalia.

As scientists attempted to find specific causal agents in the pathogenesis of cancer and CVD throughout most of the twentieth century, a new approach emerged. Unable to find single causes of diseases, attention shifted to the role of environment and host in the pathogenesis of chronic diseases. Whereas single cause-and-effect models proved successful in studying the genesis of infectious diseases, an understanding of the basis of chronic diseases turned to models based on the presence of risk factors. The identification of risk factors makes prediction of chronic diseases more likely, but individual risk factors cannot be identified as necessary and sufficient causes for many diseases. In this respect, interactions among agent, host, and the environment have now taken center stage.

At the beginning of the risk-factor revolution, it was widely believed that the causes of chronic diseases such as CHD could be explained in terms of a few biological (e.g., high cholesterol, high blood pressure) and lifestyle (e.g., smoking) risk factors; this turned out not to be the case. Other variables contributing to CHD turned out to include physical inactivity, excess consumption of alcohol, and obesity. Still other factors under investigation include individual difference variables such as depression and hostility and sociocultural variables including low socioeconomic status, ethnic minority status, lack of social support, and occupational stress.

To achieve public health objectives, it is sometimes useful to deal with unyielding problems at multiple levels. Although behavioral interventions administered at the individual level tend to produce successful weight loss in the short term, few people maintain their weight loss over the long term. In order for individual-based interventions to succeed on a population basis, such interventions should take place in a sociocultural environment that is conducive to healthful eating and exercise. Improving the availability of healthy food choices, providing economic incentives for healthy eating by selective taxation, ensuring through the schools that children and adolescents get adequate exercise, enhancing accessibility of physical activity for the general public by providing bicycle paths and highway lanes, and initiating mass media campaigns supporting a healthy lifestyle could be useful for maintaining weight loss.

The recent successes in tobacco control in the United States provide a heartening example of how multilevel approaches to a major public health problem can lead to a decline in disease. In this case, the improvements have occurred in cardiovascular disease (CVD), some cancers including lung and esophageal cancer, and respiratory diseases. At the interpersonal level, smoking cessation interventions, sometimes in conjunction with pharmacologic treatment, have been effective. At the organizational level, smoking cessation support groups, school campaigns against smoking, restrictions on smoking in restaurants and work sites, and reductions in health insurance premiums for nonsmokers have been established. Finally, at the societal level, laws against juvenile smoking, taxation of cigarettes, and governmental sponsored antismoking campaigns have all been realized. These measures have led to a marked improvement in the nation's health. Unfortunately, the export of tobacco products outside of the USA to other countries remains a threat to improvements in global public health.

A distinction is sometimes made between clinical or high-risk approaches to disease treatment and prevention versus population-based strategies. Although there is some value in differentiating between these approaches, they should be seen as complementary because neither strategy

is effective for all behaviors or all target groups. Thus, an important public health task is to identify which risk behaviors are open to individual-based versus population-based interventions and how to make these interventions synergistic with one another. Application of the social and behavioral sciences to improve health and combat disease occurs at multiple levels and requires putting into practice different skills both within and across levels. Genetic counseling for those at familial risk of disease, family counseling to reduce substance abuse, and interfamilial violence and group counseling to help those living with HIV/AIDS are examples of interpersonal interventions at the individual level. At the organizational level, interpersonal interventions such as blood pressure screenings and smoking cessation programs, the provision of physical fitness facilities, and media communication have been used in schools, work sites, and community centers. Finally, societal-type interventions involving media and policy actions can occur at the community, state, or federal level. Seat-belt laws, public service announcements about drunk driving, and taxation of cigarettes are examples of interventions at this level.

An important cornerstone of public health is prevention. Primary prevention refers to measures taken to reduce the incidence of disease. In the case of CVD, for example, people may be encouraged to quit smoking, decrease intake of dietary fat, and increase physical activity before diseases become evident. In contrast, secondary prevention involves reducing the prevalence of disease by shortening its duration. Mortality from certain cancers, for example, prostate cancer, is decreased by early detection of the cancers when they are still treatable. Still another form of prevention is tertiary prevention. This involves reducing the complications associated with chronic diseases reducing the complications associated with chronic diseases and minimizing disability and suffering. Medication adherence training in HIV/AIDS patients is a form of tertiary prevention.

Widespread social disorganization and the growing disparity in income within and between nations also pose a global threat to public health. Because public health is a global matter that is closely tied to international policies, hope for future improvements in public health will largely depend on global improvements in public policy.

Cross-References

- ▶ Cancer Prevention
- ▶ Cardiovascular Disease Prevention
- ▶ Cardiovascular Risk Factors
- ▶ Centers for Disease Control and Prevention
- ▶ HIV Prevention
- ▶ Multiple Risk Factors
- ▶ Prevention: Primary, Secondary, Tertiary
- ▶ Smoking Prevention Policies and Programs

References and Readings

Centers for Disease Control and Prevention. (1999). Achievements in public health, 1900–1999; Changes in the public health system. *Morbidity and Mortality Weekly Report, 48*, 1141–1147.

Institute of Medicine. (2001a). *Health and behaviour: The interplay of biological, behavioural and societal influences*. Washington, DC: National Academy Press.

Institute of Medicine. (2001b). *New horizons in health: An integrative approach*. Washington, DC: National Academy Press.

Kawachi, I. (1999). Social capital and community effects on population and individual health. *Annals of the New York Academy of Sciences, 896*, 120–130.

Posner, S. F. (2012). Advancing and improving preventing chronic disease: Public health research, practice and policy. *Preventing Chronic Disease, 9*, 110291. doi: http://dx.doi.org/10.5888/pcd9.110291.

Schneider, M. J. (2011). *Introduction to public health* (2nd ed.). Boston: Jones & Bartlett Learning.

Smedley, B. D., & Syme, S. L. (Eds.). (2000). *Promoting health: Intervention strategies from social and behavioural research*. Washington, DC: National Academy Press.

Turnock, B. J. (2012). *Essentials of public health* (2nd ed.). Boston: Jones & Bartlett Learning.

World Health Organization. (2000). *The world health report 2000: Executive summary*. Geneva: Author.

Public Health Education

- ▶ MPH (Masters of Public Health)

Public Interest Advertising

► Social Marketing

Public Service Advertising

► Social Marketing

Pulmonary Disorders, COPD: Psychosocial Aspects

Akihisa Mitani
Department of Respiratory Medicine, Mitsui Memorial Hospital, Chiyoda-ku, Tokyo, Japan

Synonyms

Chronic obstructive pulmonary disease

Definition

Patients with COPD often suffer from anxiety and depression. These psychological distresses cause low quality of life, social isolation, increased hospitalization rates, and might increase mortality. Treatments include pharmacotherapy, psychotherapy, and pulmonary rehabilitation.

Description

Patients pulmonary disorders are more likely to be of lower self-esteem and downgrade the significance of life in their depression, and chronic obstructive pulmonary disease (COPD) is no exception. However, in spite of the high prevalence of the disease, psychosocial aspects of COPD and other pulmonary diseases have not been fully investigated, compared to cancers or cardiovascular diseases (Hill & Geist, 2008; Hynninen & Breitve, 2005; Kaptein & Scharloo, 2008; Kaptein & Scharloo, 2009; Maurer & Rebbapragada, 2008; von Leupoldt & Dahme, 2007).

(COPD) is a disease characterized by airflow obstruction. The respiratory symptoms are dyspnea, chronic cough, and sputum production. The treatments of COPD include medications focusing on bronchodilators, pulmonary rehabilitation, and oxygen administration.

Psychiatric disorders often appear in patients with COPD. Among them, anxiety and depression are most frequent, but the accurate prevalence is hard to say. In previous reports, the prevalence of anxiety ranges between 10% and 100% and the prevalence of depression, between 10% and 80%. However, it is an indisputable fact that anxiety and depression are often underdiagnosed and/or undertreated. Other disorders include panic disorder, hypochondriasis, and hysteria.

Variables, such as physical disability, smoking, long-term oxygen therapy (LTOT), low body mass index, and severe dyspnea, are associated with anxiety and depression.

As for anxiety, major presumed underlying mechanisms are smoking and dyspnea. Smoking is the most important environmental risk factor for the development of COPD. Adolescents with high levels of anxiety tend to have smoking habits, and smokers with a history of an anxiety-related disorder also experience more symptoms of nicotine withdrawal on cessation of smoking, resulting in tendency to nicotine addiction. Therefore, patients with COPD caused by smoking are likely to show higher levels of anxiety than the general population. Dyspnea is the most common symptom of COPD. The severity of dyspnea changes in response to air temperature or exercise and notably increases during acute exacerbations. For the patients, such episodes of increased dyspnea during exacerbations are associated with anxious feelings, although dyspnea at rest or on exertion does not correlate so much with anxiety. Furthermore, anxiety, in turn, increases the sensation of dyspnea. These bidirectional relationships between dyspnea and anxiety contribute to the increased prevalence of

anxiety-related disorders in COPD (Kaptein & Scharloo, 2008).

Depression in COPD could be caused by various factors such as low body mass index. However, some of them are confounders, making the analysis difficult. The lack of social support for elderly people, their past medical history, and their low socioeconomic status are mixed as risk factors for depression. The use of systemic corticosteroids for treatment, although the long-term use of it is not a standard medication, might cause depression. In addition, there is a relationship between smoking and depression, but little is known about it. Cigarette smoking might prompt a feeling of relaxation for some patients, and smoking cessation could be associated with an increased rate of depression. Chronic hypoxemia and LTOT are also closely related to symptoms of depression, although the underlying mechanisms remain unclear. It is true that LTOT improves survival and exercise capacity, but it may, at the same time, reduce social interactions because the use of oxygen therapy makes the patients lose in several areas of their lives.

Psychological stress typically plays a considerable role in the life of COPD patients because of the interaction of somatic and psychological factors. The emotional response to chronic pulmonary disorders results in further inactivity and social isolation. The patient may feel useless and lose interest in future project. In addition, COPD patients with psychological disorder tend to feel fatigue and short of breath more than patients without such a distress, although psychological distress has not been proved to worsen objective measures of functional exercise capacity. Inadequate perception of dyspnea contributes to a progressive avoidance of activities, resulting in a vicious circle where a physical deconditioning leads to more dyspnea.

As a result, anxiety in COPD patients lowers quality of life and increases hospitalization rates and the economic burden. Intensities of anxiety are correlated with measures of social isolation, suggesting that COPD patients with anxiety have impaired social interactions. The impact of anxiety on the physical disability and mortality of COPD patients is less clear. Depression also causes low quality of life, decreased adherence to treatment, increased frequency of hospital admissions, and prolonged length of stay, resulting in higher medical cost. In addition, depression negatively affects physical function in COPD patients, and the mortality rate among depressed patients is increased. Furthermore, depressed patients tend to make a preference for "do not resuscitate" decisions.

Treatments of psychological distress in COPD patients consist of pharmacotherapy, psychotherapy, and pulmonary rehabilitation.

In pharmacological therapy, antidepressants are commonly used, although evidence for their use in COPD patients is inadequate. Still, they might benefit COPD patients with symptom of anxiety and depression. Selective serotonin reuptake inhibitors (SSRIs) are regarded as first-line therapy. Tricyclic antidepressants and low-dose benzodiazepines could be effective. Administration of benzodiazepines to COPD patients with hypercapnia demands extreme caution because of their respiratory depressant effect; needless to add, each drug has its own side effects and must be used carefully.

Many psychological aspects are relevant to a variety of treatments of COPD. One of the most recent focuses within behavioral treatment on COPD is on self-management. Self-management education is likely to be associated with a reduction in hospital admissions without any detrimental effects in other parameters. Other methods, such as relaxation and biofeedback training, also might have some good effect, although many questions are still not answered.

COPD patients with respiratory symptom should receive comprehensive pulmonary rehabilitation with or without psychological distress. There is growing evidence that it does improve depressive symptoms. However, there remain some questions to be solved. Further research should focus on finding effective and acceptable maintenance strategies. It also remains to be solved whether pulmonary rehabilitation is as effective in COPD patients with severe anxiety and depression. Above all, it is not clear which elements confer psychosocial benefits for COPD patients. Only improvements in exercise capacity

may be associated with improvements in anxiety and depression, or it would be better to add a specific psychological component to pulmonary rehabilitation. At present, it is considered reasonable that a comprehensive pulmonary rehabilitation programs should include at least disease education or psychosocial components as the most effective formats.

Cross-References

► Chronic Obstructive Pulmonary Disease

References and Readings

Hill, K., & Geist, R. (2008). Anxiety and depression in end-stage COPD. *European Respiratory Journal, 31*(3), 667.

Hynninen, K. M., & Breitve, M. H. (2005). Psychological characteristics of patients with chronic obstructive pulmonary disease: A review. *Journal of Psychosomatic Research, 59*(6), 429.

Kaptein, A. A., & Scharloo, M. (2008). Illness perceptions and COPD: An emerging field for COPD patient management. *The Journal of Asthma, 45*(8), 625.

Kaptein, A. A., & Scharloo, M. (2009). 50 years of psychological research on patients with COPD–road to ruin or highway to heaven? *Respiratory Medicine, 103*(1), 3.

Maurer, J., & Rebbapragada, V. (2008). Anxiety and depression in COPD: Current understanding, unanswered questions, and research needs. *Chest, 134* (Suppl. 4), 43S.

von Leupoldt, A., & Dahme, B. (2007). Psychological aspects in the perception of dyspnea in obstructive pulmonary diseases. *Respiratory Medicine, 101*(3), 411.

Pulmonary Function

Valerie Sabol
School of Nursing, Duke University, Durham, NC, USA

Synonyms

Lung function

Definition

Pulmonary function testing (PFT) is a process for assessing functional status of the lungs, for obstructive and restrictive lung disease screening, and for evaluating treatment response (e.g., medications, chest physical therapy). Spirometry, using a spirometer, is the most common of the pulmonary function tests and measures the amount (volume) and the speed (flow) of air that can be inhaled and exhaled. It is designed to measure changes in lung volume and can only measure lung volume compartments that exchange gas with the atmosphere. Graphical measurement of gas movement (in and out of the chest) is referred to as a spirograph, and the tracing is called a spirogram. Spirogram tracings typically include both predicted and observed values to aid in clinical diagnostic evaluation.

The spirometry procedure is highly dependent on individual effort and cooperation and to ensure reproducibility, is generally repeated three times. Typically, individuals are asked to take the deepest breath they can and then exhale into the sensor as hard as possible, for as long as possible (preferably at least 6 s). It is sometimes directly followed by a rapid inhalation (inspiration) to evaluate for upper airway obstruction. The most common parameters measured in spirometry are vital capacity (VC); forced vital capacity (FVC); forced expiratory volume (FEV) at timed intervals of 0.5, 1.0 (FEV1), 2.0, and 3.0 s; forced expiratory flow 25–75% (FEF 25–75); and maximal voluntary ventilation (MVV). Airflow patency is estimated by measuring the flow of air an individual can exhale as hard and as fast as possible. Reduced airflow or obstruction can be a result of narrowed airways (e.g., asthma, bronchial inflammation, emphysema, tumor external compression). Airflow restriction is a decrease in lung volumes and is a measure of both FVC and residual volume. There are a variety of causes of restrictive lung disorders and include pneumonia, scoliosis, kyphosis, pleural effusion, obesity, tumors, and neuromuscular diseases.

As a person ages, the natural elasticity of the lungs decreases, and this translates into smaller

lung volumes and capacities. Males typically have larger lung volumes and capacities than females. Subsequently, PFT interpretation should be based on results of a normal person of the same age and gender. Body height and size also have an impact of PFT results. As an individual ages, their body mass may increase as a result of increased body fat to lean body mass ratio changes. For example, if an individual becomes obese, the abdominal mass prevents the diaphragm from descending as far as it could, and PFT observed (measured) results will be less than predicted (from preestablished normal data tables). Ethnicity may also impact PFT results, and interpretation of observed data should be compared to normal data of similar ethnic groups. Results are usually given in both raw data (liters per second) and percent predicted (i.e., the test result as a percent of the predicted values for the patients of similar characteristics). Generally, results nearest to 100% of the predicted value are the most normal, and results over 80% are often considered normal.

Cross-References

- ▶ Bronchitis
- ▶ Chronic Obstructive Pulmonary Disease
- ▶ Emphysema

Pulse Rate

- ▶ Heart Rate

Purpose

- ▶ Meaning (Purpose)

Q

Q Wave Myocardial Infarction

► Acute Myocardial Infarction

qEEG

► Quantitative EEG Including the Five Common Bandwidths (Delta, Theta, Alpha, Sigma, and Beta)

QTL

► Quantitative Trait Locus (QTL)

Qualitative Research Methods

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

Qualitative research methods collect and analyze qualitative data, which are often expressed in words rather than numbers. Qualitative data can be distinguished from quantitative data, which are expressed in numbers (e.g., a systolic blood pressure of 120 millimeters of mercury [mmHg]).

One example of a qualitative characteristic is skin coloration (at least as traditionally described). While normal skin colors vary from pinkish white to black, medical conditions and diseases can cause alterations of an individual's skin color. Anemia can lead to a white color (in those not normally so white). Various inflammations can lead to a red color (e.g., a severe rash), and cyanosis resulting from cardiac failure or lung failure leads to a blue color. Such assessments are not recorded and reported in numbers but in descriptive text.

Some researchers regard categorical data as qualitative data, even though numerical representations do occur when presenting the data. Consider the variables sex and blood group. Each of us falls into a mutually exclusive category for each one, e.g., male or female, and O, A, B, and AB. None of us falls into more than one category in each case. Additionally, there is no order to the categories: blood group B neither comes before or after blood group O. In a group of individuals of interest, data could be presented as counts of the number of individuals falling into each category in each case, thereby using numbers.

There are categories that can be ordered. For example, ordered categories of a pain that is experienced would be mild, moderate, and severe. Again, for a group of individuals of interest (perhaps subjects undergoing mindful meditation therapy or another behavioral intervention for pain), it is possible to describe how many individuals reported a pain falling in each of the categories.

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

Cross-References

► Data

References and Readings

Piantadosi, S. (2005). *Clinical trials: A methodologic perspective* (2nd ed.). Hoboken, NJ: Wiley-Interscience.

Quality of Care

► Doctor-Patient Communication: Why and How Communication Contributes to the Quality of Medical Care

Quality of Life

Maartje de Wit and Tibor Hajos
Medical Psychology, VU University Medical Center, Amsterdam, North Holland, The Netherlands

Synonyms

Health-related quality of life

Definition

Quality of life (QoL) is a term used to refer to an individual's total well-being. There is disagreement between scientists, sociologists, and clinicians about the conceptualization of QoL, and hence, a clear definition is lacking (Hunt, 1997). However, current definitions can be categorized into three types (Farquhar, 1995): (1) global definitions, such as happiness/unhappiness; (2) definitions that break down QoL into a series of components or dimensions; and (3) focused definitions, which are often pragmatic approaches in which QoL is seen as synonymous with domains of the field of interest to the researchers (e.g., functional status is sometimes used as a measure of QoL by health researchers).

The first type of definitions has been researched since Aristotle, and still no consensus has been reached on how to define happiness and how to measure it (Fayers & Machin, 2000). The second and third types of definitions have underlying assumptions for which consensus exists about the domains encompassed by QoL. These latter two types of definitions provide a more practical approach for research purposes.

Across all definitions, considerable agreement exists that QoL is conceptualized as a multidimensional construct incorporating primarily the person's evaluation of his/her own life. The World Health Organization (WHO) defines QoL as the individuals' perception of their position in life in the context of the culture and value systems in which they live and in relation to their goals, expectations, standards, and concerns. It is a broad-ranging concept affected in a complex way by the person's physical health, psychological state, level of independence, social relationships, personal beliefs, and their relationship to salient features of their environment (World Health Organization, 1995).

The impact of health and illness on a person's QoL is referred to as health-related quality of life (HRQoL). QoL and HRQoL are often used interchangeably.

Cross-References

► Health-Related Quality of Life
► Multiple Sclerosis: Psychosocial Factors
► Quality of Life: Measurement

References and Readings

Farquhar, M. (1995). Definitions of quality of life: A taxonomy. *Journal of Advanced Nursing, 22*(3), 502–508.
Fayers, P., & Machin, D. (2000). *Quality of life assessment, analysis and interpretation*. Chichester: John Wiley & Sons.
Hunt, S. M. (1997). The problem of quality of life. *Quality of Life Research, 6*(3), 205–212.

World Health Organization. (1995). The World Health Organization Quality of Life assessment (WHOQOL): Position paper from the World Health Organization. *Social Science and Medicine, 41*(10), 1403–1409.

Quality of Life Assessments

► Measures of Quality of Life

Quality of Life Instruments

► Measures of Quality of Life

Quality of Life: Measurement

Maartje de Wit
Medical Psychology, VU University Medical Center, Amsterdam, North Holland, The Netherlands

Definition

Subjective assessment of the various aspects of quality of life.

Description

Quality of Life (QoL) is considered an important outcome variable in health-care practice and research. Consideration of QoL provides health-care providers, researchers, and policy makers insight into how variables of interest (e.g., chronic disease, medication use, living environment) impact a person's subjective well-being, which is instrumental to improving health-care practice systems.

QoL measures may be of potential value in comparing outcomes in clinical trials, evaluating interventions, commissioning programs of care, assessing the outcomes of new treatments, and in audit work. Further on, measuring health-related QoL (HRQoL) can facilitate discussion between patients and professionals in health care and stimulate the dialogue between medical outcomes and patients' subjective views (de Wit et al., 2008; Detmar, Muller, Schornagel, Wever, & Aaronson, 2002; Pouwer, Snoek, van der Ploeg, Ader, & Heine, 2001).

Measurement of QoL

Despite a clear definition of QoL, there is general agreement that health-related QoL (HRQoL) encompasses physical, cognitive, affective, psychological, social well-being, health perception, and disease- and treatment-related symptoms. Overall QoL expands upon this and incorporates non-medical-related aspects of a person's life such as the influence of work, spirituality, and other life circumstances (Koot, 2001).

QoL can include both objective and subjective perspectives in each domain (Testa & Simonson, 1996). The objective assessment of QoL focuses on what the individual can do, and is important in defining the degree of health. The subjective assessment of QoL includes the meaning to the individual; essentially it involves the translation or appraisal of the more objective measurement of health status into the experience of QoL. Differences in appraisal account for the fact that individuals with the same objective health status can report very different subjective QoL (Fayers & Machin, 2000).

Generic and Disease Specific Instruments

There are a wide variety of instruments available to obtain information on QoL. These instruments can be divided into generic and disease-specific instruments. Generic measures attempt to measure all important domains of QoL. Generic measures are most useful when comparisons or decisions have to be made for large groups of patients with disparate conditions and backgrounds (Fayers & Machin, 2000). For example, it gives the opportunity to compare the QoL of people with diabetes with their healthy peers or to people with asthma.

Disease-specific measures include domains that are designed to be valid only for a specified condition. Therefore they maximize content validity and support greater sensitivity and

specificity (Guyatt, Feeny, & Patrick, 1993), and may be particularly informative for disease management at the individual patient level. Advances in the development of QoL questionnaires for children resulted in generic questionnaires with complementary disease-specific modules (Bullinger, 2005; Ravens-Sieberer et al., 2008; Varni, Burwinkle, & Seid, 2005; Varni, Seid, & Rode, 1999). This measurement approach enables researchers and professionals to compare children with a specific disease to their healthy peers and also examine the impact of the disease at the same time.

QoL Measurement in Children

The development and validation of instruments for children have evolved over the past 30 years. Traditionally, parents have been the ones reporting on their child's HRQoL, because the child was seen as an unreliable respondent. When the child is very young or ill, the child may indeed be unable to complete questionnaires. However, relying on the parent as informant may result in incomplete assessment to the extent that the child's subjective experience and perceptions of HRQoL may be overlooked (Eiser & Morse, 2001a). Especially as the child grows older and develops his/her own life, the HRQoL reports of parents become less informative. It has been shown that parents and children agree more on objective domains of HRQoL (i.e., physical functioning) than on subjective domains, like emotional and social functioning (Eiser & Morse, 2001a; Janse, Sinnema, Uiterwaal, Kimpen, & Gemke, 2008).

As children and parents do not necessarily share similar views about the impact of illness, children are more directly involved in decisions about their own care and treatment as they mature. However, assessment of QoL in children poses unique problems (Eiser & Morse, 2001a). Children do not share adult views about the cause, aetiology, and treatment of illness. They may interpret questions differently, and adopt a different time perspective. In addition, their abilitiy to use rating scales, understand the language, and generally complete lengthy questionnaires of the type used in adult work, may be compromised by age and cognitive development. These considerations have guided the development of QoL questionnaires especially for children (Eiser & Morse, 2001a; Solans et al., 2008).

Choosing an Instrument

Because of the broad, multidimensional nature of QoL, it is advised that researchers who want to measure QoL define what they mean by "quality of life," or explain which domains of quality of life they want to measure using a specific instrument. For example, the terms "health-related QoL" (HRQoL), "functional status," and "health status" are often used interchangeably. However, although health or functional status can be part of HRQoL, it does not take the patient's perspective into account.

The Patient-Reported Outcome and Quality Of Life Instrument Database (PROQOLID) (www.proqolid.org, Mapi Research Institute, 2001–2011) provides an outline of available instruments to measure patient-reported outcomes or QoL. It should be noted, however, that identification of an instrument in this database does not necessarily mean it is valid and reliable in view of the question at hand. Thus, instruments should be selected with care (Bradley, 2001; Eiser & Morse, 2001b; Fayers & Machin, 2000; Koot & Wallander, 2001; Solans et al., 2008).

Cross-References

- ▶ Health-Related Quality of Life
- ▶ Quality of Life

References and Readings

Bradley, C. (2001). *Handbook of Psychology and Diabetes*. Amsterdam: Harwood Academic.

Bullinger, M. (2005). *Translations DISABKIDS*. Retrieved February, 2006, from http://kidscreen.diehauptstadt.de/disabkids/master/translations/index.html

de Wit, M., Delemarre-van de Waal, H. A., Bokma, J. A., Haasnoot, K., Houdijk, M. C., Gemke, R. J., et al. (2008). Monitoring and discussing health-related quality of life in adolescents with type 1 diabetes improve

psychosocial well-being: A randomized controlled trial. *Diabetes Care, 31*(8), 1521–1526.
Detmar, S. B., Muller, M. J., Schornagel, J. H., Wever, L. D., & Aaronson, N. K. (2002). Health-related quality-of-life assessments and patient-physician communication: A randomized controlled trial. *Journal of the American Medical Association, 288*(23), 3027–3034.
Eiser, C., & Morse, R. (2001a). Can parents rate their child's health-related quality of life? Results of a systematic review. *Quality of Life Research, 10*(4), 347–357.
Eiser, C., & Morse, R. (2001b). A review of measures of quality of life for children with chronic illness. *Archives of Disease in Childhood, 84*(3), 205–211.
Fayers, P., & Machin, D. (2000). *Quality of life. Assessment, analysis and interpretation*. Chichester, UK: Wiley.
Guyatt, G. H., Feeny, D. H., & Patrick, D. L. (1993). Measuring health-related quality of life. *Annals of Internal Medicine, 118*(8), 622–629.
Janse, A. J., Sinnema, G., Uiterwaal, C. S., Kimpen, J. L., & Gemke, R. J. (2008). Quality of life in chronic illness: Children, parents and paediatricians have different, but stable perceptions. *Acta Paediatrica, 97*, 1118–1124.
Koot, H. M. (2001). The study of quality of life: Concepts and methods. In J. L. Wallander & H. M. Koot (Eds.), *Quality of life in child and adolescent illness. Concepts, methods and findings* (pp. 3–17). East Sussex, UK: Brunner-Routledge.
Koot, H. M., & Wallander, J. L. (2001). Challenges in child and adolescent quality of life research. In H. M. Koot & J. L. Wallander (Eds.), *Quality of life in child and adolescent illness. Concepts, methods and findings* (pp. 431–456). East Sussex: Brunner-Routledge.
Mapi Research Institute. (2001–2011). *PROQOLID*. Retrieved January 25, 2011, from www.proqolid.org
Pouwer, F., Snoek, F. J., van der Ploeg, H. M., Ader, H. J., & Heine, R. J. (2001). Monitoring of psychological well-being in outpatients with diabetes: Effects on mood, HbA(1c), and the patient's evaluation of the quality of diabetes care: A randomized controlled trial. *Diabetes Care, 24*(11), 1929–1935.
Ravens-Sieberer, U., Gosch, A., Rajmil, L., Erhart, M., Bruil, J., Power, M., et al. (2008). The KIDSCREEN-52 quality of life measure for children and adolescents: Psychometric results from a cross-cultural survey in 13 European countries. *Value in Health, 11*(4), 645–658.
Solans, M., Pane, S., Estrada, M.-D., Serra-Sutton, V., Berra, S., Herdman, M., et al. (2008). Health-related quality of life measurement in children and adolescents: A systematic review of generic and disease-specific instruments. *Value in Health, 11*(4), 742–764.
Testa, M. A., & Simonson, D. C. (1996). Assessment of quality-of-life outcomes. *The New England Journal of Medicine, 334*(13), 835–840.
Varni, J. W., Burwinkle, T. M., & Seid, M. (2005). The PedsQL™ as a pediatric patient-reported outcome: Reliability and validity of the PedsQL™ Measurement Model in 25,000 children. *Expert Review of Pharmacoeconomics & Outcomes Research, 5*, 705–719.
Varni J. W., Seid M., & Rode C. A. (1999). The PedsQL: measurement model for the pediatric quality of life inventory. *Med Care* 37(2):126–139.

Quality of Work

▶ Job Performance

Quality-Adjusted Life Years (QALYs)

M. Bryant Howren
Department of Psychology, The University of Iowa & VA Iowa City Healthcare System, Iowa City, IA, USA

Definition

The *Quality-Adjusted Life Year* (QALY) is a standardized measure of disease burden which combines both survival and health-related quality of life into a single index. The QALY is primarily used in cost-effectiveness analyses to guide decisions regarding the distribution of limited health care resources among competing health programs or interventions for a population of interest, but has also been used to aid decisions regarding clinical management and individual patient care.

Conceptually based in expected utility theory, the QALY rests on the assumption that preference-weighted values may be attached to specific health states relative to the time spent in those states. Because the QALY incorporates both quantity and quality of life, it therefore provides a reasonable estimate of the amount of quality time (i.e., health benefit) an individual may experience as a result of a particular health program or intervention. Furthermore, comparisons between programs or interventions may be made both within a single disease and across different diseases. Consequently, the QALY has been widely used as an outcome measure in medicine, psychology, public health, and economics.

Calculation of the QALY requires various health states be assigned a value ranging from zero to one, with zero representing death and one representing ideal health. Health states considered worse than death (i.e., a health state valuation less than zero) can also exist and are assigned a negative value. In order to determine the values of each particular health state, respondents are asked to rate them relative to one another, or using an anchor point such as death. Several methods may be used to ascertain valuations, including preference measurement techniques such as the standard gamble or time trade-off, or through the use of rating scales, such as the EQ-5D, Health Utilities Index, Quality of Well-Being Scale, and SF-6D.

A number of limitations have been described with respect to the measurement and incorporation of QALYs. In particular, QALYs may be less useful in the context of preventive health programs (i.e., when health effects may not materialize for some time), or chronic diseases (i.e., where quality of life may be more important than survival). Other concerns include inconsistencies among valuations due to method variance, situations in which population-level preferences about a particular disease differ from preferences of the subgroup afflicted with the disease and, more generally, decisions regarding whose preferences should matter most when assigning values to specific health states. These limitations notwithstanding, the QALY serves as a straightforward, intuitive measure of disease burden and remains an important tool in health care decision-making.

Cross-References

- ▶ Benefit Evaluation in Health Economic Studies
- ▶ Decision Making
- ▶ Health-Related Quality of Life

References and Readings

Drummond, M. F., Sculpher, M. J., Torrance, G. W., O'Brien, B. J., & Stoddart, G. L. (2005). *Methods for the economic evaluation of health care programmes*. Oxford: Oxford University Press.

Gold, M. R., Stevenson, D., & Fryback, D. G. (2002). HALYs and QALYs and DALYs, oh my: Similarities and differences in summary measures of population health. *Annual Review of Public Health, 23*, 115–134.

Kaplan, R. M., & Frosch, D. L. (2005). Decision making in medicine and health care. *Annual Review of Clinical Psychology, 1*, 525–556.

Kaplan, R. M., & Groessl, E. J. (2002). Applications of cost-effectiveness methodologies in behavioral medicine. *Journal of Consulting & Clinical Psychology, 70*, 482–493.

Kind, P., Lafata, J. E., Matuszewski, K., & Raisch, D. (2009). The use of QALYs in clinical and patient decision-making: Issues and prospects. *Value in Health, 12*, S27–S30.

Nord, E. (1999). *Cost-value analysis in health care: Making sense out of QALYs*. New York: Cambridge University Press.

Weinstein, M. C., Torrance, G., & McGuire, A. (2009). QALYs: The basics. *Value in Health, 12*, S5–S9.

Quantitative EEG Including the Five Common Bandwidths (Delta, Theta, Alpha, Sigma, and Beta)

Salvatore Insana
Western Psychiatric Institute and Clinic, Pittsburgh, PA, USA

Synonyms

Frequency analysis; Power spectral analysis; qEEG; Spectral analysis

Definition

Quantitative electroencephalography (qEEG) is an analytical technique that can be used to objectively describe the frequency and power of electroencephalography data.

Description

Quantitative electroencephalography (qEEG) can be used in any application where electroencephalography (EEG) is applied. In this entry, qEEG will be described in the context of its application

in the measurement of brain activity patterns that can be used to infer sleep. Sleep can be measured with polysomnography (PSG), which is currently considered the "gold standard" measure of sleep-wake states. EEG is an integral component to PSG, along with electrooculography (EOG), and electromyography (EMG). In order to assure standardized PSG measurement procedures, there are uniform practice parameters established by the American Academy of Sleep Medicine (AASM [Iber, Ancoli-Israel, Chesson, Quan, & AASM, 2007]). The AASM sleep-monitoring practice parameters include specified criteria for electrode placement, equipment calibration, and PSG acquisition. Once PSG sleep signals (i.e., EEG, EOG, and EMG) are appropriately recorded, the signals can be used together for visual analyses; additionally or alternatively, EEG can be independently used for quantitative analyses.

Visual PSG Analyses: When sleep is measured with PSG, the measured signals are typically sectioned into consecutive 30-s intervals throughout the entire PSG recording period. These 30-s intervals are termed "epochs." Within each epoch, the PSG measured signals (i.e., EEG, EOG, and EMG) are cumulatively used to differentiate sleep from wake, and to further classify sleep into different categories that are known as sleep stages. According to the AASM (Iber et al., 2007), sleep can be classified into four stages that include N1, N2, N3, and Rapid Eye Movement (REM) sleep. Sleep stage scoring is completed by a trained technician who visually interprets the PSG signals in accordance with the aforementioned standard practice parameters. Once visually scored, sleep can be described by variables that include, but not limited to, time spent in particular stages, latency to particular stages, arousals from sleep, and specific physiological events during sleep. Visually scored sleep can be displayed on a hypnogram plot.

Quantitative EEG analyses: When sleep is measured with EEG, the measured signals can be analyzed and described quantitatively by their frequency and power. There are mathematically detailed components to quantitative EEG analysis; these components will be broadly described (see reviews, Campbell, 2009; Thakor & Tong, 2004). Preliminary processing includes digitization and prefiltering. Typically, EEG signals are digitized at a rate of 256 Hz, are band-limited using a low-frequency and high-frequency filters (e.g., signals $\leq$0.5 and $\geq$64 Hz are removed) to remove irrelevant signals, and are decimated (e.g., halved to128 Hz) for the analyses. Low-frequency artifacts are removed; for example, epochs that were visually scored as wakefulness or movement artifact could be excluded. High-frequency EEG artifacts are removed; for example, an algorithm can be implemented that excludes a predetermined bin length (e.g., 4 s) if that bin exceeds a predetermined high-frequency threshold relative to the frequency in adjacent bins. Once artifacts are removed, a fast Fourier transformation (FFT) is implemented to analyze wave frequency (in Hertz) within the epochs; that is, epochs are analyzed according to moving windows that partially overlap with preceding and succeeding windows. For example, a 30-s epoch that is analyzed according to a 4-s window can have a 2-s overlap among windows, thus yielding 15 windows for the epoch. Common FFT window functions include Hanning, Hamming, Bartlett, and Welch; these functions differ in duration and window overlap lengths (Campbell, 2009; Thakor & Tong, 2004). Wave amplitude is sampled in microvolts and is squared to convert the wave amplitude to power; power is calculated within each frequency band, described below.

Power is grouped according to frequency within specific broad bands or frequency ranges. There are five commonly used frequency bands that are examined with spectral analysis. The frequency bands typically fall within the range of 0.5–32 Hz; however, these frequency bands can slightly vary by laboratory and can be further broken down into narrower components as guided by the research or clinical question. The behavioral functions that correspond to each frequency band during sleep have not been clearly elucidated; however, current theories are briefly mentioned. The delta band encompasses the frequency range of 0.5–4 Hz. Delta activity is positively associated with the homeostatic sleep

drive in such a manner that delta activity increases in correspondence to increased awake time (Dijk, Brunner, Beersma, & Borbély, 1990). The theta band encompasses the frequency range of 4–8 Hz. Similar to delta activity, theta activity is positively associated with the homeostatic sleep drive (Dijk et al., 1990). The alpha band encompasses the frequency range of 8–12 Hz. Alpha activity is positively associated with relaxed wakefulness and drowsiness associated with sleep onset (Cantero, Atienza, & Salas, 2002). Alpha activity is also present during REM sleep and is theorized as being a micro-arousal that can then lead to a full arousal during sleep; the purpose(s) of alpha associated arousals during REM sleep is unknown (Cantero & Atienza, 2000). The sigma band encompasses the frequency range of 12–16 Hz. Sigma activity is positively associated with sleep spindles and has been linked to learning, memory, and intelligence (Fogel & Smith, 2011; Geiger et al., 2011). The beta band encompasses the frequency range of 16–32 Hz. Beta activity is positively associated with physiological arousal and psychological stress (Hall et al., 2007).

Spectral EEG power within particular bandwidths changes throughout development (Gaudreau, Carrier, & Montplaisir, 2001). Spectral EEG power is highly variable across individuals, but demonstrates a trait-like "fingerprint" that is stable within individuals across nights (De Gennaro, Ferrara, Vecchio, Curcio, & Bertini, 2005), as well as across both sleep and wake states (Ehlers et al., 1998). Spectral EEG power has been implicated in a wide range of sleep research topics including stress (Hall et al., 2000), intelligence (Geiger et al., 2011), memory (Fogel & Smith, 2011), psychopathology (Tekell,et al., 2005), and sleep disorders (Buysse et al., 2001; Krystal & Edinger, 2010; Perlis, Smith, Andrews, Orff, & Giles, 2001).

Spectral power can be calculated for each EEG recording site (e.g., C3/M2, and C4/M1). Total, or absolute, power is the sum of all power frequencies, within each epoch, across all bandwidths analyzed. Relative power is the power per bandwidth divided by total power times 100. Relative power density can be used to describe individual differences in variability because it is standardized by an individual's total power. qEEG calculations are commonly paired with visually scored sleep stages; this pairing can yield power in particular bands during specific sleep stages (e.g., delta power during non-REM sleep [NREM]). Once analyzed, sleep power spectra can be visually displayed on modeled power frequency curves with frequency, in Hertz, on the X-axis and the logarithmic power transformation on the Y-axis. The modeled power frequency curve can represent the entire night, or a specified sleep state such as all NREM sleep, the first NREM sleep bout, all REM sleep, etc.

To date, neither qEEG standardized analyses nor practice parameters have been established; therefore, the qEEG techniques can vary by laboratory (e.g., Vasko et al., 1997). For instance, laboratories use different methods for identifying low- and high-frequency artifacts, choice of window for weighting epochs, definition of frequency broad band ranges, and the specific electrode placement used for analyses (e.g., C3/M2, C4/M1, or average of the two locations). Thus, when reporting qEEG values, a thorough description of the acquisition, processing, and interpretation techniques are of paramount importance.

Cross-References

- ▶ Non-REM Sleep
- ▶ Polysomnography
- ▶ REM Sleep
- ▶ Sleep
- ▶ Sleep Architecture

References and Readings

Buysse, D. J., Hall, M., Begley, A., Cherry, C. R., Houck, P. R., Land, S., et al. (2001). Sleep and treatment response in depression: New findings using power spectral analysis. *Psychiatry Research, 103*, 51–67.

Campbell, I. G. (2009). EEG recording and analysis for sleep research. *Current Protocols in Neuroscience, 49*, 10.2.1–10.2.19.

Cantero, J. L., & Atienza, M. (2000). Alpha burst activity during human REM sleep: Descriptive study and

functional hypotheses. *Clinical Neurophysiology, 111*, 909–915.
Cantero, J. L., Atienza, M., & Salas, R. M. (2002). Human alpha oscillations in wakefulness, drowsiness period, and REM sleep: Different electroencephalographic phenomena within the alpha band. *Clinical Neurophysiology, 32*, 54–71.
De Gennaro, L., Ferrara, M., Vecchio, F., Curcio, G., & Bertini, M. (2005). An electroencephalographic fingerprint of human sleep. *NeuroImage, 26*, 114–122.
Dijk, D. J., Brunner, D. P., Beersma, D. G., & Borbély, A. A. (1990). Electroencephalogram power density and slow wave sleep as a function of prior waking and circadian phase. *Sleep, 13*, 430–440.
Ehlers, C. L., Kupfer, D. J., Buysse, D. J., Cluss, P. A., Miewald, J. M., Bisson, E. F., & Grochocinski, V. J. (1998). The Pittsburgh study of normal sleep in young adults: Focus on the relationship between waking and sleeping EEG spectral patterns. *Electroencephalography and Clinical Neurophysiology, 106*, 199–205.
Fogel, S. M., & Smith, C. T. (2011). The function of the sleep spindle: A physiological index of intelligence and a mechanism for sleep-dependent memory consolidation. *Neuroscience and Biobehavioral Reviews, 35*, 1154–1165.
Gaudreau, H., Carrier, J., & Montplaisir, J. (2001). Age-related modifications of NREM sleep EEG: From childhood to middle age. *Journal of Sleep Research, 10*, 165–172.
Geiger, A., Huber, R., Kurth, S., Ringli, M., Jenni, O., & Achermann, P. (2011). The sleep EEG as a marker of intellectual ability in school age children. *Sleep, 34*, 181–189.
Hall, M., Buysse, D. J., Nowell, P. D., Nofzinger, E. A., Houck, P., Reynolds, C. F., 3rd, & Kupfer, D. J. (2000). Symptoms of stress and depression as correlates of sleep in primary insomnia. *Psychosomatic Medicine, 62*, 227–230.
Hall, M., Thayer, J. F., Germain, A., Moul, D., Vasko, R., Puhl, M., Miewald, J., & Buysse, D. J. (2007). Psychological stress is associated with heightened physiological arousal during NREM sleep in primary insomnia. *Behavioral Sleep Medicine, 5*, 178–193.
Iber, C., Ancoli-Israel, S., Chesson, A., Quan, S. F., & American Academy of Sleep Medicine. (2007). *The AASM manual for the scoring of sleep and associated events: Rules, terminology and technical specifications* (1st ed.). Westchester, IL: American Academy of Sleep Medicine.
Krystal, A. D., & Edinger, J. D. (2010). Sleep EEG predictors and correlates of the response to cognitive behavioral therapy for insomnia. *Sleep, 33*, 669–677.
Perlis, M. L., Smith, M. T., Andrews, P. J., Orff, H., & Giles, D. E. (2001). Beta/Gamma EEG activity in patients with primary and secondary insomnia and good sleeper controls. *Sleep, 24*, 110–117.
Tekell, J. L., Hoffmann, R., Hendrickse, W., Greene, R. W., Rush, A. J., & Armitage, R. (2005). High frequency EEG activity during sleep: Characteristics in schizophrenia and depression. *Clinical EEG and Neuroscience, 36*, 25–35.
Thakor, N. V., & Tong, S. (2004). Advances in quantitative electroencephalogram analysis methods. *Annual Review of Biomedical Engineering, 6*, 453–495.
Vasko, R. C., Brunner, D. P., Monhan, J. P., Doman, J., Boston, R. J., El-Jaroudi, A., et al. (1997). Power spectral analysis of EEG in a multiple-bedroom, multiple-polygraph sleep laboratory. *International Journal of Medical Informatics, 46*, 175–184.

Quantitative Trait Locus (QTL)

Matthew A. Simonson
Institute for Behavioural Genetics, Boulder, CO, USA

Synonyms

QTL

Definition

A quantitative trait locus (QTL) is a region of DNA that influences, or is otherwise associated with, a quantitative trait (Mackay, 2001). Unlike "Mendelian" traits, most traits that vary within a population are influenced by a large number of genes. The genetic effects of Mendelian traits are predominantly influenced by a single genetic locus that is inherited through simple and predictable patterns. Some examples of such traits in humans include blood type, albinism, and Huntington's disease (Dipple & McCabe, 2000). Alternatively, traits such as height, intelligence, risk for most forms of illness, as well as many others, are usually due to the combined effect of many genes and regulatory regions. Such traits vary over a continuous range and are quantifiable by the degree to which the trait is expressed (Weiss, Pan, Abney, & Ober, 2006).

To identify genetic regions that harbor QTLs, genetic markers are first identified across an organism's chromosomes. By examining whether a marker co-segregates with a trait through

a pedigree more often than expected by chance, the regions of the genome near those markers are thought to have some effect on the trait (Morgan, 1911). This method of mapping traits to regions of genomes has been applied to several model organisms, both plant and animal, for roughly 100 years. The mapping of QTL regions, called "linkage analysis," has enabled the identification of genes and causal polymorphisms for several human traits (Almasy & Blangero, 2009).

By locating which QTLs (and associated genes or regulatory regions) contribute to a trait, a better understanding of the genetic architecture of a phenotype is gained (Eaves 1994). Through an increased understanding of the genetic architecture of traits, more refined methods of analysis can be employed. By combining information from genome-wide linkage studies with genome-wide association studies (GWAS), susceptibility alleles that would have previously been overlooked can be identified (Howrigan, Laird, Smoller, Devlin, & McQueen, 2011). Also, if a region is identified as a QTL relevant to a phenotype, it can then be sequenced (Feltus et al., 2011). By analyzing the sequence in a region, a better understanding of the underlying biology of a trait can then be ascertained.

Cross-References

- ▶ Admixture
- ▶ Allele
- ▶ Benefit Evaluation in Health Economic Studies
- ▶ DNA
- ▶ Phenotype
- ▶ Single Nucleotide Polymorphism (SNP)

References and Readings

Almasy, L., & Blangero, J. (2009). Human QTL linkage mapping. *Genetica, 136*(2), 333–340.

Dipple, K. M., & McCabe, E. R. (2000). Phenotypes of patients with "simple" Mendelian disorders are complex traits: thresholds, modifiers, and systems dynamics. *American Journal of Human Genetics, 66*(6), 1729–1735.

Eaves, L. J. (1994). Effect of genetic architecture on the power of human linkage studies to resolve the contribution of quantitative trait loci. *Heredity, 72*(Pt 2), 175–192.

Feltus, F. A., Saski, C. A., Mockaitis, K., Haiminen, N., Parida, L., Smith, Z., et al. (2011). Sequencing of a QTL-rich region of the Theobroma cacao genome using pooled BACs and the identification of trait specific candidate genes. *BMC Genomics, 12*, 379.

Howrigan, D. P., Laird, N. M., Smoller, J. W., Devlin, B., & McQueen, M. B. (2011). Using linkage information to weight a genome-wide association of bipolar disorder. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics, 156B*(4), 462–471.

Mackay, T. F. (2001). Quantitative trait loci in Drosophila. *Nature Reviews Genetics, 2*(1), 11–20.

Morgan, T. H. (1911). Random segregation versus coupling in mendelian inheritance. *Science, 34*(873), 384.

Weiss, L. A., Pan, L., Abney, M., & Ober, C. (2006). The sex-specific genetic architecture of quantitative traits in humans. *Nature Genetics, 38*(2), 218–222.

Quiet Sleep

- ▶ Non-REM Sleep

Quit Smoking

- ▶ Smoking Cessation

R

RA

- ▶ Degenerative Diseases: Joint
- ▶ Rheumatoid Arthritis: Psychosocial Aspects

Racial Inequality in Economic and Social Well-Being

Kristine M. Molina
Department of Psychology, University of Miami, Miami, FL, USA

Synonyms

Racial/ethnic disparities; Social health

Definition

Racial inequalities in economic and social well-being refer to disparities or disproportionate differences between racial/ethnic groups in key areas (e.g., educational achievement, health status, housing quality) that shape a person's/groups' opportunities and chances for optimal functioning in society (Blank, 2001).

Description

Generally, indicators of economic and social well-being in the literature have included educational attainment, labor markets, economic status, health status, crime and criminal justice, and housing. As in many other aspects of our society, race/ethnicity remains a significant predictor of well-being. Economic and social well-being are socially patterned. In comparison with Whites, racial/ethnic minorities experience disproportionate lower (i.e., worse) rates on some of the aforementioned indicators. For example, racial/ethnic inequalities in educational attainment (e.g., high school completion, college graduation) are prevalent for African Americans/Blacks and Hispanics/Latinos compared with Whites, even though educational attainment for African Americans/Blacks has increased considerably in recent years. Of significance is that no improvements have been noted in racial inequalities for family income, the most widely used measure of overall economic well-being (Blank, 2001).

Although racial inequalities in economic and social well-being exist, with certain racial/ethnic groups (e.g., African Americans/Blacks; Latinos/Hispanics) being persistently disadvantaged in a number of areas, it has generally been assumed that all racial/ethnic groups experience these inequalities in a uniform manner. In fact, however, not only are there differences between racial/ethnic minority groups and majority racial/ethnic groups, there are also differences between and within racial/ethnic minority groups (Nazroo, 2003). For example, although also considered to be racial/ethnic minorities, Asian and Pacific Islanders, unlike African Americans/Blacks and Hispanics/Latinos, typically tend to do as well as

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

Whites on many measures of well-being (Blank, 2001). Likewise, although Hispanics/Latinos tend to experience inequalities in most measures of economic well-being, some subgroups of Hispanics/Latinos (e.g., Cubans) tend to fare better than others (e.g., Mexican Americans and Puerto Ricans) on these same measures.

Limited data exist to account for the heterogeneity that exists within racial/ethnic groups when conducting health research. Nonetheless, as a whole, racial/ethnic minorities (primarily African Americans/Blacks, Hispanics/Latinos, and American Indians/Alaskan Natives) tend to lag behind their White counterparts on most measures of economic well-being (e.g., educational achievement, personal and family income, employment) as well as being disproportionately represented in measures of social well-being (e.g., crime victims, being arrested and incarcerated) (Blank, 2001).

Racially structured inequalities threaten the well-being of racial/ethnic minority groups. Indeed, inequalities in any of the economic and social well-being measures may come to adversely affect health, either directly or indirectly (Nazroo, 2003). Racial inequalities in any one area of well-being tend to be closely linked to inequalities in other areas. For example, racial/ethnic groups that are less likely to complete high school are also less likely to have high earnings, and may therefore be less likely to have health insurance or access to quality health care. Thus, racial inequalities in economic and social well-being may come to create a cycle of accumulated disadvantages that can eventually take a toll on health (Blank, 2001; Braveman, Sadegh-Nobari, & Egerter, 2008).

At every stage of life, moreover, these social inequalities are linked to health. Indeed, from a life-course perspective, racial inequalities experienced in childhood, particularly those related to socioeconomic status (e.g., poverty, parental education, and income level), may also translate into accumulated disadvantage and can have significant effects on human development. Significantly, the health impacts of racial inequalities in economic and social well-being can be transmitted across generations (Braveman et al., 2008).

Without a doubt, racial inequalities in economic and social well-being have significant implications for population health. As noted by Williams and Jackson (2005, p. 331), persistent racial/ethnic disparities "violate widely shared U.S. norms of equality of opportunity and the dignity of each person" and as such, eliminating existing disparities is critical for the overall well-being of the entire society.

Cross-References

▶ Racism
▶ Socioeconomic Status (SES)

References and Readings

Blank, R. M. (2001). An overview of trends in social and economic well-being, by race. In N. J. Smelser, W. J. Wilson, & F. Mitchell (Eds.), *America becoming: Racial trends and their consequences* (pp. 21–39). Washington, DC: National Research Council/National Academies Press.

Braveman, P., Sadegh-Nobari, T., & Egerter, S. (2008). *Early childhood experiences: Laying the foundation for health across a lifetime*. Robert Wood Johnson Foundation, Commission to Build a Healthier America. Issue Brief 1: Early Childhood Experiences and Health.

Nazroo, J. Y. (2003). The structuring of ethnic inequalities in health: Economic position, racial discrimination, and racism. *American Journal of Public Health, 93*, 277–284.

Williams, D. R., & Jackson, P. B. (2005). Social sources of racial disparities in health. *Health Affairs, 24*(2), 325–335.

Racial/Ethnic Discrimination

▶ Racism

Racial/Ethnic Disparities

▶ Racial Inequality in Economic and Social Well-Being

Racism

Kristine M. Molina
Department of Psychology, University of Miami, Miami, FL, USA

Synonyms

Racial/ethnic discrimination

Definition

Racism can manifest itself in multiple forms and at multiple levels. Jones (2000) noted there are three levels by which racism can occur: (1) institutional, (2) personally-mediated, and (3) internalized. Institutional-level ("structural") racism refers to discriminatory policies, practices, laws, and procedures embedded within an organizational infrastructure that disproportionately affect a particular racial/ethnic group. At the institutional level, racism can manifest itself in terms of material conditions (e.g., differential access to and quality of health care) or access to power (e.g., differential access to resources and information). Personally-mediated (individualized) racism refers to differential treatment based on race/ethnicity, resulting from negative attitudes, assumptions, and beliefs about a racial/ethnic group. Examples of personally-mediated racism include receiving poor or no service at restaurants, being devalued, and followed around in stores.

Internalized racism refers to a person's perception of themselves, including acceptance of negative societal belies and stereotypes about one's own racial/ethnic group. In contrast to institutional and personally-mediated racism, which generally do not have an identifiable source for its perpetrator, internalized racism does (Jones, 2000). Significantly, Jones (2000) added that by the time individuals experience personally-mediated or internalized racism, they will most likely have already encountered some level of institutional racism.

In general, ethnic minorities are more likely to experience and perceive racism (Shavers & Shavers, 2006). For example, racial/ethnic minority groups disproportionately experience structural racism, such as residing in segregated neighborhoods with concentrated levels of poverty, which is a result of discriminatory housing practices. These racialized contexts put racial/ethnic minority groups at risk for a number of deleterious health outcomes, since racial segregation and the adverse conditions experienced within this context generally include crowded housing and exposure to pollutants, which can affect both mental and physical health (Shavers & Shavers, 2006). Likewise, living in structurally and economically deprived communities also means that children have less access to high-quality public schools, which limits opportunities for social mobility and may indirectly have an effect on one's life chances and achieving optimal health (Shavers & Shavers, 2006). Considering the significant implications that institutional racism has on health (and a number of social outcomes), burgeoning research in public health is now focused on examining how structural factors influence health.

Interpersonal racial/ethnic discrimination, which is a direct outgrowth of racism (Williams, Lavizzo-Mourey, & Warren, 1994), has been one of the most widely studied in health research, although studies range in its measurement (Brondolo, Gallo, & Myers, 2010; Paradies, 2006). For example, many studies have measured the construct with single-item measures such as whether a person has been treated differently or unfairly due to their race or ethnicity, and many others have measured it with scales tapping into unfair treatment that could be attributed to race, ethnicity, gender, weight, among other factors. (Brondolo et al., 2010; Krieger, 1999; Paradies, 2006). Significantly, this form of racism has been found to strongly (inversely) relate to a number of mental and physical health outcomes and health-related behaviors, including higher risk for psychiatric disorders, chronic health conditions, smoking, illicit substance use, and alcohol use/abuse (Krieger, 1999; Paradies, 2006; Shavers & Shavers, 2006). It has been argued that these

outcomes result from the chronicity and perceived stressfulness associated with experiences of differential treatment based on race and ethnicity (Williams et al., 1994).

Although less studied in health research, internalized racism has significant implications for health, particularly because this form of racism is associated with the way people think and feel about themselves and members of their own group. Thus, changes in affect and cognition, for example, may in turn affect health through behavioral and psychophysiological mechanisms, which are likely to contribute to racial/ethnic disparities in health status (Brondolo et al., 2010).

Moreover, in contrast to socioeconomic status, which has been widely cited as an underlying factor of health disparities, racism has only recently (in the last two decades) gained considerable attention as a contributor to health disparities. In fact, racial/ethnic discrimination accounts for health disparities even after accounting for a number of other factors, including socioeconomic status (Shavers & Shavers, 2006). Indeed, the role of racism in public health is argued to be of substantial importance given the wide-ranging effects it may have on population health. That is, racism is a central social determinant of health status and an underlying mechanism of racial/ethnic health disparities (Williams et al., 1994). Further, although racism manifests itself in multiple ways, all forms of racism have been shown to independently relate to adverse health outcomes. Thus, racism at any level can result in not just individual-level effects but in population-level effects as well (Brondolo et al., 2010).

Cross-References

- ▶ Ethnicity
- ▶ Racial Inequality in Economic and Social Well-being
- ▶ Social Epidemiology
- ▶ Social Factors

References and Readings

Brondolo, E., Gallo, L. C., & Myers, H. F. (2010). Race, racism and health: Disparities, mechanisms, and interventions. *Journal of Behavioral Medicine, 32*, 1–8.

Jones, C. P. (2000). Levels of racism: A theoretical framework and a gardener's tale. *American Journal of Public Health, 90*(8), 1212–1215.

Krieger, N. (1999). Embodying inequality: A review of concepts, measures, and methods for studying health consequences of discrimination. *International Journal of Health Services, 29*, 295–352.

Paradies, Y. (2006). A systematic review of empirical research on self-reported racism and health. *International Journal of Epidemiology, 35*, 888–901.

Shavers, V. L., & Shavers, B. S. (2006). Racism and health inequity among Americans. *Journal of the National Medical Association, 98*(3), 386–396.

Williams, D. R., Lavizzo-Mourey, R., & Warren, R. C. (1994). The concept of race and health status in America. *Public Health Reports, 109*(1), 26–41.

Radiation Therapy

- ▶ Cancer Treatment and Management

Radical Prostatectomy, Psychological Impact

Heather Honoré Goltz[1,2], Marc A. Kowalkouski[1], Stacey L. Hart[3] and David Latini[4]
[1]HSR&D Center of Excellence, Michael E. DeBakey VA Medical Center (MEDVAMC 152), Houston, TX, USA
[2]Department of Social Sciences, University of Houston-Downtown, Houston, TX, USA
[3]Department of Psychology, Ryerson University, Toronto, ON, Canada
[4]Scott Department of Urology, Baylor College of Medicine, Houston, TX, USA

Synonyms

Prostatectomy

Definition

Radical Prostatectomy

Radical prostatectomy (RP) is a technique used to surgically remove the prostate gland and, commonly, the seminal vesicles, lymph nodes, and surrounding tissue. Surgeons have used RP to treat benign and malignant prostate disease for over 100 years. Advances in knowledge of pelvic anatomy, technology, and surgical techniques have positioned RP as a definitive treatment for localized prostate cancer. Currently, RP refers to several surgical approaches that vary, based on site of surgical entry and whether the surgery is "open" or uses minimally invasive "laparoscopic" techniques. These options include open retropubic (RRP), perineal (RPP), and laparoscopic and robotic-assisted laparoscopic (LRP) surgical approaches. Additionally, RP approaches may be used as first-line treatment for prostate cancer or second treatment after "biochemical recurrence" (i.e., rising prostate-specific antigen levels posttreatment). Other options for treatment include radiation, hormone therapy, or surveillance instead of active treatment.

Factors Influencing Receipt of Radical Prostatectomy

A number of factors influence patients' decisions to undergo active prostate cancer treatment. Factors specifically influencing receipt of RP treatment include patients' age, comorbid conditions, prostate cancer stage and grade, and the presence of metastases. Men who are older, or who have poor health status (e.g., obesity, comorbidities) and advanced stage or grade at the time of diagnosis are more likely to receive treatment modalities other than RP. Research is divided on the association between race/ethnicity and receipt of RP. Some authors have reported that African Americans are less likely to receive RP, possibly because of more advanced disease at diagnosis; while others report no ethnic variations in treatment patterns.

Complications of Radical Prostatectomy

RP approaches produce similar cancer-control outcomes but vary in terms of peri- and postoperative complications. Perioperative complications include neurological, bowel, rectal, and bladder injuries; deep venous thrombosis; and pulmonary embolism. While there are less data for LRP, it appears that rates for many perioperative complications are low for LRP but somewhat higher than rates for RRP and RPP. More common perioperative complications include urine leakage into the abdominal cavity, restrictive narrowing of the bladder neck, and excessive blood loss requiring transfusion. Higher rates of complications occur during RRP and RPP than during LRP. Many perioperative complications can be corrected surgically after discovery. Patients opting for surgical intervention tend to experience postoperative complications at significantly higher rates than those opting for other prostate cancer treatment modalities (e.g., electron beam radiation).

Urinary and bowel incontinence and sexual dysfunction are well-documented postoperative complications of RP. For example, RP causes changes in urinary function (i.e., urgency, frequency, control, incomplete emptying), irrespective of surgical approach. Urinary effects are usually immediate, and recovery times may vary substantially for men who develop stress urinary incontinence. Men may take as long as 2–3 years before regaining some measure of continence after RP, though patients rarely experience continuous or complete incontinence. Post-RP continence rates at 12 months are between 40% and 95%, with patients at high-volume facilities achieving better continence rates. Hence, it is recommended that patients be monitored for 1 year post-RP before introducing medical or surgical interventions for urinary incontinence, as this issue may resolve with time. Findings from previous trials suggest that Kegel exercises and biofeedback prior to surgery may improve post-RP continence rates. In addition to provider- and facility-level factors, factors such as older age at the time of surgery, higher body mass index, higher prostate volume, perioperative RP bladder injury, and previous history of lower urinary tract symptoms or radiation therapy also contribute to post-RP urinary continence outcomes.

Post-RP sexual dysfunction is a complex phenomenon, with erectile dysfunction occurring in 25–85% of patients. This postoperative complication is more likely the result of a combination of the aging process, the disease itself, and RP treatment; but preoperative sexual function, comorbid health conditions, and lifestyle or behavioral factors (e.g., tobacco and alcohol use) are also contributing factors. Patients experiencing erectile difficulties immediately after surgery may gradually regain sexual function, particularly those with higher sexual functioning pre-RP; men have reported partial recovery of sexual function upwards of 2–4 years after surgery. Additional post-RP sexual effects include sterility, changes in penile length, and changes in orgasms resulting from concurrent removal of the prostate and seminal vesicles during RP.

Postoperative changes in bowel functioning are another potential complication of the RP surgical approach. Symptoms include increased frequency or urgency, diarrhea, and rectal bleeding. Men with post-RP bowel incontinence may report decline in bowel function at 4 months after surgery. As with urinary continence, many patients experiencing bowel dysfunction may recover function within the first year post-RP, while others may struggle with symptoms for years after treatment.

The Psychological Impact of Radical Prostatectomy

Advances in prostate cancer screening and detection are contributing to increasingly early prostate cancer diagnoses. A related trend concerns the often direct linkage between diagnosis and active treatment. Combined, these trends virtually ensure that younger men and those with low-risk prostate cancer will be offered treatment that they may or may not take and experience related side effects potentially for decades. In addition to medical complications, there are mental health and psychological complications.

A number of studies have examined psychosocial outcomes after prostate cancer treatment, including general, cancer-specific, and disease-specific health-related quality of life (HRQOL). General HRQOL encompasses a number of health domains related to mental/emotional, social, and physical/functional well-being. Cancer-specific instruments broadly assess the impact of having cancer across various health domains, while disease-specific instruments evaluate aspects of specific cancers. Commonly used general and cancer-specific HRQOL measures include the Medical Outcomes Survey Short Form-36 (or shorter versions) and the European Organization for Research and Treatment of Cancer Quality of Life Questionnaire (EORTC-QLQ-30), respectively. The Prostate Cancer Index (PCI) and Expanded Prostate Cancer Index: Composite (EPIC) are widely used disease-specific instruments.

Peer-reviewed literature provides strong evidence concerning the relationship between receiving RP and reduced HRQOL from chronic urinary, sexual, and bowel complications. Based on longitudinal studies, men receiving RP initially experience problems across general HRQOL domains, such as vitality/energy and role-physical well-being that substantially resolve over the course of the first year; most will return to preoperative general HRQOL levels across many domains during that time period. In terms of cancer-specific or disease-specific HRQOL, receiving RP is linked to immediate decrements in urinary and sexual function. Urinary function reaches its nadir around 3 months post-RP and gradually improves until years 2–3. At year 2, a substantial number of men's urinary function scores have not returned to preoperative levels, and they continue to report some degree of daily urine leakage. RP patients also experience decreased sexual functioning, reaching its nadir at 6 months post-RP and gradually improving through years 2–4. Yet return to presurgery levels of sexual function may not be possible for substantial numbers of patients; only about 34% of men undergoing RP return to preoperative levels by year 5. Men experiencing persistent erectile dysfunction may experience radical shifts in body image and confidence in initiating sexual intimacy, causing them to question their masculine and sexual identities.

Thus, post-RP complications cause a number of psychological, emotional, and social concerns that have lasting impact on men's HRQOL, including changes in masculine self-image and confidence and anxiety about cancer recurrence. The RP-HRQOL relationship is influenced by a number of demographic and psychosocial factors. Younger men undergoing RP experience greater recovery in terms of urinary and sexual function than their older male counterparts at 1-year post-prostatectomy. They also report lower urinary and sexual bother scores on average than older men. Men who are married or white are significantly more likely to return to pre-RP general HRQOL levels than their unmarried or racial/ethnic minority counterparts. Married men experience greater recovery in general health and social well-being domains; while those who are white experience gains in the physical, social well-being, and role-physical domains. Moderating or mediating psychosocial factors include health literacy, perceived stress, treatment satisfaction, and fear of recurrence.

Interventions exist that ameliorate many of the post-prostatectomy physical complications. For example, nonpharmacological interventions such as Kegel exercises or erectile aides (e.g., penile pumps) are available. Some RP patients may also experience enhanced sexual functioning with PDE-5 inhibitor usage; over one half of patients who initiate pharmacological penile rehabilitation recover natural erectile function by 18 months post-RP. Interestingly, a number of men with erectile difficulties will discontinue treatment after the first failed intervention, despite evidence suggesting that men attempting two or more options are more likely to find effective treatment.

A growing number of randomized controlled trial-tested interventions are targeting prostate cancer patients and caregivers in clinically modifiable areas, such as physical and psychological symptom management, literacy and culturally appropriate educational materials, decisional aids, marital communication and adjustment, and other areas. Integrative psychological and behavioral approaches to prostate cancer treatment-related complications are warranted for improving HRQOL after RP. Behavioral medicine researchers and clinicians will increasingly be called upon to lend their expertise as part of multidisciplinary teams or in disseminating study results within clinical settings.

References and Readings

Bokhour, B. G., Clark, J. A., Inui, T. S., Silliman, R. A., & Talcott, J. A. (2001). Sexuality after treatment for early prostate cancer: Exploring the meanings of "erectile dysfunction." *Journal of Internal Medicine, 16*, 649–655.

Cooperberg, M. R., Moul, J. W., & Carroll, P. R. (2005). The changing face of prostate cancer. *Journal of Clinical Oncology, 23*(32), 8146–8151.

Eton, D. T., & Lepore, S. J. (2002). Prostate cancer and health-related quality of life: A review of the literature. *Psycho-Oncology, 11*, 307–326.

Knight, S. J., & Latini, D. M. (2009). Sexual side effects and prostate cancer treatment decisions: Patient information needs and preferences. *Cancer Journal, 15*(1), 41–44.

Le, J. D., Cooperberg, M. R., Sadetsky, N., Hittleman, A. B., Meng, M. V., Cowan, J., et al. (2010). Changes in specific domains of sexual function and sexual bother after radical prostatectomy. *British Journal of Urology International, 106*(7), 1022–1029.

Michaelson, M. D., Cotter, S. E., Gargollo, P. C., Zietman, A. L., Dahl, D. M., & Smith, M. R. (2008). Management of complications of prostate cancer treatment. *CA: Cancer Journal for Clinicians, 58*, 196–213.

Muller, A., Parker, M., Waters, B. W., Flanigan, R. C., & Mulhall, J. P. (2009). Penile rehabilitation following radical prostatectomy: Predicting success. *Journal of Sexual Medicine, 6*(10), 2806–2812.

Namiki, S., & Arai, Y. (2010). Health-related quality of life in men with localized prostate cancer. *International Journal of Urology, 17*, 125–138.

Sandhu, J. S., & Eastham, J. A. (2010). Factors predicting early return of continence after radical prostatectomy. *Current Urology Reports, 11*, 191–197.

Schover, L. R., Fouladi, R. T., Warneke, C. L., Neese, L., Klein, E. A., Zippe, C., et al. (2002). The use of treatments for erectile dysfunction among survivors of prostate carcinoma. *Cancer, 95*(11), 2397–2407.

Shavers, V. L., & Brown, M. L. (2002). Racial and ethnic differences in the receipt of cancer treatment. *Journal of the National Cancer Institute, 94*(5), 334–357.

Sriprasad, S., Feneley, M. R., & Thompson, P. M. (2009). History of prostate cancer treatment. *Surgical Oncology, 18*, 185–191.

Wilt, T. J., MacDonald, R., Rutks, I., Shamliyan, T. A., Taylor, B. C., & Kane, R. L. (2008). Systematic review: Comparative effectiveness and harms of treatments for clinically localized prostate cancer. *Annals of Internal Medicine, 148*(6), 435–448.

Wittman, D., Northouse, L., Foley, S., Gilbert, S., Wood, D. P., Balon, R., et al. (2009). The psychosocial aspects of sexual recovery after prostate cancer treatment. *International Journal of Impotence Research, 21*, 99–106.

Wright, J. L., Lin, D. W., Cowan, J. E., Carroll, P. R., Litwin, M. S., & The CaPSURE Investigators. (2007). Quality of life in young men after radical prostatectomy. *Prostate Cancer and Prostatic Diseases, 11*(1), 67–73.

Random-Coefficient Model

▶ Hierarchical Linear Modeling (HLM)

Random-Coefficient Regression Modeling

▶ Multilevel Modeling

Random-Effects Modeling

▶ Multilevel Modeling

Randomization

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

Randomization is a process of randomly assigning experimental subjects to one of the treatment groups so that many potential influences that cannot be controlled for (e.g., height, weight) or cannot be determined by observation (e.g., specific metabolic pathway influences in pharmaceutical clinical trials) are likely to be as frequent in one treatment group as they are in the other. The goal of randomization is to eliminate bias.

Randomization occurs after a subject's eligibility for a clinical trial has been determined and before any experimental data are collected. The process facilitates the random assignment of subjects to different treatment groups with the intent of avoiding any selection bias in subject assignment.

As discussed in the entry titled "▶ Hypothesis Testing," inferential statistics requires the random assignment of subjects to different treatment groups to allow differences in responses between treatment groups to be connected to the treatments received. Randomization means that other potential sources of influence on the data have been randomly allocated to each treatment group. That is, subjects have an independent (and usually, but not necessarily, equal) chance of being in the different groups. While subjects are typically randomized to two treatment groups in a 1:1 ratio, generating the same number of subjects in each group, other randomization ratios can be used. For example, a ratio of 2:1 for an active treatment versus a placebo treatment would mean that two thirds of the subjects would be randomized to the treatment group and one third to the placebo group. Such an unequal randomization ratio has various implications, including the consequence that the statistical power to detect a difference between the groups is not as high as it would be if the same number of subjects had been used and the number of subjects in each group had been equal.

As noted, the goal of randomization is to eliminate bias. This includes subject bias, based on their knowledge of which treatment group they have been assigned to (this is not possible on all occasions, and can be particularly difficult in studies of behavioral medicine interventions), and investigator bias. Investigator bias is eliminated by preventing researchers from deliberately assigning subjects to one treatment group or the other. Two possible unconscious or conscious biases on the part of the researcher that are thus removed are an inclination to place less healthy subjects in the treatment group receiving the intervention they believe to be most beneficial, and an inclination

to place the more healthy subjects in the group receiving a "favored" intervention to demonstrate its superiority.

Cross-References

- ▶ Bias
- ▶ Hypothesis Testing
- ▶ Randomized Clinical Trial

Randomized Clinical Trial

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Randomized concurrently controlled clinical trial; Randomized controlled clinical trial

Definition

A randomized clinical trial is one in which the subjects in each treatment group have been placed in that group by a randomization procedure. Randomization involves randomly assigning experimental subjects to one of the treatment groups so that many potential influences that cannot be controlled for (e.g., height, weight) or cannot be determined by observation (e.g., specific metabolic pathway influences that may be relevant to the efficacy or safety of interventions in the trial) are likely to be as frequent in one treatment group as they are in the other (Turner, 2010).

Description

The fundamental goal of randomization is to eliminate bias (or, pragmatically, to reduce it as much as possible). This includes subject bias, based on their knowledge of which treatment/intervention group they have been assigned to, and investigator bias. Investigator bias is eliminated by preventing investigators from deliberately assigning patients to one treatment group or the other. Two possible unconscious or conscious biases on the part of the investigators that are thus removed are an inclination to place less healthy subjects in the treatment group receiving the intervention they believe to be most beneficial, and an inclination to place the more healthy subjects in the investigational intervention group to demonstrate its superiority.

Randomized concurrently controlled clinical trials are generally regarded as the "gold standard" for providing compelling evidence that an intervention is effective. The treatment can be of various kinds, including pharmacological and behavioral. In the realm of behavioral medicine, two examples of interventional treatments are physical exercise and cognitive behavioral therapy. These interventions would be tested against one or more control interventions. These can be "active controls," i.e., an intervention that is established to be efficacious in this context. They can also be "placebo controls," i.e., interventions that are not able to be efficacious in this context, but whose implementation involves as many as possible of the same demands and benefits that the interventional treatment makes on, and provides to, subjects in the interventional group. Matthews (2006) commented as follows:

"Over the last two to three decades randomized concurrently controlled clinical trials have become established as the method which investigators must use to assess new treatments if their claims are to find widespread acceptance. The methodology underpinning these trials is firmly based in statistical theory, and the success of randomized clinical trials perhaps constitutes the greatest achievement of statistics in the second half of the twentieth century."

Compelling evidence is sought that the investigational intervention is statistically significantly (and then clinically significantly) more effective than the comparator intervention. Such evidence is generated via the use of inferential hypothesis testing and a resultant p-value of less than 0.05.

(Despite its attained prominence, the value of 0.05 was not ordained, but conceived by the renowned statistician Sir Ronald Fisher. Had he decided, for example, that odds of 1 in 25, with an analogous p-value of 0.04, were more suitable for this purpose than odds of 1 in 20, modern science might be held to a different standard.)

Randomization occurs after a subject's eligibility for a clinical trial has been determined and before any experimental data are collected. The process of randomization is facilitated by the generation of a randomization list. This list is generated (often by a random-number generator) in advance of recruiting the first subject. The list is generated under the direction of the trial statistician, but, to maintain confidentiality, is not released to the statistician until the completion of the study.

Inferential statistics requires the random assignment of subjects to different treatment groups to allow differences in responses between treatment groups to be connected to the treatments administered. Randomization means that other potential sources of influence on the data have been randomly allocated to each treatment group. That is, subjects have an independent (and usually, but not necessarily, equal) chance of receiving either the investigational intervention or a control intervention.

Kay (2007) highlighted several methods of randomization, including simple randomization, block randomization, and stratified randomization. Simple randomization involves assigning treatments to subjects in a completely random way. While this strategy is attractively simple, it is not advisable in the case of small trials. In the example of a trial involving 30 subjects randomized to two treatment groups, the probability of a 15–15 split, the most powerful from a statistical analysis point of view, is only 0.144, while the probability of a split of 11–19 or even more unbalanced is 0.20. While 30 subjects is a small number that is used for illustrative purposes here, some researchers advocate not using simple randomization schedules in trials with less than 200 participants. In such trials the stratified randomization approach is recommended. Many clinical trials in behavioral medicine are likely to involve such numbers of subjects, in contrast to therapeutic confirmatory trials in the pharmaceutical industry, where several thousands of subjects are typically employed.

Concurrent control is a critical feature of these trials. The term "control" has already been discussed here. The term "concurrent" simply means that the control treatment should be given at the same point in time (and also under the same conditions) as the intervention of interest.

Cross-References

▶ Mode

References and Readings

Kay, R. (2007). *Statistical thinking for non-statisticians in drug regulation*. Chichester, UK: Wiley.

Matthews, J. N. S. (2006). *Introduction to randomized controlled clinical trials* (2nd ed.). Boca Raton, FL: Chapman & Hall/CRC.

Turner, J. R. (2010). *New drug development: An introduction to clinical trials* (2nd ed.). New York: Springer.

Randomized Concurrently Controlled Clinical Trial

▶ Randomized Clinical Trial

Randomized Controlled Clinical Trial

▶ Randomized Clinical Trial

Randomized Controlled Trial

▶ Clinical Trial
▶ Randomized Clinical Trial

Randomized Experimental Design

▶ Experimental Designs

Raynaud's Disease and Stress

Leah Rosenberg[1] and Sarah Piper[2]
[1]Department of Medicine, School of Medicine, Duke University, Durham, NC, USA
[2]Institute of Metabolic Science, Addenbrookes Hospital, Metabolic Research Laboratories, University of Cambridge, Cambridge, UK

Synonyms

Idiopathic Raynaud's Phenomenon; Primary Raynaud's Phenomenon

Definition

Raynaud's disease is a reversible vasospastic phenomenon triggered in susceptible patients by exposure to cold and/or emotional stress. The condition is characterized by arterial and arteriolar vasoconstriction, most commonly affecting the distal extremities such as the hands or, less commonly, the feet. The vasoconstriction produces a characteristic pallor and cyanosis of the involved digits (ischemic phase) followed by subsequent reperfusion and digit erythema, or redness.

Description

General Information

Raynaud's disease, or primary Raynaud's phenomenon, was first described in 1862 by Maurice Raynaud, who observed the characteristic color changes of the hands of affected patients during episodes of vasospasm. Modern medicine differentiates Raynaud's disease, or primary Raynaud's phenomenon, which is an idiopathic disorder, from secondary Raynaud's phenomenon, which is commonly associated with rheumatologic or autoimmune causes and which often has a more severe clinical course.

Raynaud's disease predominantly affects females, with an estimated prevalence of 6–20% of women and 3–12.5% of men, and is more common in colder climates. The symptoms in Raynaud's disease typically begin prior to the age of 30, may involve associated pain or numbness during episodes of vasoconstriction, and tend to affect the extremities symmetrically. They can vary considerably in severity, with most individuals experiencing mild to moderate symptoms and others experiencing symptoms that are intermittently debilitating. Rarely, episodes of vasospasm in Raynaud's disease can progress to ulceration or necrosis of the involved digits.

Role of Stress

The exact relationship between emotional stress and Raynaud's disease is not clear. While most episodes of vasospasm in Raynaud's disease are caused by exposure to cold temperatures, emotional stress has long been thought to play a role in precipitating symptoms as well. In fact, management of Raynaud's disease during the mid-1900s fell under the purview and management of psychiatry and psychoanalysis, treated with questionable success with psychotherapeutic techniques. In more recent studies, patients have reported a role for stress in triggering Raynaud's attacks in up to 33% of episodes, experienced by a self-reported 21% of Raynaud's disease sufferers. Initially, it was hypothesized that Raynaud's patients who were susceptible to stress-triggered attacks may be more likely to manifest higher levels of anxiety in general, termed "trait anxiety," although the accuracy of this characterization is unclear. Studies in the 1980s failed to show any significant differences in measures of anxiety or "neuroticism" based on psychologic questionnaires. A study of the quality-of-life impact of primary Raynaud's phenomenon suggested that patients suffering from the disease reported worse quality of life than control subjects and were more likely to report the subjective experience of moderate to severe anxiety symptoms. It remains uncertain, however, whether these findings inform an underlying psychiatric predisposition among Raynaud's sufferers to stress-triggered episodes or if the results are simply commonly seen complications of long-standing chronic disease.

Suspected Pathogenesis

The proposed pathogenesis of Raynaud's disease is not fully elucidated but seems to be closely related to the sympathetic nervous system, a physiologic mechanism triggered frequently by physiologic or emotional stress. Scientists have found that Raynaud's disease sufferers show an increased peripheral responsiveness to circulating catecholamines, or stress hormones, which are increased in all individuals in response to cold temperatures or emotional stress. People diagnosed with Raynaud's disease have been shown to have an increased sensitivity of the alpha-2 adrenergic receptors in peripheral blood vessels that detect these hormones, perhaps either through altered characteristics of the receptors themselves or through an increased density of receptors. By this model, levels of circulating catecholamines caused by emotional stress that would not typically trigger vasoconstriction in normal individuals could understandably trigger vasospasm in patients who are more sensitive to their effects, either with or without a predisposition to higher anxiety states.

Researching Stress and Raynaud's Disease

Efforts to study the effects of stress in Raynaud's disease have frequently produced equivocal and at times conflicting results. Early research in the 1940s and 1950s focused on measuring finger temperature in relation to emotional states to determine if emotional distress could cause vasospasm. While some initial studies documented correlative evidence of markedly decreased finger temperatures in Raynaud's patients exposed to emotional stressors, this correlation was not consistently replicated over the next decades. Some studies even documented an increased finger temperature among Raynaud's sufferers in response to a provoked stress responses.

More recently, efforts to delineate the effect of stress in triggering Raynaud's attacks have revealed the possibility of a patient cohort that shows an increased susceptibility to emotional stress-triggered Raynaud's attacks, characterized by a higher trait anxiety scores than other Raynaud's sufferers. The Raynaud's Treatment Study in 2001 suggested that, while perceived stress alone did not seem to predict an increase in Raynaud's disease, increased anxiety scores were associated with more frequent attacks, particularly at warmer temperatures, as well as a higher reported severity of attacks at all temperatures and increased pain at temperatures greater than 40°. The observed effects were small but significant. It has been proposed that the effects of stress carry greater impact at higher temperatures than lower temperatures because of the absolute increase in cold-induced vasospasm below 40°. The effects of stress may only be observable at higher temperatures, where vasospasm is less universally triggering for Raynaud's sufferers.

Treatment

Understandably, the possible role of stress in triggering Raynaud's symptoms has led to the development and study of stress reduction-based interventions. Early recommendations of relaxation techniques have not shown any statistical benefit in studies. Cognitive-behavior treatments have been tested in small populations, again without any demonstrated benefit. Among nonpharmacologic treatments for Raynaud's disease, behavioral treatments seem to be the most effective in diminishing symptoms. For an in-depth discussion of behavioral treatments in Raynaud's disease, refer to "Raynaud's Disease and Behavioral Treatments."

References and Readings

Bakst, R., Merola, J. F., Franks, A. G., & Sanchez, M. (2008). Raynaud's phenomenon: Pathogenesis and management. *Journal of the American Academy of Dermatology, 49*(4), 633–653.

Brown, K. M., Middaugh, S. J., Haythornthwaite, J. A., & Bielory, L. (2001). The effects of stress, anxiety, and outdoor temperatures on the frequency and severity of Raynaud's attacks: The Raynaud's treatment study. *Journal of Behavioral Medicine, 24*(2), 137–153.

De Angelis, R., Salaffi, F., & Grassi, W. (2008). Health-related quality of life in primary Raynaud phenomenon. *Journal of Clinical Rheumatology, 14*(4), 206–210.

Goreczny, A. (Ed.). (1995). *Handbook of health and rehabilitative psychology*. New York: Plenum Press.

Mittelmann, B., & Wolff, H. G. (1939). Affective states and skin temperature: Experimental study on subjects with "Cold Hands" and Raynaud's syndrome. *Psychosomatic Medicine, 1*, 271–292.

Raynaud's Disease: Behavioral Treatment

Leah Rosenberg[1] and Sarah Piper[2]
[1]Department of Medicine, School of Medicine, Duke University, Durham, NC, USA
[2]Institute of Metabolic Science, Addenbrookes Hospital, Metabolic Research Laboratories, University of Cambridge, Cambridge, UK

Synonyms

Idiopathic Raynaud's Phenomenon; Primary Raynaud's Phenomenon

Definition

Raynaud's disease is a reversible vasospastic phenomenon triggered in susceptible patients by exposure to cold or emotional stress. The condition is characterized by arterial and arteriolar vasoconstriction, most commonly affecting the distal extremities, that produces a characteristic pallor and cyanosis of the involved digits (ischemic phase) followed by subsequent reperfusion and digit erythema, or redness.

Description

Symptoms of Raynaud's disease, including vasospasm and associated pain or numbness, are frequently amenable at least in part to conservative approaches to treatment. These initial approaches focus on avoidance of potential environmental triggers which can vary according to individual patients but most often relates to exposure to cold temperatures. To minimize the risk of cold exposure, patients are advised to practice full body insulation since other areas of exposed skin besides the hands can precipitate attacks. Patients are advised to avoid sudden changes in temperature as well. Discontinuation of known vasoconstrictive medications, smoking cessation, avoidance of vibration, and discontinuation of caffeine consumption have also been counseled. Some individuals also benefit from a swing-arm maneuver that causes pooling of blood in the distal extremities to counteract the effects of vasoconstriction, shown in some people to abort the onset of a Raynaud's attack. While a majority of Raynaud's disease sufferers respond well to these recommendations with at least some reduction in the severity and frequency of symptoms, others require more aggressive pharmacologic or even behavioral interventions, described below.

For patients who do not respond to conservative interventions, the next step in treatment is frequently the initiation of a medication to inhibit vasospasm, such as calcium channel blockers. This class of medication has been shown in multiple studies to help decrease the rate of Raynaud's attacks and to mitigate the severity of attacks in a significant number of patients. Reports of side effects from these medications are common, including orthostatic hypotension, edema, headache, tachycardia, and constipation. Given poor tolerance of pharmacotherapy in some patients, behavioral treatments have been concurrently developed and researched to provide an alternative to medications. These interventions, most notably thermal biofeedback, autogenic training, and classical conditioning, have been tested head to head with medical treatments in some studies and have been shown to have potential benefit.

Behavioral treatments have been developed alongside medical therapies in treating Raynaud's disease, in part due to the known side effects of many medications but also due to early evidence that principles of biofeedback may be very effective in controlling patient's symptoms.

Biofeedback is a behavioral technique developed in the last 40 years that links involuntary physiologic processes, such as heart rate, which often occur below the level of conscious awareness, to sensory stimuli that are easily perceptible and able to be tracked to note variations in these unconscious processes. Patients then use this information to manipulate the target physiologic response (e.g., slowing heart rate through conscious focus).

This technique has been applied to Raynaud's disease in several forms, the best-researched of which is thermal biofeedback. Thermal biofeedback (also called finger temperature biofeedback) is a behavioral treatment first studied in Raynaud's patients in the 1970s. The technique instructs individuals in the use of sensory feedback to convey an increase in finger temperature by facilitating peripheral vasodilation. This technique has been shown in several small studies to have strong efficacy in reducing Raynaud's attacks by up to 92.5%, particularly when the initial training was conducted during exposure to cold temperatures. Improvements in symptoms have been observed as long as 3 years from the time of initial training. Further studies, including those comparing the effectiveness of thermal biofeedback to standard medical therapies, have been inconclusive. The wide variability in study results is thought to be attributable to the underlying difficulty in teaching the technique, which often requires intensive instruction to achieve mastery. Patients who do achieve mastery are frequently able to facilitate hand rewarming through practiced conscious intervention in high-risk environments that would previously have trigged episodes of vasospasm. Despite these results, a recent head-to-head comparison of nifedipine, a standard medical treatment for Raynaud's, and thermal biofeedback was unimpressive, although authors highlighted the limitations in assessing the behavioral technique due to strong evidence that participants had not achieved mastery of the technique during the study, which likely significantly reduced its apparent effectiveness.

Another technique that may be in some part considered a relative of biofeedback is autogenic training. This technique was first introduced in the early 1930s within the mental health community to promote stress management and relief from somatic symptoms often related to anxiety. AT is a relaxation method consisting of passive self-monitoring of physiologic sensations such as heart rate to then inform self-initiated verbal cues intended to cause a desired change in physiologic effect or modification of the initial sensation. This technique has been applied to treatment models in Raynaud's disease through cultivation of an awareness of finger temperature and the application of cuing to trigger peripheral vasodilatation. The effectiveness of this technique is unclear. Some studies have shown an increase in finger temperature (often a measured outcome in Raynaud's research) that equals the observed efficacy of thermal biofeedback interventions, with reports of similar clinical efficacy. Combining the two techniques does not appear to convey any additional benefit, however.

Classical conditioning is a principle that pairs a behavior with a predictable outcome, such as salivation in response to the presentation of food, to an alternate stimulus to link the alternate stimulus with the outcome through repetitive training. In application to Raynaud's disease, Raynaud's disease sufferers have been conditioned to respond to exposure to cold temperatures with peripheral vasodilatation after serial exposures to cold ambient temperatures with concurrent immersion of the person's hands in warm water. Over time, patients were observed to have increased blood flow simply with exposure to cold temperatures even without the added exposure of warm water immersion. This technique, while not as well studied as thermal biofeedback, also has been shown to have a possible effect in decreasing the frequency and severity of Raynaud's attacks.

Less well-studied interventions have included progressive relaxation and guided imagery. These techniques focus on relieving emotional stress and providing improved relaxation. Progressive relaxation is a tool that involves the progressive contraction of muscle groups followed by sequential release of muscle tone to achieve a global state of relaxation. Guided imagery is a technique that teaches patients to directed imagining of calming or peaceful imagery to facilitate relief from anxiety and stress. Both techniques been studied in conjunction with

other behavioral interventions such as thermal biofeedback, but not as a stand-alone treatment.

Behavioral treatments such as thermal biofeedback, autogenic training, and classical conditioning have been shown to have variable effects on the symptom frequency and severity of Raynaud's disease sufferers, although evidence overall is supportive for a role of these treatments among this patient population. Certainly, further research will help to clarify the role of these interventions and to elucidate the mechanism by which their effect is mediated.

Cross-References

▶ Raynaud's Disease and Stress

References and Readings

Bakst, R., Merola, J. F., Franks, A. G., & Sanchez, M. (2008). Raynaud's phenomenon: Pathogenesis and management. *Journal of the American Academy of Dermatology, 49*(4), 633–653.

Baum, A. (Ed.). (2001). *Handbook of health psychology*. Mahwah, NJ: Lawrence Erlbaum.

Freedman, R., Ianni, P., & Wenig, P. (1983). Behavioral treatment of Raynaud's disease. *Journal of Consulting and Clinical Psychology, 51*, 539–549.

Garcia-Carrasco, M., Jimenez-Hernandez, M., & Escarcega, R. (2008). Treatment of Raynaud's phenomenon. *Autoimmunity Reviews, 8*, 62–68.

Jobe, J. B., Sampson, J. B., Roberts, D. E., & Kelly, J. A. (1986). Comparison of behavioral treatments for Raynaud's disease. *Journal of Behavioral Medicine, 9*(1), 89–96.

Karavidas, M. K., Tsai, P. S., Yucha, C., McGrady, A., & Lehrer, P. M. (2006). Thermal biofeedback for primary Raynaud's phenomenon: A review of the literature. *Applied Psychophysiology and Biofeedback, 31*(3), 203–216.

Middaugh, S. J., Haythornthwaite, J. A., Thompson, B., Hill, R., Brown, K. M., Freedman, R. R., Attanasio, V., Jacob, R. G., Scheier, M., & Smith, E. A. (2001). The Raynaud's treatment study: Biofeedback protocols and acquisition of temperature biofeedback skills. *Applied Psychophysiology and Biofeedback, 26*(4), 251–278.

Stetter, F., & Kupper, S. (2002). Autogenic training: A meta-analysis of clinical outcome studies. *Applied Psychophysiology and Biofeedback, 27*, 45–98.

Reactivity

▶ Psychophysiologic Reactivity

Readiness for Return-to-Work (RRTW)

Timothy Wolf
Department of Occupational Therapy and Neurology, Program in Occupational Therapy, St. Louis, MO, USA

Synonyms

Vocational assessment

Definition

Readiness for return to work is the complex decision-making behavior related to returning to work after an injury or illness. In this process, individuals must consider their self-efficacy surrounding their ability to return to work; the interaction of the many third parties involved in returning to work including the employer and the healthcare and insurance providers; as well as the state of their disability as it interacts with the demands of the job. This term was originally conceived by Franche and Krause (2002) in their conceptual model for readiness for return to work.

Description

Work disability is often determined by an evaluation of impairments following injury or illness. This most often focuses on physical impairments which is apparent in most work evaluation methodologies, i.e., functional capacity evaluation. The limitation in this approach is that the

healthcare community has long known that work disability involves a lot of factors beyond what impairment can explain. Workers who are seemingly more impaired may return to work very quickly and without difficulty while workers who are seemingly less impaired may struggle with returning to work or never attempt to return to work at all. The notion of readiness for return to work is used to help address this limitation in current work assessment methodologies. The concept for readiness for return to work was developed by Franche and Krause (2002) to provide a framework for work disability research and practice that would account for the multiple factors that influence work disability. These factors include physical (i.e., impairments), psychological (i.e., self-efficacy related to completing the demands of the job), and social (i.e., the employee's interactions in the work environment, with healthcare providers/health insurance companies). They developed the Readiness for Return to Work model in order to combine these factors to explain return to work behaviors. The Readiness for Return to Work model incorporates two well-known theoretical models to explain a work-disabled person's behavior regarding return to work. First, the Phase Model of Occupational Disability provides a framework for understanding the developmental component of disability (Krause & Ragland, 1994). Second, the Readiness for Change Model provides a framework for understanding the motivation to change behavior relative to returning to work (Prochaska & Diclemente, 1983).

Further work with the Readiness for Return to Work model has focused on the application of the Readiness for Change Model to work behavior to help understand work-disabled individuals' motivation to return to work following injury or illness. There are five stages within the Readiness for Change Model that an individual will progress through (Prochaska & Diclemente, 1983):

- *Precontemplation*: Workers are not thinking about returning to work and are not making plans to do so either.
- *Contemplation*: Workers are considering return to work but are still not engaging in the planning process to do so.
- *Preparation for action*: Workers are seeking information about returning to work and are also looking for feedback on their ability to meet the demands of the workplace. In addition, workers are making plans to return to work.
- *Action*: Workers return to work in some capacity.
- *Maintenance*: Workers use their skills and social support to help cope with adversity they encounter in returning to work in order to maintain their ability to work.

Within this model, it is important to note that workers can relapse anytime and start regressing through these stages. The Readiness for Return-to-Work Scale (RRTW) (Franche, Corbiere, Lee, Breslin, & Hepburn, 2007) is used to determine the worker's present stage. The results might be helpful for healthcare providers to determine support and resources needed for the worker to be able to return to work. This information can also be use to tailor interventions for the worker in order to improve the effectiveness of rehabilitation efforts.

Cross-References

▶ Functional Versus Vocational Assessment

References and Readings

Franche, R. L., Corbiere, M., Lee, H., Breslin, F. C., & Hepburn, C. G. (2007). The readiness for return-to-work (RRTW) scale: Development and validation of a self-report staging scale in lost-time claimants with musculoskeletal disorders. *Journal of Occupational Rehabilitation, 17*, 450–472.

Franche, R. L., & Krause, N. (2002). Readiness for return to work following injury or illness: Conceptualizing the interpersonal impact of health care, workplace, and insurance factors. *Journal of Occupational Rehabilitation, 12*, 233–256.

Krause, N., & Ragland, D. R. (1994). Occupational disability due to low back pain: A new interdisciplinary classification based on a phase model of disability. *Spine, 19*, 1011–1020.

Prochaska, J. O., & Diclemente, C. C. (1983). Stages and process of self-change of smoking: Toward an integrative model of change. *Journal of Consulting and Clinical Psychology, 51*, 390–395.

RE-AIM Guidelines

Paul A. Estabrooks[1], Samantha M. Harden[2] and Kacie C. Allen[2]
[1]Translational Obesity Research Program, Virginia Tech Riverside, Roanoke, VA, USA
[2]Human Nutrition, Foods, and Exercise, Virginia Tech, Roanoke, VA, USA

Synonyms

Evaluation of potential public health impact; Internal and external validity issues

Definition

RE-AIM is an acronym for an assessment and planning framework that addresses individual and setting-level factors that are related to the overall public health impact of health behavior interventions (Glasgow, Vogt, & Boles, 1999). Interventions are defined in a broad sense to include community programs, clinical practices, environmental changes, and policy approaches intended to promote healthy lifestyle behaviors. The acronym represents the following dimensions: reach, effectiveness/efficacy, adoption, implementation, and maintenance. Reach is an individual-level measure and is defined as the number of participants, participation rate among of the target population, and representativeness of the participants. Efficacy or effectiveness is also an individual-level measure and is defined as change in the primary outcome, impact on quality of life, and assessment of potential negative outcomes. Efficacy is assessed under optimal circumstances, while effectiveness is assessed under more context relevant circumstances (Flay, 1986). Adoption is a setting-level indicator and is defined as the number, percent and representativeness of settings and educators who agree to deliver an intervention. Another setting-level dimension is implementation which determines the cost of and extent to which the intervention is delivered as intended and, in some cases, is defined as the degree of participant adherence to intervention protocol (Estabrooks & Gyurcsik, 2003). Finally, maintenance is conceptualized at both the individual and setting level. At the setting level, maintenance is defined as the extent to which an intervention is sustained over time (Glasgow et al., 1999). At the individual level, maintenance is defined as the degree to which behavior changes are sustained 6 months or longer after the intervention is complete (Glasgow et al.).

Description

The RE-AIM framework was developed in response to the need to expand the evaluation of health behavior interventions beyond simply assessing efficacy or effectiveness to include criteria related to external validity. The underlying proposition of the RE-AIM framework is that evidence which is contextual, practical, and robust is more likely to facilitate the translation of health behavior interventions into typical clinical or community practice. Further, considering each of the RE-AIM dimensions during the planning phase is hypothesized to result in interventions that can be widely adopted by different sites and personnel, having the ability for sustained and consistent implementation at a reasonable cost, reaching large numbers of people (especially those who can most benefit), and producing replicable and long-lasting effects with minimal negative impacts (Glasgow & Emmons, 2007; Klesges, Estabrooks, Dzewaltowski, Bull, & Glasgow, 2005). Unfortunately, a number of reviews of literature investigating the degree to which RE-AIM information is reported across behavioral interventions suggest that data across the dimensions related to external validity are rarely reported and that inconsistent techniques have been used to operationalize RE-AIM assessments (Glasgow, Klesges, Dzewaltowski, Bull, & Estabrooks 2004). The remainder of this chapter will be dedicated to outlining guidelines for assessing each RE-AIM dimension.

Three general data elements are necessary to assess reach: the number of participants, the number of potential participants, and comparative information on the target population. Determining the number of individuals who participate in or are exposed to a given intervention is relatively straightforward. However, determining an appropriate denominator to calculate participation rate can be more difficult. For example, in one research project, the goal was to promote physical activity following a health maintenance visit at a local health care clinic using a 24-week program (Almeida et al., 2005). There were a number of possible denominators to choose from: the total number of adult patients, the total number of adult patients who were not meeting recommended physical activity guidelines, and the total number of patients who were not meeting recommended guidelines and came in for a visit. The key determining factor for selecting the appropriate denominator was fairly simple: the denominator that reflected insufficiently active patients who were exposed to recruitment activities. The total number of patients that attended a health maintenance visit over the course of the study recruitment was 1,518. Of those 1,518 individuals, 607 were eligible, 218 were referred, and 115 participated. Thus, the participation rate was calculated as 19% based on the sample size ($n = 115$) and the number of eligible individuals exposed to recruitment activities ($n = 607$). To determine the representativeness of the sample, the study sample was compared to the demographic profile of the catchment area of the clinic. Nonparticipants were more likely to be older females. However, the participants were representative of the racial and ethnic composition of the community (Almeida et al.).

Methods to determine the efficacy or effectiveness of a behavioral intervention have been exhaustively covered by other authors (e.g., Flay, 1986). Briefly, a comparison of the ability of an intervention(s) to change a primary study outcome either under optimal or more real-world conditions provides estimates of efficacy or effectiveness, respectively. The comparison of changes in quality of life adds a dimension to effectiveness that allows a more patient-centered perspective and standardization for comparison across interventions targeting different primary outcomes. The focus on unintended negative consequences can range from issues related to safety or changes the reduction of one health behavior in favor of increasing another (e.g., participants who quit smoking and replace that behavior with eating unhealthy snack foods). Other issues related to improving the external validity of documenting effectiveness include assessment of differential attrition across participants with different demographic profiles and the use intention-to-treat analyses.

Adoption is conceptualized similarly to reach, but addresses participating settings or delivery personnel rather than intervention participants. As with reach, three data elements are necessary: the number of participating settings, the number of potential settings where the intervention could be delivered, and comparative information on the population of potential settings. Adoption can also be assessed within a given setting and based on the staff who would ultimately deliver an intervention. A simple example of adoption at the setting level is provided in a paper on Walk Kansas, a statewide nutrition and physical activity intervention intended for delivery by a county Cooperative Extension agent in each of the 105 (i.e., denominator) counties in Kansas (Estabrooks, Bradshaw, Dzewaltowski, & Smith-Ray, 2008). Forty-eight county agents agreed to participate, reflecting an adoption rate of 46%. To determine representativeness, a survey that was administered to all Cooperative Extension agents prior to Walk Kansas was used. The results indicated that less physically active county agents were less likely to deliver the program and smaller population counties were less likely to deliver the program.

Implementation can be assessed in a number of ways: observation of the delivery of intervention components, checklists for those delivering the intervention, or the degree to which participants engage in intervention components. Further, cost of implementation can be collected using self-reported or direct observation of the resources and time needed for delivery. Schillinger,

Handley, Wang, and Hammer (2009) provide an example of reporting on the dimension of implementation of an automated telephone intervention to support diabetes self-management. They assessed the degree to which automated telephone support calls were completed across the 9-month intervention and found that 94% of the participants completed at least one automated call. Of those participants, the range of calls completed was from 22 to 39 (out of 39 possible calls). Costs of implementation were assessed at for the automated telephone intervention at $782 per participant (Schillinger et al., 2009).

Assessment of effectiveness or efficacy indicators 6 months after an intervention is completed and the degree to which an intervention is sustained in a delivery setting post research funding are the key components to evaluating the maintenance. Thus the data necessary to determine efficacy or effectiveness of an intervention (i.e., primary outcome, quality of life, and potential negative outcomes) are identical to the data necessary to determine maintenance with the only caveat being the time of assessment. As continued concerns with recidivism and poor adherence to newly acquired health behaviors, the prevalence of research on individual-level maintenance has increased. Less available in the literature is information on the degree to which programs are sustained once research is completed. This is not surprising as most research studies are not designed to lead to a sustained program over time. However, researchers embedded within delivery systems like the Veterans Administration are beginning to examine the degree to which new behavioral interventions can be sustained once the formal testing of effectiveness is completed (Stetler, Mittman, & Francis, 2008).

A common question related to the RE-AIM framework is: Which dimension is most important? Some argue that the answer is obvious – effectiveness. However, one could also argue that reach is equally important. For example, an intervention with no potential for broad reach is just as irrelevant as one with no potential for effectiveness. From a setting level, if an intervention cannot be adopted in typical delivery settings, or implemented as intended, or sustained, it will not achieve a strong public health impact even if research studies indicate it is highly effective. Still, there is some interest in understanding how the dimensions of RE-AIM may interact to generate a significant public health impact (Glasgow, Klesges, Dzewaltowski, Estabrooks, & Vogt, 2006). Future research into the interplay between reach, effectiveness, cost or adoption, implementation, and sustainability will help to inform public health practice related to health behavior interventions. Other areas of promise include examining cost across RE-AIM dimensions, rather than just as it is associated with implementation, and cost-effectiveness and the role of adaptation and local innovation in real-world implementation studies.

Cross-References

- ▶ Generalizability
- ▶ Public Health

References and Readings

Almeida, F. A., Smith-Ray, R. L., Van Den Berg, R., Schriener, P., Gonzales, M., Onda, P., et al. (2005). Utilizing a simple stimulus control strategy to increase physician referrals for physical activity promotion. *Journal of Sport & Exercise Psychology, 27*(4), 505–514.

Estabrooks, P. A., Bradshaw, M., Dzewaltowski, D. A., & Smith-Ray, R. L. (2008). Determining the impact of Walk Kansas: Applying a team-building approach to community physical activity promotion. *Annals of Behavioral Medicine, 36*(1), 1–12.

Estabrooks, P. A., & Gyurcsik, N. C. (2003). Evaluating the impact of behavioral interventions that target physical activity: Issues of generalizability and public health. *Psychology of Sport and Exercise, 4*(1), 41–55.

Flay, B. R. (1986). Efficacy and effectiveness trials (and other phases of research) in the development of health promotion programs. *Preventive Medicine, 15*(5), 451–474.

Glasgow, R. E., & Emmons, K. M. (2007). How can we increase translation of research into practice? Types of evidence needed. *Annual Review of Public Health, 28*, 413–433.

Glasgow, R. E., Klesges, L. M., Dzewaltowski, D. A., Bull, S. S., & Estabrooks, P. (2004). The future of health behavior change research: What is needed to improve translation of research into health promotion practice? *Annals of Behavioral Medicine, 27*(1), 3–12.

Glasgow, R. E., Klesges, L. M., Dzewaltowski, D. A., Estabrooks, P. A., & Vogt, T. M. (2006). Evaluating the impact of health promotion programs: Using the RE-AIM framework to form summary measures for decision making involving complex issues. *Health Education Research, 21*(5), 688–694.

Glasgow, R. E., Vogt, T. M., & Boles, S. M. (1999). Evaluating the public health impact of health promotion interventions: The RE-AIM framework. *American Journal of Public Health, 89*(9), 1322–1327.

Klesges, L. M., Estabrooks, P. A., Dzewaltowski, D. A., Bull, S. S., & Glasgow, R. E. (2005). Beginning with the application in mind: Designing and planning health behavior change interventions to enhance dissemination. *Annals of Behavioral Medicine, 29*, 66–75.

Schillinger, D., Handley, M., Wang, F., & Hammer, H. (2009). Effects of self-management support on structure, process, and outcomes among vulnerable patients with diabetes: A three-arm practical clinical trial. *Diabetes Care, 32*(4), 559–566.

Stetler, C. B., Mittman, B. S., & Francis, J. (2008). Overview of the VA quality enhancement research initiative (QUERI) and QUERI theme articles: QUERI series. *Implementation Science, 3*, 8. Retrieved from www.re-aim.org

Real-Life Blood Pressure Monitoring

► Ambulatory Blood Pressure

Reasons

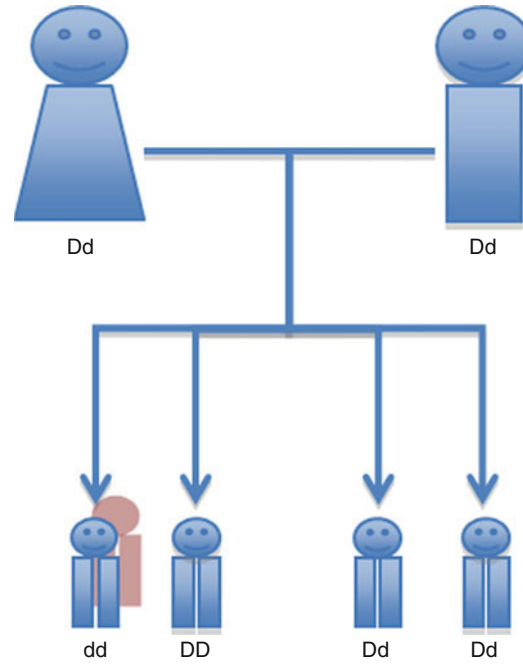
► Attribution Theory

Recessive Inheritance

Laura Rodriguez-Murillo[1] and Rany M. Salem[2]
[1]Department of Psychiatry, Columbia University Medical Center, New York, NY, USA
[2]Broad Institute, Cambridge, MA, USA

Definition

A trait that is inherited in a recessive fashion only manifests phenotypically in homozygous individuals, i.e., when the individual has two copies of the same recessive allele. Humans have two versions of all autosomal genes, called alleles, one from each parent. The recessive trait is hidden in the heterozygous individual (Dd) if the other allele is inherited in a dominant fashion, and so this person is a called a "carrier" of the recessive allele, but does not manifest the disease or trait. A recessive trait can only be passed to the offspring if both parents carry (Dd or dd) and transmit the recessive allele to their offspring. In the scenario where both parents are heterozygous carriers of the recessive trait, the children have 25% chance of inheriting two copies (dd) of the recessive allele and exhibiting the recessive trait (see pedigree figure, Fig. 1). An example of a disease with a recessive inheritance is Tay-Sachs disease that occurs when a child has two defective copies of the HEXA gene, neither of which can be successfully transcribed and form a functional enzyme product (Myerowitz & Costigan, 1988).



Recessive Inheritance, Fig. 1 Recessive inheritance diagram. Highlighted individuals are affected by the disease after inheriting one recessive allele from each carrier parent (dd)

Cross-References

- ▶ Allele
- ▶ Dominant Inheritance
- ▶ Gene
- ▶ Genotype
- ▶ Heterozygous
- ▶ Homozygous

References and Readings

Lewis, R. (2005). *Human genetics. Concepts and applications* (7th ed.). Boston: McGraw-Hill Science/Engineering/Math.

Myerowitz, R., & Costigan, F. C. (1988). The major defect in Ashkenazi Jews with Tay-Sachs disease is an insertion in the gene for the alpha-chain of beta-hexosaminidase. *Journal of Biological Chemistry, 263*(35), 18587–18589.

Strachan, T., & Read, A. P. (2003). *Human molecular genetics* (3rd ed.). London/New York: Garland Science/Taylor & Francis Group.

Recovery

- ▶ Cardiovascular Recovery
- ▶ Psychophysiologic Recovery
- ▶ Resilience

Recruitment and Retention of Research Subjects

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Recruitment of research participants

Definition

Recruitment of research subjects is the process by which individuals are recruited as potential subjects in a research study. Upon learning full details of the study by reading the Informed Consent Form, eligible individuals may or may not decide to participate: Nonetheless, they have participated in the process of recruitment.

Description

While individual investigators can play an important role in subject recruitment – their access to potential subjects likely having been a reason for their being selected to participate in the trial – the primary responsibility for subject recruitment can fall on the principal investigator/holder of the grant that is funding the behavioral medicine research study. For all but the smallest studies, creation and implementation of a recruitment strategy and plan is an important and potentially critical operational step. This plan should be a formal written document that carefully considers best-case and worst-case scenarios, takes into account various timing influences (e.g., national holidays and typical vacation periods, seasons of the year, likely onset of flu season), and has contingencies built in to address all anticipated potential recruitment barriers.

Spilker (2009) provided the following operational definition of recruitment:

- Identifying potential pools of subjects and specific individual subjects who may be eligible to enroll in the trial.
- Attracting those individuals to consider participation.
- Discussing the trial with them.
- Prescreening subjects.
- Having subjects read an informed consent form, answering any questions they may have, and then having the subjects sign the consent form.
- Conducting a more complete and formal screening procedure.
- Being able to state that subjects are now enrolled in the trial.

Recruitment is of ongoing interest to a principal investigator throughout a trial, and there are several common metrics addressing

this. One is "first subject first visit," the date on which the first subject completes his or her first visit to the investigational site. In cases where there are multiple investigational sites another metric of interest is the date when 50% of the sites have each enrolled at least one subject.

Traditional subject recruitment strategies include the following:

- Advertisements on radio and television, and in print media.
- Mailing flyers to individuals and patient advocacy groups.
- Data mining from insurance company databases.
- Using web sites to publicize the trial.
- Posting information on a hospital's bulletin board.
- Hospital staff wearing lapel pins that bring attention to the trial.

Cabell (2009) recently discussed another approach that focuses on understanding and utilizing the "patient pathway," the route by which patients receive treatment. By understanding and documenting the pathway via which patients with the disease or condition of clinical concern receive their current medical care, it becomes possible for recruitment specialists to reach out to clinicians and other behavioral medicine specialists who provide the care. This allows the specialists to access the appropriate patients and, should they be willing, recruit them as subjects for the trial.

While this entry focuses primarily on subject recruitment, it has become widely accepted that principal investigators need to devote considerable resources to subject retention as well as recruitment: The ultimate goal is for an enrolled subject to complete participation in the trial. Many characteristics of a subject's participation in the trial can influence the likelihood that he or she will complete the trial, including:

- The length of the treatment period. All subjects have personal and professional lives, and events that preclude trial completion – personal or family illness, relocating, financial difficulties – do and will happen. Some trials have relatively long treatment periods, and the researcher cannot alter this, but the length should alert the researcher to the need for appropriate contingency plans.
- How onerous participation is. If the trial's protocol requires subjects to undergo many procedures at many visits, withdrawal rate may increase. The burden of the trial should be considered at the protocol development phase, with an eye to decreasing noncritical subject tasks, such as filling out a large number of questionnaires that do not address the primary objective.
- Ease of traveling to the investigational site(s). If many subjects participating at a given site have to travel a considerable distance, and/or at considerable expense, each time they visit the site, the PI is well advised to address this proactively.

Specific retention strategies include:

- Providing reimbursement for expenses such as parking, meals, and babysitting.
- Calling subjects shortly before each visit to remind and assure them that provisions for their attendance are in place.
- Paying for, or actually providing transportation to and from the site.
- Providing escorts from and back to a parking lot, particularly if it is not well lighted, and/or in bad weather.
- Optimizing the environment in the subject waiting area. This can include having sufficient and appropriate magazines that are not 5 years out of date, providing toys for children who accompany subjects out of necessity, and ensuring that all trial staff treat the subjects courteously at all times.
- Reminding the subjects throughout the trial that their participation is extremely valuable in evaluating a new behavioral medicine intervention that, if successful, may provide considerable benefit to many patients. This can be done verbally and also by sending out regular newsletters.

Cross-References

▶ Informed Consent

References and Readings

Cabell, C. (2009). Patient recruitment: Are we looking in the right place? *International Pharmaceutical Industry, Summer issue*, 38–41.
Spilker, B. (2009). *Guide to drug development: A comprehensive review and assessment*. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins.

Recruitment of Research Participants

▶ Recruitment and Retention of Research Subjects

Recurrence Risk Ratio

Jennifer Wessel
Public Health, School of Medicine, Indiana University, Indianapolis, IN, USA

Definition

Recurrence risk ratio is a measure of familial aggregation of disease. When a genetic factor(s) cannot be measured directly, information on disease status among family members of an affected individual can be used to derive a genetic risk ratio. It can provide a rationale for performing genetic studies to identify the susceptibility alleles and has been used extensively in the mapping of complex diseases. The recurrence risk ratio, λ_R, is the ratio of disease manifestation in other family members to the affected individual compared with the disease prevalence in the general population.

The sibling recurrence risk ratio is commonly used, where the numerator would represent the risk of manifesting the disease given that one's sibling is affected:

$$\lambda S = \frac{\Pr(\text{D in a sibling}/\text{affected case})}{K}$$

where K is the prevalence of the disease in the general population.

Cross-References

▶ Allele
▶ Gene

References and Readings

Risch, N. (1990a). Linkage strategies for genetically complex traits. I. Multilocus models. *American Journal of Human Genetics, 46*, 222–228.
Risch, N. (1990b). Linkage strategies for genetically complex traits. II. The power of affected relative pairs. *American Journal of Human Genetics, 46*, 229–241.

Regional Enteritis

▶ Crohn's Disease (CD)

Regression Analysis

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Linear regression; Multiple regression; Regression modeling

Definition

In regression analysis it is assumed that a change in variable x will lead to a change in variable y. It is often the case that the researcher wishes to predict the value of y for a given value of x (Campbell, Machin, & Walters, 2007).

Consider the case of linear regression and the plotting of a fitted regression line. In cases where linear regression is appropriate there is only one independent variable. Conventionally, the dependent variable is plotted on the vertical axis (y-axis) and the independent variable is plotted on the horizontal axis (x-axis). The equation

$y = \alpha + \beta x$ is defined as the linear regression equation. Alpha (α) is the intercept, and Beta (β) is the regression coefficient (Greek letters are used to clarify that these are population parameters). The regression equation is a model that is used to describe or model the relationship between y and x. When the graph is plotted, alpha is the value of the equation when $x = 0$, and beta is the slope of the line drawn (plotted). An increase of x of one unit will be associated with a change in y of beta units.

A set of data is used to create the plot, and then to draw the regression line. Imagine a set of data describing hemoglobin levels and age in a set of 20 women. The individual subjects' data can be represented as a set of 20 paired observations, and these points can be plotted on the graph. The best fitting linear regression line is then drawn. This line enables the determination of alpha and beta. Then, for any value of x, the appropriate value of y as given by this model can be calculated.

Since outcomes in behavioral medicine research are often influenced by more than one independent variable, the linear regression model can easily be extended to the technique of multiple regression. Multiple regression can address various questions of interest, including examination of the relationship between continuous variables while allowing for a third variable, and adjusting for differences in confounding factors between experimental groups (Machin et al., 2007). Examples are provided in the "References and Readings" section of this entry.

Cross-References

- ▶ Data
- ▶ Experimental Group
- ▶ Multivariate Analysis
- ▶ Univariate Analysis

References and Readings

Campbell, M. J., Machin, D., & Walters, S. J. (2007). *Medical Statistics: A textbook for the health sciences* (4th ed.). Chichester, UK: Wiley.

Naicker, N., Richter, L., Mathee, A., Becker, P., & Norris, S. A. (2011). Environmental lead exposure and socio-behavioral adjustment in the early teens: The birth to twenty cohort. *Science of the Total Environment, 414*, 120–125.

Smith, K. J., Blizzard, L., McNaughton, S. A, Gall, S. L., Dwyer, T., Venn, A. J. (2011, December 7). Takeaway food consumption and cardio-metabolic risk factors in young adults. *European Journal of Clinical Nutrition*, (Epub ahead of print).

Soureti, A., Hurling, R., van Mechelen, W., Cobain, M., & Chinapaw, M. (2011, December 5). Moderators of the mediated effect of intentions, planning, and saturated-fat intake in obese individuals. *Health Psychology*, (Epub ahead of print).

Regression Modeling

▶ Regression Analysis

Regulation of Expression

▶ Gene Expression

Rehabilitation

Bengt H. Sjölund
University of Southern Denmark, Odense, DK, Denmark

Synonyms

Habilitation

Definition

According to the World Report on Disability, rehabilitation is "a set of measures that assist individuals who experience, or are likely to experience, disability to achieve and maintain optimal functioning in interaction with their environments" (World Health Organization [WHO], 2011).

Description

Rehabilitation literally means "redressing" (*Latin habitat – dress*). While there are many definitions of this concept, the world health organization (WHO) has defined rehabilitation as "a process aimed at enabling disabled persons to reach and maintain their optimal physical, sensory, intellectual, psychological and social functional levels. Rehabilitation provides disabled people with the tools they need to attain independence and self-determination." Thus, the aim of rehabilitation has traditionally been seen as facilitating the normalization of human functioning after injury, disease, or due to congenital defects. It is usually said to comprise a number of coordinated measures of medical, psychological, social, educational and vocational nature. The Convention of the Rights of Persons with Disabilities, in its article 26 calls for " appropriate measures . . . to enable persons with disabilities to attain and maintain their maximum independence, full physical, mental, social and vocational ability, and full inclusion and participation in all aspects of life" (United Nations, 2006).

Background. Modern rehabilitation originated in British experiences of people with spinal cord injuries during the Second World War, where the neurosurgeon Dr. Ludwig Guttman at Stoke-Mandeville pioneered the development of reliable rehabilitation programs for those with paraplegia and tetraplegia. Apart from crisis intervention, these programs placed strong emphasis on conservative treatment of the spinal fractures and on preventive measures, such as prophylactic bed positioning to avoid pressure sores, to empty the urinary bladder at regular intervals, and to train adequate techniques for breathing and coughing in high injuries. Effective techniques for independently taking care of personal hygiene as well as transferring were also developed, e.g., between a bed, a chair, and a wheelchair, including the effective handling of transport vehicles. After post-acute rehabilitation, a lifelong follow-up ensued. The result was that the remaining number of life years increased from 1 to 2 years after the injury (even in young people), to today's normal life span for a paraplegic person, usually at an independent level, and a moderately reduced life span for the tetraplegic person, as a rule partly dependent.

In the mid-twentieth century, Physical and Rehabilitation Medicine (PRM) was established as an independent specialty in Western health care. It currently defines itself as "concerned with the promotion of physical and cognitive functioning, behavior, quality of life (activities and participation) and with the prevention, diagnosis, treatment and rehabilitation management of people with disabling medical conditions and co-morbidity across all ages" (Gutenbrunner, Ward, & Chamberlain, 2007).

Rehabilitation services have gradually been expanded to include people disabled by stroke, neurological disease, traumatic brain injury, chronic pain, cardiac and pulmonary insufficiency, cancer, mental disorders, and other disabling conditions. Generally speaking, rehabilitation can be considered a re-adaptive process, where the disabled person adapts his/her set of values to a different, more restricted life situation. Originally, rehabilitation was based on a biomedical model, where problems in functioning of an individual were seen as due to a deviation from "normal" organ function. In the 1970s, the social model of disability was launched by disabled people's organizations, where problems in functioning were instead viewed as society's exclusion (by its design and organization) of such persons. These models have more recently been replaced by the biopsychosocial model of disability, recognizing biological as well as social factors (ICF, WHO, 2001). This model is now acknowledged among both organizations of people with disabilities and among professional organizations.

The Interdisciplinary Team

It was recognized that many different health professions apart from physicians and nurses were necessary in the daily work to fulfill the multiple needs of people receiving rehabilitation services. This led to the development of *rehabilitation teams*, supervised by the rehabilitation physician. Today, these teams ideally contain psychologists (with cognitive-behavioral or

neuropsychological background), physiotherapists, occupational therapists, social workers, nurses and sometimes vocational specialists, speech therapists, and recreational therapists. The expected norm in such *interdisciplinary* teams is group decision making, both in assessment, rehabilitation planning, and treatment. In the team, the disabled person is considered the most important member, both in planning and in decision making.

Rehabilitation Strategies

Common rehabilitation strategies are used, where the various team professionals contribute important components. The team members share responsibility for the rehabilitation given. The team conference, led by anyone of the team members, is the important forum for lateral communication and for decision making. A comprehensive rehabilitation plan is formulated, where time frames, types of intervention, and responsible therapists are given, all communicated to and agreed with the disabled person. This concept allows a free exchange of ideas, may benefit from group synergies in problem solving, and facilitates the use of common strategies and coordination. However, it can be time consuming and the team members need training in the team process.

Disability Assessment, Rehabilitation and the ICF

An important part of the work is disability assessment, usually with a team approach, and greatly helped by the development of the International Classification of Functioning, Disability and Health (ICF) (WHO, 2001). Special rating instruments provide supplementary information in this task. The ICF is based on the biopsychosocial model and is a *components of health* classification. It has advanced the understanding and assessment of disability in that it classifies both problems in human functioning (health conditions) and the context in which people with different levels of functioning live and act. The problems are characterized in three interconnected areas:

Impairments are problems in body function or structure such as a significant deviation or loss.

Activity limitations are difficulties an individual may have in executing activities.

Participation restrictions are problems an individual may experience in involvement in life situations.

It is the *interaction* between a health condition and contextual factors, both environmental and personal that may be experienced as a *disability*. *Environmental factors* make up the physical, social, and attitudinal environment in which people live and conduct their lives, whereas p*ersonal factors* are the particular background of an individual's life and living, and may include gender, race, age, other health conditions, fitness, lifestyle, habits, upbringing, coping styles, social background, education, profession, past and current experience, overall behavior pattern and character style, individual psychological assets, and other characteristics.

Disability and Rehabilitation. Defining disability as an interaction means that disability is no longer the attribute of the person (World Report on Disability; WHO & World Bank, 2011).The prevalence of disability in different countries varies greatly, not only due to differences in definitions but also in context, including cultural values. The current definition of rehabilitation (supra), "a set of measures that assist individuals who experience, or are likely to experience, disability to achieve and maintain optimal functioning in interaction with their environments," emphasizes firstly that disability is something experienced, i.e., subjective, and secondly that changing an individual's environment, e.g., by housing adaptation or by educating the family to change attitudes, is also part of rehabilitation. Consequently, rehabilitation is cross-sectional and often carried out by health professionals in cooperation with specialists on education, social welfare and employment. The fact that disability is seen as a subjective experience may also explain the core role of cognitive therapy as an important component in many rehabilitation programs, where modifications of thought patterns along with influencing the links between the individual's thoughts, emotions, and behaviors is vital.

ICF and Rehabilitation. It is obvious with the evolvement of the ICF as a conceptual model and

Rehabilitation, Table 1 ICF-based conceptual description of rehabilitation strategy, modified version (ICF terms are marked in bold; from Meyer et al., 2011 with permission)

Rehabilitation is the health strategy which, based on WHO's **integrative model** of **functioning, disability and health** applies and integrates
Approaches to assess **functioning** in light of **health conditions**
Approaches to optimize a **person's capacity**
Approaches that build on and strengthen the resources of the **person**
Approaches that provide a **facilitating environment**
Approaches that develop a **person's performance**
Approaches that enhance a person's health-related quality of life in partnership between person and provider and in appreciation of the person's perception of his or her position in life over the course of a **health condition** and in all age groups; along and across the continuum of care, including hospitals, rehabilitation facilities and the community, and across sectors, including health, education, labor and social affairs; with the goal to enable persons with **health conditions** experiencing or likely to experience **disability** to achieve and maintain optimal **functioning**

the disability concept that rehabilitation should be defined in relation to these notions. An attempt to do so was elaborated in cooperation with several professional rehabilitation organizations by Stucki, Cieza, and Melvin (2007) and importantly, further developed by integrating the perspective of the disabled person to describe rehabilitation as a health strategy (Meyer et al., 2011) (Table 1).

Community-Based Rehabilitation

If there are few health professional resources such as in developing countries, *community-based rehabilitation* (CBR) performed locally by community groups, health assistants, families, and friends may be an alternative (Chatterjee, Patel, Chatterjee, & Weiss, 2003; Gona, Xiong, Muhit, Newton, & Hartley, 2010; Dalal, Zawada, Jolly, Moxham, & Taylor, 2010). CBR started in Europe as rehabilitation in the home or in the work place in the 1970s as a cheaper alternative to institution-based rehabilitation and has spread rapidly in low-income countries, often advocated and performed by human rights activists and non-health professionals working with development with the additional goal to transform and strengthen local communities in third world countries.

The Effectiveness of Rehabilitation

The evidence base for the effectiveness of modern rehabilitation is now considerable, e.g., for traumatic brain injury and stroke (Cicerone, et al., 2011), spinal cord injury (Martin Ginis, Jetha, Mack, & Hetz, 2010), chronic musculoskeletal pain (Norlund, Ropponen, & Alexanderson, 2009; Schonstein, Kenny, Keating, & Koes, 2003), cardiac insufficiency (Dalal et al., 2010), breast cancer (Duijts, Faber, Oldenburg, van Beurden, & Aaronson, 2011), and some mental diseases (Twamley, Jeste, & Lehman, 2003).

In summary, rehabilitation as the health strategy of functioning represents a unique health service delivery system that contributes markedly to restoring and maintaining health for people with disabilities in most developed countries, but has insufficient resources in others. It is often substituted by voluntary community-based services in developing countries. Since many of the functional disturbances treated emanates from the nervous system, the rapidly expanding new neurobiology, having demonstrated plasticity, regeneration, and adult-born neural stem cells in adult man, will provide fascinating new therapeutical possibilities for the rehabilitation of persons with disabilities in the years to come.

Cross-References

- ▶ Occupational Therapy
- ▶ Physiotherapy

References and Readings

Chatterjee, S., Patel, V., Chatterjee, A., & Weiss, H. A. (2003). Evaluation of a community-based rehabilitation model for chronic schizophrenia in rural India. *The British Journal of Psychiatry, 182*, 57–62.

Cicerone, K. D., Langenbahn, D. M., Braden, C., Malec, J. F., Kalmar, K., Fraas, M., Felicetti, T., et al. (2011). Evidence-based cognitive rehabilitation: Updated review of the literature from 2003 through. *Archives of Physical Medicine and Rehabilitation, 92*, 519–530.

Dalal, H. M., Zawada, A., Jolly, K., Moxham, T., & Taylor, R. S. (2010). Home based versus centre based cardiac rehabilitation: Cochrane systematic review and meta-analysis. *British Medical Journal, 340*, b5631. Review. Erratum in: BMJ. 2010;340:c1133.

Duijts, S. F., Faber, M. M., Oldenburg, H. S., van Beurden, M., & Aaronson, N. K. (2011). Effectiveness of behavioral techniques and physical exercise on psychosocial functioning and health-related quality of life in breast cancer patients and survivors – a meta-analysis. *Psycho-Oncology, 20*, 115–126.

Gona, J. K., Xiong, T., Muhit, M. A., Newton, C. R., & Hartley, S. (2010). Identification of people with disabilities using participatory rural appraisal and key informants: A pragmatic approach with action potential promoting validity and low cost. *Disability and Rehabilitation, 32*(1), 79–85.

Gutenbrunner, C., Ward, A. B., Chamberlain, M. A., (Eds.) (2007). White book on physical and rehabilitation medicine in Europe. *Journal of Rehabilitation Medicine*, *39*(Suppl. 45), 1–48.

Martin Ginis, K. A., Jetha, A., Mack, D. E., & Hetz, S. (2010). Physical activity and subjective well-being among people with spinal cord injury: A meta-analysis. *Spinal Cord, 48*, 65–72.

Meyer, T., Gutenbrunner, C., Bickenbach, J., Cieza, A., Melvin, J., & Stucki, G. (2011). Towards a conceptual description of rehabilitation as a health strategy. *Journal of Rehabilitation Medicine, 43*, 765–769.

Norlund, A., Ropponen, A., & Alexanderson, K. (2009). Multidisciplinary interventions: review of studies of return to work after rehabilitation for low back pain. *Journal of Rehabilitation Medicine, 41*, 115–121.

Schonstein, E., Kenny, D. T., Keating, J., & Koes, B. W. (2003). Work conditioning, work hardening and functional restoration for workers with back and neck pain. *Cochrane Database of Systematic Reviews, 1*, CD001822.

Stucki, G., Cieza, A., & Melvin, J. (2007). The international classification of functioning, disability and health: A unifying model for the conceptual description of the rehabilitation strategy. *Journal of Rehabilitation Medicine, 39*, 279–285.

Twamley, E. W., Jeste, D. V., & Lehman, A. F. (2003). Vocational rehabilitation in schizophrenia and other psychotic disorders: A literature review and meta-analysis of randomized controlled trials. *The Journal of Nervous and Mental Disease, 191*, 515–523.

United Nations. (2006). *Convention on the rights of people with disabilities*. New York: Author. Retrieved November 14, 2006, from http://www.un.org/disabilities/convention/conventionfull

World Health Organization (WHO). (2001). *ICF: International classification of functioning, disability and health*. Geneva: Author. September 3, 2011, Also available online, http://www.who.int/classifications/icf/en/

World Health Organization (WHO), World Bank. (2011). *World report on disability*. Geneva: Author. Retrieved November 14, 2011, from http://www.who.int/disabilities/world_report/2011/en/

Rehabilitation Psychology

▶ Psychosocial Adjustment

Relapse, Relapse Prevention

M. Kathleen B. Lustyk
School of Psychology, Family, and Community, Seattle Pacific University, University of Washington, Seattle, WA, USA

Synonyms

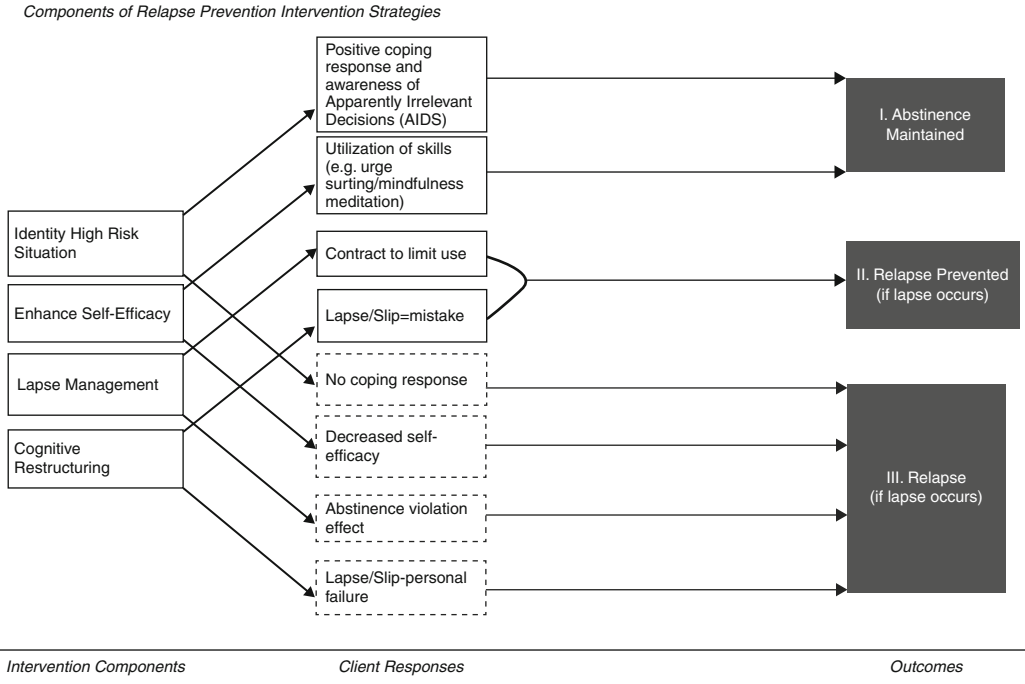
Remission and remission prevention

Definition

Relapse, as a clinical term, is the loss of ground gained toward health or the return of a bad habit following a period of cessation. Not unlike a lapse, which is a single occurrence of the habitual behavior, relapse is often associated with feelings of guilt and shame, and accompanied by self-degrading thoughts. Relapse prevention is an approach to therapy or education that teaches ways to indentify and anticipate triggers or precipitators of relapse. Relapse prevention also teaches behavioral coping strategies for managing high-risk situations in which triggers are present as well as coping strategies for urges, cravings, and negative emotional states that often precipitate relapse.

Description

In the model of addiction, relapse is the return to problematic substance use following a period of abstinence. Relapse is a major roadblock to treatment efficacy, as the majority of addicts who attempt abstinence will relapse. Given what we know about the neurobiology of addiction, the



Relapse, Relapse Prevention, Fig. 1 During the early stages of relapse prevention treatment, the client and clinician collaborate to (a) identify high-risk situations, (b) enhance client self-efficacy, (c) prepare for possible lapses or slips (d), and undergo cognitive restructuring (see *Intervention Components boxes* on *left*). Implementation of these four primary intervention components results in a reduced probability of relapse by increasing positive responses from the client. For example, abstinence is maintained through positively responding to high-risk situations and utilizing skills such as urge surfing to cope with cravings (see first two *Client Responses boxes*). In the case that a lapse or slip occurs, clients are encouraged to adhere to a contract to limit use and avoid full-scale relapse (see third and fourth *Client Responses boxes*). In addition to skills that promote increased self-efficacy, cognitive aspects of relapse are also addressed with the client. Lapses are reframed to equate with mistakes rather than personal failures. Subsequently, the guilt and shame of a "personal failure" are supplanted by a learning experience. Through lapse management and cognitive restructuring, the abstinence violation effect is avoided, and relapse is prevented. Poor client responses and potential outcomes are illustrated via the *dashed Client Responses boxes* and pathways from *Intervention Components* to *Outcomes* (Adapted from Marlatt & Donovan, 2005)

high occurrence of relapse is not surprising. With addiction, the brain's pleasure circuit is usurped such that what was once perceived as a pleasurable experience (e.g., drug use) becomes necessary to prevent a painful experience (i.e., withdrawal). With addiction, habit circuits involved in behavior, become entrained. For example, with habitual drug use, a trigger can produce a small amount of activity in the pleasure circuit and this pleasure "appetizer" can increase the desire or craving for the abused substance as well as the motivation to seek out and use the abused substance. Thus triggers can tap the habit circuitry, which may ultimately result in relapse.

Triggers or precipitators of relapse often arise in interpersonal situations where people or the environment can serve as powerful reminders of the pleasure and behaviors that once sustained the addiction. For example, an abstinent alcoholic may find the pub they used to frequent a powerful cue for drinking. Stress and other negative emotional states may also serve as triggers, especially if the habitual behavior served to reduce stress or improve negative affect.

An abstinence violation effect (AVE) may compound the impact of negative affect on relapse. In the presence of a trigger, relapse is not inevitable. The way in which a person approaches the trigger can have profound effects on the outcome. If, for example, people find themselves in high-risk situations whereby triggers result in a lapse, they may respond by removing themselves from that situation and chalk it up to a learning experience such that they will need to face similar situations differently in the future. Another response would be to suffer an AVE whereby the person berates himself or herself for failing to remain abstinent. They may convince themselves that they will never be successful in managing their addiction and give in to full relapse.

Relapse prevention therapy targets these high-risk situations by helping people recognize them while offering helpful coping skills and management strategies to avoid relapse. In terms of changing habits, willpower as the motivation to change needs to be linked with "skillpower" – as the saying goes, "where there's a will, there's a way." Coping skills provide the "way" to change addictive behavior in relapse prevention. The components of relapse prevention therapy along with client responses and potential outcomes are shown in Fig. 1. By identifying high-risk situations and engaging in behaviors aimed at enhancing self-efficacy, managing lapses, and restructuring thought processes, the substance abuser may prevent relapse. As shown in Fig. 1, client responses that are associated with relapse prevention and maintained abstinence include positive coping responses and skill utilization which may involve overt behaviors such as removing oneself from a high-risk situation to engaging in mental training aimed at reducing the negative consequences of craving. An innovative therapy known as mindfulness-based relapse prevention builds upon the basic tenets of relapse prevention therapy while incorporating mindfulness training as a way of tapping one's metacognitive abilities to further cope with urges and cravings. Specifically, mindfulness teaches one how to cultivate nonjudgmental, nonreactive present moment awareness. This type of awareness can allow for a kind of "space" between a person's sensations or feelings and their ultimate reaction to them. This space may ultimately break the habit cycle. Readers interested in learning more about mindfulness-based relapse prevention therapy are referred to the Bowen, Chawla, and Marlatt (2011) reference listed below.

Cross-References

- ▶ Addiction
- ▶ Mindfulness

References and Readings

Bowen, S., Chawla, N., & Marlatt, A. G. (2011). *Mindfulness-based relapse prevention for addictive behaviors: A clinician's guide*. New York: Guilford Press.

Lustyk, M. K. B., Chawla, M., Nolan, R., & Marlatt, G. A. (2009). Mindfulness meditation research: Issues of participant screening, safety procedures, and researcher training. *Advances in Mind-Body Medicine, 24*(1), 20–30.

Marlatt, G. A., & Donovan, D. M. (2005). *Relapse prevention* (2nd ed.). New York: Guilford Press.

Relational Distress

- ▶ Social Conflict

Relationship Conflict

- ▶ Social Conflict

Relationship Processes

- ▶ Interpersonal Relationships

Relationship Stress

► Family Stress

Relative Risk

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

Relative risk is best defined in conjunction with absolute risk. For this example, we can define risk as the likelihood of an adverse consequence in two behavioral medicine interventions, Treatment A and Treatment B. Imagine that the risk is 1 in 10 for Treatment A and 2 in 10 for Treatment B. In this case, a relative risk statement can be made, saying that the probability of the event occurring following Treatment B is twice the probability of the event occurring following Treatment A. However, the same relative risk statement can be made for probabilities of 1 in 1,000,000 and 2 in 1,000,000. However, the absolute risks are vastly different: 1 and 2 in 10; and 1 and 2 in a million.

Description

Relative risk is often captured by a relative risk ratio. Consider a hypothetical trial in which the interventional treatment group receives a stress-reduction program intended to become an add-on treatment for patients with a serious disease that leads to heart attacks. This group of subjects is already receiving certain medication that is the "standard of care" for this condition. Subjects in the control group do not receive the stress-reduction intervention, but they too are receiving the standard of care medication. The trial is designed to investigate whether the standard of care medication plus the add-on intervention leads to fewer heart attacks than the standard of care medication alone.

The number of heart attacks is determined for each treatment group, and a relative risk ratio is calculated by considering the number of heart attacks in the treatment group as the numerator and the number of heart attacks in the control group as the denominator. If the number of events in each treatment group were to be precisely the same (the likelihood of which is vanishingly small), the value of the risk ratio will be unity, represented here as 1.00. If the number of events in the treatment group is less than that for the control treatment group, the value will be less than 1.00. Conversely, if the number of events in the treatment group is greater than that for the control treatment group, the value will be greater than 1.00.

Imagine that the value of the relative risk ratio is 0.80. This value conveys that, in this single trial, the addition of the stress-reduction program to the standard of care medication led to a 20% reduction in risk of heart attack. To estimate the true but unknown relative risk for the general patient population, confidence intervals (CIs) are placed around this value of 0.80, which in this context is called the relative risk point estimate. Let us calculate a two-sided 95% CI, which can be defined as a range of values that we are 95% confident that will cover the true but unknown population risk ratio. Imagine that the lower and upper limits of the 95% CI are 0.70 and 0.92, respectively. (The calculation of a ratio means that these limits do not fall symmetrically around the point estimate.) The confidence interval provides insight into the likely occurrence of heart attacks in the general population receiving the stress-reduction program. The following statement can now be made:

- The data obtained from this single trial are compatible with as little as an 8% decrease and as much as a 30% decrease in the risk of a heart attack in the general population, and our best estimate is a decrease of 20%.

Consider now a scenario in which the relative risk point estimate is 1.50, and the lower and upper bounds of a two-sided 95% CI placed around this point estimate are 1.35 and 1.68,

respectively. Such data would show that adding the stress-reduction intervention to the standard of care medication actually *increased* the risk of heart attack. The interpretation of such results is as follows:

- The data obtained from this single trial are compatible with as little as a 35% increase and as much as a 68% increase in risk of heart attack in the general population, and our best estimate is an increase in risk of 50%.

Cross-References

► Absolute Risk

Relaxation

► Psychophysiologic Recovery

Relaxation Techniques

► Stress Management

Relaxation: Techniques/Therapy

Hiroe Kikuchi
Department of Psychosomatic Research, National Institute of Mental Health, National Center of Neurology and Psychiatry, Tokyo, Japan

Definition

Relaxation techniques/therapy are techniques for reducing stress and inducing relaxation. Relaxation techniques/therapy increase parasympathetic nervous system activity, thereby decreasing the opposing sympathetic nervous system activity such as the fight or flight phenomenon and decreasing arousal. There are a variety of techniques for inducing relaxation. Probably, the most common are variants of Jacobson's progressive muscle relaxation training. Other methods include autogenic training, use of relaxation imagery, biofeedback, and practices derived from meditation and yoga techniques (Jorm, Morgan, & Hetrick, 2008).

With regard to psychiatric illnesses, relaxation techniques/therapy are often used as a competing stimulus during systematic desensitization and as part of a comprehensive intervention strategy for the reduction of anxiety. They are more effective at reducing self-rated depressive symptoms than no or minimal treatment, while data on clinician-rated depressive symptoms are less conclusive. However, they are not as effective as psychological treatment (Jorm et al., 2008). In addition, progressive muscle relaxation training has significant therapeutic efficiency in chronic insomnia in adult patients (Taylor & Roane, 2010).

With regard to physical symptoms or diseases, relaxation techniques/therapy have positive effects on pain reduction (Palermo, Eccleston, Lewandowski, Williams, & Morley, 2010). In addition, they have a mild to moderate effect on reducing hot flushes in women with a history of breast cancer (Rada et al., 2010). Although many studies tried to investigate the effect of relaxation techniques/therapy on primary hypertension, the evidence in favor of causal association between relaxation and blood pressure reduction is weak due to the poor quality of the studies (Dickinson et al., 2008). Therefore, further research is required to investigate the possibility of relaxation on physical symptoms or diseases.

Cross-References

► Biofeedback
► Meditation
► Yoga

References and Readings

Dickinson, H. O., Campbell, F., Beyer, F. R., Nicolson, D. J., Cook, J. V., Ford, G. A., et al. (2008). Relaxation therapies for the management of primary hypertension

in adults. *Cochrane Database of Systematic Reviews, 23*, CD004935.
Jorm, A. F., Morgan, A. J., & Hetrick, S. E. (2008). Relaxation for depression. *Cochrane Database of Systematic Reviews, 8*, CD007142.
Palermo, T. M., Eccleston, C., Lewandowski, A. S., Williams, A. C., & Morley, S. (2010). Randomized controlled trials of psychological therapies for management of chronic pain in children and adolescents: An updated meta-analytic review. *Pain, 148*, 387–397.
Rada, G., Capurro, D., Pantoja, T., Corbalán, J., Moreno, G., Letelier, L. M., et al. (2010). Non-hormonal interventions for hot flushes in women with a history of breast cancer. *Cochrane Database of Systematic Reviews, 8*, CD004923.
Taylor, D. J., & Roane, B. M. (2010). Treatment of insomnia in adults and children: A practice-friendly review of research. *Journal of Clinical Psychology, 66*, 1137–1147.

Reliability and Validity

Yori Gidron
Faculty of Medicine and Pharmacy,
Free University of Brussels (VUB), Jette,
Belgium

Definition

These two concepts are the basis for assessment in most scientific work in medical and social sciences. Reliability refers to the degree of consistency in measurement and to the lack of error. There are several types of indices of reliability. Internal reliability (measured by Cronbach's alpha) is a measure of repeatability of a measure. In psychometrics, a questionnaire of, for example, 10 items, is said to be reliable if its internal reliability coefficient is at least 0.70. This reflects approximately the mean correlation between each score on each item, with all remaining item scores, repeated across all items. Methodologically, this reflects a measure of repeatability, a basic premise of science. Another type of reliability is inter-rater reliability, which refers to the degree of agreement between two or more observers, evaluating a patient's behavior, for example. Thus, in the original type A behavior interview, which currently places more emphasis on hostility, researchers provide a measure of inter-rater reliability, reflecting their agreement on an observed behavior. Finally, test-retest reliability refers to stability of a measure over time, for example, over 2 weeks, 1 month, or 4 years. For example, hostility has a strong test-retest reliability over 1 year of $r = 0.85$, $p < .0001$ (Barefoot, Dahlstrom, & Williams, 1983). This reliability can indicate stability of a measure and of the psychological phenomenon being assessed.

Validity of instruments refers to the degree to which instruments assess the construct they aim to measure. To test this, researchers have developed several types of validity indices. Face validity reflects the degree to which items of a scale "appear" to assess the construct they claim to, based on the meaning of items. This can be judged by participants or experts rating the relevance of each item to the construct being investigated. Construct validity refers to the degree to which scores on a scale correlate with scores on other measures of other constructs with which they should be theoretically correlated. For example, a scale of anxiety would be expected to correlate with life events in one's past, the latter being a possible trigger of current anxiety levels. Concurrent validity refers to correlations between scores of two different measures of the same construct, for example, between two anxiety scales. Criterion validity occurs when scores on an instrument predict or correlate with an accepted criterion, for example, anxiety being associated with observed behavioral responses to a stressor. Alternatively, criterion validity can be measured by showing that anxiety scores of a group of patients with clinically high anxiety levels (post-traumatic stress disorder) are higher than those of a healthy control group, also referred to as discriminant validity. Finally, predictive validity reflects the degree to which scores on an instrument predict in time the outcome of people in future. For example, an anxiety scale has predictive validity if it predicts health-care utilization over the next 12 months.

Another type of validity, a very important one, is internal validity. This reflects the confidence a researcher may have in his or her inferences from a study. This can be achieved by choosing

an adequate study design and methodological and statistical control over confounders. However, it also results from close consideration and measurement of all possible variables in a study, from sampling, to reliability and validity of tests, to control over confounders. To the extent that these are achieved, the attribution of observed results to an independent variable or predictor variable may be internally valid. Internal validity is perhaps the outmost important issue in scientific investigation, and a study can be scientifically valid if its internal validity is maintained.

Cross-References

- ▶ Psychometrics
- ▶ Psychometric Properties

References and Readings

Barefoot, J. C., Dahlstrom, W. G., & Williams, R. B., Jr. (1983). Hostility, CHD incidence, and total mortality: A 25-year follow-up study of 255 physicians. *Psychosomatic Medicine, 45*, 59–63.

Del Greco, L., Walop, W., & McCarthy, R. H. (1987). Questionnaire development: 2. Validity and reliability. *CMAJ, 136*, 699–700.

Religion

- ▶ Religiousness/Religiosity
- ▶ Spirituality

Religion/Spirituality

Stephen Gallagher and Warren Tierney
Department of Psychology, Faculty of Education & Health Sciences, University of Limerick, Castletroy, Limerick, Ireland

Synonyms

Religiosity; Religiousness; Spiritual; Spirituality

Definition

Even though there is much dispute among researchers regarding accurate definitions of spirituality and religion, it is generally accepted that spirituality concerns the exploration for sacred, celestial, or the transcendent side of life, while religion can be defined as a perception, influence, and behavior which emerges from a consciousness of, or alleged contact with, metaphysical entities which are deemed to perform an essential role in human life. Moreover, in contemporary society, the formation of a dichotomy is being witnessed: with spirituality representing the personal, subjective, inner-directed, unsystematic, liberating expression, and religion signifying a formal, authoritarian, institutionalized inhibiting expression. Yet the concept of religion may further be separated into two categories: intrinsic and extrinsic religion. Intrinsic religion is an internalized faith which becomes an innate outlook on life. Extrinsic is focused more on individual practices driven by external motives, such as improved social standing or acceptance. Although religion and spirituality at times appear to be polarized concepts, for the moment, at least, it is impossible to completely segregate the two concepts as the search for the sacred occurs within various faith traditions, and most researchers agree that they are in fact overlapping constructs.

Description

Associations Between Spirituality, Religion, and Health

Historically, it was argued that the study of the natural and explicable phenomena belonged to science whereas the relatively unexplained belonged to religion. This was very pertinent to the field of medicine. Nowadays, however, there is a strong realization that health involves the interaction of mind, body, and more. In fact, health is far more than a physical matter. Much of this realization has been attributed to a substantial body of literature which has accumulated over the last number of decades connecting

spirituality, religion, and communal religious involvement to a variety of health outcomes (Hill & Pargament, 2003; Koenig, 2009; McCullough & Willoughby, 2009; Powell, Shahabi, & Thoresen, 2003). The links between spirituality and health are discussed elsewhere in this encyclopedia (see ▶ Spirituality). Even though the vast majority of studies report a beneficial effect of religion on health, this research has been the subject of considerable controversy. For example, religious involvement is strongly correlated with health-related factors, such as functional status, lifestyle, and social support, which may confound associations between religious observance, beliefs, and health (Sloan, Bagiella, & Powell, 1999). Further, longitudinal studies have found differential stress buffering effects: protective against multiple events but not discrete events (Schnittker, 2001). It could be that religious coping is used as a last resort when individuals feel overwhelmed when dealing with multiple stressful episodes, especially when their personal coping resources fail or prove to be inadequate. However, what adds to this controversy is that the precise mechanisms behind the links between religion and health are not yet clear and are the subject of much research(Powell, Shahabi, & Thoresen, 2003; Seeman, Dubin, & Seeman, 2003).

A number of possible psychological, social, and physiological mediators have been proposed to account for this connection. For example, it could be that prayer may help people deal with unpleasant situations or that faith promotes a positive disposition which facilitates coping. It is also becoming increasingly evident that religious/spiritual coping strategies can be divided into positive (e.g., seeking support from clergy, forgiveness, reappraisal) and negative (e.g., spiritual discontent, pleading for direct intercession, punishing God reappraisal) forms; positive forms typically relate to more positive outcomes, whereas negative religious coping strategies are generally related to more negative outcomes (Hill & Pargament, 2003). Moreover, religious affiliations are associated with the practice of healthy lifestyles and the social aspect of attending religious rituals, i.e., church, promotes social integration, all of which are linked to better health (Sloan & Ramakrishnan, 2006). Further, religious attendance has been found to be inversely related to inflammatory cytokines high interleukin −6 levels (>5 pg/ml), providing supportive evidence of a direct link between religious observance and health (Koenig, et al., 1997); this observed association was somewhat attenuated after controlling for age, sex, race, education, chronic illnesses, and physical functioning, implying that other factors may be driving the observed effects. In addition, the proponents of these links between religion and health would argue that the strongest evidence comes from intervention studies, demonstrating the positive effect of intercessory prayer on cardiovascular health (Townsend, Kladder, Ayele, & Mulligan, 2002). However, in a very recent Cochrane Review, it was found that this particular evidence was rather weak and that trials of this type of intervention should not be undertake, stating that they "would prefer to see any resources available for such a trial used to investigate other questions in health care (Roberts, Ahmed, Hall & Davidson, 2009)." Another issue that needs to be addressed is the differential effects of intrinsic and extrinsic religion on psychological distress. Studies have indicated that intrinsic religious individuals who attend religious services demonstrate reduced anxiety whereas extrinsically motivated individuals who attend services tend to portray anxiety (Koenig, 2009). How these different religious concepts relate to physical health outcomes is still unclear, but the link between psychological distress and ill-health is well established (Roberts, Ahmed, Hall & Davidson, 2009; Rugulies, 2002).

How to Measure Spirituality and Religion

Measuring spirituality and religion has proven to be a very difficult task due to researcher's inability to completely differentiate between religion and spirituality. This inadequacy has unlocked Pandora's Box, due to the paradoxical question: is one measuring/or has one measured spirituality or religion? Or are these religious measurements tapping into other constructs such as social

support? Nonetheless, there are a number of measures available for measuring spirituality (see entry on ▶ Spirituality) and religion: the Religious Involvement Scale (Piedmont, Ciarrochi, Dy-Liacco, & Williams, 2009) is a useful scale to test how involved individuals are in religious activities; the Multi-Religion Identity Measure (Abu-Rayya, Abu-Rayya, & Khalil, 2009) is a scale which can be used accurately in order to distinguish between members of different religions; and the religious coping scale (RCOPE) (Pargament, Koenig, & Perez, 2000) can be used to assess religious coping. If one wants to measure both spirituality and religion, the Assessment of Spirituality and Religious Sentiments ASPIRES (Piedmont, 2004) scale can be used, which has proved to have reliability and discriminant validity. Finally, and more recently, an Implicit Christian Humanist Implicit Association Test (Ventis, Ball, & Viggiano, 2010) has been developed to measure both religion and spirituality which may have stronger behavioral correlates.

Cross-References

▶ Religiousness/Religiosity
▶ Spirituality

References and Readings

Abu-Rayya, H., Abu-Rayya, M., & Khalil, M. (2009). The multi-religion identity measure: A new scale for use with diverse religions. *Journal of Muslim Mental Health, 4*, 124–138.

Allport, G. W., & Ross, J. M. (1967). Personal religious orientation and prejudice. *Journal of Personality and Social Psychology, 5*, 432–443.

Hill, P. C., & Pargament, K. I. (2003). Advances in the conceptualization and measurement of religion and spirituality: Implications for physical and mental health research. *American Psychologist, 58*, 64–74.

Koenig, H. G. (2009). Research on religion, spirituality, and mental health: A review. *Canadian Journal of Psychiatry, 54*, 283–291.

Koenig, H. G., Cohen, H. J., George, L. K., Hays, J. C., Larson, D. B., & Blazer, D. G. (1997). Attendance at religious services, interleukin-6, and other biological parameters of immune function in older adults. *International Journal of Psychiatry in Medicine, 27*, 233–350.

Koenig, H. G., McCullough, M. E., & Larson, D. B. (Eds.). (2001). *Handbook of religion and health*. New York: Oxford University Press.

McCullough, M. E., & Willoughby, B. B. (2009). Religion, self-regulation, and self-control: Associations, explanations, and implications. *Psychological Bulletin, 135*, 69–93.

Pargament, K. I., Koenig, H. G., & Perez, L. M. (2000). The many methods of religious coping: Development and initial validation of the RCOPE. *Journal of Clinical Psychology, 56*, 519–543.

Piedmont, R. L. (2004). *Assessment of Spirituality and Religious Sentiments (ASPIRES) technical manual*. Columbia: Author.

Piedmont, R. L., Ciarrochi, J. W., Dy-Liacco, G. S., & Williams, J. G. (2009). The empirical and conceptual value of the spiritual transcendence and religious involvement scales for personality research. *Psychology of Religion and Spirituality, 1*(3), 162–179.

Powell, L. H., Shahabi, L., & Thoresen, C. E. (2003). Religion and spirituality: Linkages to physical health. *American Psychologist, 58*, 36–52.

Roberts, L., Ahmed, I., Halls, S., & Davison, A. (2009). Intercessory prayer for the alleviation of ill health. *Cochrane Database Systematic Review*, (2), CD000368. doi:10.1002/14651858.CD000368.pub3.

Rugulies, R. (2002). Depression as a predictor for coronary heart disease a review and meta-analysis. *American Journal of Preventive Medicine, 23*, 51–61.

Schnittker, J. (2001). When is faith enough? the effects of religious involvement on depression. *Journal for the Scientific Study of Religion, 40*, 393–411.

Seeman, T. E., Dubin, L. F., & Seeman, M. (2003). Religiosity/spirituality and health: A critical review of the evidence for biological pathways. *American Psychologist, 58*, 53–63.

Sloan, R. P., Bagiella, E., & Powell, T. (1999). Religion, spirituality, and medicine. *Lancet, 353*, 664–667.

Sloan, R. P., & Ramakrishnan, R. (2006). Science, medicine, and intercessory prayer. *Perspectives in Biological Medicine, 49*, 504–514.

Townsend, M., Kladder, V., Ayele, H., Mulligan, T., et al. (2002). Systematic review of clinical trials examining the effects of religion on health. *Southern Medical Journal, 95*, 1429–1434.

Ventis, W., Ball, C. T., & Viggiano, C. (2010). A Christian humanist implicit association test: Validity and test – retest reliability. *Psychology of Religion and Spirituality, 2*, 181–189.

Religiosity

▶ Religion/Spirituality
▶ Spirituality

Religious Beliefs

▶ Spiritual Beliefs

Religious Ceremony

▶ Religious Ritual

Religious Coping

Jennifer Wortmann
Department of Psychology, University of Connecticut, Storrs, CT, USA

Synonyms

Negative religious coping; Religious struggle; Spiritual coping; Spiritual struggle

Definition

Religious coping is religiously framed cognitive, emotional, or behavioral responses to stress, encompassing multiple methods and purposes as well as positive and negative dimensions.

Description

Religion and spirituality translate into coping responses to stress insofar as they serve as available and compelling orienting systems and especially when stressors test "the limits of personal powers" (Pargament, 1997, p. 310). Religion can provide a framework for understanding emotional and physical suffering and can facilitate perseverance or acceptance in the face of stressors. Religious coping encompasses religiously framed cognitive, emotional, or behavioral responses to stress. It may serve many purposes, including achieving meaning in life, closeness to God, hope, peace, connection to others, self-development, and personal restraint (Pargament, 1997). Who uses religious coping depends on individual (e.g., degree of personal religious commitment), situational (e.g., stressfulness of the event), and cultural factors (Harrison, Koenig, Hays, Eme-Akwari, & Pargament, 2001). The outcomes of religious coping depend on the appropriateness of the coping method to the stressor (Pargament, 1997).

Early measures of religious coping were limited in breadth and depth. For instance, Ways of Coping (Lazarus & Folkman, 1984) measures one religious practice: prayer. The COPE (Carver, Scheier, & Weintraub, 1989) assesses religious coping with four items: "I seek God's help," "I put my trust in God," "I try to find comfort in my religion," and "I pray." The RCOPE religious coping scale by Pargament and colleagues expands measurement of religious coping to include methods to find meaning, to gain control, to gain comfort and closeness to God, and to achieve a life transformation (Pargament, Koenig, & Perez, 2000). Measurement of multiple methods of religious coping permits researchers and clinicians to assess specific beliefs, experiences, or practices that differentially relate to health.

Religious coping may be active or passive in nature. In fact, a collaborative religious coping style in which a person considers him or herself partners with God in resolving a problem was found to be more common and more effective than either a self-directing or passive style (Pargament, 1997). Religious coping methods that have been shown to have a generally positive relationship with psychological outcomes (Ano & Vasconcelles, 2005), hence *positive religious coping*, include the collaborative style, benevolent reappraisal of the stressor, and seeking spiritual support from God, clergy, or members of one's religious group. Religious coping is increasingly being researched in the contexts of multiple illnesses, and suspected mechanisms through which religious coping can influence health include relaxation, sense of control, and the promotion of healthy behaviors. Although research has failed to show consistent

relations between positive religious coping methods and physical health, future research may incorporate sophisticated design and proposed psychosocial (e.g., meaning, social support, meditation) and physiological mediation pathways (Park, 2007; Seybold, 2007).

Research has demonstrated more consistent relations between negative religious coping methods and poorer mental and physical health (Ano & Vasconcelles, 2005; Powell, Shahabi, & Thoresen, 2003). Negative religious coping, also known as spiritual struggle, encompasses interpersonal, intrapersonal, and divine categories, including conflict with religious others, questioning, guilt, and perceived distance from or negative views of a higher power (Pargament, 2007). Religious coping may also be a negative force in health if it interferes with receipt of necessary treatments (e.g., passive religious deferral). Although generally less common than positive coping in response to stress, negative religious coping is common in people facing serious or life-threatening illness. As such, researchers have recommended incorporating assessment of positive and negative religious coping in clinical practice to identify those at risk for the negative impacts of spiritual struggles and have begun to develop recommendations regarding and interventions targeted at spiritual struggles (Pargament, 2007).

Cross-References

- ▶ Coping
- ▶ Religion/Spirituality
- ▶ Spirituality

References and Readings

Ano, G. G., & Vasconcelles, E. B. (2005). Religious coping and psychological adjustment to stress: A meta-analysis. *Journal of Clinical Psychology, 61*, 461–480.

Carver, C. S., Scheier, M. F., & Weintraub, J. K. (1989). Assessing coping strategies: A theoretically based approach. *Journal of Personality and Social Psychology, 56*, 267–283.

Harrison, M. O., Koenig, H. G., Hays, J. C., Eme-Akwari, A., & Pargament, K. I. (2001). The epidemiology of religious coping: A review of recent literature. *International Review of Psychiatry, 13*, 86–93.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.

Pargament, K. I. (1997). *The psychology of religion and coping: Theory, research, practice*. New York: Guilford Press.

Pargament, K. I. (2007). *Spiritually integrated psychotherapy: Understanding and addressing the sacred*. New York: Guilford Press.

Pargament, K. I., Ano, G. G., & Wachholtz, A. B. (2005). The religious dimension of coping: Advances in theory, research, and practice. In R. F. Paloutzian & C. L. Park (Eds.), *Handbook of the psychology of religion and spirituality* (pp. 479–495). New York: Guilford Press.

Pargament, K. I., Koenig, H. G., & Perez, L. M. (2000). The many methods of religious coping: Development and initial validation of the RCOPE. *Journal of Clinical Psychology, 56*, 519–543.

Park, C. L. (2007). Religiousness/spirituality and health: A meaning systems perspective. *Journal of Behavioral Medicine, 30*, 319–328.

Powell, L. H., Shahabi, L., & Thoresen, C. E. (2003). Religion and spirituality: Linkages to physical health. *American Psychologist, 58*, 36–52.

Seybold, K. (2007). Physiological mechanisms involved in religiosity/spirituality and health. *Journal of Behavioral Medicine, 30*, 303–309.

Religious Practice

▶ Religious Ritual

Religious Ritual

Login S. George and Crystal L. Park
Department of Psychology, University of Connecticut, Storrs, CT, USA

Synonyms

Church attendance; Prayer; Religious ceremony; Religious practice; Religious service; Service attendance

Definition

A religious ritual is any repetitive and patterned behavior that is prescribed by or tied to a religious institution, belief, or custom, often with the intention of communicating with a deity or supernatural power. Rituals may be performed individually or collectively during predetermined times (e.g., praying at specific times of day), elicited by events (e.g., mourning rituals performed after a death), or performed sporadically (e.g., praying at various times throughout the day).

Description

Rituals are an important aspect of religion because they allow believers to express and reaffirm their belief systems. One of the primary purposes of rituals is communication. Rituals communicate or are intended to communicate to self, others, or deities. They convey information regarding the commitments, beliefs, and values of the individuals performing the ritual and link them to the larger religious tradition. They reaffirm the religious frameworks individuals use to conceptualize and understand life and, thus, endow a sense of meaning. Religious rituals also serve other psychosocial functions such as emotional control, social support, and community cohesion. By structuring and prescribing behavior, rituals may ease anxiety and uncertainty and promote stability and understanding in social interactions. It is thought that religious rituals may affect physical and mental health through the psychosocial functions that they serve.

In the context of health, prayer is one of the most common religious rituals examined. Studies of relationships between prayer and health have produced contradictory findings. Some have demonstrated favorable links between the two. For example, a longitudinal study of healthy elderly adults found that prayer was related to reduced mortality rates even after many potential confounds such as demographics, health practices, and social support were controlled for (Helm, Hays, Flint, Koenig & Blazer, 2000). In contrast, cross-sectional studies have found unfavorable relationships between prayer and health such that higher levels of prayer are related to more disability and pain. A possible explanation for this negative relationship is that those who are ill and suffering may turn to prayer as a way to deal with their distress. Prayer may become particularly relied upon when individuals are facing stress or illness in that prayer can be an important coping resource. The contradictory findings regarding prayer and health may also be due to the failure of many studies to examine factors such as frequency and content of prayer. Prayer can be categorized into different types based on its content, and evidence suggests that different types of prayer may have differing relationships with health and well-being. As of now, it seems that the relationship between prayer and health is a complex one and one that is yet to be fully explored or understood.

Numerous studies have also looked at relationships between frequency of service attendance and health. Ample evidence points to a strong positive relationship between the two. For example, a review of the literature concluded that there was a 25% reduction in mortality rates among those who attended religious services even after confounds and risk factors (such as demographics, healthy lifestyle, social support, and depression) were taken into account (Powell, Shahabi, & Thoresen, 2003). A meta-analysis that included data from over 125,000 participants found a similar 25% reduction in mortality rates even after potential confounding variables were taken into account (McCullough, Hoyt, Larson, Koenig, & Thoresen, 2000). Many pathways through which service attendance affects health have been proposed; service attendance may affect health by generating strong positive emotions, encouraging healthy behaviors, and providing access to resources and social support.

Other forms of religious rituals exist besides religious service and prayer, and such forms of rituals may be related to health as well. For example, religious mourning rituals have been postulated to play a role in constructively dealing with grief. Mourning rituals may provide a sense

of mastery and control during a highly stressful and uncertain time by prescribing to individuals what ought to be done. Other collective rituals such as participation in religious festivals or ceremonies may foster social bonding and yield a sense of community and social support. Although it can be reasoned that such forms of rituals may affect health as it is related to many salutary psychosocial functions such as social support and a sense of control, currently there is very little empirical literature linking them to health.

In conclusion, although scholars have theorized that religious rituals serve a wide array of functions such as social cohesion, sense of meaning, emotional control, and personal control, all of which have been shown to be related to better health, very little research has examined the full linkages. With the exception of participation in formal worship services as strongly linked to better health, few solid conclusions regarding links between religious rituals and physical health can be drawn at this point. These questions await further empirical attention.

Cross-References

- ▶ Beliefs
- ▶ Coping
- ▶ Emotional Control
- ▶ Grieving
- ▶ Prayer
- ▶ Religion/Spirituality
- ▶ Social Support

References and Readings

Helm, H. M., Hays, J. C., Flint, E. P., Koenig, H. G., & Blazer, D. G. (2000). Does private religious activity prolong survival? A six-year follow-up study. *Journals of Gerontology A: Biological and Medical Science, 55*, 400–405.

Lee, B. Y., & Newberg, A. B. (2005). Religion and health: A review and critical analysis. *Zygon, 40*, 443–468.

Masters, K. S., & Spielmans, G. I. (2007). Prayer and health: Review, meta-analysis, and research agenda. *Journal of Behavioral Medicine, 30*, 329–338.

McCullough, M. E., Hoyt, W. T., Larson, D. B., Koenig, H. G., & Thoresen, C. (2000). Religious involvement and mortality: A meta-analytic review. *Health Psychology, 19*, 211–222.

Powell, L. H., Shahabi, L., & Thoresen, C. E. (2003). Religion and spirituality: Linkages to physical health. *American Psychologist, 58*, 36–52.

Pruyser, P. (1968). *A dynamic psychology of religion*. New York: Harper & Row.

Rappaport, R. (1999). *Ritual and religion in the making of humanity*. Cambridge, England: Cambridge University Press.

Spilka, B. (2005). Religious practice, ritual, and prayer. In R. F. Paloutzian & C. L. Park (Eds.), *Handbook of the psychology of religion and spirituality* (pp. 435–459). New York: Guilford.

Religious Service

▶ Religious Ritual

Religious Social Support

Chad Barrett
Department of Psychology, University of Colorado, Denver, CO, USA

Synonyms

Church-based support

Definition

Religious social support can be described as the social support individuals receive as a result of their religious beliefs and participation in religious activities. Social support refers to the size of one's social network and the perception of belonging to one or more groups. It also includes perceptions of received, provided, and expected emotional and tangible support from one's social network (Cohen, Underwood, & Gottlieb, 2000). Thus, religious social support refers to the emotional and tangible support that one receives, provides, and expects from one's religious

community. It may also refer to the size of one's social network as a result of participation in religious and spiritual activities (Debnam, Holt, Clark, Roth, & Southward, 2011; Krause, Ellison, Shaw, Marcum, & Boardman, 2001). Religious support is a multidimensional construct that has sometimes been operationalized differently across various studies. Most definitions of religious social support include the amount of religious involvement (usually indicated by frequency of attending religious services and related activities). Religious involvement is intended as a proxy measure of the social interaction and supportive relationships that one may form with fellow members of a religious community. Religious social support can be further specified according to type (emotional or spiritual) and source (clergy or laity members) of the support (Debnam et al., 2011). Occasionally, religious support includes a person's perception of emotional and tangible support received from supernatural entities as a result of religious or spiritual beliefs and activities. A four-dimensional model of religious social support has been described in the Brief Multidimensional Measure of Religiousness/Spirituality for Use in Health Research (NIA working group/Fetzer Institute, 1999). The four dimensions are emotional support provided, emotional support received, negative interaction, and anticipated support.

Description

There is a broad literature linking religiosity and spirituality to mental and physical health (George, Ellison, & Larson, 2002; Koenig, 2009; Koenig, McCullough, & Larson, 2001; Oman & Thoresen, 2005). In general, though not always, the large number of studies examining the relationship between religiosity and spirituality and mental and physical health suggest that religious and spiritual activities have a salutary influence on a variety of variables related to mental and physical health. Numerous studies involving participants in a variety of settings, from different ethnic backgrounds, in different age groups, and in different countries, some of which used longitudinal designs or random controlled trials, have found evidence that religious and spiritual activities are mostly related to better coping with stress and less depression, suicide, anxiety, and substance abuse (Koenig, 2009). The positive influence of religion and spirituality goes beyond the mere absence of psychopathology to include greater general happiness, satisfaction with life, and sense of meaning and purpose (Miller & Kelley, 2005). They are also related to better physical health, better health behaviors, and longer survival (George, Ellison, & Larson, 2002; Oman & Thoresen, 2005; McCullough, Hoyt, Larson, Koenig, & Thoresen, 2000; Powell, Shahabi & Thorensen, 2003). People who attend religious services and activities once a week or more tend to have fewer illnesses, recover more quickly from illness, and live longer than those who attend less frequently (George et al., 2002). There is also evidence that they have lower blood pressure and reduced risk of hypertension. Many religious communities encourage health-promoting behaviors. Individuals who attend religious activities tend to engage in more exercise and less smoking and heavy drinking, and they tend to wear their seat belts more often. Further, they are more likely to seek preventative medical care and comply with medical treatment. At least one longitudinal study of over 2,500 community participants spanning 30 years found that people who attended religious services more often were also more likely to adopt and maintain healthy behaviors, such as exercising and avoiding smoking and abusing alcohol (Oman & Thoresen, 2005). At least two reviews found evidence from several large-scale studies that religious involvement is related to lower mortality rates (McCullough et al., 2000; Powell et al., 2003). In both studies, on average, increased religious activity reduced mortality rates by about 25%.

It is likely that the influence religion and spirituality have on mental and physical health is partially mediated by religious social support. Social support in general, (i.e., social support that is not specific to religiously based social support) has often been related to better physical and mental health (Taylor, 2011). Involvement in

religious and spiritual communities seems to provide individuals with opportunities to develop greater social support. Individuals who participate in religious and spiritual communities may have additional, and/or unique, opportunities to benefit from social support. People who regularly attend religious and spiritual activities, compared to those who attend less frequently, typically have larger social networks and engage in more activities in which they provide or receive social support (Ellison, Hummer, Burdette, & Benjamins, 2010; Oman & Thoresen, 2005). Increased participation in religious community activities is associated with larger and more stable social networks and with more perceived social, and emotional, support (George et al., 2002; Strawbridge, Shema, Cohen, & Kaplan, 2001). Further, the quality of religious social support may be better. Members of religious and spiritual communities tend to share similar beliefs, worldviews, values, backgrounds, and experiences. They may also have more opportunities to share intimate moments, significant life experiences, and develop long-term friendships and bonds. They may feel more closely connected as a result of participating in common worship, prayer, services, or social groups and activities. In addition, religious and spiritual teachings that promote prosocial values can encourage and facilitate providing and receiving emotional and tangible support, especially during times of stress (Ellison & George, 1994).

Religious social support may help individuals cope with stress by encouraging healthy coping strategies and may help them construct a sense of meaning and purpose. Religious social support can also provide individuals with a sense of shared burden and comfort. Another way religious social support may influence physically and mentally is by encouraging healthy behaviors. Most religious and spiritual communities encourage their members to abstain from harmful behaviors such as drug and alcohol abuse, smoking, and unsafe sex. Dimensions of religious social support have been related to better eating and exercising habits (Debnam et al., 2011; Oman & Thoresen, 2005; McCullough et al., 2000; Powell et al., 2003). However, the evidence for the mediating role of religious social support is mixed with some studies finding evidence for the mediating role of religious social support and some not finding this (George et al., 2002).

It is also important to note that increased involvement in religious and spiritual communities can sometimes have negative consequences. Religious and spiritual participation can sometimes be a source of stress rather than comfort. Interactions may be more negative than positive. For example, in religious communities where certain things such as divorce or homosexuality are strongly condemned, individuals and families experiencing these issues may encounter a variety of negative interactions with community members. In some cases, religious communities may reinforce unhealthy beliefs and behaviors. They can promote perfectionism, negative self-views, and exacerbate feelings of guilt, worry, and anxiety. They can perpetuate mental illness by interpreting pathological symptoms in religious and moralistic terms. Further, some religious communities may discourage members from seeking and adhering to medical treatment for physical and mental health issues (Exline & Rose, 2005; Miller & Kelley, 2005). Quite often, the negative reality of religious and spiritual activity exists alongside the positive benefits. Thus, religious social support includes both positive and negative elements.

Elements of religious and spiritual activities likely to influence health and mental health include public participation (e.g., attendance at religious services and related activities), religious affiliation (i.e., belonging to religious groups or denominations), private religious practices (e.g., prayer, meditation, reading religious literature), and religious coping (relying on religion to help cope with stressful situations) (George, Ellison, & Larson, 2002).

Cross-References

- ▶ Religion/Spirituality
- ▶ Religious Coping
- ▶ Social Support

References and Readings

Cohen, S., Underwood, L., & Gottlieb, B. H. (2000). *Social support measurement and intervention: A guide for health and social scientists*. New York: Oxford University Press.

Debnam, K., Holt, C. L., Clark, E. M., Roth, D. L., & Southward, P. (2011). Relationship between religious social support and general social support with health behaviours in a national sample of African Americans. *Journal of Behavioural Medicine*. Retrieved December 1, 2011, from http://0-www.springerlink.com.skyline.ucdenver.edu/content/243t686161407262/

Ellison, C. G., & George, L. K. (1994). Religious involvement, social ties, and social support in a southeastern community. *Journal for the Scientific Study of Religion, 33*, 46–61.

Ellison, C., Hummer, R., Burdette, A., & Benjamins, M. (2010). Race, religious involvement, and health: The case of African Americans. In C. G. Ellison & R. A. Hummer (Eds.), *Religion, families, and health: Population-based research in the United States* (pp. 321–348). New Brunswick, NJ: Rutgers University Press.

Exline, J. J., & Rose, E. (2005). Religious and spiritual struggles. In R. F. Paloutzian & C. L. Park (Eds.), *Handbook of the psychology of religion and spirituality* (pp. 435–459). New York: Guilford.

Fetzer Institute, National Institute on Aging Working Group. (1999). *Multidimensional measurement of religiousness/spirituality for use in health research*. Kalamazoo, MI: John E. Fetzer Institute.

George, L. K., Ellison, C. G., & Larson, D. B. (2002). Explaining the relationships between religious involvement and health. *Psychological Inquiry, 13*, 190–200.

Koenig, H. G. (2009). Research on religion, spirituality, and mental health: A review. *Canadian Journal of Psychiatry, 54*, 283–291.

Koenig, H. G., McCullough, M. E., & Larson, D. B. (2001). *Handbook of religion and health*. New York: Oxford University Press.

Krause, N., Ellison, C., Shaw, B. A., Marcum, J. P., & Boardman, J. D. (2001). Church-based social support and religious coping. *Journal for the Scientific Study of Religion, 40*, 637–656.

McCullough, M. E., Hoyt, W. T., Larson, D. B., Koenig, H. G., & Thoresen, C. (2000). Religious involvement and mortality: A meta-analytic review. *Health Psychology, 19*, 211–222.

Miller, L., & Kelley, B. S. (2005). Relationships of religiosity and spirituality with mental health and psychopathology. In R. F. Paloutzian & C. L. Park (Eds.), *Handbook of the psychology of religion and spirituality* (pp. 435–459). New York: Guilford.

Oman, D., & Thoresen, C. E. (2005). Do religion and spirituality influence health? In R. F. Paloutzian & C. L. Park (Eds.), *Handbook of the psychology of religion and spirituality* (pp. 435–459). New York: Guilford.

Powell, L. H., Shahabi, L., & Thorensen, C. E. (2003). Religion and spirituality: Linkages to physical health. *American Psychologist, 58*, 36–52.

Strawbridge, W. J., Shema, S. J., Cohen, R. D., & Kaplan, G. A. (2001). Religious attendance increases survival by improving and maintaining good health practices, mental health, and stable marriages. *Annals of Behavioral Medicine, 23*, 68–74.

Taylor, S. E. (2011). Social support: A Review. In M. S. Friedman (Ed.), *The handbook of health psychology* (pp. 189–214). New York: Oxford University Press.

Religious Struggle

▶ Religious Coping

Religiousness

▶ Religion/Spirituality
▶ Spirituality

Religiousness and Health

▶ Spirituality and Health

Religiousness/Religiosity

Stephen Gallagher and Warren Tierney
Department of Psychology, Faculty of Education & Health Sciences, University of Limerick, Castletroy, Limerick, Ireland

Synonyms

Religion; Spirituality

Definition

The terms religiousness/religiosity are used interchangeably but often defined as an individual's conviction, devotion, and veneration towards a divinity. However, in its most comprehensive use, religiosity can encapsulate all dimensions of religion, yet the concept can also be used in a narrow sense to denote an extreme view and over dedication to religious rituals and traditions. This rigid form of religiosity in essence is often viewed as a negative side of the religious experience, it can be typified by an over involvement in religious practices which are deemed to be beyond the social norms of one's faith.

Description

Religiousness/Religiosity and Health

As the use of the concept religiosity has a certain ambiguity in its definition, the lack of clarification can lead to much confusion among researchers. Further, adding to this complexity is the fact that research has found both negative and positive relationships between religiosity and a variety of health outcomes (Miller & Kelley, 2005). Thus, future researchers may need to investigate not only linear patterns, but to test for nonlinear associations between these particular constructs and need to appreciate that religiosity can often be used to represent an extreme view and over dedication to religious rituals and traditions that are positioned outside social norms. Indeed, such obsessive behaviors are frequently viewed as pathological, and perhaps it is these excessive practices that underlie the negative ill-health associations that have been observed. Moreover, some researchers have proposed a bidirectional and interaction model to explain these complex relationships (Erwin-Cox, Hoffman, Grimes, & Fehl, 2007).

Measuring Religiosity/Religiousness

When one is measuring religiosity and religiousness, they are in fact measuring how religious an individual is. The Faith Maturity Scale (Benson, Donahue, & Erikson, 1993) is an 11-item self-report questionnaire which is segregated into two subscales: horizontal and vertical faith maturity. The vertical scale assesses closeness to God, while the horizontal subscale reviews the existent to which faith has motivated an individual to assist another person. Also, religiousness can be measured using a number of scales such as the Religiosity Index, (Piedmont, 2001) and the Religion Schema Scale (Streib, Hood, & Klein, 2010). However, as researchers neglect to consider that religiousness may also denote religiosity, one must analyze specific questions from the scales in order to assess the religiosity of an individual. Similarly, is not yet clear whether these scales allow for assessment of the more extreme aspects of religiosity which have been associated with ill health.

Cross-References

- ▶ Religion
- ▶ Spirituality

References and Readings

Benson, P. L., Donahue, M. J., & Erikson, J. A. (1993). The faith maturity scale: Conceptualization, measurement, and empirical validation. *Research in the Social Scientific Study of Religion, 5*, 1–26.

Erwin-Cox, B., Hoffman, L., Grimes, C. S. M., & Fehl, S. (2007). Spirituality, health, and mental health: A holistic model. In K. Rockefeller (Ed.), *Psychology, spirituality and healthcare*. Westport, CT: Praiger Books.

Miller, L., & Kelley, B. (2005). Relationships of religiosity and spirituality with mental health and psychopathology. In R. F. Paloutzian & C. L. Park (Eds.), *Handbook of the psychology of religion and spirituality*. New York: Guilford Press.

Piedmont, R. L. (2001). Spiritual transcendence and the scientific study of spirituality. *Journal of Rehabilitation, 67*, 4–14.

Streib, H., Hood, R. R., & Klein, C. (2010). The religious schema scale: Construction and initial validation of a quantitative measure for religious styles. *The International Journal for the Psychology of Religion, 20*, 151–172.

REM Sleep

Salvatore Insana
Western Psychiatric Institute and Clinic, Pittsburgh, PA, USA

Synonyms

Active sleep; Paradoxal sleep; Phasic REM; Tonic REM

Definition

Rapid eye movement (REM) sleep is a behavioral classification of sleep that is characteristic to rapid jerky eye movements and is associated with increased incidence of dream activity. REM is present in humans from birth throughout the life span and is expressed by all terrestrial mammals as well as birds.

Description

Rapid eye movement (REM) sleep is widely known by its shortened acronym "REM" sleep – when pronounced, REM rhymes with "gem." Sleep can be measured with polysomnography (PSG). Polysomnographically measured sleep can be classified into distinct categories that include N1, N2, N3, and REM – the focus of this entry. REM can be classified as defined by the 2007 American Academy of Sleep Medicine sleep scoring manual (Iber, Ancoli-Israel, Chesson, & Quan, 2007) and is similarly classified according to the Rechtschaffen and Kales "classic criteria" (Rechtschaffen & Kales, 1968). REM sleep was given its name due to the rapid and jerky eye movements that occur during this behavioral state. Another common term used to describe REM sleep is "paradoxal sleep" because brain activity during REM sleep has a striking resemblance to that observed during wakefulness, hence the paradox. The term "paradoxal sleep" was coined in the late 1950s by Michel Jouvet, M.D.

REM sleep was discovered and first reported in the early 1950s by Eugene Aserinsky, Ph.D. and his mentor Nithaniel Kleitman, Ph.D. The discovery was made through Aserinsky's doctoral dissertation study, serendipitously, when Aserinsky observed rapid eye movements among his sleeping participants (Aserinsky & Kleitman, 1953). During the late 1950s and early 1960s, William C. Dement, M.D., Ph.D. began researching REM sleep in greater detail (e.g., Dement & Kleitman, 1957), which initiated his to-be legendary career in sleep science, and provided a platform for sleep science to advance. During the 1950s and 1960s era, the groundbreaking discovery of REM sleep lead to the acknowledgment of brain activity during sleep, which countered the conventional wisdom that the brain was inactive during this behavioral state. In sum, the discovery of REM sleep propagated the investigations that built the foundation for the development of modern sleep medicine as it stands today (Dement, 2005).

A characteristic component of REM sleep is an unstructured pattern of low amplitude (4–7 Hz) mixed frequency neurological activity, as measured by electroencephalography. The neurological activity during REM sleep displays sawtooth wave forms or trains of sharp triangular waves that occur at 2–6 Hz. Behavioral and physiological changes occur during REM sleep, including rapid eye movements, poikilothermia, atonia (i.e., low or absent muscle tone), fast bursts of transient muscle activity, shallow irregular breathing, increased heart rate and blood pressure, increased genital blood flow, and increased neurological oxygen and glucose metabolism. Poikilothermia is the loss of thermoregulatory control, resulting in the body temperature gravitating toward the ambient environmental temperature; sweating and shivering also cease. Atonia is when the skeletal musculature essentially enters a state of paralysis, which likely functions to avoid acting out the dreams that typically occur during REM sleep. Transient increases in heart rate, blood pressure, and irregular breathing typically occur during dream content and co-occur with rapid eye movements. Increased genital blood flow leads to erections that are unrelated

to dream content for men and increased vaginal blood flow for women. Dreams typically occur during REM sleep (Dement & Kleitman, 1957); brain activity associated with dreams increases, which propagates increased neurological oxygen and glucose metabolism. Due to the multiple physiological and behavioral expressions during REM sleep, REM sleep can be further subdescribed as phasic or tonic. Phasic is the occurrence of rapid eye movements and muscle twitches, whereas tonic is the more constant state of REM between the phasic events.

Typically, sleep transitions from wake into N1, followed by N2, followed by N3, and then followed by REM – this progressive series is termed a "sleep cycle." Sleep cycles typically occur throughout sleep at approximately 50-min intervals among infants and approximately 90–110-min intervals among adults. Several sleep cycles occur throughout the night, and the time spent in the different sleep stages changes within each sleep cycle. During the first sleep cycle, REM may only last several minutes; REM episodes within each subsequent sleep cycle become progressively longer throughout the night. Thus, typically, the first portion of the night primarily consists of non-REM sleep (i.e., N1, N2, and N3), and the second portion of the night primarily consists of REM sleep.

Although the true function of sleep, and particularly REM sleep, is unknown, to date several functions of REM sleep have been delineated (e.g., Rector, Schei, Van Dongen, Belenky, & Krueger, 2009; Siegel, 2009). Dreams typically occur during REM sleep; although, the purpose of dreaming remains unknown. REM sleep may stimulate the brain during sleep through dreaming. REM sleep appears central to memory formation, where REM sleep and non-REM sleep (N1, N2, N3) complement each other to process and consolidate various memory forms (Diekelmann & Born, 2010; Stickgold & Walker, 2007). REM sleep also appears to be central to emotion regulation (Walker & van der Helm, 2009). Despite the unknown purpose of REM sleep, it appears necessary to obtain. For example, total sleep deprivation, as well as experimentally induced REM sleep deprivation, leads to a compensatory increase in time spent in REM sleep during the following sleep episode, a phenomenon known as REM rebound. Furthermore, REM sleep has been observed among all terrestrial mammals and birds, but not reptiles.

Entering directly into REM sleep from wake, instead of into N1 first, is associated with a sleep disorder called narcolepsy. Several parasomnias are particularly linked to REM sleep, including nightmares, isolated sleep paralysis, and REM sleep behavior disorder. REM sleep disturbances are implicated in and are associated with numerous psychiatric disorders, including depression, schizophrenia, bipolar disorder, and posttraumatic stress disorder.

Human Development: From birth to approximately 6 months post-term, infant sleep patterns are classified into either REM or NREM; during the birth–6-month postterm period, these classifications are also referred to as active sleep and quiet sleep, respectively. Following 6 months post-term, infant NREM sleep patterns become more complex and can be categorized into N1, N2, and N3 sleep stages – in addition to REM sleep. During the first few postterm months, infants spend approximately 50% of time in both REM and NREM sleep. During childhood, the percentage of time spent in REM sleep drops off, levels out to approximately 25% of the entire sleep time, and then gradually decreases across the life span. Across the life span, generally, the percentages of N1 and N2 sleep increase, whereas N3 and REM sleep decrease (Ohayon, Carskadon, Guilleminault, & Vitiello, 2004).

Cross-References

- ▶ Non-REM Sleep
- ▶ Polysomnography
- ▶ Sleep
- ▶ Sleep Architecture

References and Readings

Aserinsky, E., & Kleitman, N. (1953). Regularly occurring periods of eye motility, and concomitant phenomena, during sleep. *Science, 118*, 273–274.

Dement, W. C. (2005). History of sleep medicine. *Neurologic Clinics, 23*, 945–965.
Dement, W., & Kleitman, N. (1957). Cyclic variations in EEG during sleep and their relation to eye movements, body motility, and dreaming. *Electroencephalography and Clinical Neurophysiology, 9*, 673–90.
Diekelmann, S., & Born, J. (2010). The memory function of sleep. *Nature Reviews. Neuroscience, 11*, 114–126.
Iber, C., Ancoli-Israel, S., Chesson, A., & Quan, S. F., for the American Academy of Sleep Medicine. (2007). *The AASM manual for the scoring of sleep and associated events: Rules, terminology and technical specifications* (1st ed.). Westchester, IL: American Academy of Sleep Medicine.
Ohayon, M. M., Carskadon, M. A., Guilleminault, C., & Vitiello, M. V. (2004). Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: Developing normative sleep values across the human lifespan. *Sleep, 27*, 1255–1273.
Rechtschaffen, A., & Kales, A. (1968). *A manual of standardized, techniques and scoring system for sleep stages in human subjects*. Washington, DC: NIH Publication No. 204, US Government Printing Office.
Rector, D. M., Schei, J. L., Van Dongen, H. P., Belenky, G., & Krueger, J. M. (2009). Physiological markers of local sleep. *The European Journal of Neuroscience, 29*, 1771–1778.
Siegel, J. M. (2009). Sleep viewed as a state of adaptive inactivity. *Nature Reviews: Neuroscience, 10*, 747–753.
Stickgold, R., & Walker, M. P. (2007). Sleep-dependent memory consolidation and reconsolidation. *Sleep Medicine, 8*, 331–343.
Walker, M. P., & van der Helm, E. (2009). Overnight therapy? The role of sleep in emotional brain processing. *Psychological Bulletin, 135*, 731–748.

Remission and Remission Prevention

► Relapse, Relapse Prevention

Renin

George J. Trachte
Academic Health Center, School of Medicine-Duluth Campus, University of Minnesota, Duluth, MN, USA

Definition

General Background

Renin is an aspartyl protease secreted primarily from the kidney that raises blood pressure, retains sodium, and produces various changes in gene transcription that are usually associated with pathological damage, particularly to the kidney and heart. Specifically renin cleaves a 10 amino acid peptide (angiotensin I) from a large protein (angiotensinogen) secreted by the liver. The angiotensin I is subsequently modified by removal of two amino acids by angiotensin converting enzyme to form an eight amino acid product, angiotensin II. Angiotensin II is the primary mediator of renin effects. Pathological conditions involving this system include the following: congestive heart failure, myocardial infarction, hypertension, and diabetes mellitus (renal damage). Inhibitors of the system are beneficial in treating these pathologies and include direct renin inhibitors (aliskerin), converting enzyme inhibitors (lisinopril and other "prils"), and angiotensin receptor antagonists (candesartan and other "artans").

Physiological Relevance

The renin-angiotensin system is thought to be important in adaptations to low-salt diets and normal development of the kidneys in utero. Inhibition of the system with converting enzyme inhibitors has resulted in greater fetal mortality and children born with malformed kidneys.

Control of Renin Release

Renin is secreted in response to the following: a drop in pressure in the renal artery, reduced dietary sodium intake, sympathetic nerve activation leading to the stimulation of ß1 receptors, and prostaglandins. Renin release is inhibited by: elevated renal artery pressure, increased sodium consumption, ß receptor antagonists, and atrial natriuretic peptide. The drop in renal artery pressure is the specific stimulus for renin release leading to hypertension in renal artery stenosis. Dietary sodium status is primarily responsible for the daily fluctuations in renin levels. Prostaglandins are the specific stimulus leading to excessive activity of this system resulting in potassium depletion in Bartter's syndrome.

Renin Tissue Localization and Molecular Biology

In addition to the kidney, renin (REN) mRNA is found in the following: adrenal gland, testis, ovary, liver, brain, hypothalamus, salivary gland, adipose tissue and, in lower levels, lung, spleen, thymus, and prostate. The REN genes code for a large 406 amino acid protein (pre-prorenin). The removal of 23 amino acids from the carboxyl terminal results in the formation of prorenin. The final step in the process involves removal of 43 amino acids from the amino terminal to form the 340 amino acid active protein known as renin. Both renin and prorenin are secreted into the circulation. Prorenin can be activated by binding to its receptor even in the absence of conversion to renin. The enzyme converting prorenin to renin has not been identified but is believed to be specific to the kidney because nephrectomized patients or animals contain no renin in their circulation but prorenin is present. Inhibitors of the prorenin receptor have been shown to be beneficial in diabetic nephropathy.

Behavioral Actions of Renin

Renin and other components of the renin-angiotensin system are present in the brain and are believed to impact memory negatively and to exacerbate neurogenic diseases such as Alzheimer's disease and other neurodegenerative disorders.

Cross-References

- ▶ Blood Pressure
- ▶ Diabetes
- ▶ Heart Failure
- ▶ Hypertension

Repeated Measures Design

- ▶ Crossover Design

Repetitive Thinking

- ▶ Perseverative Cognition
- ▶ Worry

Repression

- ▶ Defensiveness

Repressive Coping

- ▶ Coping
- ▶ Defensiveness

Reproductive Health

Ulrike Ehlert[1] and Simona Fischbacher[2]
[1]Department of Psychology, University of Zurich, Binzmuehlestrasse, Zurich, Switzerland
[2]Klinische Psychologie und Psychotherapie, Universität Zürich, Zürich, Switzerland

Definition

As a part of general health and development, reproductive health, which refers to the reproductive processes and functions, is essential in adolescence and adulthood and also affects one's well-being beyond the reproductive years. Healthy development of the reproductive system in the fetus and during early childhood is required to achieve reproductive capacity. Reproductive maturity is usually reached after puberty, a phase characterized by biologically based growth and change processes modulated by psychosocial factors. In adulthood, when reproductive capacity is completely developed, a healthy menstrual

cycle and male reproductive functioning are biological requirements for reproduction. Furthermore, individual reproductive goals need to be developed and pursued. For women, the fertile phase ends rapidly with menopause, whereas men usually experience a gradual decrease in fertility with increase in age.

Description

A Biopsychological Approach to Reproductive Health

From a biopsychological perspective, the association between psycho-endocrinological or psycho-immunological factors and reproductive processes is of interest. Deviations from the healthy sex hormone secretion are associated with the development and maintenance of various chronic diseases and modulate neurotransmitter systems associated with psychiatric disorders. On the other hand, infertility and diseases of the reproductive system affect one's psychological well-being and may lead to impairments in social life, work productivity, interpersonal relationships, and sexuality (Stanton, Lobel, Sears, & Stein De Luca, 2002).

Stress is one of the most potent factors affecting reproductive health and is related to several reproductive disorders, such as cycle length disorders, infertility, premenstrual syndrome, or erectile dysfunction. The bidirectional interaction between the hypothalamic-pituitary-adrenal (HPA) axis, as the most prominent stress-associated hormone system, and the hypothalamic-pituitary-gonadal (HPG) axis is well documented. The HPA axis exerts an inhibitory effect on the HPG axis, due to the inhibiting action of corticotropin-releasing hormone (CRH), arginine-vasopressin (AVP), and CRH-induced beta-endorphin release. In addition, cortisol suppresses secretion of the gonadotropin-releasing hormone (GnRH) and the luteinizing hormone (LH), and inhibits ovarian sex steroids (estrogen and progesterone) and testosterone. Sex steroids, on the other hand, have a modulating effect on the HPA axis (Creatsas, Mastorakos, & Chrousos, 2006).

Women's Reproductive Health

Women's reproductive health includes not only reproductive processes, a healthy reproductive system, and reproductive functioning, but also psychological well-being and a safe and satisfying sexual life. The biological basis of women's reproductive health is a healthy and ovulatory menstrual cycle. This entry will give a brief overview on the endocrinological processes of a healthy menstrual cycle.

Endocrinology of female reproduction and the menstrual cycle: The most important physiological function of the hypothalamic-pituitary-ovarian (HPO) axis is the control of reproduction. This hormonal system includes the hypothalamic GnRH, which is controlled by the GnRH-pulse generator, the pituitary gonadotropes, and sex-hormones produced by the ovaries. The menstrual cycle can be divided into three successive phases: the follicular phase, the ovulatory phase, and the luteal phase. Menstrual bleeding marks the first day of the follicular phase and occurs after the decrease in levels of progesterone and estrogen at the end of the previous cycle. At the beginning of the follicular phase, the follicle-stimulating hormone (FSH) stimulates the development of several follicles, resulting in a slow increase in a woman's estrogen level. As the estradiol level increases, its negative feedback on the pituitary inhibits secretion of FSH; consequently, the FSH level decreases. Shortly before ovulation occurs, maturation of the dominant follicle and the corresponding peak in estrogen level lead to a positive feedback on LH, generating a preovulatory LH surge. This surge causes the follicle to swell and rupture. Ovulation usually occurs after the LH surge. The following luteal phase is characterized by the formation of the corpus luteum and its production of progesterone and estrogen, which help to prepare the endometrium for implantation and maintenance of pregnancy. High progesterone level exerts negative feedback on the GnRH pulse frequency, and consequently, FSH and LH secretions decrease. Lacking stimulation by FSH and LH, the corpus luteum regresses after approximately 14 days. The following decline in estrogen and progesterone levels remove the negative

feedback control on FSH, and its level increases again to initiate the next menstrual cycle.

The first day of menstrual bleeding is counted as the beginning of the menstrual cycle, which lasts until the day before the next onset of menses. Between the ages of 20 and 40 years, the average cycle length ranges from 27 to 30 days. Whereas the follicular phase can vary in length (13–15 days), the luteal phase lasts approximately 14 days, correlating with the life span of the corpus luteum. Menstrual cycle lengths vary most after menarche and before menopause, mainly because of anovulatory cycles (Rees, Hope, & Ravnikar, 2005).

Women's Reproductive Disorders

Reproductive health is influenced by a number of different factors, such as age, genetics, lifestyle, and exposure to environmental toxins. Thus, women's reproductive health not only includes the healthy aspects of reproduction but also diseases and conditions that negatively affect the female reproductive system. Reproductive disorders, defined as abnormalities in the reproductive system, reduce reproduction and often affect overall health, psychological well-being, and sex life. This section will provide a brief outline of selected menstrual cycle–related reproductive disorders from the perspective of behavioral medicine.

Menstrual Cycle–Related Disorders

Cycle Length Disorders and Bleeding Disorders: Cycle length disorders and bleeding disorders are deviations from normative patterns of menstrual functioning in terms of menstruation frequency or bleeding pattern. Cycle length disorders and bleeding disorders are often associated with anovulatory cycles and infertility. The most common are oligomenorrhea, amenorrhea, menorrhagia, and spotting or breakthrough bleeding. Oligomenorrhea is defined as infrequent menstruation, with 5–9 periods within 1 year; amenorrhea is the absence of a menstrual period. Primary amenorrhea (or the absence of a menstrual period until the age of 16) is a relatively rare disorder, whereas secondary amenorrhea (i.e., the absence of a menstrual period for 3 or more months) affects about 3% of women during the reproductive years (Rees et al., 2005). Menorrhagia is defined as prolonged and/or abnormally heavy periods, with a blood loss of more than 80 ml/period. Spotting or breakthrough bleeding usually affects young or perimenopausal women. Cycle length disorders or bleeding disorders can have organic, endocrine, or psychosocial causes. Exposure to chronic stress or traumatic events seems to influence the hypothalamic pulse generator, leading to a disturbance of reproductive function. Behavioral factors like restraint eating or excessive exercise may contribute to the development of menstrual disorders. In addition, there is a close association between eating disorders, such as anorexia nervosa or obesity, and cycle length disorders (Creatsas et al., 2006).

Dysmenorrhea: Dysmenorrhea is defined as pelvic pain associated with the bleeding phase of the menstrual cycle and can be classified as either primary or secondary, depending on the absence or presence of an identifiable pelvic pathology. Painful cramping is felt in the lower abdomen or back area and is often accompanied by somatic or psychological symptoms, for example, headache, nausea, vomiting, dizziness, restlessness, fatigue, and depressed mood. Dysmenorrhea often interferes with daily activities and may result in absenteeism from school or work (Stanton et al., 2002). The cause of dysmenorrhea can be either organic (e.g., endometriosis) or functional (associated with excessive production of prostaglandines). Studies have shown that affected women show a learning history of enhanced body vigilance and anticipated pain-avoidance behavior. The widely used therapy options for dysmenorrhea are nonsteroidal anti-inflammatory drugs (NSAIDs) to inhibit prostaglandin production and oral contraceptives to suppress ovulation. Pain relief can also be achieved by behavioral therapy methods, such as relaxation techniques or an increase in (physical) activity.

Premenstrual Syndrome and Premenstrual Dysphoric Disorder: Up to 90% of women of reproductive age experience some degree of premenstrual symptoms. A smaller group of women

report symptoms meeting criteria of premenstrual syndrome (PMS), and less than 10% of women are classified as having premenstrual dysphoric disorder (PMDD). According to the *Diagnostic and Statistical Manual of Mental Health Disorders* 5th edition (in development process; www.dsm5.org), indications of PMDD include depressive symptoms (decreased interest in usual activities, depressed mood, self-deprecating thoughts, difficulty concentrating, hopelessness, affective lability, and change in sleep and appetite), anxiety symptoms (feeling of tension or anxiety, irritability or anger, and feeling overwhelmed), and physical symptoms (bloating, breast tenderness, and joint or muscle pain). Symptoms begin in the late luteal phase and remit after the onset of the follicular phase. Women suffering from PMDD often experience impairment of work productivity and interpersonal relationships and increased healthcare utilization. Although the etiology of PMS/PMDD has not been definitely established, studies suggest that the cause of PMS/PMDD is allopregnanolone and $GABA_A$ receptor dysfunction, serotonin abnormalities, and a special sensitivity to the withdrawal or fluctuation of estrogen and progesterone (Freeman, 2003). Depending on the predominant symptoms, different treatment options seem to be effective: oral contraceptives, stress management training, exercise, dietary regulation, vitamins, herbal preparations, cognitive behavioral therapy (CBT), and selective serotonin reuptake inhibitors (SSRIs) (Rapkin, 2003).

Endometriosis: Endometriosis is a common gynecological condition that affects women of reproductive age. It is a condition in which extrauterine, glandular, and stromal endometrial-like tissue grows in physiologically abnormal locations, often within the fallopian tubes, on the outside of the tubes and ovaries, or on the surface of the uterus and intestines. These cells are influenced by monthly hormonal changes; they build up during the menstrual cycle and are shed as levels of sex steroids decrease. Endometriosis is associated with several symptoms, including chronic pelvic pain, dysmenorrhea, menstrual disorders, infertility, dyspareunia (painful intercourse), and dysuria (painful or difficult urination). The exact cause of endometriosis remains unknown; several factors have been suggested, for example, the presence of estrogen, retrograde menstruation, hereditary factors, immune system function, and environmental influence. In addition, discrepancies between the reported location of pain and endoscopic findings make it difficult for women and their gynecologists to handle the symptoms. Affected women present greater susceptibility to mood disturbances, and depressive or anxiety symptoms may play a role in the maintenance of pelvic pain (Gao et al., 2006).

Polycystic Ovary Syndrome: Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders, affecting about 5–10% of women of childbearing age. Symptoms are heterogeneous and include oligo- or anovulation, biochemical or clinical hyperandrogenism (hirsutism, acne, and alopecia), polycystic ovaries, hyperinsulinemia, infertility, and obesity. The exact etiology has not yet been clarified, but considerable knowledge of the underlying pathophysiologic mechanism and management has been gained. Various lines of evidence suggest a genetic component, but the candidate genes have yet to be identified. Several hypotheses have been proposed to explain the pathogenesis of PCOS:

(a) An alteration in LH pulse frequency and amplitude, resulting from an increased frequency of GnRH pulses and leading to increased androgen synthesis in the theca cells within the ovary
(b) Hyperinsulinemia, resulting from insulin resistance and acting synergistically with LH to stimulate ovarian testosterone production and decrease serum sex hormone–binding globulin (SHBG) concentrations
(c) A primary defect of sex steroid synthesis, leading to increased androgen production and anovulation

The treatment of PCOS depends on the predominant symptoms and the women's stage of life (Jones, Hall, Balen, & Ledger, 2008). The management (e.g., lifestyle management and exercise) of PCOS is directed toward improving the woman's health-related quality of life.

A growing body of evidence suggests that affected women are at increased risk for cardiovascular disease, type II diabetes mellitus, dyslipidemia, metabolic syndrome, and affective disorders (Azziz, 2007).

Reproductive Health Issues Not Related to the Menstrual Cycle

The following disorders are not related to the endocrinological processes of the menstrual cycle but to female sexual health.

Vulvodynia: Vulvodynia is defined as vulvar discomfort (burning, irritation, or sharp pain) often accompanied by sexual dysfunction and affective distress in the absence of anatomic or neurologic findings. Many factors have been proposed to be possible causes (e.g., genetic, immune, or hormonal factors), yet the etiology remains unknown. Comorbidity with psychosomatic disorders, such as irritable bowel syndrome or fibromyalgia, points to a potential psychosomatic cause. Vulvar discomfort affects one's psychological well-being, relationship quality, and sexuality; however, only a few randomized controlled studies have been conducted to study vulvodynia. Apart from numerous medical treatment options (including topical, oral, and injectable medications), biofeedback and relaxation techniques are helpful, especially when vaginismus is concomitant. There is growing evidence that CBT for pain management reduces pain severity and improves sexual functioning in women suffering from vulvodynia.

Pruritus Vulva: Pruritus vulva is characterized by severe itching of the external female genitalia. It can be caused by infections, systemic diseases, or mechanical stimulation. Scratching, which relieves the physical discomfort, may eventually lead to chronic painful sensations in the vulva area. Therapeutic interventions, such as psychoeducation, diverting attention, and development of new coping skills, seem to be promising approaches.

Other Common Reproductive Diseases/Disorders

- Ovarian cancer
- Cervical cancer
- Neoplasia and dysplasia of the cervix
- Uterine cancer
- Sexually transmitted diseases
- Pelvic inflammatory disease/pelvic pain
- Ovarian cysts
- Benign/malignant breast ailments
- Medical complications during gestation
- Low birth weight
- Reduced fertility/infertility

Male Reproductive Health

The endocrinology of male reproduction and a healthy reproductive system are biological requirements for successful reproduction and an important part of overall health in men. The following section will describe the endocrinology of male reproduction and the healthy male reproductive system.

Endocrinology of Male Reproduction and the Male Reproductive System: The male morphologic reproductive system includes the hypothalamic-pituitary-testicular (HPT) axis, penis, scrotum, epididymis, vas deferens, accessory glands, seminal vesicles, prostate, and urethra. The testes have two important functions: production of sperm and synthesis of androgen (testosterone). Both functions are regulated by an endocrine feedback mechanism of the hypothalamus and the pituitary. The pulsatile hypothalamic release of GnRH causes the secretion of FSH and LH. Whereas FSH primarily acts on Sertoli cells to facilitate spermatogenesis, LH acts on Leydig cells in the testicular interstitium to stimulate steroidogenesis, particularly of testosterone. Testosterone is necessary for spermatogenesis, and together with inhibin (which is secreted by the Sertoli cells), it downregulates LH and FSH release via a negative feedback loop (Nieschlag & Behre, 2001).

Male Reproductive Disorders

The above-mentioned endocrinological functioning of male reproduction describes healthy male reproduction. However, male reproduction can be diminished by a variety of diseases and conditions. In the following section, selected male reproductive disorders will be discussed.

Hypogonadism: Primary or secondary hypogonadism is a defect of the testes, resulting in inadequate production of androgen (causing androgen deficiency) and germ cells. Primary hypogonadism is caused by abnormal testicular function. Reasons for primary hypogonadism include genetic disorders (e.g., Klinefelter's syndrome), viral orchitis, and the influence of toxins, among others. Secondary hypogonadism results from a malfunction in the hypothalamus or pituitary gland, which inhibits secretion of pituitary hormones. Symptoms of hypogonadism can be observed in the developing fetus (e.g., improperly formed external genitals), during puberty (e.g., absence of testicular growth and secondary sexual characteristics), and adulthood (e.g., decreased fertility). Hypogonadism is associated with loss of libido, erectile dysfunction, concentration problems, decreased interest in activities, depression, irritability, sleep disorders, loss of muscle strength, and loss of bone density. The most commonly used therapy for hypogonadism is testosterone replacement therapy (Nieschlag & Behre, 2001).

Erectile Dysfunction: Erectile dysfunction is defined as the "consistent inability to achieve or maintain an erection sufficient for satisfactory sexual performance." The prevalence rate ranges from 7% for men aged between 18 and 29 years to 18% for men aged between 50 and 59 years. The cause of erectile dysfunction can be organic or psychogenic. Several risk factors have been detected, including cardiovascular disease, hypertension, smoking, stress, and alcohol or drug abuse. Psychosocial distress, relationship problems, depression, and performance anxiety seem to be potential psychosocial causes. The management of erectile dysfunction includes lifestyle modification, medication (sildenafil citrate), psychotherapy, and surgery. There is evidence that psychotherapy improves erectile functioning, interpersonal assertiveness, and relationship satisfaction and reduces anxiety in men suffering from erectile dysfunction (Nieschlag & Behre, 2001).

Chronic Prostatitis/Chronic Pelvic Pain Syndrome: Chronic prostatitis/chronic pelvic pain syndrome (CP/CPPS) is characterized by pain in the pelvis or perineum, which may be accompanied by ejaculatory disturbances, dysuria, fatigue, low libido, and erectile dysfunction. Chronic prostatitis/chronic pelvic pain syndrome can result either from a bacterial infection (5–10% of all cases) or may not be associated with an identifiable cause, which is the most common type of CP/CPPS. Several different causes have been suggested (including immunological, neurological, endocrine, and psychological causes), yet the actual cause remains unknown. Recently published studies have shown that several traditionally used medications (antimicrobials, anti-inflammatories, and alpha-blockers) do not significantly ameliorate symptoms of CP/CPPS (Tripp, Nickel, Landis, Wang, & Knauss, 2004). Affected men often report helplessness, catastrophic thinking about pain, pain-contingent resting for coping with pain, and a low perceived control over pain. There is primary evidence that psychotherapy, which focuses on decreasing negative thoughts and emotional responses related to pain while building up self-management skills, is predictive of positive treatment outcomes for patients with CP/CPPS (Nickel, Mullins, & Tripp, 2007).

Other Common Reproductive Diseases/Disorders

- Infertility
- Sexually transmissible diseases
- Testicular cancer

Reproductive Health in Middle-Aged and Elderly Men

Whereas women show an unavoidable clear cessation of reproductive capacity, men usually maintain fertility through old age. Similar to women, men may also experience physical and psychological symptoms due to changing concentrations of steroid hormones from midlife onward. Age-related decline in insulin, growth hormone, thyroid hormones, aldosterone, melatonin, and progesterone has been demonstrated. Cortisol, on the other hand, increases with advancing age. Testosterone and bioavailable testosterone show a decline of 35–50% between

the ages of 20 and 80 years, whereas the LH level increases slightly with age. In addition to age-related variations in hormone concentrations, loss of circadian rhythm of LH and testosterone levels has been demonstrated. Furthermore, sex hormone–binding globulin (SHBG) increases with age, leading to reduced bioavailable testosterone (Hermann, Untergasser, Rumpold, & Berger, 2000).

Partial Androgen Deficiency in Aging Men: If decline in testosterone levels is associated with symptoms of androgen deficiency, the condition is called andropause, androgen deficiency in aging men, or partial androgen deficiency in aging men. The incidence and prevalence of androgen deficiency (or unequivocally low serum testosterone levels) in middle-aged men and elderly men are difficult to determine, particularly because of controversial diagnostic criteria and because some of the symptoms are also experienced by normal aging men. Androgen deficiency is associated with symptoms similar to those experienced by menopausal women or men suffering from hypogonadism. Several studies have indicated that testosterone replacement therapy can improve many of these parameters in middle-aged and elderly men. However, the risks of testosterone replacement therapy (e.g., cardiovascular disease) have not been fully assessed.

Other Age-Associated Disorders: Several age-associated histomorphological alterations of the testes have been observed. Age seems to have a negative impact on spermiogenesis, and there is an association between chromosomal abnormalities in spermatozoa and age. One of the most common age-associated morbid changes is prostate hyperplasia (benign enlargement of the prostate); it is sometimes accompanied by erectile dysfunction, which affects quality of life and can cause mood disorders and relationship problems. The major risk factor for erectile dysfunction is age, with an incidence rate of 5–15% for complete impotence and a prevalence of 52% (of erectile dysfunction of any grade) in men between the ages of 40 and 70 years.

Summary

As a part of general health, reproductive health, which refers to the processes and functions of the reproductive system, is essential across the life span. From a biopsychological perspective, a close association between the endocrinology of the reproductive system and psychosocial well-being is of interest. Normal and healthy functioning of the reproductive system and processes is one of the biological fundaments of psychosocial well-being. Nevertheless, reproductive health includes the diseases, disorders, and conditions that affect the functioning of the male and female reproductive system, their sexual life, and mental health.

Cross-References

- Androgen
- Breast Cancer
- Endometriosis
- Erectile Dysfunction
- Estrogen
- Family Planning
- Gestation
- Menopause
- Puberty
- Sex Hormones
- Sexual Functioning
- Women's Health

References and Readings

Azziz, R. (Ed.). (2007). *The polycystic ovary syndrome. Current concepts on pathogenesis, and clinical care*. New York: Springer.

Creatsas, G., Mastorakos, G., & Chrousos, G. P. (Eds.). (2006). *Women's health and disease. Gynecologic, endocrine, and reproductive issues*. Boston: Blackwell.

Freeman, E. W. (2003). Premenstrual syndrome and premenstrual dysphoric disorder: Definitions and diagnosis. *Psychoneuroendocrinology, 28*, 25–37.

Gao, X., Yeh, Y. C., Outley, J., Simon, J., Botteman, M., & Spalding, J. (2006). Health-related quality of life

burden of women with endometriosis: A literature review. *Current Medical Research and Opinion, 22*(9), 1787–1797.
Hermann, M., Untergasser, G., Rumpold, H., & Berger, P. (2000). Aging of the male reproductive system. *Experimental Gerontology, 35*, 1267–1279.
Jones, G. L., Hall, J. M., Balen, A. H., & Ledger, W. L. (2008). Health-related quality of life measurement in women with polycystic ovary syndrome: A systematic review. *Human Reproduction Update, 14*, 15–25.
Nickel, J. C., Mullins, Ch, & Tripp, D. A. (2007). Development of an evidence-based cognitive behavioral treatment program for men with chronic prostatitis/chronic pelvic pain syndrome. *World Journal of Urology, 26*, 167–172.
Nieschlag, E., & Behre, H. M. (Eds.). (2001). *Andrology. Male reproductive health and dysfunction*. Berlin: Springer.
Rapkin, A. (2003). A review of treatment of premenstrual syndrome and premenstrual dysphoric disorder. *Psychoneuroendocrinology, 28*, 39–53.
Rees, M., Hope, S., & Ravnikar, V. (Eds.). (2005). *The abnormal menstrual cycle*. Oxfordshire: Taylor & Francis Group.
Stanton, A. L., Lobel, M., Sears, S., & Stein De Luca, R. (2002). Psychosocial aspects of selected issues in women's reproductive health: Current status and future directions. *Journal of Consulting and Clinical Psychology, 70*(3), 751–770.
Tripp, D. A., Nickel, J. C., Landis, J. R., Wang, Y. L., & Knauss, J. S. (2004). Predictors of quality of life and pain in chronic prostatitis/chronic pelvic pain syndrome: Findings from the National Institutes of Health Chronic Prostatitis Cohort Study. *British Journal of Urology International, 94*, 1279–1282.

Research Benefits

▶ Research Participation, Risks and Benefits of

Research Ethics Committee

▶ Ethics Committee
▶ Human Subjects Committee
▶ Institutional Review Board (IRB)

Research Hypothesis

▶ Hypothesis Testing

Research Methodology

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Study methodology

Definition

Research methodology falls in between study design (including the design of a clinical trial) and data management and analysis. It is concerned with the acquisition of optimum quality data.

Three factors are of central importance in determining the attainment of statistical significance:

1. Variation *between* the two sets of data (one receiving the behavioral intervention of interest, the test treatment group, and one receiving a control treatment, the control treatment group). This is between-groups variation.
2. Variation *within* the two sets of data, or within-groups variation.
3. The total number of subjects participating in the study (the sum of subjects in each treatment group), sometimes expressed as the size of the trial.

The following statements can then be made for each of these factors, assuming in each case that the other two factors remain constant:

- The greater the between-groups variation (operationalized as the greater the difference between the group means, i.e., the greater the

treatment effect), the more likely it is that the difference will attain statistical significance.
- The greater the within-groups variation, the less likely it is that the difference will attain statistical significance.
- The greater the number of subjects participating in the trial, the more likely it is that the difference will attain statistical significance.

Notice that two of these three factors address variation. Consider the first statement, which is intuitively the most straightforward. The larger the treatment effect, the more likely it is to attain statistical significance. That is, all other factors being constant, the greater the treatment's efficacy the more likely it is that compelling evidence for the use of the new treatment is provided.

The influence of the treatment can be considered a systematic influence. It is the signal in which we are interested. In contrast, in the present context, the within-groups variation can be regarded as nonsystematic variation, or the noise against which we desire to detect the signal. This noise has two components: biological variation and random error. Since we all have unique biological systems, including those pertinent to a treatment's influence on the body (a statement that is true for identical twins and other identical births given the gene-environment interactions they experience), biological variation is inevitable and perfectly normal.

In contrast, random error can be addressed. Indeed, minimizing random error is a key focus of research methodology. There are multiple instances where such error can occur, with the following being just a few examples:
- Administering the wrong behavioral treatment to a subject on one or more occasions during the trial.
- Employing measurement equipment that is substandard (e.g., blood pressure monitors).
- Conducting blood pressure measurements in an inconsistent manner (e.g., using the dominant arm sometimes and the nondominant arm at others).
- Recording a measurement incorrectly.
- Misplacing data.

Optimum quality research methodology contributes to optimum quality data. All researchers must strive to the greatest extent possible to execute their role in a study flawlessly to reduce random error, thus allowing the trial the best opportunity of accurately assessing the test treatment's characteristics.

Cross-References

► Efficacy

Research Participation, Risks and Benefits of

Marianne Shaughnessy
School of Nursing, University of Maryland, Baltimore, MD, USA

Synonyms

Research benefits; Research risks

Definition

Voluntary decision to engage in a process of *organized scientific inquiry (research)*.

Description

The conduct of human subjects research relies on the voluntary participation of persons in research studies. The design of a particular study and definition of the population of interest will define which person or populations will be sought for recruitment into a research study. The decision to participate in a research study begins with a process known as informed consent. Informed consent is referred to as a process because continued review and education must be offered throughout every stage of participation in a study, and consent to continue participation is ongoing.

Through this process, participants are continually informed about the requirements of their participation, any known risks associated with participation, and any potential benefits (to themselves or to a larger community) that may result from their participation. Prior to the start of research projects, an institutional review board will review the risks and benefits of proposed research from a scientific and human subject's perspective. However, before actually enrolling in a research study, every participant (or legally authorized representative, in the event the participant cannot consent on his/her own behalf) should understand the potential risks and benefits to the individual associated with participation in the research. The risks and benefits of participation must be explained completely to all potential participants and, ideally, the participant communicates a thorough understanding of the research procedures, risks, and benefits prior to providing initial consent. In some cases, not all the risks of participation may be known. It is not uncommon for researchers to include a statement in the consent form advising potential participants that there may be unknown risks to participation.

Examples of some of the risks involved in research participation may include physical injury, as may occur when testing the safety of a new drug or medical device, or psychological injury, such as the diagnosis of a mental health problem during testing or data collection. There may also be a financial cost associated with research participation, such as treatment of research related illnesses or injuries not covered by the research study or the participant's medical insurance. However, generally speaking, the most common risk encountered is the breach of confidential information collected during the course of participation in human subjects research. The Health Insurance Portability and Accountability Act of 1996 (HIPAA) has had a significant impact on the way all protected health information (PHI) is collected and stored in both clinical and research arenas; therefore, researchers are required to make provisions to safeguard an individual's health information collected as part of their research participation (U.S. Department of Health & Human Services [DHHS], 2011).

Examples of some of the benefits of research participation may include direct physical benefit, such as relief from disease symptoms from the use of an experimental drug or other intervention. Psychological benefits may evolve through a targeted mental health intervention. Often, subjects will agree to participate in research with the knowledge that there may be no direct benefit to self but that they are making a contribution to science and therefore the greater good of society. Some research studies offer direct financial compensation for participation, but this practice causes concern regarding the potential for payment to unduly influence participation and thus obscure risks, impair judgment, or encourage misrepresentation (Grady, 2005). The research application and consent form should state how any risks will be minimized, and the institutional review board or regulatory body overseeing research should carefully evaluate the research study protocol to ensure that overall, benefits outweigh the risks.

Cross-References

- ▶ Confidentiality
- ▶ Protection of Human Subjects

References and Readings

U.S. Department of Health & Human Services. (2011). Health information privacy. Accessed May 2, 2011, from http://www.hhs.gov/ocr/privacy/

Grady, C. (2005). Payment of clinical research subjects. *Journal of Clinical Investigation, 115*(7), 1681–1687.

Research Risks

▶ Research Participation, Risks and Benefits of

Research to Practice Translation

Bonnie Spring, Alex Pictor, Andrew DeMott, Molly Ferguson and Arlen C. Moller
Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Synonyms

Dissemination and implementation; Knowledge translation; Translational research

Definition

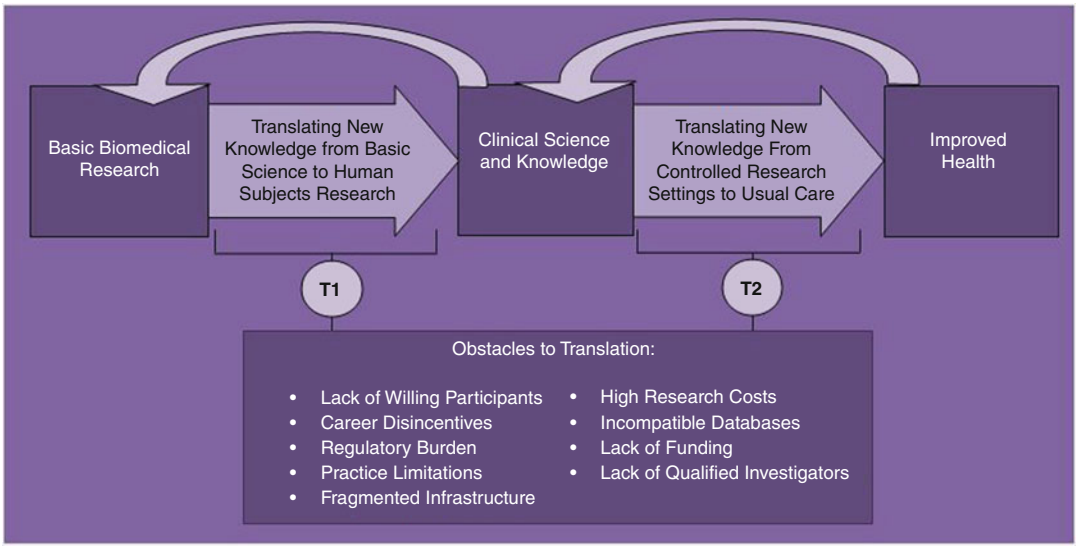
Research to practice translation is the process of adapting principles and findings from scientific investigation in order to apply them in real world practice (Sung et al., 2003; Woolf, 2008). The translational process typically proceeds through a series of phases: T1 (translation of fundamental research findings to develop new practical applications), T2 (adaptation of efficacious treatments into a form that is effective in usual practice settings), and T3 (dissemination and implementation of research-tested interventions so that they are taken up widely by care systems and become usual practice). Although T1, T2, and T3 are all part of research to practice translation, the phrase connotes an emphasis on the later stages, particularly T3. The problem addressed is the slow and incomplete uptake of scientific discoveries into clinical and public health practice. Barriers that impede translation include lack of resources for practitioner training, resistance to change, competing institutional priorities, and lack of infrastructure to support new practices.

Description

Background

A bifurcation between basic and applied research has characterized US science policy since the mid-1940s. In the wake of World War II, Vannevar Bush, Science Advisor to President Franklin D. Roosevelt, wrote an advisory entitled "Science: The Endless Frontier." In it, he advocated for major federal investment in basic science research as the engine that would drive the post-war economy. Bush drew a distinction between basic research, motivated fundamentally by curiosity and a quest for understanding, versus applied research, motivated by the need to solve practical problems. He contended that new insights from basic research are the prime mover of technological and medical progress. Because major advances result from discoveries in remote, unexpected scientific domains, it is virtually impossible to predict which basic scientific inquiries will produce major advances. Consequently, Bush aimed to protect unfettered, curiosity-driven pursuit of scientific understanding from being constrained by worries about whether the knowledge to be gained had any practical use. Arguing that industrial and medical progress would stagnate if basic research were neglected, he succeeded in prompting major federal investment in basic research, including creation of what was to become the National Science Foundation.

Over the next half century, it gradually became apparent that the insights emerging from basic science research were not being translated into practical applications. An analysis by Balas and Boren (2000) indicated that, even after 17 years, only 14% of research knowledge is adopted into practice. By 2001, the Institute of Medicine (IOM) used the term "chasm" to describe the gap between scientific knowledge and actual clinical practice. There was also growing realization that Vannevar Bush's contention that "applied research invariably drives out pure" drew too sharp a dichotomy. In a 1996 book entitled, *Pasteur's Quadrant*, Donald Stokes presented a fourfold table, whereby research could be either low or high on both quest for fundamental understanding and considerations about use. Stokes argued that the most generative, valuable research falls into *Pasteur's Quadrant*, inspired simultaneously *both* by the need to solve a practical problem *and* by curiosity to understand how nature works. A consequence of these



Research to Practice Translation, Fig. 1 Translating basic biomedical research into clinical practice and improved health outcomes (Adapted from Sung et al. (2003))

realizations has been some realignment of budget allocations at the National Institutes of Health (NIH) to support greater investment in translational and applied research. In fiscal year 1998, the NIH allocated 31% of its budget to applied research and 57% to basic science (Institute of Medicine [IOM], 1998). By fiscal year 2007, the amount allocated to applied research was 41%, increasing to 46% by fiscal year 2010 (National Science Board, 2004).

New Research Approaches to Facilitate Translation

In 2003, the Institute of Medicine's Clinical Round Table presented a schema to conceptualize blockades within the translational pipeline (Sung et al., 2003). The identified obstacles are shown in Fig. 1 (adapted from Sung et al., 2003).

Although T1 translation remains a concern, attention has focused increasingly on blockages later in the pipeline. There has been major federal investment in Comparative Effectiveness Research to address a T2 blockade: determining which treatments work best in usual practice, as contrasted with research settings. Moreover, a further translational phase (T3 – Implementation) has been recognized, acknowledging the need to overcome system-level obstacles that impede uptake of best research-tested practices into institutions and health care systems (Westfall, Mold, & Fagnan, 2007) (see also entry on "▶ Translational Behavioral Medicine"). Practice-Based Research and Community-Based Participatory Research (CBPR) are two research approaches now being applied to learn how to overcome implementation barriers (Woolf, 2008).

An insight that is driving attention toward later phase translation is the desire to scale evidence-based interventions to bring their benefits to more of the population. A highly efficacious treatment available to very few will have less public health impact than a less effective treatment available to many (Glasgow, Klesges, Dzewaltowski, Estabrooks, & Vogt, 2006). Stated differently, population level impact is the product of efficacy and reach (Abrams et al., 1996).

Comparative Effectiveness Research

The U.S. Agency for Health Research and Quality (AHRQ) defines Comparative Effectiveness Research (CER) as the conduct and synthesis of research that compares the benefits and harms of

different strategies to prevent, diagnose, treat, and monitor health conditions in "real world" settings. CER is a T2 research strategy that addresses practitioners' need to know the relative merits of the treatment options available to them. CER takes two main forms: (1) systematic evidence reviews that evaluate benefits and harms of treatment options for different groups of people on the basis of preexisting research and (2) new studies that generate evidence about the effectiveness of health care practices in usual practice. The most common CER methodologies have been Randomized Controlled Trials (RCTs) (60% of total CER), followed by systematic reviews (14%), and retrospective observational studies (6%). The most common CER interventions have been: pharmacological (34% of total CER), delivery system (20%), and behavioral (16%) (Department of Health and Human Services [DHHS] & Federal Coordinating Council for Comparative Effectiveness Research, 2009).

Implementation Research

The aim of T3 or Implementation Research (IR) is to learn how to overcome barriers that limit the uptake of evidence-based practices into usual care. A core insight is that "If we want more evidence-based practice, we need more practice-based evidence" (Green & Kreuter, 2005). A common implementation barrier is that few practitioners appreciate being admonished by academic researchers for not following scientific practices. Practitioners note that translational processes need to be bidirectional, so that an understanding of the practice context informs the genesis of relevant research.

Practice-Based Research

Because primary care is the gateway health care provider for most of the population, it offers a major channel to deliver evidence-based care to the public. In 1999, Congress enabled the AHRQ to establish Practice-Based Research Networks (PBRNs) that engage groups of experienced, practicing clinicians in framing research questions whose answers could improve the practice of primary care (Green & Hickner, 2006). By helping practices collaborate to address quality improvement questions with rigorous research methods, AHRQ hopes the PBRN can produce research findings that are immediately relevant to the clinician and readily assimilated into everyday practice.

Community-Based Participatory Research

Community-Based Participatory Research (CBPR) steps beyond the clinical practice to engage the entire community as a co-equal partner in research collaboration. Guided by a core set of values, CBPR builds on community strengths and resources, facilitates collaboration, co-learning, and capacity building, and balances research with long-term commitment to action that benefits the community and eliminates disparities (Israel, Schulz, Parker, & Becker, 2008). A guiding principle of CBPR is that culture, religious values, and economic factors specific to a community are integral to any decision about best practices.

Educating Researchers and Practitioners About Translation

Because research to practice translation is still an evolving field, efforts to educate researchers and practitioners about this area will continue to play a role in improving healthcare. Several training resources are available. First, the NIH Office of Behavioral and Social Science Research (OBSSR) offers a 5-day residential summer training institute on Dissemination and Implementation Research in Health (http://conferences.thehillgroup.com/OBSSRinstitutes/TIDIRH2011/index.html) Another OBSSR-funded resource that can be accessed remotely and free of charge is the Evidence-Based Behavioral Practice (EBBP) site available at www.ebbp.org. Among the interactive online learning modules available at ebbp.org are several dedicated to: (a) shared decision making with communities, (b) stakeholder perspectives about research and evidence-based practice, and (c) implementation. Finally, a third set of resources involve several new scholarly journals: *The Journal of Translational Medicine* (2003), *Science: Translational Medicine* (2009), and *Translational Behavioral Medicine* (2011).

Cross-References

- ▶ Evidence-Based Behavioral Medicine (EBBM)
- ▶ Translational Behavioral Medicine

References and Readings

Abrams, D. B., Orleans, C. T., Niaura, R., Goldstein, M. G., Prochaska, J. O., & Velicer, W. (1996). Integrating individual and public health perspectives for treatment of tobacco dependence under managed care: A combined stepped care and matching model. *Annals of Behavioral Medicine, 18*, 290–304.

Balas, E. A., & Boren, S. A. (2000). Managing clinical knowledge for health care improvement. In J. Bemmel & A. T. McCray (Eds.), *Yearbook of medical informatics* (pp. 65–70). Stuttgart, Germany: Schattauer Publishing Company.

Bush, V. (1945). *Science, the endless Frontier: A report to the president by Vannevar Bush, director of the office of scientific research and development*. Washington, DC: United States Government Printing Office.

Department of Health and Human Services, Federal Coordinating Council for Comparative Effectiveness Research. (2009, June 30). *Report to the president and the congress* [Internet]. Washington, DC: HHS; [cited 2011 Jan 11]. Available from http://www.hhs.gov/recovery/programs/cer/cerannualrpt.pdf

Glasgow, R. E., Klesges, L. M., Dzewaltowski, D. A., Estabrooks, P. A., & Vogt, T. M. (2006). Evaluating the impact of health promotion programs: Using the RE-AIM framework to form summary measures for decision making involving complex issues. *Health Education Research, 21*(3), 688–694.

Green, L. A., & Hickner, J. (2006). A short history of primary care practice-based research networks: From concept to essential research laboratories. *The Journal of the American Board of Family Medicine, 19*(1), 1–10.

Green, L. W., & Kreuter, M. W. (2005). *Health program planning: An educational and ecological approach* (4th ed.). Boston: McGraw Hill.

Institute of Medicine Committee on the NIH Research Priority-Setting Process. (1998). *Scientific opportunities and public needs: Improving priority setting and public input at the national institutes of health*. Washington, DC: National Academies Press.

Israel, B. A., Schulz, A. J., Parker, E. A., & Becker, A. B. (2008). Review of community-based research: Assessing partnership approaches to improve public health. *Annual Review of Public Health, 19*, 173–202.

National Science Board. (2004). *Science and engineering indicators 2004* (NSB 04–01). Arlington, VA: National Science Foundation, Division of Science Resources Statistics

Stokes, D. (1997). *Pasteur's quadrant: Basic science and technological innovation*. Washington, DC: Brookings Institute.

Sung, N. S., Crowley, W. F., Jr., Genel, M., Salber, P., Sandy, L., Sherwood, L. M., et al. (2003). Central challenges facing the national clinical research enterprise. *Journal of the American Medical Association, 289*, 1278–1287.

Westfall, J. M., Mold, J., & Fagnan, L. (2007). Practice-based research-"blue highways" on the NIH roadmap. *Journal of the American Medical Association, 297*(4), 403–406.

Woolf, S. H. (2008). The meaning of translational research and why it matters. *Journal of the American Medical Association, 299*(2), 211–213.

Residential Treatment

- ▶ Substance Abuse: Treatment

Resilience

Judith Carroll[1] and Chris Dunkel Schetter[2]
[1]Cousins Center for Psychoneuroimmunology, University of California, Los Angeles, CA, USA
[2]Department of Psychology, UCLA, Los Angeles, CA, USA

Synonyms

Adaptation; Hardiness; Personal growth; Psychological thriving; Recovery

Definition

Resilience is a process of "bouncing back" or recovering quickly from adversity, and is not considered a trait disposition. As a dynamic process, resilience involves sustaining psychological functioning during stress, recovering from stressors as rapidly as possible to resume normal functioning, and, in some cases, experiencing psychological growth in the aftermath (Zautra, Arewasikporn, & Davis, 2010). One could think

of a person's resilience as akin to a trampoline with the forces jumping on it as various stressful demands and the strength of the trampoline as the degree of resilience. In this scenario, a major stressor would be a substantial force pressing down on the trampoline, which may or may not cause permanent damage to the trampoline and allow it to remain resistant to stress. In essence, resilience is a process by which a person is able to appraise stressful circumstances in an accurate and constructive way. It involves cognitions, behaviors, and resources that are available or learned and that can support coping and growth from the stressful or traumatic experience. Although traditionally studied in the context of traumatic or major stressful life events such as major illnesses, childhood adversity, or loss of a loved one, resilience can also be examined in the context of chronic stressors – constant or recurrent demands – that are known to have a pronounced biological impact on health (Schneiderman, Ironson, & Siegel, 2005). In these circumstances, individuals often experience psychological and physiological exhaustion if their resources are inadequate to cope with stressful demands over the long term. Inversely, a person who has strong resilience resources can sustain normal functioning in the face of chronic stress. Dunkel Schetter and Dolbier (2011) developed a taxonomy of resilience resources at the individual level to foster comprehensive assessments of resilience in research that are pertinent to behavioral medicine. This taxonomy involves individual difference resources (i.e., positive affectivity, optimism), self and ego-related resources (i.e., self-efficacy, self-esteem), interpersonal/social resources (i.e., social support, social networks), worldviews and cultural beliefs (i.e., religiosity, life purpose, collectivism), behavioral and cognitive skills (emotion regulation, problem solving skills), and endowed or acquired resources (i.e., intelligence, health behaviors). Similarly, the American Psychological Association (APA, 2012) has described key factors that foster the development of resilience, including developing self-worth and self-efficacy, meaning making and positive cognitions, constructive coping, emotion regulation, social support, self-care behaviors (e.g., getting exercise, eating well, sleeping), expressive writing, spirituality, and meditation (www.apa.org). While much remains to be learned about resilience, scientific understanding has grown substantially in the wake of major disasters in the last decade.

Cross-References

- ▶ Active Coping
- ▶ Coping
- ▶ Hardiness and Health
- ▶ Optimism, Pessimism, and Health
- ▶ Posttraumatic Growth
- ▶ Religiousness/Religiosity
- ▶ Resilience: Measurement
- ▶ Salutogenesis
- ▶ Self-efficacy
- ▶ Self-esteem
- ▶ Self-regulation Model
- ▶ Social Support
- ▶ Stress, Posttraumatic
- ▶ Stress: Appraisal and Coping
- ▶ Williams LifeSkills Program

References and Readings

American Psychological Association (APA) (2012). The road to resilience. Available on the worldwide web. http://www.apa.org/helpcenter/road-resilience.aspx. Accessed 23 September, 2011.

Bonanno, G. A. (2005). Clarifying and extending the construct of adult resilience. *The American Psychologist, 60*(3), 265–267.

Dunkel Schetter, C., & Dolbier, C. (2011). Resilience in the context of chronic stress and health in adults: Resilience resources conceptualization. *Social & Personality Psychology Compass, 5*, 634–652.

Epel, E. S., McEwen, B. S., & Ickovics, J. R. (1998). Embodying psychological thriving: Physical thriving in response to stress. *Journal of Social Issues, 54*(2), 301–322.

Schneiderman, N., Ironson, G., & Siegel, S. (2005). Stress and health: Psychological, behavioral, and biological determinants. *Annual Review of Clinical Psychology, 1*, 607–628.

Zautra, A. J., Arewasikporn, A., & Davis, M. C. (2010). Resilience: Promoting well-being through recovery, sustainability, and growth. *Research in Human Development, 7*(3), 1–119.

Resilience Training

▶ Williams LifeSkills Program

Resilience: Measurement

Susanne Fischer
Department of Psychology, Clinical Biopsychology, Philipps-University of Marburg, Marburg, Germany

Synonyms

Hardiness; Resiliency; Sense of coherence – measurement

Definition

Resilience is defined as the capability of successfully restoring, sustaining, or enhancing adaptive competences in the face of significant adversity.

Description

Several core elements are part of the definition of resilience: (a) Occurrence of a stressor is an essential antecedent of resilience. (b) There is a beneficial outcome as a consequence of negative experiences. (c) A life span perspective is required, encompassing varying trajectories such as short-term recovery, delayed reactions, and chronic dysfunction (Bonanno, 2004; Bonanno & Mancini, 2008). Despite general consent on these conceptual core elements, there are ambiguities in defining type, severity, and frequency of stress experience as well as domain and level of possible subsequent competence.

Regarding *stress experiences*, operationalization usually depends on individual study aims and populations of interest. This results in numerous highly heterogeneous measurements of stressors. In studies on children and adolescents, focus is on socioeconomic deprivation as a variant of prolonged stress or aversive early life events (such as childhood trauma), whereas isolated critical life events or traumatization occurring during the adult life span are subject of numerous studies in adults. Most of these studies adopt self-report measurement approaches using structured interviews or administering checklists and questionnaires to retrospectively assess negative experiences. These subjective measures may be complemented by biological parameters, such as brain activity as well as endocrinological, autonomic, or immunological parameters which may constitute valuable indicators of often long-lasting physiological changes in stress-response systems precipitated by adverse experiences (McEwen, 2008).

In order to measure *adaptive competences* after previous exposure to stress, researchers examining children and adolescents often generate composite indices across multiple behavioral domains of functioning (e.g., school or recreational activities). Also, achievement of developmental milestones can be used as a measurement of successful adaptation (Atkinson, Martin, & Rankin, 2009). These measurements commonly rely on evaluations and ratings provided by parents or teachers. In contrast, studies investigating adults are mostly designed within a clinical context. As a consequence, definition of resilience usually equals absence of somatic symptoms, physical illness, psychological distress or psychopathology, and measurement of resilience is reflected by this. The assessment of physical and mental well-being usually relies on experiential statements made by the individual under investigation himself or herself (Atkinson, Martin, & Rankin, 2009).

Several psychometric instruments have been developed on the basis of rather comprehensive theoretical conceptualizations, such as sense of coherence (Antonovsky, 1979), hardiness (Kobasa, 1979), or ego-resiliency (Block & Block, 1980). Apart from these broader concepts, a number of more specific characteristics have been commonly associated with resilience: cognitive flexibility, internal locus of control, self-efficacy, positive affectivity, optimism,

self-esteem, active coping, and social support, among many others (Earvolino-Ramirez, 2007; Mancini & Bonanno, 2009). At present, a number of self-report questionnaires assessing at least some of these characteristics have gained considerable attention (Ahern, Kiehl, Sole, & Byers, 2006; Windle, Bennett, & Noyes, 2011). Prominent examples (in terms of good psychometric properties and/or international use in empirical studies) are the Resilience Scale (RS; Wagnild & Young, 1993), the Connor-Davidson Resilience Scale (CD-RISC; Connor & Davidson, 2003), and the Resilience Scale for Adults (RSA; Friborg, Hjemdal, Rosenvinge, & Martinussen, 2003). The RS was developed based on interviews with community-dwelling older women after experience of critical life events, conceptualizing resilience as an individual trait. Principal component analysis of the 25 items revealed two factors labeled "personal competence" and "acceptance of self and life." The scale has been employed in population-based as well as clinical contexts. The CD-RISC, by contrast, predominantly consists of questions addressing stress-coping behavior. A preliminary validation study conducted by the authors yielded a structure of five factors based on the 25 items of the test. Application of the instrument has so far mostly been limited to clinical trials. Finally, the RSA incorporates items on external protective resources such as social support. Overall, five dimensions were empirically found to adequately describe the resilience concept as suggested by the authors, namely, personal competence, social competence, family coherence, social support, and personal structure. The scale has been used in studies in both healthy participants and patient samples.

While resilience has been traditionally examined using psychometric assessments, more recent research has sought to evaluate the biological underpinnings and physiological correlates of resilience in both animals and humans (Charney, 2004; Cicchetti & Blender, 2006; Feder, Nestler, & Charney, 2009; McEwen, 2008; Stein, 2009; Yehuda, Flory, Southwick, & Charney, 2006). Studies in this area of research usually utilize experimental designs directed at exploring the dynamic biological processes involved in the promotion of general well-being, with the goal of identifying genetic, neuronal, and physiological mechanisms in resilient humans. Research incorporating multidisciplinary approaches and addressing the role of biopsychosocial mechanisms of resilience holds substantial promises for a better understanding of how individuals are capable of thriving in the face of adversity.

Cross-References

- ▶ Active Coping
- ▶ Coping
- ▶ Hardiness
- ▶ Life Events
- ▶ Locus of Control
- ▶ Optimism and Pessimism: Measurement
- ▶ Positive Affectivity
- ▶ Positive Psychology
- ▶ Salutogenesis
- ▶ Self-Efficacy
- ▶ Self-Esteem
- ▶ Social Support
- ▶ Stress
- ▶ Stress, Early Life
- ▶ Stress, Posttraumatic

References and Readings

Ahern, N. R., Kiehl, E. M., Sole, M. L., & Byers, J. (2006). A review of instruments measuring resilience. *Issues in Comprehensive Pediatric Nursing, 29*(2), 103–125.

Antonovsky, A. (1979). *Health, stress, and coping: New perspectives on mental and physical well-being*. San Francisco: Jossey-Bass.

Atkinson, P. A., Martin, C. R., & Rankin, J. (2009). Resilience revisited. *Journal of Psychiatric and Mental Health Nursing, 16*(2), 137–145.

Block, J. H., & Block, J. (1980). The role of ego-control and ego-resiliency in the organization of behavior. In W. A. Collins (Ed.), *Development of cognition, affect, and social relations: The Minnesota symposia on child psychology* (Vol. 13). Hillsdale, NJ: Erlbaum.

Bonanno, G. A. (2004). Loss, trauma, and human resilience: Have we underestimated the human capacity to thrive after extremely aversive events? *American Psychologist, 59*(1), 20–28.

Bonanno, G. A., & Mancini, A. D. (2008). The human capacity to thrive in the face of potential trauma. *Pediatrics, 121*(2), 369–375.
Charney, D. S. (2004). Psychobiological mechanisms of resilience and vulnerability: Implications for successful adaptation to extreme stress. *The American Journal of Psychiatry, 161*(2), 195–216.
Cicchetti, D., & Blender, J. A. (2006). A multiple-levels-of-analysis perspective on resilience: Implications for the developing brain, neural plasticity, and preventive interventions. *Annals of the New York Academy of Sciences, 1094*, 248–258.
Connor, K. M., & Davidson, J. R. (2003). Development of a new resilience scale: The Connor-Davidson Resilience Scale (CD-RISC). *Depression and Anxiety, 18*(2), 76–82.
Earvolino-Ramirez, M. (2007). Resilience: A concept analysis. *Nursing Forum, 42*(2), 73–82.
Feder, A., Nestler, E. J., & Charney, D. S. (2009). Psychobiology and molecular genetics of resilience. *Nature Reviews Neuroscience, 10*(6), 446–457.
Friborg, O., Hjemdal, O., Rosenvinge, J. H., & Martinussen, M. (2003). A new rating scale for adult resilience: What are the central protective resources behind healthy adjustment? *International Journal of Methods in Psychiatric Research, 12*(2), 65–76.
Kobasa, S. C. (1979). Stressful life events, personality, and health: An inquiry into hardiness. *Journal of Personality and Social Psychology, 37*(1), 1–11.
Mancini, A. D., & Bonanno, G. A. (2009). Predictors and parameters of resilience to loss: Toward an individual differences model. *Journal of Personality, 77*(6), 1805–1832.
McEwen, B. S. (2008). Central effects of stress hormones in health and disease: Understanding the protective and damaging effects of stress and stress mediators. *European Journal of Pharmacology, 583*(2–3), 174–185.
Stein, D. J. (2009). The psychobiology of resilience. *CNS Spectrums, 14*(2 Suppl. 3), 41–47.
Wagnild, G. M., & Young, H. M. (1993). Development and psychometric evaluation of the resilience scale. *Journal of Nursing Measurement, 1*(2), 165–178.
Windle, G., Bennett, K. M., & Noyes, J. (2011). A methodological review of resilience measurement scales. *Health and Quality of Life Outcomes, 9*, 8.
Yehuda, R., Flory, J. D., Southwick, S., & Charney, D. S. (2006). Developing an agenda for translational studies of resilience and vulnerability following trauma exposure. *Annals of the New York Academy of Sciences, 1071*, 379–396.

Resiliency

► Resilience: Measurement

Resistance Training

Roland Thomeé
Department of Rehabilitation Medicine, Sahlgrenska University Hospital, Öjersjö, Göteborg, Sweden

Definition

Resistance training has an impact on many-body systems such as the muscular, respiratory, skeletal, and neural, but also the immune, endocrine, and metabolic (Deschenes & Kraemer, 2002).

Description

Resistance training has been used for a very long time. There is documentation from 700 B.C. of the very strong Bybon in Greece lifting a 140 kg rock over his head with only one hand. The legendary Milon from Italy lifted a new born calf every day until the calf was a full-grown bull. This is the first documentation of progressive resistance training (Fry & Newton, 2002). Among the pioneers in research on resistance training Thomas L. De Lorme needs to be mentioned. In 1946 he presented a method for resistance training using the one repetition maximum (1RM) (De Lorme, 1946). This concept is still widely used today describing the intensity used in resistance training programs.

In order to achieve the desired response on the body it is important to understand the basic principles of resistance training. Resistance training can, according to the American Sports Medicine Institute (http://www.asmi.org/), be defined as a gradual overload of the musculoskeletal system resulting in improved capacity. In general resistant training is often synonymous with strength training. Depending on the design the resistance training program can result in improved muscle strength, muscle volume, muscle power, or muscle endurance. Motor control can improve with better balance, coordination, and technique. Also tendons, ligaments, articular cartilage, and bones

can improve their capacity as a result of resistance training (Deschenes & Kraemer, 2002).

There are several methods available and various effects of the resistance training program are achieved depending on the frequency, intensity, and volume of the training (Wernbom, Augustsson, & Thomee, 2007). The program needs to specify the number of weeks of training, the number of training sessions performed per week, the number of exercises used for each muscle group, the number of sets and repetitions used for each exercise, and how long time of rest is given in between sets and exercises. For general strength training it can be recommended that the program consists of two session per week, two exercises per major muscle group, two to three sets of 8–12 repetitions per exercise with 30 s of rest in between sets and exercises (Haskell et al., 2007). In order to improve muscle power, i.e., the ability to produce a high force rapidly, a moderate load moved as fast as possible is recommended. For muscular endurance the number of repetitions should exceed 20 and the rest between sets and exercises decreased to a minimum (Wernbom et al., 2007).

One mode of strength training can be classified as dynamic external resistance, including free weights and weight machines, where the resistance is moved through the range of motion of the joint. Another mode can be classified as accommodating resistance, for example, isokinetic (the speed of movement is constant and the resistance offered by the machine accommodates to the applied force through the whole range of motion). A third mode can be classified as isometric resistance where no movement occurs during muscle force development. For the first two modes one usually differs concentric from eccentric muscle action. In a concentric muscle action the muscle shortens while developing force and in the eccentric muscle action the muscle lengthens while developing force. There is no evidence as of now for the superiority of any mode and/or type of muscle action over other modes and types (Wernbom et al., 2007). Given sufficient frequency, intensity, and volume of work, all three types of muscle actions can induce significant increased muscle volume at an impressive rate.

Cross-References

▶ Isometric/Isotonic Exercise

References and Readings

De Lorme, T. L. (1946). Heavy resistance exercises. *Archives of Physical Medicine and Rehabilitation, 27*, 607–630.

Deschenes, M. R., & Kraemer, W. J. (2002). Performance and physiologic adaptations to resistance training. *American Journal of Physical Medicine & Rehabilitation, 81*(11 Suppl), S3–S16. doi:10.1097/01.PHM.0000029722.06777.E9.

Fry, A. C., & Newton, R. U. (2002). A brief history of strength training and basic principles and concepts. In W. Kraemer & K. Häkkinen (Eds.), *Strength training for sport* (pp. 1–19). Oxford: Blackwell Scientific.

Haskell, W. L., Lee, I. M., Pate, R. R., Powell, K. E., Blair, S. N., Franklin, B. A., & Bauman, A. (2007). Physical activity and public health: Updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Medicine and Science in Sports and Exercise, 39*(8), 1423–1434. doi:10.1249/mss.0b013e3180616b27.00005768-200708000-00027 [pii].

Wernbom, M., Augustsson, J., & Thomee, R. (2007). The influence of frequency, intensity, volume and mode of strength training on whole muscle cross-sectional area in humans. *Sports Medicine, 37*(3), 225–264. doi:3734 [pii].

Respiratory Sinus Arrhythmia

Randall Steven Jorgensen
Department of Psychology, Syracuse University, Syracuse, NY, USA

Synonyms

Heart rate variability (HRV)

Definition

In mammals, heart rate (HR) ordinarily accelerates during inspiration and decelerates during expiration, and the tenth cranial nerve (i.e., vagus nerve) exerts a profound influence on this heart rate

variability (HRV). This oscillation of R-wave (the sharp upward spike of the QRS complex associated with the contraction of the heart) to R-wave intervals (RRI) is a cardiorespiratory phenomenon called respiratory sinus arrhythmia (RSA); since RRI, which are inversely related to moment-to-moment HR, reflect vagal stimulation, noninvasive measures of RSA are commonly used to estimate autonomic cardiovascular control (Grossman & Taylor, 2007). The "peak to valley" time-domain estimate, usually quantified in milliseconds (ms), corresponds to the inspiratory-expiratory difference in RRI. For estimates based on spectral analysis and other frequency-domain approaches, RRI variation is estimated within the respiratory frequency range (.15–.4 Hz); to reflect statistical units of variance, ms^2 is commonly used. For steady-state conditions (viz., respiratory parameters, momentary physical activity, metabolic activity, and autonomic tone remain nearly constant), these estimates are almost perfectly correlated (Grossman and Taylor). These noninvasive measures' covariation with cardiac vagal tone, however, can be confounded by such factors as respiratory rate and volume, changes in physical and metabolic activity, body weight, age, and sympathetic nervous system tone; this confounding is more pronounced in between-group designs (e.g., high blood pressure vs. normal blood pressure groups) than within-subject designs (e.g., examining within-person changes in RSA following atropine administration) (Grossman & Taylor, 2007).

The vagus has been posited to be a key factor in emotional regulation and health (Thayer & Lane, 2009). Researchers, therefore, have used noninvasive measures of HRV and RSA to predict health and psychological well-being. These ostensible markers of cardiac vagal tone are reported to covary with cardiovascular disease, anxiety, depression, and childhood behavior disorders (Berntson, Cacioppo, & Grossman, 2007; Thayer & Lane, 2007). Even when the predicted associations are revealed, the (a) correlational designs; (b) paucity of multivariate/multilevel studies cutting across anatomical, physiological, and behavioral domains; and (c) inadequate controls of confounds (e.g., bodily motion and respiration rate) make interpretation of underlying causal and multiply determined pathways ambiguous (Berntson et al., 2007). At minimum, it is important to take into account and address the confounding variables discussed earlier (for guidelines, see Grossman & Taylor, 2007).

Cross-References

- ▶ Heart Rate Variability
- ▶ Parasympathetic

References and Readings

Berntson, G. G., Cacioppo, J. T., & Grossman, P. (2007). Whither vagal tone. *Biological Psychology, 74*, 297–300. doi:10.1016/j.biopsycho.2006.08.006.

Grossman, P., & Taylor, E. W. (2007). Toward understanding respiratory arrhythmia: Relations to cardiac vagal tone, evolution, and biobehavioral functions. *Biological Psychological, 74*, 263–285. doi:10.1016/j.biopsycho.2005.11.014.

Thayer, J. F., & Lane, R. D. (2007). The role of vagal function in the risk for cardiovascular disease and mortality. *Biological Psychology, 74*, 224–242. doi:0.1016/j.biopsycho.2005.11.013.

Thayer, J. F., & Lane, R. D. (2009). Claude Bernard and the heart-brain connection: Further elaboration of a model of neurovisceral integration. *Neuroscience and Biobehavoioral Reviews, 33*, 81–88. doi:0.1016/j.neubiorev.2008.08.004.

Response Bias

- ▶ Bias

Response Inhibition

- ▶ Behavioral Inhibition

Response to Disability

- ▶ Psychosocial Adjustment

Responses to Stress

▶ General Adaptation Syndrome

Responsibility

▶ Self-Blame

Rest Pain

▶ Peripheral Arterial Disease (PAD)/Vascular Disease

Retrospective Study

Jane Monaco
Department of Biostatistics, The University of North Carolina at Chapel Hill, Chapel Hill, NC, USA

Definition

A study in which the outcome of interest has already occurred when the study initiated is commonly referred to as retrospective study.

Some investigators have used the terms retrospective and case control interchangeably. This usage is misleading since study designs other than case-control studies can be retrospective. For example, in a retrospective cohort study (Okasha, McCarron, McEwen, & Davey Smith, 2002) examining the relationship between body mass index (BMI) during early adulthood and cancer death, both the exposure and the outcome had occurred at the initiation of the study. University students' weight and height information was obtained from annual physical records at the University of Glasgow health service from 1948 to 1968. Cancer mortality was determined based on a central registry in Scotland that identifies the cause of death (National Health Service Central Register). Subjects with BMI in the highest quartile were found to be at higher risk of cancer death than individuals in the lowest BMI quartile.

In a retrospective study, data may be collected based on clinical records, employment records, or memory. These retrospective data can be more prone to bias, such as recall bias, than prospective designs studying the same association.

Some investigators may also use the term "retrospective" in a different way, referring to the timing of the measurements of the exposure and outcome; studies in which the exposure is measured after the outcome is measured can be referred to as retrospective (Rothman & Greenland, 1998, pp. 74–75).

Cross-References

▶ Case-Control Studies
▶ Cohort Study

References and Readings

Kleinbaum, D. G., Sullivan, K. M., & Barker, N. D. (2007). *A pocket guide to epidemiology*. New York: Springer.
Okasha, M., McCarron, P., McEwen, J., & Davey Smith, G. (2002). Body mass index in young adulthood and cancer mortality: A retrospective cohort study. *Journal of Epidemiology and Community Health, 56*, 780–784.
Rothman, K. J., & Greenland, S. (1998). *Modern epidemiology*. Philadelphia: Lippencott-Raven.

Return to Baseline

▶ Psychophysiologic Recovery

Revised Life Orientation Test (LOT-R)

▶ Optimism and Pessimism: Measurement

Rheumatoid Arthritis: Psychosocial Aspects

Toshihide Hashimoto
Department of Rehabilitation, Graduate School of Medicine, Gunma University, Maebashi, Gunma, Japan

Synonyms

RA

Definition

Rheumatoid arthritis is a chronic, systemic, progressive inflammatory disease characterized by swelling, pain, and deformity of the joints. It affects not only synovial joints but also many tissues and organs of the body.

Description

Rheumatoid Arthritis (RA)

RA affects about 0.5–1.0% of the general population. The prevalence shows similarity by region and race worldwide. Women are afflicted two to three times more often than men. Individuals between 40 and 60 years of age are most liable to RA, but people at any age can be affected. Although the cause of RA has not been fully elucidated, the abnormal autoimmune response which attacks the body's own tissue plays a major role in the onset and progression of RA.

The first symptoms of RA generally occur in the smaller joints (e.g., finger or wrist joints) and gradually spread symmetrically to the larger joints (e.g., elbow or knee joints). In affected joints, inflammation first occurs in the synovial membrane. Abnormal synovium, called "pannus," multiplies gradually and results in cartilage damage and bone erosion and eventually the destruction of the joints. Consequently, RA patients suffer from joint symptoms such as pain, swelling, stiffness, deformity, and restriction of motion.

In addition, inflammation involved in RA may affect various organs, including fibrosis or pleuritis in the lungs, pericarditis or cardiovascular disease in the heart, osteoporosis in the bones, vertebral malalignment or subluxation in the cervical spine, and rheumatoid nodules in the skin. RA patients also experience some general symptoms due to chronic inflammation, such as anemia, fatigue, low-grade fever, sleep disorder, and lack of appetite.

Such symptoms cause multiple joint pains, physical disability, and various comorbidities in RA patients. As a result, many RA patients experience loss of income, loss of recreational and social activities, and disability in doing housework and taking care of children. These physical and social disabilities are likely to disturb interpersonal relationships and lead to psychological disorders. Depression and anxiety have also been known to cause increased pain and other RA-related symptoms (Firestein, 2009; Harris et al., 2009).

Psychological Distress of RA Patients

Depression is significantly more common among RA patients than healthy individuals. The prevalence of depression in patients with RA varies from 20% to 40%. This varying range may be due to both the RA patient population and the evaluation method of depression. RA-related symptoms (i.e., fatigue, sleep disturbance, or loss of appetite) correspond to physical symptoms of patients with general depression. When prevalence is measured satisfactorily independent of the severity of RA symptoms, it is more likely to be about 20%. This figure is presumed to be similar to those of other chronic conditions and two or three times higher than that of the general population.

Many factors have been associated with depression of RA patients. In terms of RA-related symptoms, the increase in subjective self-reported pain, fatigue, and physical disability is likely to lead to a more depressive state. The causal relationship between pain, fatigue, disability, and depression is complex and considered multidirectional. On the other hand, disease

factors that are indices of inflammation (e.g., C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), swollen joint count, grip strength, and radiological status) are generally not associated with depression.

Recently, psychosocial factors have been emphasized as important variables associated with depression. Generally, increased depression of RA patients is related to being female, younger age, lower income, less employment, greater work disability, more social stress, less social support, dysfunctional familial background, maladaptive coping strategy, and distorted cognition.

In clinical practice, objective clinical variables (CRP, ESR, or joint count) and subjective patient-reported physical symptoms are regarded as important indices of treatment effects. However, clinicians do not tend to pay close attention to mood states of RA patients. Depressed RA patients may not verbally express their emotion, because they judge that their emotional and physical conditions are a part of their RA symptoms. Accordingly, depression in RA patients is most likely underestimated by clinicians. All health-care professionals should thus try to observe whether RA patients have latent depression (Sheehy, Murphy, & Barry, 2006; Travis, 2008).

Relationship Between RA-Related Symptoms and Psychosocial Factors

Pain, fatigue, and physical disability are RA-related symptoms that need to be addressed by all health-care professionals. Pain is the most common and basic symptom in RA patients. Fatigue is also common and the most burdensome symptom. The prevalence of fatigue ranges greatly from 40% to 80%, most likely because the definition and method of measurement are not uniform. Fatigue is perceived as a frustrating or exhausting state, impacts every situation of life, and persists for a long time. Pain, fatigue, and physical disability influence psychological well-being and social participation of RA patients. Moreover, psychosocial factors, e.g., perception of symptoms, self-efficacy, coping strategy, social stress, and social support, play important roles in modifying the process and outcome of RA-related symptoms and psychological distress.

Perception of symptoms is an individual belief in how RA will influence oneself, how long RA will last, whether RA can be controlled, and what the outcomes of RA are. RA patients with negative beliefs, such as "catastrophizing" (the tendency to focus on symptoms and predict extremely negative outcomes) or "helplessness" (the belief that nothing can be done by oneself to deal with symptoms), may demonstrate increased symptoms and poor psychological well-being. On the other hand, self-efficacy (the confidence in one's ability to accomplish sufficiently a desired outcome) and an adaptive coping strategy (the suitable and effective process to deal with stresses and difficulties of life) are considered to improve symptoms and psychological distress.

All health-care professionals should make efforts to inform RA patients of the cause, treatment, clinical course, and result of RA at the early stage of their disease before they have poor perception of symptoms and acquire maladaptive coping strategies. In patients who have already experienced physical and psychological distress, several interventions (e.g., cognitive behavioral therapy, patient education, and physical exercise) have been implemented, but the evidence of these interventions has not been clarified. All health-care professionals should take note of any psychological distress of RA patients, accurately evaluate the patients' social stress and social support, and provide them with information about available care and support, in order to promote better self-efficacy and more adaptive coping strategies (Backman, 2006; Repping-Wuts, van Riel, & van Achterberg, 2009; Sheehy et al., 2006; Travis, 2008).

Relevance to Clinical Practice

In the past decade, drug treatment of RA has improved greatly. Disease-modifying antirheumatic drugs (DMARDs) and biological DMARDs (drugs to block or modify the effect of factors of the immune system, such as cytokines and lymphocytes) have been introduced to RA patients at the early stage. These new medications effectively reduce inflammation of RA and prevent the advancement of joint erosion and deformities. These drug therapies have

possibility of reducing not only pain but also fatigue or depression. As a result, the clinical course of RA could change significantly, leading to a decrease in the number of patients who suffer from fatigue and depression in the future.

However, such medications have several problems, including potential side effects (e.g., infection or blood disease), ineffectiveness for some patients, and high cost of treatment. Some, but not all, RA patients are expected to benefit from these medical advancements in RA, and they will probably experience new psychosocial problems. Health-care professionals need to further examine psychosocial factors that accompany new therapies to evaluate both the positive and negative influences on patients (Genoves, 2009; Saag et al., 2008; Smolen et al., 2010).

Cross-References

- ▶ Psychosocial Characteristics
- ▶ Psychosocial Factors

References and Readings

American College of Rheumatology (ACR). Retrieved from http://www.rheumatology.org/practice/index.asp

Backman, C. L. (2006). Arthritis and pain. Psychosocial aspects in the management of arthritis pain. *Arthritis Research & Therapy, 8*, 221–227.

Firestein, G. S. (2009). Etiology and pathogenesis of rheumatoid arthritis. In G. S. Firestein, R. C. Budd, E. D. Harris, I. B. McInnes, S. Ruddy, & J. S. Sergent (Eds.), *Kelley's textbook of rheumatology* (8th ed., pp. 1035–1086). Philadelphia: Saunders.

Genoves, M. C. (2009). Treatment of rheumatoid arthritis. In G. S. Firestein, R. C. Budd, E. D. Harris, I. B. McInnes, S. Ruddy, & J. S. Sergent (Eds.), *Kelley's textbook of rheumatology* (8th ed., pp. 1119–1143). Philadelphia: Saunders.

Harris, E. D., Jr., et al. (2009). Clinical features of rheumatoid arthritis. In G. S. Firestein, R. C. Budd, E. D. Harris, I. B. McInnes, S. Ruddy, & J. S. Sergent (Eds.), *Kelley's textbook of rheumatology* (8th ed., pp. 1087–1118). Philadelphia: Saunders.

Repping-Wuts, H., van Riel, P., & van Achterberg, T. (2009). Fatigue in patients with rheumatoid arthritis: What is known and what is needed. *Rheumatology, 48*, 207–209.

Saag, K. G., Gim, G. T., Patkar, N. M., Curtis, J. R., Mudano, A., Pisu, M., Elkins-Melton, M., et al. (2008). American College of Rheumatology 2008 recommendations for the use of nonbiologic and biologic disease-modifying antirheumatic drugs in rheumatoid arthritis. *Arthritis & Rheumatism (Arthritis Care & Research), 59*, 762–784.

Sheehy, C., Murphy, E., & Barry, M. (2006). Depression in rheumatoid arthritis – Underscoring the problem. *Rheumatology, 45*, 1325–1327.

Smolen, J. S., Landewé, R., Breedveld, F. C., Dougados, M., Emery, P., Gaujoux-Viala, C., et al. (2010). EULAR recommendations for the management of rheumatoid arthritis with synthetic and biological disease-modifying antirheumatic drugs. *Annals of the Rheumatic Disease, 69*, 964–975.

The European League Against Rheumatism (EULAR). Retrieved from http://www.eular.org/

Travis, O. (2008). Comorbid depression in rheumatoid arthritis: Pathophysiology and clinical implications. *Current Psychiatry Reports, 10*, 258–264.

Ribosomal RNA

- ▶ RNA

Rief, Winfried

Alexandra Martin
Friedrich-Alexander University Erlangen-Nürnberg; University Hospital, Erlangen, Germany

Biographical Information

Winfried Rief was born in Ellwangen, Germany, on May 12, 1959. He is married to Sabine and has

a son. Rief studied physics at the University of Karlsruhe, Germany (1978–1979), and psychology at the University of Trier, Germany (1979–1984), receiving a Diploma in psychology in 1984. His doctoral thesis presented EEG-based research about "Visual Information Processing in Chronic Schizophrenics" and led to the awarding of the Ph.D. degree at the University of Konstanz in 1987.

Rief completed postgraduate training in cognitive-behavioral therapy (1988) and is a licensed Psychological Psychotherapist. He gained excellent clinical experiences and was Head of the psychology department at the Roseneck Hospital in Prien (1989–2000), a behavioral medicine center specialized in the inpatient treatment of eating disorders, anxiety, somatoform disorders, and other conditions, which is affiliated with the Medical Faculty of the University of Munich. He accomplished the "Habilitation" – the regular German academic qualification for professorship – at the University of Salzburg, Austria in 1994.

Rief was appointed as Professor of Clinical Psychology and Psychotherapy at the University of Marburg, Germany, in 2001 and since then has chaired the division for clinical psychology. He is also Head of the Outpatient Clinic for Psychological Interventions and Head of the postgraduate training program in cognitive-behavior therapy. He has been an invited guest professor for research visits at the University of Auckland Medical School, New Zealand (2002), Harvard Medical School Institute of Psychiatry (2004), and at the University of California, San Diego, Department of Psychiatry (2009).

Rief is an active participant in many professional organizations. He was President of the German Society of Behavioral Medicine and Behavior Modification (DGVM; 2001–2005). He served as Member and as Chair of the speaker group of the section for "Clinical Psychology and Psychotherapy" in the German Psychological Society (Fachgruppe Klinische Psychologie und Psychotherapie in der Deutschen Gesellschaft für Psychologie DGPs; 1996–1998; 2005–2008). He was one of the founding members of the German Society of Biofeedback, and later served as President in this organization (DGBFB 2000–2004). He organized many national and international scientific meetings, for example, the "International Symposium on Somatoform Disorders" (co-sponsored by the Division of Mental Health of the World Health Organization; Prien, 1997), the Congress of the German Society of Behavioral Medicine (Prien, 1999, co-organized with Prof. Dr. Manfred Fichter), the International Congress "Somatoform Disorders" (Marburg 2002), the International Congress of Behavioral Medicine (Mainz 2004, co-organized with Prof. Dr. Wolfgang Hiller), and the Annual Meetings of the German Society of Biofeedback (2001, 2007). Rief is recipient of the Biofeedback Foundation of Europe Award for Excellent Scientific Contribution (2004).

Rief has been appointed as reviewer for several research funding organizations, including the Deutsche Forschungsgemeinschaft (DFG) (German Research Foundation), Swiss National Fonds (SNF), NIHR Biomedical Research Center for Mental Health, and the Institute of Psychiatry/King's College London. In 2011, he was elected as coordinator for DFG grants in the field of clinical and health psychology in Germany. He currently serves or has served as an editorial board member in several scientific journals (*Behavioural and Cognitive Psychotherapy; British Journal of Health Psychology;Cognitive Behaviour Therapy; Current Opinion in Psychiatry; Psychology and Health; Psychotherapie in Psychiatrie, Psychosomatik und Klinischer Psychologie; Verhaltenstherapie; Zeitschrift für Psychiatrie, Psychologie und Psychotherapie ZPPP*).

Major Accomplishments

Rief is a world-leading scientist in the field of *somatoform disorders*. For more than 20 years, his research has considerably advanced the knowledge about epidemiology, etiology, and effective treatments of somatoform disorders. He has shown that medically unexplained symptoms are not only prevalent in medical settings, but also common in the general population. With the Screening for Somatoform Symptoms (SOMS) he and Professor Wolfgang Hiller

provided a reliable and valid instrument to assess somatoform syndromes that are associated with considerable distress, disability, and health care utilization. One of his most recent research projects (funded by a DFG grant) aimed to investigate course and classification of medically unexplained physical complaints in the general population. Against the background of current proposals to improve the classification of somatoform disorders and related syndromes in the Diagnostic and Statistical Manual of Mental Disorders DSM-5 and the International Classification of Diseases ICD-11, Rief and colleagues were the first who tested the most prominent classification proposals (such as the Complex Somatic Symptom Disorder). Their results showed that classification criteria that include positive psychological features (such as somatic illness attribution and illness behavior) are advantageous in identifying people with health care needs and disability as compared to approaches that focus simply on symptom count and the absence of sufficient medical explicability (Rief, Mewes, Martin, Glaesmer, & Braehler, 2011). Based on his expertise in the classification of somatoform disorders Rief was appointed as an ICD-11 representative of the International Association for the Study of Pain IASP to coordinate revision proposals for pain diagnoses, and he was Member of the Expert Conference on Somatic Presentations of Mental Disorders (Task: Preparation of classification criteria for ICD-11 and DSM-V) under the auspices of the American Psychiatric Association (APA), the National Institute of Health (NIH), and the World Health Organization (WHO) 2006).

Rief has always been interested in processes that contribute to etiology and maintenance of somatoform disorders. He demonstrated the roles of cognitive processes such as symptom interpretation and memory biases (Rief, Heitmüller, Reisberg, & Rüddel, 2006), of illness behaviors, and especially of neurobiological and psychoneuroimmunological processes in somatoform disorders (Rief & Barsky, 2005; Rief & Broadbent, 2007).

Rief and his colleagues conducted a number of randomized clinical trials to evaluate the effects of different intervention strategies in somatoform syndromes (all funded by research grants from the DFG, the Federal Ministry of Education, Research BMBF Germany, and others). These studies demonstrated, for example, that a cognitive-behavior group–based inpatient treatment is effective in somatoform disorders, and that even a minimal intervention program designed for subjects in primary care results in symptom improvement. The evaluation of a special training program for general practitioners to improve management of patients with unexplained physical symptoms showed a reduction in health care utilization, but appeared not to be sufficient to improve disability in patients. Rief will continue to develop psychological treatments further to enhance their clinical effects in somatoform disorders.

Placebo effects and placebo and nocebo mechanisms are one of Rief's most recent research fields (Rief, Avorn, & Barsky, 2006). He has published meta-analytical results showing that close to 70% of positive effects of antidepressants are already reported for the placebo group. In line with his interest in the development of somatic symptoms, he was able to show that expectation of side effects results in higher side effect rates in clinical trials. Consequently, many subjects discontinuing drug intake because of side effects were shown to be in the placebo group. He is the spokesperson of a German Research Unit "Expectation and conditioning as basic mechanisms of placebo and nocebo effects," which covers eigth subprojects on placebo and nocebo mechanisms (total budget about 2.7 Mio. EUR; local budget for Rief's work is 640,000,– EUR). Together with colleagues of this research unit, he has published new proposals for study design in clinical trials (Rief, Bingel, Schedlowski, & Enck, 2011).

Rief's breadth of research interests in the field of behavioral medicine is demonstrated by additional studies he has conducted in obesity (e.g., BMBF grants' "Counseling of obese patients including genetic findings" and "Psychosocial, legal, and ethical aspects of genetic and molecular obesity research"), cancer (Rief et al., 2011), tinnitus (e.g., DFG grant "Psychophysiological

aspects and treatment of chronic tinnitus" (Weise, Heinecke, & Rief, 2008), chronic pain, CBT in schizophrenia, side effect assessment in clinical trials, and public health issues. In international projects, he has collaborated with distinguished members of Harvard Medical School (A. Barsky), UCSD (J. Dimsdale), Auckland Medical School (K. Petrie), and many others. His current publication record includes more than 300 publications in peer-reviewed journals, 15 books and co-edited books, and numerous book sections.

Cross-References

- ▶ Biofeedback
- ▶ International Society of Behavioral Medicine
- ▶ Nocebo and Nocebo Effect
- ▶ Placebo and Placebo Effect
- ▶ Psychophysiology: Theory and Methods
- ▶ Somatoform Disorders

References and Readings

Rief, W., Avorn, J., & Barsky, A. J. (2006). Medication-attributed adverse effects in placebo groups. Implications for assessment of adverse effects. *Archives of Internal Medicine, 166*(2), 155–160.

Rief, W., Bardwell, W. A., Dimsdale, J. E., Natarajan, L., Flatt, S. W., Pierce, J. P. for the Women's Healthy Eating and Living (WHEL) Study Group (2011). Long-term course of pain in breast cancer survivors: A four year longitudinal study. *Breast Cancer Research and Treatment*, *130*, 579–586.

Rief, W., & Barsky, A. J. (2005). A psychobiological perspective on somatoform disorders. *Psychoneuroendocrinology, 30*, 996–1002.

Rief, W., Bingel, U., Schedlowski, M., & Enck, P. (2011). Mechanisms involved in placebo and nocebo responses and implications for drug trials. *Clinical Pharmacology and Therapeutics, 90*, 722–726.

Rief, W., & Broadbent, E. (2007). Explaining medically unexplained symptoms-models and mechanisms. *Clinical Psychology Review, 27*, 821–841.

Rief, W., Heitmüller, A. M., Reisberg, K., & Rüddel, H. (2006). Why reassurance fails in patients with unexplained symptoms-an experimental investigation of remembered probabilities. *PLoS Medicine, 3*(8), e269. doi:10.1371/journal.pmed.0030269.

Rief, W., Mewes, R., Martin, A., Glaesmer, H., & Braehler, E. (2011). Evaluating new proposals for the psychiatric classification of patients with multiple somatic symptoms. *Psychosomatic Medicine, 73*, 760–768.

Weise, C., Heinecke, K., & Rief, W. (2008). Biofeedback-based behavioural treatment for chronic tinnitus-results of a randomised controlled trial. *Journal of Consulting and Clinical Psychology, 76*, 1046–1057.

Ringing in the Ears

- ▶ Tinnitus

Risk Aversion

- ▶ Risk Perception

Risk Factors

- ▶ Diathesis-Stress Model

Risk Factors and Their Management

Bernt Lindahl
Occupational and Environmental Medicine, Department of Public Health and Clinical Medicine, Umeå University, Umeå, Sweden

Definition

The term "risk factor" describes factors that are associated with an increased risk of developing a disease but that are not sufficient to cause the disease. In this topic, risk factors for our most important noncommunicable diseases, such as cardiovascular disease (CVD), type 2 diabetes, and certain forms of cancer, are discussed. Special emphasis is on explaining the link between an individual's choice of lifestyle (behaviors) and these diseases. This link is called "the metabolic syndrome" and can be described as a group of risk factors that occur together, are modifiable by lifestyle change, and increase the risk for CVD

and diabetes. Some implications of this lifestyle metabolic syndrome association may also be drawn to other diseases, such as dementia and depression. Management of these cardiometabolic risk factors is primarily behavior modification or long-term lifestyle change. Often, the lifestyle treatment needs to be complimented by pharmacologic treatment for some specific risk factors, such as LDL-cholesterol and high blood pressure.

Description

Risk Factors

During the last four decades, it has been said that three risk factors, often called the traditional risk factors, could explain 50% of the risk of getting an acute heart attack or an acute myocardial infarction (AMI). The three risk factors were smoking, serum cholesterol, and blood pressure. In 2004, the Interheart study was published, and this worldwide case–control study found nine modifiable risk factors to explain 90% of the risk of getting AMI (Yusuf et al., 2004). The study compared 15,000 cases of AMI with the same number of controls without having AMI. They found that smoking and lipid disorder, mainly high total and LDL-cholesterol, were the most powerful of these nine risk factors. The others were blood pressure, diabetes, abdominal obesity, psychosocial factors, physical activity, fruit and vegetables, and alcohol consumption. The last three of these risk factors were in reality protecting factors, since a higher level of physical activity, fruit and vegetables, and alcohol were associated with a lower rate of AMI. The strength of this worldwide study is that the risk factor pattern for AMI was the same among men and women, between different geographical regions of the world and also between different racial or ethnic groups (Yusuf et al., 2004).

Smoking

Smoking is perhaps the most important risk factor for cardiovascular disease (CVD), and if one was to be confronted with a patient with CVD and several other risk factors, who still was a smoker, the foremost advice would be to discuss how to stop smoking (Burell & Lindahl, 2008). The consequences of smoking and how to work with tobacco and smoking cessation will be dealt with elsewhere.

Lipids

Disturbances in blood lipids are strong risk factors for atherosclerosis and cardiovascular disease, such as coronary heart disease, stroke, and peripheral artery disease. There are two main forms of lipids in the blood: cholesterol and triglycerides. Total cholesterol can be subdivided into low density lipoprotein (LDL)-cholesterol, sometimes called bad cholesterol since it increases the risk of cardiovascular disease, and high density lipoprotein (HDL)-cholesterol, sometimes called good cholesterol since it reduces the risk of cardiovascular disease (Faergeman, 2008). As a rule of thumb, in order to avoid atherosclerosis and risk of CVD, an individual's total cholesterol should be below 5 mmol/L, LDL-cholesterol below 3 mmol/L, triglycerides below 2 mmol/L, and HDL-cholesterol above 1 mmol/L. For some individuals, the lipid goals need to be even more advanced. In secondary prevention, for instance, when you already have had one heart attack and you want to prevent another one, the aim for the LDL-cholesterol level may be below 2.5 mmol/L or 2.0 mmol/L. All patients with cardiovascular disease, diabetes, and familial high cholesterol belong to this group of individuals (Faergeman, 2008).

Obesity, Insulin Resistance, and the Metabolic Syndrome

Obesity has long been the main modifiable risk factor for type 2 diabetes, and during the last decade, a nearly explosive growth over the world of both obesity and diabetes has been demonstrated. This development has mostly been attributed to developed countries, but has also increasingly been found in many developing countries (Barnett & Kumar, 2004; Björntorp, 2001). Obesity is often defined as a condition of excessive fat accumulation in adipose tissue to the extent that health may be impaired and is the result of an imbalance between energy intake and

expenditure (Björntorp, 2001). If an individual consumes more energy than he/she spends, fat tissue will accumulate, and obesity may follow. According to the World Health Organization (WHO), obesity is defined as a body mass index (BMI) of 30 kg/m^2 or more. Although obesity per se is a strong risk factor for diabetes and CVD, it was discovered already in the beginning of the 1950s that the male form of obesity having most of the fat deposit localized around the waist, i.e., abdominal obesity, was an even stronger risk factor (Barnett & Kumar, 2004; Björntorp, 2001). A waist circumference of 94 cm (37 in.) in men and 80 cm (32 in.) in women has been shown to increase the risk of getting diabetes and CVD, and this risk is further increased with a waist of 102 cm (40 in.) or above in men and 88 cm (35 in.) or above in women. The first level could be seen as an alerting zone, i.e., an obligation to inform the individual about the increased risk and discuss possible solutions. The second level is often called "an action level," implying that these individuals really ought to be offered inclusion in a lifestyle program (Björntorp, 2001).

Portal-Visceral Hypothesis and Insulin Resistance

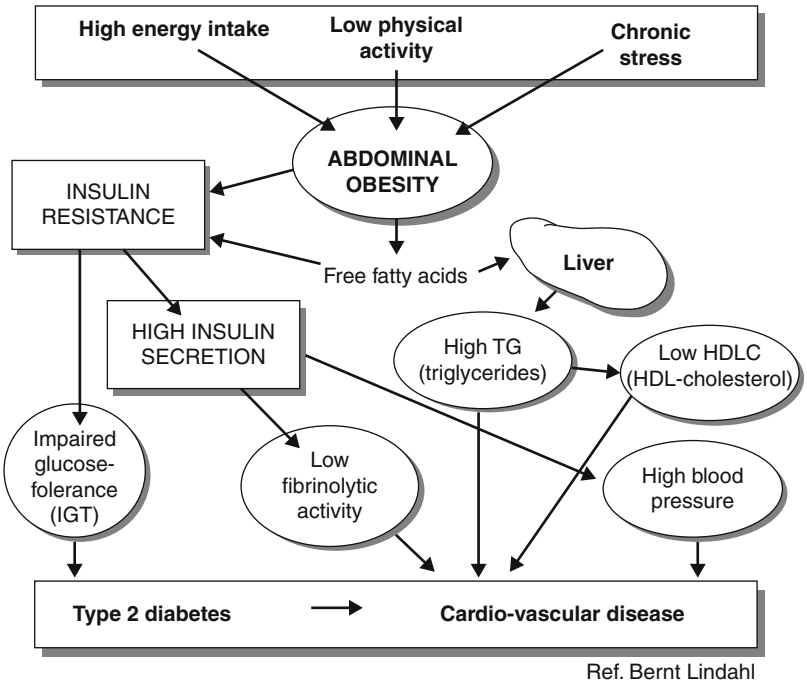
Several of the nine modifiable risk factors from the Interheart study are highly correlated to each other, i.e., they are more often than by chance found together in the same individual. As shown in Fig. 1, low physical activity in combination with high energy intake and sustained psychosocial stress will increase the risk of developing obesity and especially abdominal obesity. A large amount of abdominal fat, which is more metabolically active than subcutaneous fat, will release large amounts of fatty acids to the blood circulation. Large amounts of free fatty acids at muscular level will reduce the uptake of glucose due to a subnormal insulin action. A state of insulin resistance will follow, which may be defined as a state in which a given concentration of insulin produces a less than expected biological effect. The small increase in glucose concentration in the circulation that will follow will in turn increase the secretion of insulin from the beta cells in the pancreas. Glucose uptake at muscular level will be restored, despite that insulin resistance still exists. However, the price to pay is a larger than normal insulin secretion. This could go on for years with increasing body weight, waist circumference, insulin resistance, and high beta cell activity. The long-term problem is that the beta cells get exhausted and lose their ability to produce insulin (Kumar & O'Rahilly, 2005).

In the beginning of such a development, the amount of insulin secreted is not high enough to take care of the carbohydrates after a meal and plasma glucose often rises to levels beyond the normal. This is called a state of impaired glucose tolerance (IGT), and without a lifestyle change, this state will worsen to type 2 diabetes in about 50% of the cases within a 10-year period. IGT is a reversible state, and much attention in primary prevention of type 2 diabetes has been devoted to find and to treat this group of high-risk individuals. Impaired glucose tolerance (IGT) as well as impaired fasting glucose (IFG), i.e., having a higher than normal plasma glucose in the fasting state but not after a meal, may be called prediabetic states (Ganz, 2005; Kumar & O'Rahilly, 2005).

Other effects of insulin resistance and high insulin secretion are the appearance of risk factors such as high blood pressure and a specific lipid disturbance, characterized by high levels of triglycerides and low levels of HDL-cholesterol. This lipid combination is relatively common and considered to be a strong risk factor for CVD. The combination of these multiple risk factors for diabetes and cardiovascular disease is called "the metabolic syndrome" (Fig. 1). Other names for this risk state are syndrome X, the deadly quartet, or the insulin resistance syndrome. The description above is based upon the portal-visceral hypothesis for the mechanism of developing insulin resistance. However, other mechanisms have also been discussed (Kumar & O'Rahilly, 2005).

Adipose Tissue as an Endocrine Organ

During the last 15 years, our knowledge of fat tissue has changed tremendously, from being just a reservoir for energy to being the largest endocrine organ in the body. The handling of energy intake and expenditure and of hunger and satiety



Risk Factors and Their Management, Fig. 1 The metabolic syndrome: the link between lifestyle, type 2 diabetes, and cardiovascular disease

as well as effects on blood pressure and inflammatory systems are regulated, at least in part, by hormones or hormone-like substances (adipokines) produced and secreted from fat cells in adipose tissue (Kumar & O'Rahilly, 2005).

Ectopic Fat Accumulation

A third possible mechanism for the development of insulin resistance and increased risk for diabetes is "ectopic fat" accumulation. The definition of ectopic fat is that it is accumulation of fat cells in other tissues than adipose tissue. It has been shown that ectopic fat in liver and muscle tissues is associated with central (liver) and peripheral (muscles) insulin resistance, and accumulation of fat droplets in the neighborhood of the beta cells in the pancreas has been associated with a disturbance in insulin secretion (Kumar & O'Rahilly, 2005).

Type 2 Diabetes and Cardiovascular Disease

From population studies, using different metabolic syndrome definitions, as much as 20–25% of the adult population has been found to have a metabolic syndrome. Besides having the risk of developing diabetes, the risk of getting CVD is two- to threefold higher among those with the metabolic syndrome. Today, type 2 diabetes, due to its strong correlation with CVD (macrovascular complications of diabetes), is by many considered to be in its essence a cardiovascular disease, and among diabetes patients, there is a three- to fourfold increased risk for CVD. Importantly, much points to the fact that the CVD risk already starts to rise when the individuals start to accumulate body fat and is clearly increased in the state of the metabolic syndrome, and even more so in impaired glucose tolerance. In other words, the risks of developing type 2 diabetes and cardiovascular disease should be seen as parallel phenomena, and not as a simple case of diabetes being followed by CVD (the "common soil" hypothesis). This has of course large implications on when in this process of development it is optimal to start the prevention or treatment of these risk states or diseases (Ganz, 2005; Kumar & O'Rahilly, 2005).

The strong association between diabetes and CVD could also be demonstrated from another point of view. In a recent Swedish study on acute myocardial infarction patients submitted to

hospital care and followed up 3 months later with among other things an oral glucose tolerance test, it was demonstrated that only 35% of the AMI patients had normal glucose concentrations. A majority of the cases (65%) had either unknown diabetes or prediabetes (Norhammar et al., 2002).

Other Related Diseases

The same kind of reasoning as above, using the same risk factor pattern, has also given rise to implications for other types of diseases (outcomes), such as certain types of cancers (mainly breast, colon, and prostate), dementia, and depression. However, our knowledge is much more limited in this area of research.

Management of Risk Factors

Behavior Modification and Long-Term Lifestyle Change

As discussed above, in reality, many of our most important risk factors for noncommunicable diseases, such as type 2 diabetes and cardiovascular disease as well as certain forms of cancer, dementia, and depression, are the same and are also associated with each other in a complex pattern of interrelationships, such as in the metabolic syndrome (Fig. 1). An individual's choice of lifestyle will have great impact on these relationships, and the metabolic syndrome may be seen as the link between the lifestyle of an individual and the risk to develop type 2 diabetes and cardiovascular disease for that individual. Of course, heredity also influences many of these interrelationships, but this is a risk factor that, at least today, is unmodifiable. Behavior modification inducing long-term lifestyle change with increased physical activity and a lowered energy intake, often based upon a low-fat high-fiber diet, and sometimes also the inclusion of stress management techniques in the lifestyle change, has been the standard for an intensive lifestyle change program. Studies using this approach often achieve a weight loss of 5–10% of the initial body weight (Barnett & Kumar, 2004; Burell & Lindahl, 2008; Ganz, 2005). Although, this is a relatively modest weight loss for someone having obesity, it has been shown to generate large beneficial effects on the metabolism. However, it seems crucial that the weight loss is achieved by a lifestyle change. An intensive lifestyle program induces a multitude of effects that protect against both cardiovascular disease and type 2 diabetes. In the Finnish Diabetes Prevention Study (DPS), a weight loss difference between the intensive lifestyle group and the control group of 2.7 kg after 2 years resulted in a reduction of diabetes development of 58% at the 3-year follow-up (Tuomilehto et al., 2001).

Effects of Physical Activity

There is a strong inverse relationship between physical activity, type 2 diabetes, and cardiovascular disease, and the evidence for this relationship must be considered as solid (Burell & Lindahl, 2008). Furthermore, a large body of evidence examining the effects of physical activity on cardiovascular risk factors has also documented beneficial effects, mainly a lowering of blood pressure and a less atherogenic lipid profile. A paradigm shift concerning physical activity was launched in 1995, proclaiming that even less intensive and shorter bouts of physical activity may have health promoting effects (Pate et al., 1995). It was shown that a dose corresponding to an energy expenditure of 1,000 kcal per week significantly lowered all-cause mortality by 20–30%, and CVD mortality possibly even more. This level of energy expenditure corresponds to about 30 min of moderately intensive physical activity each day, i.e., brisk walks, bicycling, and swimming. An updated version of this physical activity statement was published in 2007 (Haskell et al., 2007).

Effects of a Healthy Diet

In the last decade, our understanding of how nutrients and foods promote cardiac health or increase the risk for diabetes and cardiovascular disease has grown substantially, but the search for an optimal composition of food intake is far from over. Diets that are higher in monounsaturated fatty acids, fiber, and low glycemic foods (slow carbohydrates) appear to improve insulin resistance, blood glucose, and blood lipids. Additionally, three food intake strategies to prevent

cardiovascular disease have been suggested. First, to increase intake of unsaturated fats and decrease intake of saturated and trans-fats, second, to increase intake of omega-3 fatty acids, and last, to increase consumption of fruits, vegetables, nuts, and whole grains (Hu & Willett, 2002).

Adherence to a Long-Term Lifestyle Change

Two key features of such an intensive lifestyle program in order to achieve adherence are goal-setting and self-monitoring. The goals should be realistic, measurable, concrete, and engaging, and there should be both short- and long-term goals. Simple ways of self-monitoring key behaviors, such as food intake and physical activity, should also be included in such a lifestyle program (Burell & Lindahl, 2008; Ganz, 2005).

Management of Specific Risk Factors: High LDL-Cholesterol and High Blood Pressure

In addition to a comprehensive lifestyle program, some specific risk factors need to be treated with medications, either because the lifestyle program is insufficient to handle the risk factor in an optimal way or because healthy lifestyle change does not impact enough on the risk factor in question. In order to lower LDL-cholesterol, lipid-modifying drugs, especially use of statins, are the standard treatment in cases with familial high cholesterol levels and in patients with cardiovascular disease and type 2 diabetes (Faergeman, 2008). In order to control high blood pressure, a lifestyle change is often not enough, and the use of antihypertensive drugs is customary.

Cross-References

► Behavior Change

References and Readings

Barnett, A. H., & Kumar, S. (Eds.). (2004). *Obesity & diabetes*. Chichester, UK: Wiley.

Björntorp, P. (Ed.). (2001). *International textbook of obesity*. Chichester, UK: Wiley.

Burell, G., & Lindahl, B. (2008). Management of specific behavioural risk factors – exercise, obesity and smoking. In I. M. Graham & R. B. D'Agostino (Eds.), *Therapeutic strategies in cardiovascular risk* (pp. 201–211). Oxford: Clinical Publishing.

Faergeman, O. (2008). Management of specific risk factors – lipids. In I. M. Graham & R. B. D'Agostino (Eds.), *Therapeutic strategies in cardiovascular risk* (pp. 213–232). Oxford: Clinical Publishing.

Ganz, M. (Ed.). (2005). *Prevention of type 2 diabetes*. Chichester, UK: Wiley.

Haskell, W. L., Lee, I.-M., Pate, R. R., Powell, K. E., Blair, S. N., Franklin, B. A., et al. (2007). Physical activity and public health. Updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Circulation, 116*, 1081–1093.

Hu, F. B., & Willett, W. C. (2002). Optimal diets for prevention of coronary heart disease. *Journal of the American Medical Association, 288*, 2569–2578.

Kumar, S., & O'Rahilly, S. (Eds.). (2005). *Insulin resistance*. Chichester, UK: Wiley.

Norhammar, A., Tenerz, Å., Nilsson, G., Hamsten, A., Efendic, S., & Rydén, L. (2002). Glucose metabolism in patients with acute myocardial infarction and no previous diagnosis of diabetes mellitus: A prospective study. *Lancet, 359*, 2140–2144.

Pate, R. R., Pratt, M., Blair, S. N., Haskell, W. L., Macera, C. A., Bouchard, C., et al. (1995). Physical activity and public health. A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. *Journal of the American Medical Association, 273*, 402–407.

Tuomilehto, J., Lindström, J., Eriksson, J. G., Valle, T. T., Hämäläinen, H., Lanne-Parikka, P., et al. (2001). Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *New England Journal of Medicine, 344*, 1343–1350.

Yusuf, S., Hawken, S., Ounpuu, S., Dans, T., Avezum, A., Lanas, F., et al. (2004). Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case–control study. *Lancet, 364*, 937–952.

Risk Perception

Catherine Darker
Public Health & Primary Care, Trinity College, The University of Dublin, Dublin, Ireland

Synonyms

Risk aversion; Risk taking

Definition

Risk perceptions are beliefs about potential harm or the possibility of a loss. It is a subjective judgment that people make about the characteristics and severity of a risk.

Description

The degree of risk associated with a given behavior is generally considered to represent the likelihood and consequences of harmful effects that result from that behavior. To perceive risk includes evaluations of the probability as well as the consequences of an uncertain outcome. There are three dimensions of perceived risk – perceived likelihood (the probability that one will be harmed by the hazard), perceived susceptibility (an individual's constitutional vulnerability to a hazard), and perceived severity (the extent of harm a hazard would cause). Risk perceptions are central to many health behavior theories. For example, models that have been developed specifically to predict health behavior such as the health belief model (Rosenstock, 1966), protection motivation theory (Rogers, 1975), and the self-regulation model (Leventhal, Meyer, & Nerenz, 1980) all contain constructs that explicitly focus on risk perceptions. In addition, other models such as the theory of reasoned action (Fishbein & Ajzen, 1975), the theory of planned behavior (Ajzen, 1985), and social cognitive theory (Bandura, 1977) also include perceptions of risk indirectly via other constructs.

Biases and Heuristics in Risk Assessment

The estimation of risk tends to be a complex process that depends on factors such as the context in which the risk information is presented, the way the risk is being described, and also on personal and cultural characteristics. Tversky and Kahneman (1973) proposed that when faced with the difficult task of judging risk, people use a limited number of strategies, called heuristics, to simplify these judgments. These heuristics can be useful shortcuts for thinking, but they may lead to inaccurate judgments in some situations in which case they become cognitive biases. There are three broad biases that can affect risk perceptions and these are the availability heuristic, the representativeness heuristic, and anchoring and adjustment heuristic.

Availability heuristic is a phenomenon in which people predict the frequency or likelihood of an event, or a proportion within a population, based on how easily an example can be brought to mind (Tversky & Kahneman, 1973). Representativeness heuristic is an occurrence in which individuals assess the frequency of a particular event based solely on the generalization of a previous similar event (Gilovich, Griffin, & Kahneman, 2002). Anchoring and adjustment heuristic is a phenomenon in which people start with one piece of known information, known as an anchor, and then adjust said information to create an estimate of an unknown risk (Epley & Gilovich, 2006).

Psychometric Paradigm of Risk Assessment

The "psychometric paradigm" developed by Slovic, Fischhoff, and Lichtenstein was a landmark in research about public attitudes toward risks (Fischhoff, Slovic, & Lichtenstein, 1983; Fischhoff, Slovic, Lichtenstein, Read, & Combs, 1978; Slovic, Fischhoff, & Lichtenstein, 1980, 1982, 1985). These studies demonstrated that the public is not irrational. Ordinary people simply use a broader definition of "risks" than experts when making their judgments about which ones are of most concern to them. "Experts" base their risk ratings on the expected number of fatalities. "Lay people," in contrast, have a richer definition of risk. This incorporates a number of more qualitative characteristics such as "voluntariness" (whether people have a choice about whether they face the risk), "immediacy of effect" (the extent to which the effect is immediate, or might occur at some later time), and "catastrophic potential" (whether many people would be killed at once). Slovic et al.(1985) identified and analyzed 18 characteristics of this kind using factor analysis and found that they could be resolved into three factors broadly defined as "dread," "unknown," and "exposure." Further,

high perceived risk, and hence a desire for societal regulation, was associated.

Research within the psychometric paradigm turned to focus on the roles of affect, emotion, and stigma in influencing risk perception. Psychometric research identified a broad domain of characteristics that may be condensed into three high order factors: (1) the degree to which a risk is understood, (2) the degree to which it evokes a feeling of dread, and (3) the number of people exposed to the risk. A dread risk elicits visceral feelings of terror, uncontrollability, catastrophe, and inequality. An unknown risk is new and unknown to science. The more a person dreads an activity, the higher its perceived risk and the more that person wants the risk reduced (Slovic et al., 1982).

Cross-References

- ▶ Bias
- ▶ Cancer Risk Perceptions
- ▶ Perceived Risk

References and Readings

Ajzen, I. (1985). From intentions to actions: A theory of planned behavior. In J. Kuhl & J. Beckman (Eds.), *Action-control: From cognition to behavior* (pp. 11–39). Heidelberg: Springer.

Bandura, A. (1977). Self efficacy: Toward a unifying theory of behavior change. *Psychological Review, 84*, 191–215.

Epley, N., & Gilovich, T. (2006). The anchoring-and-adjustment heuristic: Why the adjustments are insufficient. *Psychological Science, 17*, 311–318.

Fischhoff, B., Slovic, P., & Lichtenstein, S. (1983). The Public vs. 'the Experts'. In V. T. Covello, W. G. Flamm, J. V. Rodricks, & R. G. Tardiff (Eds.), *The analysis of actual vs. perceived risks* (pp. 235–249). New York: Plenum.

Fischhoff, B., Slovic, P., Lichtenstein, S., Read, S., & Combs, B. (1978). How safe is safe enough? A psychometric study of attitudes towards technological risks and benefits. *Policy Studies, 9*, 127–152.

Fishbein, M., & Ajzen, I. (1975). *Belief, attitude and intention and behavior: An introduction to theory and research*. Reading, MA: Addison-Wesley.

Gilovich, T., Griffin, D., & Kahneman, D. (2002). *Heuristics and biases – The psychology of intuitive judgment*. New York: Cambridge University Press.

Kahneman, D., & Tversky, A. (1973). On the psychology of prediction. *Psychological Review, 80*, 237–251.

Leventhal, H., Meyer, D., & Nerenz, D. (1980). The common sense representation of illness danger. *Medical Psychology, 2*, 7–30.

Plous, S. (1993). *The psychology of judgment and decision making*. New York: McGraw-Hill.

Rogers, R. W. (1975). A protection motivation theory of fear appeals and attitude change. *Journal of Psychology, 91*, 93–114.

Rosenstock, I. M. (1966). Why people use health services. *Milbank Memorial Fund Quarterly, 44*, 94–127.

Slovic, P. (2000). *The perception of risk*. Virginia: Earthscan.

Slovic, P., Fischhoff, B., & Lichtenstein, S. (1980). Facts and fears: Understanding perceived risk. In R. C. Schwing & W. A. Albers (Eds.), *Societal risk assessment: How safe is safe enough?* (pp. 181–216). New York: Plenum Press.

Slovic, P., Fischhoff, B., & Lichtenstein, S. (1982). Why study risk perception? *Risk Analysis, 2*, 83–93.

Slovic, P., Fischhoff, B., & Lichtenstein, S. (1985). Characterizing perceived risk. In R. W. Kates, C. Hohenemser, & J. X. Kasperson (Eds.), *Perilous progress: Managing the Hazarak of technologv*. Boulder, CO: Westview Press.

Tversky, A., & Kahneman, D. (1973). Availability: A heuristic for judging frequency and probability. *Cognitive Psychology, 5*, 207–232.

Tversky, A., & Kahneman, D. (1974). Judgments under uncertainty: Heuristics and biases. *Science, 185*, 1124–1131.

Wildavsky, A., & Dake, K. (1990). Theories of risk perception: Who fears what and why? *American Academy of Arts and Sciences (Daedalus), 119*, 41–60.

Risk Pooling

- ▶ Health Insurance: Comparisons

Risk Ratio

- ▶ Relative Risk

Risk Reduction

- ▶ Harm Reduction

Risk Taking

▶ Risk Perception
▶ Risky Behavior

Risk, Absolute

▶ Absolute Risk

Risk, Relative

▶ Relative Risk

Risk-Benefit Assessment

▶ Benefit-Risk Estimation

Risk-Benefit Ratio

▶ Benefit-Risk Estimation

Risky Behavior

Tereza Killianova
Free University of Brussels (VUB), Jette, Belgium

Synonyms

Risk taking

Definition

Risky behavior or risk-taking behavior is defined according to Trimpop (1994) as "any consciously, or non-consciously controlled behavior with a perceived uncertainty about its outcome, and/or about its possible benefits, or costs for the physical, economic or psycho-social well-being of oneself or others." In addition to this broad definition, there are other definitions of risky behavior depending on the field of research. While in the economic view, risk is defined in terms of the variability of possible monetary outcomes, in the clinical literature, the risk is generally defined as exposure to possible loss or harm (Schonberg, Fox, & Poldrack, 2011). Turner et al. (2004) described risk-taking behavior further as either a socially unacceptable volitional behavior with a potentially negative outcome in which precautions are not taken, such as speeding, drinking and driving, drugs abuse, unprotected sex, . . ., or a socially accepted behavior in which the danger is recognized (climbing, competitive sports,. . .). This description is important when looking at the relation between risk-taking behavior and injuries. It has been shown that risk-taking behavior is associated with an increased chance of sustaining an injury. However, this relation was not shown in the case of high-skilled, risk-taking sports (Turner, McClure, & Pirozzo, 2004). Risky behavior can be a direct consequence and manifestation of a risk-taking personality or of sensation-seeking personality. These can be reliably assessed by various existing questionnaires. In behavior medicine, risk behavior is an important factor to consider, since it is a predictor of various adverse health outcomes. In the domain of driving, risky behaviors, such as excessive speeding or driving under the influence of alcohol, are known predictors of traffic accidents (Jonah, 1997), one of the 10 leading causes of mortality worldwide. In infectious diseases, risky behavior in the form of unprotected sex or having multiple partners often co-occurs (Biglan et al., 1990) and can increase the risk of HIV/AIDS, a leading cause of death in Africa. Thus, identifying people likely to perform risky behaviors and developing and testing interventions to prevent risky behaviors are crucial for contributing to prevention of diseases and adverse health outcomes at the individual and social levels.

Cross-References

▶ Risk Perception

References and Readings

Biglan, A., Metzler, C. W., Wirt, R., Ary, D., Noell, J., Ochs, L., et al. (1990). Social and behavioral factors associated with high-risk sexual behavior among adolescents. *Journal of Behavioral Medicine, 13*, 245–261.

Jonah, B. A. (1997). Sensation seeking and risky driving: A review and synthesis of the literature. *Accident Analysis and Prevention, 29*, 651–665.

Schonberg, T., Fox, C. R., & Poldrack, R. A. (2011). Mind the gap: Bridging economic and naturalistic risk-taking with cognitive neuroscience. *Trends in Cognitive Sciences, 15*(1), 11–19.

Trimpop, R. (1994). *The psychology of risk taking behavior*. Amsterdam: Elsevier Science.

Turner, C., McClure, R., & Pirozzo, S. (2004). Injury and risk-taking behavior-a systematic review. *Accident Analysis and Prevention, 36*, 93–101.

Risky Drinking Episode

▶ Binge Drinking

RNA

Jana Strahler
Clinical Biopsychology, Department of Psychology, University of Marburg, Marburg, Germany

Synonyms

Coding RNA; Messenger RNA; mRNA; Noncoding RNA; Ribosomal RNA; rRNA; Transfer RNA; tRNA

Definition

Ribonucleic acid (RNA) is a chain of multiple nucleotides (a polynucleotide) consisting of a molecule of sugar (ribose), a molecule of phosphoric acid, and a nucleic base (uracil, cytosine, guanine, or adenine). In contrast to DNA, RNA is normally single-stranded. Its main function within the cell is the conversion of the genetic information into proteins, i.e., gene expression. There are different RNA molecules exerting different functions, the so-called coding and noncoding RNA. Coding RNA, also called messenger RNA (*mRNA*), copies information from the DNA and carries this information to the ribosome, the cell organelle where protein synthesis takes place. There are different forms of noncoding RNA with transfer RNA and ribosomal RNA being the most important. Transfer RNA (*tRNA*) molecules take amino acids and transport them to the ribosome. Ribosomal RNA (*rRNA*) is the fundamental part of the ribosome and catalyzes protein synthesis.

Research has demonstrated that various types of stressors modulate RNA. For instance, social isolation, chronic stress, and low socioeconomic status are associated with immunological impairment mediated via alterations in immune-related RNA expression. In contrast, there is also evidence for an RNA-regulating effect of positive psychological states.

Cross-References

▶ DNA
▶ Gene Expression

References and Readings

Cole, S. W., Hawkley, L. C., Arevalo, J. M., Sung, C. Y., Rose, R. M., & Cacioppo, J. T. (2007). Social regulation of gene expression in human leukocytes. *Genome Biology, 8*(9), R189.

Sloan, E. K., Capitanio, J. P., Tarara, R. P., Mendoza, S. P., Mason, W. A., & Cole, S. W. (2007). Social stress enhances sympathetic innervation of primate lymph nodes: Mechanisms and implications for viral pathogenesis. *Journal of Neuroscience, 27*(33), 8857–8865.

Robert Wood Johnson Foundation

Stephanie Ann Hooker
Department of Psychology, University of Colorado, Denver, CO, USA

Basic Information

The Robert Wood Johnson Foundation (RWJF) is one of the world's largest private philanthropic foundations, with a mission of improving public health in the United States. Upon his death, Robert Wood Johnson II (of Johnson & Johnson Services Inc.) dedicated virtually all of his fortune to establish this foundation.

The RWJF funds projects that are "innovative" and have "measurable impact" in seven different program areas. These program areas include childhood obesity, coverage (health care), human capital (preparation of health professionals), pioneer (innovative health-related technologies), public health, quality/equality (health care), and vulnerable populations. Within these seven program areas, the RWJF funds a variety of different types of projects including service demonstrations, gathering and monitoring of health-related statistics, public education, training and fellowship programs, policy analysis, health services research, technical assistance, communications activities, and evaluations. In 2009, the RWJF funded $350 million in grants, and approximately 20% of the funding was dedicated to research-related projects.

Major Impact on the Field

The RWJF seeks to better society and the lives of Americans through its philanthropic contributions. Some notable past efforts include the creation of the 911 emergency response system across the United States; the introduction of new methods for the research, prevention, and treatment of tobacco use; and the establishment of the field of end-of-life/palliative care.

Each year, the RWJF publishes a book entitled *To Improve Health and Health Care: The Robert Wood Johnson Foundation Anthology*. The book focuses on approximately 10 different topics every year and describes why the foundation funded certain topics, what the program activities were, what was accomplished, how the program fits with RWJF, and what lessons were learned. The most recent volume (XIV) focused on health and health care of vulnerable populations. This anthology is one way the foundation examines its impact on public health, and it is available on the foundation's website.

Cross-References

► Palliative Care
► Public Health

References and Readings

Isaacs, S. L., & Dolby, D. C. (Eds.). (2011). *To improve health and health care: The Robert Wood Johnson Foundation anthology* (Vol. XIV). San Francisco: Jossey-Bass.

Robert Wood Johnson Foundation. (2011). Retrieved July 15, 2011 from http://www.rwjf.org

rRNA

► RNA

Rumination

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

Rumination is both a state and trait tendency to focus on negative events, emotions, and

symptoms, their occurrence, causes, and consequences (Nolen-Hoeksema, 1991; Rydstedt, Cropley, & Devereux, 2011). Multiple conceptualizations of rumination exist, each having its own measure, and tested in different contexts. The majority of research has tested and shown rumination to play a key role in the onset and maintenance of depression. However, some research also shows that it causes and maintains distress in physically ill patients (see review by Soo, Burney, & Basten, 2009). Rumination is a marker of poor adaptation, since it is thought to prolong one's psychophysiological response to a stressor even long after it has ended. In a review of this domain, Brosschot (2010) views rumination as part of perseverative cognitions, where people have a sustained cognitive representation of past stressors, beyond their mere existence. Brosschot also contends that much of the effects of rumination could also be unconscious, even impacting one's well-being during sleep. Furthermore, rumination is thought to mediate the association between stress and one's psychophysiological responses. Rumination, which amplifies one's failure to achieve goals, can be contrasted with more constructive self-reflection which is more distant and less immersed in one's experiences. Supporting this, among people moderately high on rumination, promotion failure predicted depressive symptoms. In contrast, among people high on self-reflection, promotion failure was not predictive of severe increases in depressive symptoms (Jones, Papadakis, Hogan, & Strauman, 2009). Looking at physiological indices of stress, Rydstedt et al. (2011) found that rumination interacted synergistically with job ambiguity (a known stressor) in relation to morning cortisol in British workers. As such, rumination amplified the effects of job ambiguity on a physiological marker of stress, namely, morning cortisol. Thus, rumination amplifies and worsens the impact of negative events, both in terms of their "duration," and in terms of their possible psychophysiological consequences. Some research shows that meditation can reduce levels of rumination, and this requires further research.

Cross-References

- ▶ Coping Strategies
- ▶ Negative Thoughts
- ▶ Neuroticism
- ▶ Perseverative Cognition
- ▶ Psychophysiologic Reactivity
- ▶ Worry

References and Readings

Brosschot, J. F. (2010). Markers of chronic stress: Prolonged physiological activation and (un)conscious perseverative cognition. *Neuroscience and Biobehavioral Reviews, 35*, 46–50.

Jones, N. P., Papadakis, A. A., Hogan, C. M., & Strauman, T. J. (2009). Over and over again: Rumination, reflection, and promotion goal failure and their interactive effects on depressive symptoms. *Behaviour Research and Therapy, 47*, 254–259.

Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology, 100*(4), 569–582.

Rydstedt, L. W., Cropley, M., & Devereux, J. (2011). Long-term impact of role stress and cognitive rumination upon morning and evening saliva cortisol secretion. *Ergonomics, 54*, 430–435.

Soo, H., Burney, S., & Basten, C. (2009). The role of rumination in affective distress in people with a chronic physical illness: A review of the literature and theoretical formulation. *Journal of Health Psychology, 14*, 956–966.

S

Saccharide

► Carbohydrates

Saliva

► Salivary Biomarkers

Salivary Biomarkers

Douglas A. Granger[1] and Sara B. Johnson[2]
[1]Center for Interdisciplinary Salivary Bioscience Research, School of Nursing, Bloomberg School of Public Health, and School of Medicine The Johns Hopkins University, Baltimore, MD, USA
[2]School of Medicine and Bloomberg School of Public Health, Johns Hopkins School of Medicine, Baltimore, MD, USA

Synonyms

Analytes; Biomarkers; Saliva

Author Notes In the interest of full disclosure, DAG is founder and Chief Strategy and Scientific Advisor at Salimetrics LLC (State College, PA). DAG's relationship with Salimetrics LLC is managed by the policies of the Conflict of Interest Committee at the Johns Hopkins University School of Medicine.

Definition

Behavioral medicine research is increasingly being influenced by theoretical models that explain individual differences in behavior and disease risk as a function of interrelated biological, behavioral, social, and contextual forces. This multi-level theoretical approach follows technical innovations that have made measuring the activity of many biological systems straightforward, portable, and cost efficient. Saliva, in particular, has received attention as a biospecimen; sample collection is perceived as feasible, cost-efficient, and safe, and salivary assays as reliable and accurate (see Table 1). A single oral fluid specimen can provide information about a range of physiologic systems, chemical exposures, and genetic variability relevant to basic biological function, health, and disease. The purpose of this review is to provide a road map for investigators interested in integrating this unique biospecimen into the next generation of studies in behavioral medicine.

Description

Oral fluid as a biospecimen: "Saliva" is a composite of oral fluids secreted from many different glands. The source glands are located in the upper posterior area of the oral cavity (*parotid gland* area), lower area of the mouth between the cheek and jaw (*submandibular gland* area), and under the tongue (*sublingual gland* area). There are also

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

Salivary Biomarkers, Table 1 Perceived advantages of oral fluids as a research specimen compared to serum

"Minimally invasive"	Considered "acceptable and noninvasive" by research participants and patients
	Collection is quick, non-painful, uncomplicated
"Safety"	Reduces transmission of infectious disease by eliminating the potential for accidental needle sticks
	CDC does not consider saliva a class II biohazard unless visibly contaminated with blood
"Self-collection"	Allows for community- and home-based collection
	Enables specimen collection in special populations
"Economics"	Eliminates the need for a health care intermediary (e.g., phlebotomist, nurse)
	Resources for collection and processing samples are of low cost and available
"Accuracy"	Salivary levels of many analytes represent the "free unbound fraction" or biological active fraction in the general circulation

Source: US Department of Health and Human Services (2000)

many minor secretory glands in the lip, cheek, tongue, and palate. A small fraction of oral fluid (i.e., crevicular fluid) comes from serum leakage, either from the cleft area between teeth and gums, or from mucosal injury or inflammation. In the presence of significant mucosal or epithelial inflammation, however, serum constituents may contribute substantially to oral fluids. Each secretory gland produces a fluid that differs in volume, composition, and constituents. Oral fluid is water-like in composition and has a pH (acidity) between 6 and 9; it has minimal buffering capacity, so substances placed in the mouth can change salivary acidity very quickly.

An understanding of how a given analyte makes its way into oral fluid is key to interpreting individual differences in that analyte, as well as its association with outcomes of interest. Many of the salivary analytes of interest in biobehavioral research are serum constituents (e.g., steroid hormones). Serum constituent analytes are transported into saliva either by *filtration* between the tight spaces between acinus or duct cells in the salivary glands, or by *diffusion* through acinus or duct cell membranes. In contrast, some analytes found in oral fluids are synthesized, stored, and released from the granules within the secretory cells of the salivary glands (i.e., enzymes, mucins, cystatins, histatins). Still others are components of humoral immunity (antibodies, complement) or compounds (cytokines) secreted by immune cells (neutrophils, macrophages, lymphocytes). In addition to these analytes, saliva contains sufficient cellular material to obtain high quantity and quality DNA (Zimmerman, Park, & Wong, 2007).

The *rate* of saliva secretion can significantly influence levels of salivary analytes produced locally in the mouth (e.g., alpha-amylase (sAA), secretory IgA) as well as those that migrate into saliva from blood by filtration (e.g., dehydroepiandrosterone-sulfate and other conjugated steroids) (Malamud & Tabak, 1993). Oral fluid secretion is influenced by many factors, including the day-night cycle, chewing movement of the mandibles, taste and smell, medications that cause dry mouth, as well as medical conditions and treatments that affect salivary gland function (e.g., radiation therapy, Sjögren's syndrome). It is important to note, therefore, that for analytes influenced by flow rate, the measured concentration or activity of the analyte (e.g., U/mL, pg/mL) must be multiplied by the flow rate (mL/min). The resulting measure is expressed as *output as a function of time* (e.g., U/min, pg/min).

Sample Collection: Even under normative-healthy conditions, more than 250 species of bacteria are present in oral fluids (Paster et al., 2001). During upper respiratory infections, oral fluids are highly likely to contain agents of disease. Oral fluid specimens should, therefore, be handled with *universal precautions* when used in research and diagnostic applications.

Saliva collection devices have historically involved cotton-based absorbent materials. Placed in the mouth for 2–3 min, oral fluids rapidly saturate the cotton; fluids are subsequently recovered by centrifugation or compression.

Most of the time, this approach is convenient, simple, and time-efficient. However, when the sample volume is small, the specimen can be diffusely distributed in the cotton fibers, making sample recovery problematic (Harmon, Hibel, Rumyansteva, & Granger, 2007). The process of absorbing oral fluid with cotton and other materials also interferes with several salivary immunoassays (Groschl & Rauh, 2006). Further, where in the mouth oral swabs are placed may affect the measured levels or activity of some salivary analytes (e.g., Beltzer et al., 2010). Standardizing swab placement instructions and monitoring compliance can minimize this threat to measurement validity.

In early studies, saliva flow was often stimulated using techniques that involved chewing or tasting various substances (e.g., gums, waxes, sugar crystals, powdered drink mixes). When not used minimally and/or consistently, some of these methods may change immunoassay performance (Granger et al., 2007). Indirectly, stimulants also influence levels of salivary analytes that depend on saliva flow rate (SIgA; dehydroepiandrosterone-sulfate (DHEA-S); Neuropeptide Y (NPY); Vasoactive Intestinal Peptide, (VIP)). Researchers are advised to avoid these techniques.

Collecting *whole saliva* by "passive drool" (Granger et al., 2007) is an alternative collection approach that minimizes many of the threats to validity described above. Briefly, participants imagine they are chewing their favorite food, slowly moving their jaws in a chewing motion, and allowing oral fluid to pool in their mouth. Next, they gently force the specimen through a short plastic drinking straw into a vial. The advantages of this procedure include the following: (1) A large sample volume may be collected relatively quickly (3–5 min). (2) Target collection volume may be confirmed by visual inspection in the field. (3) The fluid collected is a pooled specimen mixture of the output from all salivary glands. (4) The procedure does not introduce interference related to stimulating or absorbing saliva. (5) Collection materials are of very low cost. (6) Samples can be aliquoted and archived for future assays.

Blood Leakage into Oral Fluid: Blood poses a threat to the validity of salivary analyte measurements because most analytes are present in serum in much higher levels (10–100-fold) than in saliva. Specifically, to meaningfully index *systemic* (vs oral) biological activity, analyte levels in saliva must be highly correlated with levels measured in serum. This serum-saliva association depends, in part, on circulating molecules being appropriately and consistently transported into oral fluids (Malamud & Tabak, 1993). When the integrity of diffusion or filtration is compromised (e.g., through blood leakage directly into salivary fluid), the level of the serological marker in saliva will be affected. Blood leakage into oral fluid is more common among individuals with poor oral health (i.e., open sores, periodontal disease, gingivitis), certain infectious diseases (e.g., HIV), and tobacco users. Samples visibly contaminated with blood present varying degrees of yellow-brownish hue. Kivlighan and colleagues (2004) have proposed a five-point Blood Contamination in Saliva Scale (BCSS) that rates contamination from one (no visible color) to five (deep, rich, dark yellow or brown). Under healthy conditions, BCSS ratings ($N = 42$) averaged 1.33 ($SE = .08$); after microinjury caused by vigorous tooth brushing, ratings averaged 2.42 ($SE = .19$).

Particulate Matter and Interfering Substances: As noted above, items placed in the mouth can influence the integrity of oral fluid samples. Food residue in the oral cavity may introduce particulate matter in samples, change salivary pH or composition, and/or contain substances (e.g., bovine hormones, active ingredients in medications, enzymes) that cross-react with assays. Accordingly, research participants should not eat or drink for 20 min prior to sample donation. In the event that they do eat in this time window, participants should rinse their mouths with water. Importantly, however, they must wait at least 10 min after rinsing before a specimen is collected to avoid artificially lowering estimates of salivary analytes. Access to food and drink should be carefully planned and scheduled when study designs involve repeated sample collections over long time periods.

Sample Handling, Transport, and Storage: Typically, once specimens are collected, they should be kept cold or frozen. Refrigeration prevents degradation of some salivary analytes and restricts the activity of protolytic enzymes and growth of bacteria. Conservatively, it is recommended that samples be kept frozen. At minimum, samples should be kept cold (on ice or refrigerated) and frozen later on the day of collection. Repeated freeze-thaw cycles should be avoided. DHEA, estradiol, and progesterone are very sensitive to freeze-thaw, whereas DNA, cortisol, testosterone, and sAA are robust (up to at least three cycles). Freeze-thaw cycles should be considered in the context of plans to aliquot and archive frozen samples for future assays. It should also be noted that some salivary analytes (e.g., neuropeptides) may require specimens be collected into prechilled storage vials (Carter et al., 2007) or treated with neuropeptidase inhibitors (e.g., EDTA, aprotinin) to minimize degradation (Dawidson, Blom, Lundeberg, Theodorsson, & Angmar-Mansson, 1997). For large-scale national surveys, investigators working in remote areas, or patients collecting samples at home, freezing and shipping these frozen samples can be logistically complex and cost-prohibitive. In such circumstances, the impact of the handling and storage conditions should be documented by pilot work, or alternative biospecimens should be considered.

Medications: As noted above, many medications can indirectly affect some analytes by reducing salivary flow (e.g., diuretics, hypotensives, antipsychotics, antihistamines, barbiturates, hallucinogens, cannabis, and alcohol). Further, the condition for which the medication is prescribed or taken may itself directly influence analyte levels or activity (Granger, Hibel, Fortunato, & Kapelewski, 2009). Few behaviorally oriented studies involving salivary analytes comprehensively document medication usage. Further, a lack of normative data coupled with wide individual variation in salivary analyte levels makes it impractical to identify improbable values due to medication use (unless the value is not physiologically plausible).

Medications that are applied intranasally, inhaled, or applied as oral topicals (e.g., teething gels) are of particular concern. Residue in the oral cavity left by these substances may change saliva composition and/or interfere with antibody-antigen binding in immunoassays. The name, dosage, and schedule of all medications taken (prescription and nonprescription) within 48 h should be recorded and used to statistically evaluate the possibility that medication use is driving analyte-outcome relationships.

Assays for Salivary Analytes: Immunoassays are the main laboratory techniques employed to assess levels and activity of salivary analytes. Most immunoassays share two basic steps. Antibodies prepared against a specific salivary analyte are coated to the bottom of a microtiter plate well; these antibodies are used to capture the target molecules. Conversely, antigens may be coated to the wells to capture antibodies present in the sample. Most modern assays employ a labeling design known as enzyme immunoassay (EIA), which uses enzymes coupled to antigens or antibodies (i.e., the enzyme conjugate). To measure salivary cortisol, for instance, antibodies to cortisol are fixed to the plastic surface of a microtiter well. The specimen and a cortisol-enzyme conjugate are added into the well and incubated. During the incubation, cortisol from the sample and the cortisol-enzyme conjugate compete for available antibody-binding sites. The well is then rinsed to remove unbound materials. Next, a substrate is added that reacts with the enzyme conjugate to produce a color. The degree of color in each reaction well is measured in units of optical density (OD). The more cortisol in the sample, the lower the amount of cortisol-conjugate that is bound to the plate, and the lower the OD in that reaction well. To determine concentrations of cortisol in the unknown samples, samples with known concentrations of cortisol (standards) are analyzed as part of each assay. Results from the standards are used to establish a calibration curve from which concentration/volume units can be interpolated from OD.

Operationalizing Individual Differences: A "basal level" is the level or activity of an analyte that represents the "stable state" of the host during a resting period. One approach to assessing "basal" levels is to sample early in the

morning before the events of the day are able to contribute variation. However, day-to-day variability differs across salivary analytes depending on a number of factors including inherent variation in the production/release of the analytes, rate of their metabolism/degradation, and their sensitivity to environmental influences. Therefore, a single time-point measure of salivary analytes (other than invariant genetic polymorphisms) is unlikely to yield meaningful insight into an individual's true "basal level." The reliability of "basal" estimates of salivary analytes can be enhanced by sampling at the same time of day across a number of days, then aggregating (by averaging assay results or physically pooling specimens) across days.

Most salivary biomarker/analyte studies have involved a reactivity/regulation paradigm; this approach uses repeated samples to evaluate time-dependent changes in analytes (i.e., cortisol, sAA) in response to (or in anticipation of) a discrete event. The number of samples collected depends on the research question and logistical and practical issues (e.g., participant's tolerance for sampling burden). The optimal design for the measurement of salivary cortisol and sAA reactivity and regulation involves a pre-pre-[task]-post-post-post sampling scheme with samples collected on arrival to the lab (after consent) immediately before the task, then again immediately, 10-, 20- and 40-min post-challenge. Although some studies have yielded consistent mean-level differences in the patterns of cortisol response following a stressful or novel event, there are more often significant individual differences in stress-reactivity. Some individuals exhibit unexpected patterns of change (or no change), as well as continuously increasing or decreasing analyte levels over time.

An important component of variability in salivary analyte levels, both within and between individuals, is the diurnal rhythm. Most salivary hormone levels (e.g., cortisol) are high in the morning, decline before noon, and then decline more slowly in the afternoon and evening hours (Nelson, 2005). By contrast, levels of sAA show the opposite pattern with low levels in the morning and higher levels in the afternoon (Nater, Rohleder, Scholtz, Ehlert, & Kirschbaum, 2007). The nonlinear nature of these patterns requires multiple sampling time points to create adequate statistical models. A typical sampling design for salivary cortisol would involve sampling immediately upon waking, 30-min post-waking, midday (around noon), in the late afternoon, and immediately prior to bed.

Many analytical techniques have been used to model individual differences in diurnal rhythm including mean levels, evaluating the awakening response, and calculating summary measures of analytes over time (e.g., area under the curve, Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003). Another approach, growth curve modeling (McArdle & Bell, 2000), has recently gained popularity for a number of reasons. Briefly, growth models allow the level and slope of the diurnal rhythm to be examined in the same model and their distinct effects on predictors can be evaluated; they minimize the impact of error or noise in measured values; and the presence of individual differences in the diurnal rhythm can be statistically tested (e.g., McArdle & Bell, 2000).

Another analytical approach, hierarchical linear modeling (HLM) allows investigators to estimate values across the day for an individual, based on several samples (Bryk & Raudenbush, 1992). Then, deviations from these expected values can be predicted from momentary states and feelings (e.g., mood states) about activities reported at that time of day. Documenting everyday events and emotions that help explain changes in analyte levels or activity across a time period of interest may strengthen causal inference when paired with samples across multiple days. For example, in studies focusing on cortisol, samples are collected approximately 20 min after a diary entry. Computerized handheld devices have made self-assessments quite feasible.

Analytes in Saliva of Interest to Behavioral Medicine: To date, most biobehavioral research has focused on a small number of salivary analytes, that is, cortisol, testosterone, DHEA, and sAA. In fact, however, the salivary proteome has recently been characterized, and includes

Salivary Biomarkers, Table 2 Salivary analytes of potential interest to biobehavioral research

Endocrine		
Aldosterone	Estradiol, Estriol, Esterone	
Androstenedione	Progesterone; 17-OH Progesterone	
Cortisol	Testosterone	
Dehydroepiandrosterone, and -sulfate	Melatonin	
Adiponectin, leptin, ghrelin	Oxytocin, Vassopressin	
Immune/inflammation		
Secretory immunoglobulin A (SIgA)	Beta-2-microglobulin (B_2M)	
Neopterin	Cytokines	
Soluble tumor necrosis factor receptors	C-reactive protein (CRP)	
Autonomic nervous system		
Alpha-amylase (sAA)	Neuropeptide Y (NPY)	
Vasoactive intestinal peptide (VIP)	Chromogranin A	
Nucleic acids		
Human genomic	mRNA	
Mitochondrial	Microbial	
Bacterial	Viral	
Antibodies specific for antigens		
Measles	Hepatitis A	Herpes simplex
Mumps	Hepatitis B	Epstein-Barr
Rubella	Hepatitis C	HIV
Pharmaceuticals/environmental chemicals		
Cotinine	Alcohol	Pesticides
Meth-, amphetamine	Lithium	Metals
Methadone	Cocaine	Opiods
Marijuana (THC)	Caffeine	Phenytoin
Bisphenol-A (BPA)	Barbituates	

Sources: Cone & Huestis (2007); Malamud & Tabak (1993); Tabak (2007); US Department of Health and Human Services (2000)

more than 1,000 analytes (Hu, Loo, & Wong, 2007). These analytes provide information about the following: (1) systemic body processes, (2) local oral biology, (3) surrogate markers of physiological activity, (4) antibodies, (4) medications and environmental exposures, and (5) genetic factors. Each category of analyte is briefly discussed below and summarized in Table 2.

The first group of analytes is present in saliva because oral fluid represents an ultra-filtrate of analytes found in the bloodstream (i.e., serum constituents). Because of high serum-saliva correlations, measuring these analytes in saliva enables investigators to make interferences about systemic physiological states. Adrenal and gonadal hormones are exemplars of this category of salivary markers (e.g., see Table 2).

Most analytes in oral fluid are produced locally in the oral cavity and are secreted from salivary glands. While individual differences in these salivary analytes may reflect systemic processes, a major contributor is local oral biology (e.g., local inflammatory processes, oral health and disease). Many salivary immune and inflammatory markers such as neopterin, beta-2-microglobulin, cytokines, and C-reactive protein (see Table 2) fall into this category. Markers in this group may be less interesting to investigators outside the fields of oral biology and oral health.

A third group of salivary analytes is produced locally by salivary glands, but the levels vary predictably with systemic physiological activation. For example, sympathetic nervous system activation affects the release of catecholamines from nerve endings, and these compounds' action

on adrenergic receptors influences the activity of the salivary glands. For instance, salivary alpha-amylase is considered a *surrogate marker* of ANS activation, as are salivary measures of neuropeptide Y and vasoactive intestinal peptide.

Antibodies to specific antigens (e.g., HIV antibodies) comprise another group of salivary analytes. Table 2 offers several additional examples. Antibodies in oral fluids reflect an individual's immunological history and pathogen/microbe exposure. Further, depending on the specific antibody measured, they may reflect local and/or systemic immune activity. To date, relatively few biobehavioral studies have taken advantage of the information provided by salivary antibodies.

A variety of pharmaceuticals, abused substances, and environmental contaminants can be quantitatively monitored in oral fluids (see Table 2). One example is bisphenol-A (BPA) – a constituent of polycarbonate plastic and epoxy resins used in water bottles, baby bottles, and food containers that may leach into food and drink. Daily BPA exposures below the US Human Exposure limit (50 ug/kg/day) have been linked to permanent changes in genitalia, early puberty, and reversal of sex differences in brain structure (Maffini, Rubin, Sonnenschein, & Soto, 2006).

A final group of analytes has been made possible by recent technical advances allowing high quantity and quality DNA to be extracted from whole saliva (Zimmerman et al., 2007). Saliva samples collected to assess individual differences in salivary analytes, and biomarkers can yield reliable and valid information about genetic polymorphism.

Conclusion and Future Directions: As the gateway to the body, the mouth senses and responds to the external world, and reflects what is happening inside the body. Oral fluids provide insight into environmental exposures and contaminants, and serve as an early warning system for disease and infection. Genetic analyses using oral fluids can help explain individual differences, predict outcomes of medical treatments, and identify polymorphisms that affect disease risk and resilience. As the number of substances that can be reliably measured in saliva increases, oral fluid may become an increasingly attractive alternative to collecting blood. The wealth of information provided by salivary analytes has the potential to greatly enrich behavioral medicine research.

Cross-References

- ▶ Adrenal Glands
- ▶ Adrenergic Activation
- ▶ Antibodies
- ▶ Autonomic Nervous System (ANS)
- ▶ Behavioral Immunology
- ▶ Biobehavioral Mechanisms
- ▶ Central Nervous System
- ▶ Chronobiology
- ▶ Circadian Rhythm
- ▶ Cortisol
- ▶ Genetics
- ▶ Public Health
- ▶ Stress

References and Readings

Adam, E. (2006). Transactions among adolescent trait and state emotion and diurnal and momentary cortisol activity in naturalistic settings. *Psychoneuroendocrinology, 31*, 664–679.

Arendorf, T. M., Bredekamp, B., Cloete, C. A., & Sauer, G. (1998). Oral manifestations of HIV infection in 600 South African patients. *Journal of Oral Pathology & Medicine, 27*, 176–179.

Beall, C. M., Worthman, C. M., Stallings, J., Strohl, K. P., Brittenham, G. M., & Barragan, M. (1992). Salivary testosterone concentration of Aymara men native to 3,600 m. *Annals of Human Biology, 19*, 67–78.

Beltzer, E. K., Fortunato, C. K., Guaderrama, M. M., Peckins, M. K., Garramone, B. M., & Granger, D. A. (2010). Salivary flow on alpha-amylase: Collection technique, duration, and oral fluid type. *Physiology and Behavior, 101*, 289–296.

Booth, A., Johnson, D. R., Granger, D. A., Crouter, A. C., & McHale, S. (2003). Testosterone and child and adolescent adjustment: The moderating role of parent-child relationships. *Developmental Psychology, 39*, 85–98.

Brandtzaeg, P. (2007). Do salivary antibodies reliably reflect both mucosal and systemic immunity? *Annals of the New York Academy of Science, 1098*, 288–311.

Bryk, A. S., & Raudenbush, S. W. (1992). *Hierarchical linear models*. Newbury Park, CA: Sage.

Carter, C. S., Pournajafi-Nazarloo, H., Kramer, K. M., Ziegler, T. E., White-Traut, R., Bello, D., & Schwertz, D. (2007). Oxytocin: Behavioral associations and potential as a salivary biomarker. *Annals of the New York Academy of Science, 1098*, 312–322.

Chard, T. (1990). *An introduction to radioimmunoassay and related techniques* (4th ed.). Amsterdam, NY: Elsevier.

Cone, E. J., & Huestis, M. A. (2007). Interpretation of oral fluid tests for drugs of abuse. *Annals of the New York Academy of Science, 1098*, 51–103.

Dabbs, J. M., Jr. (1991). Salivary testosterone measurements: Collecting, storing, and mailing saliva samples. *Physiology and Behavior, 49*, 815–817.

Dawidson, I., Blom, M., Lundeberg, T., Theodorsson, E., & Angmar-Mansson, B. (1997). Neuropeptides in the saliva of healthy subjects. *Life Sciences, 60*, 269–278.

Flinn, M. V., & England, B. G. (1995). Childhood stress and family environment. *Current Anthropology, 36*, 854–866.

Granger, D. A., Hibel, L. C., Fortunato, C. K., & Kapelewski, C. H. (2009). Medication effects on salivary cortisol: Tactics and strategy to minimize impact in behavioral and developmental science. *Psychoneuroendocrinology, 34*, 1437–1448.

Granger, D. A., Kivlighan, K. T., Fortunato, C., Harmon, A. G., Hibel, L. C., Schwartz, E. B., & Whembolua, G.-L. (2007). Integration of salivary biomarkers into developmental and behaviorally-oriented research: Problems and solutions for collecting specimens. *Physiology and Behavior, 92*, 583–590.

Groschl, M., & Rauh, M. (2006). Influence of commercial collection devices for saliva on the reliability of salivary steroids analysis. *Steroids, 71*, 1097–1100.

Gunnar, M., Mangelsdorf, S., Larson, M., & Hertsgaard, L. (1989). Attachment, temperament, and adrenocortical activity in infancy: A study of psychoendocrine regulation. *Developmental Psychology, 3*, 355–363.

Gunnar, M. R., & Vasquez, D. (2001). Low cortisol and a flattening of the expected daytime rhythm: potential indices of risk in human development. *Development and Psychopathology, 13*, 515–538.

Haeckel, R., & Bucklitsch, I. (1987). Procedures for saliva sampling. *Journal of Clinical Chemistry and Biochemistry, 25*, 199–204.

Harmon, A. G., Hibel, L. C., Rumyansteva, O., & Granger, D. A. (2007). Measuring salivary cortisol in studies of child development: Watch out–what goes in may not come out of saliva collection devices. *Developmental Psychobiology, 49*, 495–500.

Harmon, A. G., Towe, N. R., Fortunato, C. K., & Granger, D. A. (2008). Differences in saliva collection location and disparities in baseline and diurnal rhythms of alpha-amylase: A preliminary note of caution. *Hormones and Behavior, 54*, 592–596.

Hellhammer, J., Fries, E., Schweusthal, O. W., Schlotz, W., Stone, A. A., & Hagemann, D. (2007). Several daily measurements are necessary to reliably assess the cortisol rise after awakening: State- and trait components. *Psychoneuroendocrinology, 32*, 80–86.

Horvat-Gordon, M., Granger, D. A., Schwartz, E. B., Nelson, V., & Kivlighan, K. T. (2005). Oxytocin is not a valid biomarker when measured in saliva by immunoassay. *Physiology and Behavior, 16*, 445–448.

Hu, S., Loo, J. A., & Wong, D. T. (2007). Human saliva proteome analysis. *Annals of the New York Academy of Science, 1098*, 323–329.

Kivlighan, K. T., Granger, D. A., Schwartz, E. B., Nelson, V., Curran, M., & Shirtcliff, E. A. (2004). Quantifying blood leakage into the oral mucosa and its effects on the measurement of cortisol, dehydroepiandrosterone, and testosterone in saliva. *Hormones and Behavior, 46*, 39–46.

Kugler, J., Hess, M., & Haake, D. (1992). Secretion of salivary immunoglobulin A in relation to age, saliva flow, mood states, secretion of albumin, cortisol, and catecholamines in saliva. *Journal of Clinical Immunology, 12*, 45–49.

Maffini, M. V., Rubin, B. S., Sonnenschein, C., & Soto, A. M. (2006). Endocrine disruptors and reproductive health: The case of bisphenol-A. *Molecular and Cellular Endocrinology, 254*, 179–186.

Malamud, D., & Tabak, L. (1993). Saliva as a diagnostic fluid. *Annals of the New York Academy of Sciences, 694*, 216–233.

McArdle, J. J., & Bell, R. Q. (2000). An introduction to latent growth models for developmental data analysis. In T. D. Little, K. U. Schnabel, & J. Baumert (Eds.), *Modeling longitudinal and multiple-group data: Practical issues, applied approaches, and specific examples* (pp. 69–107). Hillsdale, NJ: Lawrence Erlbaum Associates.

McArdle, J. J., & Nesselroade, J. (1994). Using multivariate data to structure developmental change. In S. H. Cohen & H. W. Reese (Eds.), *Life-span developmental psychology* (pp. 223–267). Hillsdale, NJ: Lawrence Erlbaum Associates.

Melnick, R., Lucier, G., Wolfe, M., Hall, R., Stancel, G., Prins, G., Gallo, M., Reuhl, K., Ho, S. M., Brown, T., Moore, J., Leakey, J., Haseman, J., & Kohn, M. (2002). Summary of the national toxicology program's report of the endocrine disruptors low-dose peer review. *Environmental Health Perspectives, 110*, 427–431.

Nater, U. M., Rohleder, N., Schlotz, W., Ehlert, U., & Kirschbaum, C. (2007). Determinants of the diurnal course of salivary alpha-amylase. *Psychoneuroendocrinology, 32*, 392–401.

Nelson, R. J. (2005). *An introduction to behavioral endocrinology*. Sunderland, MA: Sinauer Associates.

Nemoda, Z., Horvat-Gordon, M., Fortunato, C. K., Beltzer, E. K., Scholl, J. L., & Granger, D. A. (2011). Assessing genetic polymorphisms using DNA extracted from cells present in saliva samples. *BMC Med Res Methodol, 11*, 170.

Nieuw Amerongen, A. V., Ligtenberg, A. J. M., & Veerman, E. C. I. (2007). Implications for diagnostics

in the biochemistry and physiology of saliva. *Annals of the New York Academy of Science, 1098*, 1–6.
Paster, B. J., Boches, S. K., Galvin, J. L., Ericson, R. E., Lau, C. N., Levanos, V. A., Sahasrabudhe, A., & Dewhirst, F. E. (2001). Bacterial diversity in human subgingival plaque. *Journal of Bacteriology, 183*, 3770–3783.
Pruessner, J., Kirschbaum, C., Meinlschmid, G., & Hellhammer, D. H. (2003). Two formulas for computation of the area under the curve represent measures of total hormone concentration versus time-dependent change. *Psychoneuroendocrinology, 28*, 916–931.
Raff, H., Homar, P. J., & Skoner, D. P. (2003). New enzyme immunoassay for salivary cortisol. *Clinical Chemistry, 49*, 203–204.
Rees, T. D. (1992). Oral effects of drug abuse. *Critical Reviews in Oral Biology and Medicine, 3*, 163–184.
Reibel, J. (2003). Tobacco and oral diseases. Update on the evidence, with recommendations. *Medical Principles and Practice, 12*(Suppl. 1), 22–32.
Santavirta, N., Konttinen, Y. T., Tornwall, J., Segerberg, M., Santavirta, S., Matucci-Cerinic, M., & Bjorvell, H. (1997). Neuropeptides of the autonomic nervous system in Sjogren's syndrome. *Annals of Rheumatoid Disease, 56*, 737–740.
Scannapieco, F. A., Papandonatos, G. D., & Dunford, R. G. (1998). Associations between oral conditions and respiratory disease in a national sample survey population. *Annals of Periodontology, 3*, 251–256.
Schwartz, E. B., Granger, D. A., Susman, E. J., Gunnar, M. R., & Laird, B. (1998). Assessing salivary cortisol in studies of child development. *Child Development, 69*, 1503–1513.
Shirtcliff, E. A., Granger, D. A., Schwartz, E. B., & Curran, M. J. (2001). Use of salivary biomarkers in biobehavioral research: Cotton based sample collection methods can interfere with salivary immunoassay results. *Psychoneuroendocrinology, 26*, 165–173.
Smyth, J. M., Ockenfels, M. C., Gorin, A. A., Cately, D., Porter, L. S., Kirschbaum, C., Hellhammer, D. H., & Stone, A. A. (1997). Individual differences in the diurnal cycle of cortisol. *Psychoneuroendocrinology, 22*, 89–105.
Sreebny, L. M., & Schwartz, S. S. (1997). A reference guide to drugs and dry mouth – 2nd edition. *Gerodontology, 14*, 33–47.
Stone, A. A., Broderick, J. E., Schwartz, J. E., Shiffman, S., Litcher-Kelly, L., & Calvanese, P. (2003). Intensive momentary reporting of pain with an electronic diary: Reactivity, compliance, and patient satisfaction. *Pain, 104*, 343–351.
Tabak, L. A. (2007). Point-of-care diagnostics enter the mouth. *Annals of the New York Academy of Science, 1098*, 7–14.
U.S. Department of Health and Human Services. (2000). *Oral health in America: A report of the surgeon general*. Rockville, MD: U.S. Department of Health and Human Services, National Institute of Dental and Craniofacial Research, National Institutes of Health.
Veerman, E. C. I., Van Den Keijbus, P. A. M., Vissink, A., & Nieuw Amerongen, A. V. (1996). Human glandular saliva: Their separate collection and analysis. *European Journal of Oral Science, 104*, 346–352.
Zimmerman, B. G., Park, N. J., & Wong, D. T. (2007). Genomic targets in saliva. *Annals of the New York Academy of Science, 1098*, 184–191.

Salt, Intake

Kelly Flannery
School of Nursing, University of Maryland Baltimore, Baltimore, MD, USA

Synonyms

Sodium; Sodium chloride

Definition

Salt is a dietary element made up of sodium and chlorine (U.S. National Library of Medicine & National Institutes of Health, 2011a).

Description

A majority (90%) of sodium consumed comes from salt (Centers for Disease Control & Prevention, 2011). The body needs a small amount of sodium for fluid regulation, nerve impulse transmission, and muscle function. The kidneys are responsible for retaining sodium (if body stores are low) or excreting sodium through urine (if body stores are too high). However, if the kidneys do not excrete enough sodium the excess sodium will accumulate in the blood. This can lead to high blood pressure, from an increase in fluid volume in the arteries, ultimately putting additional stress on the heart (Mayo Clinic, 2011a; U.S. National Library of Medicine & National Institutes of Health, 2011a).

Recommendations

There are no specific sodium recommendations for those under 18 years of age. However, it is suggested these individuals consume a moderate intake of sodium (U.S. National Library of Medicine & National Institutes of Health, 2011b). There are two different recommendations for adult sodium intake based on specific characteristics. African-Americans, people with diabetes, people with high blood pressure, people with chronic kidney disease, and anyone over the age of 51 are more susceptible to blood pressure elevation from sodium; therefore, these individuals should limit sodium intake to less than 1,500 mg of sodium a day. The remaining American population should consume less than 2,300 mg of sodium a day. However, the general population will benefit by reducing their sodium to less than 1,500 mg a day (U.S. Department of Agriculture, & U.S. Department of Health and Human Services, 2010). In fact, the American Heart Association recommends the general public reduce their sodium intake to no more than 1,500 mg per day (American Heart Association Presidential Advisory, 2011). In addition, those with certain diseases (e.g., cirrhosis and congestive heart failure) may be recommended lower sodium intake levels by their primary care providers (U.S. National Library of Medicine & National Institutes of Health, 2011b).

A half of a teaspoon of salt is approximately 1,200 mg of sodium and one teaspoon of salt is approximately 2,300 mg of sodium (American Heart Association, 2011). More than 85% of Americans consume 2,300 mg of sodium or more a day; the average intake of sodium for Americans over 2 years of age is 3,400 mg per day (Centers for Disease Control & Prevention, 2011; U.S. Department of Agriculture, & U.S. Department of Health and Human Services, 2010). Diets high in sodium have been associated with an increased risk for high blood pressure, heart disease, and stroke (American Heart Association, 2011). Generally, when salt intake is reduced it only takes a few weeks for blood pressure to decrease (Centers for Disease Control & Prevention, 2011).

Identifying Sources of Sodium

Most foods naturally contain sodium (U.S. National Library of Medicine & National Institutes of Health, 2011b); however, this form of sodium only accounts for about 12% of daily sodium intake. An additional 11% of sodium intake comes from cooking at home and adding salt while eating. A majority (77%) of the sodium Americans consume comes from processed foods, foods bought at stores, packaged foods, and foods cooked at restaurants (Centers for Disease Control & Prevention, 2010). Sodium is added to foods to act as a preservative, cure meat, retain moisture, and enhance color and flavor (American Heart Association, 2011; U.S. Department of Agriculture, & U.S. Department of Health and Human Services, 2010). When food and beverages were grouped in 96 categories, the top six categories that contributed the most sodium to Americans' diets included yeast breads, chicken and chicken mixed dishes, pizza, pasta and pasta dishes, cold cuts, and condiments (National Cancer Institute, 2010).

Reading food labels is important for determining sodium intake because milligrams of sodium in food can vary even for the same type of food. For instance, a slice of frozen pizza can range from 450 to 1,200 mg of sodium (Centers for Disease Control & Prevention, 2011). However, caution should be used reading the %DV (daily value) on the food label because the percentage is based on 2,400 mg, which is 100 or 900 mg higher than the recommended daily sodium intake depending on recommended group (U.S. Department of Agriculture, & U.S. Department of Health and Human Services, 2010; U.S. Food and Drug Administration, 2011). Food packaging messages can be confusing (Centers for Disease Control & Prevention, 2011). For example, a package message titled unsalted or no salt added simply means no salt was added while processing the food; yet, reading the label is important because some of the ingredients may contain sodium (Mayo Clinic, 2011b). Additionally, looking at the ingredients list can help determine if sodium was added. Sodium is sometimes called different names; some examples include baking soda, monosodium

glutamate, and sodium nitrite (U.S. National Library of Medicine & National Institutes of Health, 2011b; Mayo Clinic, 2011b).

Methods for Reducing Sodium

Some methods for reducing the amount of sodium consumed can include (Mayo Clinic, 2011b; National Heart, Lung and Blood Institute, n.d.; National Library of Medicine & National Institutes of Health, 2010; U.S. Department of Agriculture, & U.S. Department of Health and Human Services, 2010; American Heart Association, 2011):

- Following specific heart-healthy diets (e.g., dietary approaches to stop hypertension, which is also called the DASH diet)
- Eating fresh foods
- Using food labels to purchase items low in sodium
- Ordering lower sodium items when eating out
- Using healthy salt substitutes to replace salt

Cross-References

► Hypertension

References and Readings

American Heart Association. (2011). *Sodium (salt or sodium chloride)*. Retrieved April 15, 2011, from http://www.heart.org/HEARTORG/GettingHealthy/NutritionCenter/HealthyDietGoals/Sodium-Salt-or-Sodium-Chloride_UCM_303290_Article.jsp

American Heart Association Presidential Advisory. (2011). *Population-wide reduction in salt consumption recommended*. Retrieved April 15, 2011, from http://www.newsroom.heart.org/index.php?s=43&item=1237

Centers for Disease Control & Prevention. (2010). *Sodium and food sources*. Retrieved April 15, 2011, from http://www.cdc.gov/salt/food.htm

Centers for Disease Control & Prevention. (2011). *Sodium fact sheet*. Retrieved April 15, 2011, from http://www.cdc.gov/dhdsp/data_statistics/fact_sheets/fs_sodium.htm

Mayo Clinic. (2011a). *Sodium: How to tame your salt habit now*. Retrieved April 15, 2011, from http://www.mayoclinic.com/health/sodium/NU00284

Mayo Clinic. (2011b). *Sodium: How to tame your salt habit now (continued)*. Retrieved April 15, 2011, from http://www.mayoclinic.com/health/sodium/NU00284/NSECTIONGROUP=2

National Cancer Institute. (2010). Sources of Sodium Among the US Population, 2005-06. Risk Factor Monitoring and Methods Branch Website. *Applied Research Program*. Retrieved March 22, 2012, from http://riskfactor.cancer.gov/diet/foodsources/sodium/

National Library of Medicine, & National Institutes of Health. (2010). Tasty stand-ins for salt. *NIH Medline Plus, 5*, 15.

National Heart, Lung and Blood Institute. (n.d.). *Your guide to lowering high blood pressure: Healthy eating*. Retrieved April 15, 2011, from http://www.nhlbi.nih.gov/hbp/prevent/h_eating/h_eating.htm

U.S. Department of Agriculture, & U.S. Department of Health and Human Services. (2010). *Dietary guidelines for Americans 2010* (7th ed.). Washington, DC: U.S. Government Printing Office.

U.S. Food and Drug Administration. (2011). *How to understand and use the nutrition facts label*. Retrieved April 15, 2011, from http://www.fda.gov/food/labeling-nutrition/consumerinformation/ucm078889.htm

U.S. National Library of Medicine, & National Institutes of Health. (2011a). *Dietary sodium*. Retrieved April 15, 2011, from http://www.nlm.nih.gov/medlineplus/dietarysodium.html

U.S. National Library of Medicine, & National Institutes of Health. (2011b). *Sodium in diet*. Retrieved April 15, 2011, from http://www.nlm.nih.gov/medlineplus/ency/article/002415.htm

Salutogenesis

Sefik Tagay
Department of Psychosomatic Medicine and Psychotherapy, University of Duisburg-Essen, Essen, North Rhine-Westphalia, Germany

Synonyms

Hardiness; Resilience; Self-efficacy; Sense of coherence

Definition

The medical sociologist Aaron Antonovsky (1923–1994) introduced the term "salutogenesis" which derives from the Latin "salus = health" and the Greek "genesis = origin." Antonovsky was mainly interested in the question of what creates

and what sustains health rather than explaining the causes of disease in the pathogenic direction (Antonovsky, 1979, 1987, 1993). In his salutogenetic model, he described health as a continuum between total ease (health) and total disease rather than a health-disease dichotomy. Therefore, his most important research question was: What causes health (salutogenesis)? (rather than what are the reasons for disease (pathogenesis)). The core concepts of salutogenesis show great conceptual overlap with the theory of "hardy personality" (Kobasa, 1979, 1982), the theory of "self-efficacy" (Bandura, 1977), and with the theory of "resilience" (Werner & Smith, 1982).

The central terms of the salutogenetic theory are sense of coherence (SOC) and general resistance resources (GRRs). Antonovsky postulates that the status of health and well-being depends on these personal and environmental resources (Antonovsky, 1979, 1987).

Description

Sense of Coherence (SOC)

Sense of coherence explains why humans in stressful situations stay well and are even able to improve their physical, mental, and social well-being. Antonovsky (1993) suggested that SOC depicts a stable and long-lasting way of looking at the world. He postulated that SOC is mainly formed in the first three decades of life and then becomes relatively stable.

Antonovsky defined SOC as a:

"Global orientation that expresses the extent to which one has a pervasive, enduring though dynamic feeling of confidence that (1) the stimuli, deriving from one's internal and external environments in the course of living are structured, predictable, and explicable; (2) the resources are available to one to meet the demands posed by these stimuli; and (3) these demands are challenges, worthy of investment and engagement" (Antonovsky, 1987, p.19).

The SOC consists of three dimensions (Antonovsky, 1987) as follows:

1. *Comprehensibility (cognitive component)*: The internal and external environments are interpreted as understandable, consistent, structured, and predictable.
2. *Manageability (behavioral component)*: Individuals consider resources to be personally available to help them cope adequately with demands or problems.
3. *Meaningfulness (motivational component)*: This dimension refers to the extent to which a person feels that life makes sense, and that problems and demands are worth investing energy in. Additionally, it determines whether a situation is appraised as challenging, and whether it is worth making commitments and investments in order to cope with it.

According to Antonovsky (1987) the third component is the most important aspect of SOC.

General Resistance Resources (GRRs)

The general resistance resources (GRRs) are biological and psychosocial factors that make it easier for people to perceive their lives as predictable, controllable, and understandable. Typical GRRs are money, intelligence, social support, self-esteem, ego-strength, healthy behavior, traditions, and culture. These types of resources can help the person to deal in a better way with the challenges of life. In general, the GRRs lead to life experiences that promote a better SOC (Antonovsky, 1987).

Measuring Sense of Coherence

With the Sense of Coherence (SOC) Scale, there is only one instrument that measures sense of coherence worldwide. Antonovsky (1987) developed the SOC as a self-report questionnaire with Likert-type items; higher scores indicate a better SOC. This instrument exists in a long form (SOC-29) and in a short form (SOC-13). In the long form, 11 items refer to "comprehensibility," 8 items refer to "meaningfulness," and 10 items refer to "manageability." The SOC scale is a reliable, valid, and cross-culturally applicable instrument. SOC seems to be a multidimensional concept rather than a unidimensional one (Eriksson & Lindström, 2005). By 2007, the SOC questionnaire has been used in at least 44 languages all over the world (Singer & Brähler, 2007).

Sense of Coherence and Health (Empirical Evidence)

Empirical evidence shows a strong association between SOC and mental health. A large number of studies consistently reveal a negative relationship of SOC with depression, anxiety, and posttraumatic symptoms (Antonovsky, 1993; Eriksson & Lindström, 2007; Tagay, Erim, Brähler, & Senf, 2006; Tagay, Mewes, Brähler, & Senf, 2009). In a recent review, Eriksson and Lindström (2007) synthesized empirical findings on SOC and examined its capacity to explain health and its dimensions. SOC was strongly related to perceived health. The stronger the SOC, the better the perceived health in general. This relation was manifested in study populations regardless of age, sex, ethnicity, nationality, and study design. Therefore, numerous authors assert that there is substantial empirical support for the idea that SOC promotes health. A strong SOC is associated with successful coping with the inevitable stressors that individuals encounter in the course of their daily lives, and therefore, with better outcomes (Antonovsky, 1993; Eriksson & Lindström, 2007). All in all, SOC seems to have a main, moderating, or mediating role in the explanation of health, and it seems to be able to predict health (Schnyder, Büchi, Mörgeli, Senky, & Klaghofer, 1999; Tagay et al., 2011).

Cross-References

- ▶ Coping
- ▶ Health
- ▶ Optimism
- ▶ Self-Esteem
- ▶ Stress
- ▶ Well-Being

References and Readings

Antonovsky, A. (1979). *Health, stress, and coping*. San Francisco/Washington/London: Jossey-Bass.

Antonovsky, A. (1987). *Unraveling the mystery of health*. San Francisco/London: Jossey-Bass.

Antonovsky, A. (1993). The structure and properties of the sense of coherence scale. *Social Science & Medicine, 36*, 725–733.

Bandura, A. (1977). Self-efficacy: Toward a unifying theory of behavioral change. *Psychological Review, 84*, 191–215.

Eriksson, M., & Lindström, B. (2005). Validity of Antonovsky's sense of coherence scale. *Journal of Epidemiology and Community Health, 59*, 460–466.

Eriksson, M., & Lindström, B. (2007). Antonovsky's sense of coherence scale and it's relation with quality of life – a systematic review. *Journal of Epidemiology and Community Health, 61*, 938–944.

Kobasa, S. C. (1979). Stressful life events, personality, and health. *Journal of Personality and Social Psychology, 37*, 1–11.

Kobasa, S. C. (1982). The hardy personality: Toward a social psychology of stress and health. In G. S. Sanders & J. Suls (Eds.), *Social psychology of health and illness* (pp. 3–32). Hillsdale, NJ: Erlbaum.

Schnyder, U., Büchi, S., Mörgeli, H., Senky, T., & Klaghofer, R. (1999). Sense of coherence-a mediator between disability and handicap? *Psychotherapy and Psychosomatics, 68*, 102–110.

Singer, S., & Brähler, E. (2007). *Die "Sense of coherence scale". Testhandbuch zur deutschen Version*. Göttingen, LS: Vandenhoeck & Ruprecht.

Tagay, S., Düllmann, S., Schlegl, S., Nater-Mewes, R., Repic, N., Hampke, Ch, Brähler, E., Gerlach, G., & Senf, W. (2011). Effects of inpatient treatment on eating disorder symptoms, health-related quality of life and personal resources in anorexia and bulimia nervosa. *Psychotherapie, Psychosomatik, Medizinische Psychologie, 61*(7), 319–327.

Tagay, S., Erim, Y., Brähler, E., & Senf, W. (2006). Religiosity and sense of coherence – protective factors of mental health and well-being? *Zeitschrift für Medizinische Psychologie, 4*, 165–171.

Tagay, S., Mewes, R., Brähler, E., & Senf, W. (2009). Sense of coherence in female patients with bulimia nervosa: A protective factor of mental health? *Psychiatrische Praxis, 36*, 30–34.

Werner, E., & Smith, R. (1982). *Vulnerable but invincible. A longitudinal study of resilient children and youth*. New York: McGraw Hill.

Sample Size Estimation

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Sample-size calculation; Sample-size determination; Study size

Definition

Sample-size estimation is the process by which a researcher decides how many subjects to include in a given clinical trial. Sample-size estimation is a critical part of the design of clinical trials, and, like all design issues, this must be addressed in the study protocol before the trial commences.

Description

Many sources use the terms sample-size determination or sample-size calculation when discussing this issue. The term sample-size estimation emphasizes that deciding on the sample size that will be employed in a clinical trial is a process of estimation that involves both statistical and clinical informed judgment and not a process of simply calculating the "right" answer. It is true that mathematical calculations are made in this process, and, for a given set of values that are placed into the appropriate formula in any given circumstance, a precise answer will be given. However, the values that are placed into the formula are chosen by the researcher.

Some of the values that need to be entered into the formula are typically chosen from a standard set of possibilities, with the researcher deciding which of several generally acceptable values is best suited for the intentions of a given trial. Other values are estimates based on data that may be available in existing literature or may have been collected in an earlier trial. These include the estimated treatment effect and the variability associated with the estimated treatment effect.

The likelihood of a successful outcome (at least from the point of view that "success" means obtaining a statistically significant result) can be increased by increasing the sample size. When designing a study, the researcher wants to ensure that a large enough sample size is chosen to be able to detect an important difference that does in fact exist. It is certainly possible that a trial can fail to demonstrate such a difference simply because the sample size chosen was too small. Therefore, it might appear reasonable to think that a very big sample size is a good idea. However, increasing the sample size increases the expenses, difficulties and overall length of a trial. Somewhere, for each researcher and each study, an acceptable sample size needs to be chosen that balances the likelihood of a statistically significant result with the cost and time involved in conducting the clinical trial.

Several variables need to be considered in the process of sample-size estimation. The values of these variables in any given case can be chosen by the researcher based on several considerations. Relevant terms include:

- Type I errors and Type II errors. A Type I error occurs when a significant result is "found" when it does not really exist, and a Type II error occurs when one fails to find a significant difference that actually exists.
- The probability of making a Type I error, α. This is also the level of statistical significance chosen, typically 0.05, but it is possible to choose 0.01 or even more conservative values.
- The probability of making a Type II error, β. A probability value must be between 0 and 1: therefore, β will be between 0 and 1.
- Power, calculated as $1 - \beta$. Since the probability represented by β will be between 0 and 1, power will also be between 0 and 1 since it is defined as $1 - \beta$. The power of a statistical test is the probability that the null hypothesis is rejected when it is indeed false. Since rejecting the null hypothesis when it is false is extremely desirable, it is generally regarded that the power of a study should be as great as practically feasible.

Sample-size estimation can be performed for any study design. In each case, the respective formula will be used to estimate the sample size required. For the formula used in the type of study design discussed in some entries in the Methodology section (i.e., a comparison of a new behavioral medicine treatment or intervention with an existing one), each of the variables we have discussed will have certain influences on the sample size, N, that will be given by the formula. These influences, i.e., their relationships with

N given that all of the others remain the same, can be summarized as follows:

- The smaller the chosen value of α, the larger the value of N that will be given.
- The smaller the chosen value of β, the larger the value of N that will be given. This is because power is defined as 1 − β. As β decreases, power increases; as power increases, the larger the value of N that will be given.
- The larger the standardized effect size, the smaller the value of N that will be given. The standardized treatment effect is the estimated treatment effect divided by the variability associated with it.

Cross-References

- ▶ False-Negative Error
- ▶ False-Positive Error
- ▶ Probability
- ▶ Study Protocol

Sample-Size Calculation

- ▶ Sample Size Estimation

Sample-Size Determination

- ▶ Sample Size Estimation

Sarcopenia

Oliver J. Wilson and Anton J. M. Wagenmakers
School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Anabolic resistance; Disuse atrophy; Skeletal muscle atrophy

Definition

Sarcopenia is the progressive involuntary loss of skeletal muscle mass, strength, and function with age. Sarcopenia is derived from Greek "sarx" and "penia," literally meaning "loss of flesh."

Description

The global population aged 60 years or older is expected to double from 600 million in the year 2000 to 1.2 billion by 2025 and reaching nearly 2 billion by 2050 (United Nations, Department of Economic & Social Affairs, 2002). In the UK, the number of people aged >65 years will nearly double 61% from 10 million in 2010 to 19 million by 2050 (Office of National Statistics, 2012). However, a healthy life expectancy does not follow in parallel, suggesting that the global population is living longer with detrimental aging-related diseases such as the metabolic syndrome, type 2 diabetes, cardiovascular disease, and inflammatory diseases (WHO, 2008). A common result of the aging process is the loss of an independent lifestyle. Although the causes are varied, an important contributor is the loss of mobility and the associated risk of accidental falls. At present, 30% of people aged >65 years fall each year in the UK, and the estimated annual cost to the National Health Service in the UK is £1 billion. Consequently, meeting the future demand and associated costs of aging will prove challenging for health-care provision.

A key contributor to the increased risk of accidental (fatal) falls, dependency, and self-reported disability is sarcopenia, the progressive involuntary loss of skeletal muscle mass, strength, and function with age. Sarcopenia is derived from Greek "sarx" and "penia," literally meaning "loss of flesh." It is estimated that 20% of 70-year-olds and 50% of people aged 80 years and over have sarcopenia (Narici & Maffulli, 2010). The loss of muscle mass and strength with age is evident even in those who engage in regular aerobic or resistance exercise. Sarcopenia is more notable in lower than upper limbs and is generally more prevalent in women than men.

Sarcopenia, Table 1 Summary of the potential mechanisms underpinning sarcopenia

Whole body
Reduced physical activity and muscle disuse
Loss of motor neurons
Reduced growth hormone and insulin-like growth factor-I production
Chronic pro-inflammatory state
Increased glucocorticoid production and receptor activity
Malnutrition
Muscular
Reduced number and proliferative capacity of skeletal muscle satellite cells
Mitochondrial DNA mutations and apoptosis
Increased intracellular production of glucocorticoids
Impaired insulin mediated increase in (micro)vascular blood flow
Blunting of the effects of insulin and amino acids on muscle protein synthesis
Poor transcriptional responses of muscle to exercise and nutrition

It is also highly associated with osteoporosis and hip fractures in women. Consequently, the higher life expectancy in women coupled with the higher prevalence of sarcopenia means women are more likely than men to spend their final years of life receiving institutionalized care (Narici & Maffulli). The genesis of sarcopenia can be attributable to physical inactivity, inadequate nutrition, and a chronic pro-inflammatory state among other factors (Table 1).

Sarcopenia can be masked by weight stability due to a concomitant increase in fat mass. However, the prolonged accumulation of fat mass can lead to obesity, resulting in "sarcopenic obesity." This presents further complications as excess fat mass which, in association with chronic low-level pro-inflammatory cytokines, can result in the development of insulin resistance in a variety of tissues. These include the endothelium of the microvasculature and skeletal muscle, leading to periods of hyperglycemia, hyperlipidemia, and the development of non-insulin-dependent diabetes mellitus (type 2 diabetes) and cardiovascular disease (Wagenmakers, van Riel, Frenneaux, & Stewart, 2006).

Aging Muscle

In young lean adults, skeletal muscle mass accounts for nearly 60% of total fat-free body mass but accounts for just 45% in elderly individuals. The loss of muscle cross-sectional area is ~40% between 20 and 60 years of age, with men demonstrating greater decreases in muscle mass than women. Consequently, there is a corresponding reduction in knee extensor muscle strength between 20% and 40% in both groups and up to 50% in those aged >90 years. There is also a loss of muscle power, declining at a greater rate than the loss of muscle strength.

The loss of muscle mass is linked to whole muscle and individual muscle fiber atrophy. Aging slow-twitch type I fibers atrophy in size to about 75% of younger individuals, but fast twitch type II fibers but fast twitch type II fibres atrophy in size to about 43% of their younger counterparts (Andersen, 2003). Further changes include the infiltration of fat and connective tissue within the muscle, fiber necrosis, fiber-type grouping, and a reduction in type 2 fiber satellite cell content (Koopman & van Loon, 2009).

A reduction in the oxidative capacity of skeletal muscle is also observed with age and is likely attributable to a reduction in mitochondrial content and/or function. This results in poor endurance and increased fatigability, compromising the ability to live an independent lifestyle. Poor oxidative capacity is also mechanistically linked to the development of insulin resistance and type 2 diabetes.

Protein Metabolism in the Elderly

The maintenance of muscle mass depends on the balance between muscle protein synthesis (MPS) via anabolic stimuli (feeding, muscle contraction, anabolic hormones) and muscle protein breakdown (MPB) via catabolic stimuli (fasting, disuse, pro-inflammatory cytokines). Where muscle protein synthesis exceeds protein breakdown, hypertrophy occurs, whereas the reverse is true for muscle atrophy. Sarcopenia is therefore associated with a removal of

anabolic stimuli, an increase in catabolic stimuli, or a combination of the two.

Early studies suggested basal MPS rates were lower in the elderly compared with young, but when the results are extrapolated across a year, a substantial loss in muscle mass will occur that far exceeds the modest decline observed with healthy aging. It is now generally accepted that in healthy physically active elderly individuals, rates of basal MPS and MPB equal that of younger individuals (Koopman & van Loon, 2009). Instead, sarcopenia might be the result of a blunted response to anabolic stimuli, termed "anabolic resistance" (Rennie et al., 2010).

The ingestion of amino acids and/or protein increases plasma insulin concentrations which independently and additively stimulates MPS and inhibits MPB, leading to a positive net protein balance. However, some studies suggest the anabolic response to ingested essential amino acids is blunted in the elderly compared with young controls (Rennie et al., 2010). Interestingly, some studies have suggested that by increasing the leucine content of the meal, it is possible to increase elderly MPS further (Rennie, 2005).

The current recommended daily allowance (RDA) for habitual protein intake in the elderly is 0.8 g/kg body mass per day, but this might not be sufficient for optimizing exercise-induced gains in muscle mass. It has been suggested that increasing the daily dietary intake of protein (and thus amino acid availability) beyond the RDA might overcome the anabolic resistance and further increase exercise-induced gains in muscle mass. However, when daily protein intake was set at either 0.9 g/kg body mass (adequate) or 1.2 g/kg body mass (moderately high) during a 3-month resistance training study, the gains in exercise-induced muscle mass did not differ between the high and low dose (Koopman & van Loon, 2009).

At the molecular level, the anabolic resistance to plasma insulin and amino acid availability might be attributed to an age-associated reduction in the content, phosphorylation, and activity of key insulin-signaling proteins within the Akt-mTOR pathway (Rennie, 2005). These defects may contribute toward sarcopenia by impairing the sensing and transduction of insulin and/or amino acid-dependent signaling, thereby limiting the stimulation of MPS and inhibition of MPB.

Resistance Training in the Elderly

Resistance exercise training is a potent stimulator of MPS, leading to increased muscle mass and strength in the young and the elderly, frail elderly, and in older individuals presenting with comorbidities. Indeed, the elderly respond to resistance programs training with similar relative gains in limb muscle mass, size, strength, and power as the young. Resistance training therefore offers an effective strategy to counteract sarcopenia and improve functional muscle capacity.

The American College of Sports Medicine (ACSM) Position Stand for Exercise and Physical Activity in Older Adults (Chodzko-Zajko et al., 2009) recommends progressive resistance training at least 2 days per week using 8–10 exercises involving the major muscle groups over 8–10 repetitions. However, even a single weekly bout of resistance training has been shown to increase muscle strength in elderly individuals. After a 3-month resistance training program, an increase in muscle strength of more than 100% has been reported in the elderly with similar relative improvements in muscle strength observed in men aged 90 years and older. This is associated with an increase in muscle cross-sectional area attributable to hypertrophy of type I and type II fibers (Burton & Sumukadas, 2010).

Gains in muscular endurance ranging from 34% to 200% have also been reported after completing moderate to high-intensity resistance training (Chodzko-Zajko et al., 2009). This likely stems from an increase in mitochondrial number, size, and function and has the result of improving skeletal muscle insulin sensitivity and glucose uptake (Dela & Kjaer, 2006).

Conclusion

A growing global population of elderly individuals presents a number of health-care challenges

that will strain health-care provision and increase socioeconomic costs. The development of sarcopenia contributes to the increased risk of falls, fractures, poor mobility, and a reduced ability to lead an independent lifestyle. The resulting reduction in physical activity contributes to the development of insulin resistance, type 2 diabetes, hypertension, and cardiovascular disease. Although aging skeletal muscle can respond to protein intake, peak rates of MPS are blunted compared with the young, suggesting an anabolic resistance, and this may contribute to sarcopenia. However, resistance exercise is a potent stimulator of MPS, and progressive resistance training results in improved muscle mass, strength, and metabolic health.

Cross-References

- ▶ Atrophy
- ▶ Cytokines
- ▶ Glucocorticoids
- ▶ Insulin
- ▶ Physical Inactivity

References and Readings

Andersen, J. L. (2003). Muscle fibre type adaptation in the elderly human muscle. *Scandinavian Journal of Medicine & Science in Sports, 13*, 40–47.

Burton, L. A., & Sumukadas, D. (2010). Optimal management of sarcopenia. *Clinical Interventions in Aging, 5*, 217–228.

Chodzko-Zajko, W. J., Proctor, D. N., Fiatarone Singh, M. A., Minson, C. T., Nigg, C. R., & Salem, G. J. (2009). Exercise and physical activity for older adults. *Medicine& Science in Sports & Exercise, 41*, 1510–1530.

Dela, F., & Kjaer, M. (2006). Resistance training, insulin sensitivity and muscle function in the elderly. *Essays in Biochemistry, 42*, 75–88.

Office of National Statistics. (2012). National Population Projections, 2010-based projections; Executive Summary. http://www.ons.gov.uk/ons/dcp171776_253890.pdf. Accessed 8 March 2012.

Koopman, R., & van Loon, L. J. (2009). Aging, exercise, and muscle protein metabolism. *Journal of Applied Physiology, 106*, 2040–2048.

Narici, M. V., & Maffulli, N. (2010). Sarcopenia: Characteristics, mechanisms and functional significance. *British Medical Bulletin, 95*, 139–159.

Rennie, M. J. (2005). Body maintenance and repair: How food and exercise keep the musculoskeletal system in good shape. *Experimental Physiology, 90*, 427–436.

Rennie, M. J., Selby, A., Atherton, P., Smith, K., Kumar, V., Glover, E. L., et al. (2010). Facts, noise and wishful thinking: Muscle protein turnover in aging and human disuse atrophy. *Scandinavian Journal of Medicine & Science in Sports, 20*, 5–9.

United Nations, Department of Economic and Social Affairs. (2002). *Population division. World population ageing 1950–2050*. New York: United Nations.

Wagenmakers, A. J., van Riel, N. A., Frenneaux, M. P., & Stewart, P. M. (2006). Integration of the metabolic and cardiovascular effects of exercise. *Essays in Biochemistry, 42*, 193–210.

WHO. (2008). *WHO global report on falls prevention in older age*. Geneva: Author.

Saturated Fats

▶ Fat, Dietary Intake

Saturated Fatty Acids

▶ Fat: Saturated, Unsaturated

SBM

▶ Society of Behavioral Medicine

Scale Development

Yori Gidron
Faculty of Medicine and Pharmacy,
Free University of Brussels (VUB), Jette,
Belgium

Definition

Scale development is an essential stage in the assessment of constructs and variables in behavior medicine, and in any social and biomedical

science. Scales are used for assessment of self-reported variables including mood, daily disability, various types of symptoms, adherence to recommended diet, etc. Though there is no explicit "rule" for the stages of scale development, certain steps need to be included for claiming that a scale is reliable and valid. The reliability of a scale is very important and refers to its repeatability and lack of measurement error. This is tested by internal-reliability tests (Cronbach's α) and by a test-retest reliability of scores over time. Validity is an essential aspect of a scale and refers to the extent to which it measures what it claims to measure. This is tested by several manners including "face validity," concurrent validity, construct validity, and criterion validity. When developing a scale, it is essential to have a clear definition of the concept it refers to. Thus, for example, an anxiety scale should not have items assessing depression since these are not the same construct. After choosing an acceptable definition for the construct, a group of "experts" on the construct meets to provide items or even topics related to the construct, from which the researcher creates items. The chosen items will reflect the most common topics or items suggested by the expert panel. The panel can be experts from the field (psychologists, physicians, etc.) and patients who experienced the issue under investigation, thus reflecting experienced "experts." Then, the investigator can ask another group of experts or patients to rate the relevance of each item to the construct, reflecting face validity. The items with a mean relevance above a chosen criterion will be selected for the preliminary scale. Next, the researcher administers the scale to a larger sample, with theoretically relevant additional tests. This will enable to test the internal reliability, concurrent validity against another scale assessing the same construct, and the construct validity against scales assessing theoretically related constructs. Finally, an acceptable criterion (e.g., ill vs. healthy sample) will enable to test the scale's criterion validity. Predictive validity can also be tested by examining whether the scale's scores predict a certain event or outcome in future, beyond the effects of known confounders. These steps are needed for scale development, to verify a scale's reliability and validity, for use in research and clinical evaluations.

Cross-References

▶ Reliability and Validity

References and Readings

Clark, L. A., & Watson, D. (1995). Constructing validity: Basic issues in objective scale development. *Psychological Assessment, 3*, 309–319.

Scatter

▶ Dispersion

Schneiderman, Neil

Neil Schneiderman
Department of Psychology, Behavioral Medicine Research Center, University of Miami, Coral Gables, FL, USA

Biographical Information

Neil Schneiderman

Neil Schneiderman was born in Brooklyn, New York, on February 24, 1937. He has been married to his wife Eleanor since 1960 and is the father of three children and grandfather of five. Schneiderman received his A.B. degree from Brooklyn College, spent 2 years in the US Army, earned his Ph.D. degree in Psychology from Indiana University, and received postdoctoral training in neurophysiology and neuropharmacology in the Physiological Institute of the University of Basel, Switzerland. Schneiderman was appointed as assistant professor at the University of Miami, Coral Gables, Florida, in 1965, rising through the ranks to become professor in 1974. He subsequently received secondary appointments as professor of Medicine, Psychiatry and Behavioral Sciences, and Biomedical Engineering. In 1989, he was awarded an endowed chair as the James L. Knight Professor of Health Psychology. Since 1986, he has served as the director of the Division of Health Psychology in the Department of Psychology and as director of the University of Miami Behavioral Medicine Research Center. He also served extensively as chair of the NIH-funded University of Miami General Clinical Research Center Advisory Committee. Schneiderman has directed pre- and postdoctoral NIH training grants involving cardiovascular disease from the National Heart, Lung, and Blood Institute (NHLBI) since 1979 and HIV/AIDS from the National Institute of Mental Health (NIMH) since 1993.

Schneiderman was the second editor in chief of the journal *Health Psychology* before becoming founding editor in chief of the *International Journal of Behavioral Medicine*. Within the NIH, he served as a member of the Biopsychology Study Section, NHLBI Research Training Review Committee, and NIMH Health Behavior and Prevention Review Committee. In the American Psychological Association (APA), he was chair of the Board of Scientific Affairs and is a fellow in the Divisions of Experimental Psychology (3), Behavioral Neuroscience and Comparative Psychology (6), and Health Psychology (38) as well as a former president of Division 38. A founding fellow of the Academy of Behavioral Medicine Research, Schneiderman later served as president of that organization. Schneiderman also served as president of the International Society of Behavioral Medicine (ISBM). He is a fellow of the Society of Behavioral Medicine and of the American College of Clinical Pharmacology. He is also the recipient of the APA Distinguished Scientific Contribution Award (1994), Society of Behavioral Medicine Distinguished Scientist Award (1997), and ISBM Outstanding Scientific Achievement Award (2004).

Major Accomplishments

Schneiderman's first two empirical research articles were published in *Science* in 1962. Written with his academic mentor, Isadore Gormezano, the papers described animal models of eyelid and nictitating membrane Pavlovian conditioning in rabbits. These preparations were suitable for concomitantly studying behavioral and neurophysiological processes in conscious, minimally restrained animals. Subsequently, Schneiderman added heart rate conditioning to the repertoire of animal models, and for the next several decades, he and his colleagues traced neuronal pathways involved in Pavlovian conditioning of cardiovascular responses in rabbits. This began with identifying the cells of origin of vagal cardioinhibitory motoneurons in the rabbit medulla, using histochemistry, microstimulation, and single neuron extracellular electrophysiological recordings, and continued with mapping the central nervous system pathways that mediated conditioned and unconditioned cardiovascular adjustments. The study of central nervous system pathways mediating differentiated patterns of cardiovascular adjustments also led Schneiderman and his colleagues including Marc Gellman, Barry Hurwitz, Maria Llabre, and Pat Saab to conduct an important series of psychophysiological studies in humans. They described differentiated patterns of neurohormonal and cardiovascular responses to separate behavioral stressors as a function of race, gender, and hypertensive status. These responses were also shown to be influenced by such psychosocial factors as harassment and hostility. In recent years, Schneiderman has

collaborated with Philip McCabe, Armando Mendez, and other Miami scientists in documenting the psychosocial prevention of atherosclerosis progression in a rabbit model of coronary artery disease.

Because of Schneiderman's interest in relationships among biological regulation, psychosocial factors, and disease processes, it was not surprising that he also joined with colleagues including MaryAnn Fletcher, Gail Ironson, and Nancy Klimas relatively early in the HIV/AIDS epidemic to study relationships between psychosocial variables and endocrine-immune regulation in HIV infected patients, when AIDS was beginning to ravage the Miami community. This, in turn, led Schneiderman, Michael Antoni, and their collaborators to begin to use group-based cognitive behavior therapy and relaxation training in randomized controlled trials to influence psychosocial, endocrine, and immune factors and even to reduce HIV viral load to undetectable levels in patients who were failing their regimen of highly active antiretroviral drugs.

Schneiderman's broad research experience, including intervention studies with clinical patients, prepared him to serve as principal investigator of the Miami Field Center for the NIH/NHLBI "Enhancing Recovery in Coronary Heart Disease Patients (ENRICHD)" randomized trial. The trial compared cognitive behavior therapy and usual care in post-myocardial infarction patients. Although that trial produced null results in terms of morbidity and mortality, Schneiderman and colleagues conducted a secondary analysis that suggested that the trial appeared to decrease morbidity and mortality in White men, but not in women or minority patients. Based on the supposition that the null result in the ENRICHD trail was due to the protocol not being sufficiently tailored to women, Schneiderman joined with Kristina Orth-Gomér and other Swedish colleagues to conduct the "Stockholm Women's Intervention Trial for Coronary Heart Disease (SWITCHD)." This trial, which used group-based cognitive behavior therapy and relaxation training in women previously hospitalized for myocardial infarction or coronary revascularization, showed a significant decrease in mortality rate for the intervention compared with a usual care group. Similar results have now also been reported by others.

In addition to Schneiderman's contributions in basic science and in clinical trials research, he has been actively involved in population-based observational studies such as those with Ronald Goldberg and Jay Skyler. As the principal investigator of the Miami Field Center of the NIH/NHLBI multicenter Hispanic Community Health Study/Study of Latinos, Schneiderman and his colleagues in Miami, including Frank Penedo and David Lee, as well as investigators in the Bronx, Chicago, and San Diego are characterizing the health status and disease burden of Hispanic adults living in the United States, describing the positive and negative consequences of immigration and acculturation in relation to lifestyle and access to health care and assessing likely causal factors of disease in this diverse population. The study has been examining 16,000 Hispanic men and women who have now completed a rigorous 6.5-h baseline exam and participated in an overnight sleep study. Longitudinal follow-up is currently documenting the incidence of acute myocardial infarction, heart failure, resuscitated cardiac arrest, cardiac revascularization, stroke, transient ischemia attack, asthma, and mortality. In summary, Schneiderman's major contributions have been in terms of basic science, population-based observational studies, and randomized clinical trials.

Cross-References

► International Society of Behavioral Medicine

References and Readings

Ironson, G. H., Gellman, M. D., Spitzer, S. B., Llabre, M. M., Pasin, R. D., Weidler, D. J., et al. (1989). Predicting home and work blood pressure measurements from resting baselines and laboratory reactivity in black and white Americans. *Psychophysiology, 26*, 174–184.

Orth-Gomér, K., Schneiderman, N., Wang, H., Walldin, C., Blom, M., & Jernberg, T. (2009). Stress reduction prolongs life in women with coronary disease: The Stockholm women's intervention trial for coronary heart

disease (SWITCHD). *Circulation. Cardiovascular Quality and Outcomes, 2*, 25–32. doi:10.1161/CIRCOUTCOMES.108.812859. PMID 20031809.

Schneiderman, N., Fuentes, I., & Gormezano, I. (1962). Acquisition and extinction of the classically conditioned eyelid responses in the albino rabbit. *Science, 136*, 650–652.

Schneiderman, N., Saab, P. G., Catellier, D. J., Powell, L. H., DeBusk, R. F., Williams, R. B., et al. (2004). Psychosocial treatment within sex by ethnicity subgroups in the enhancing recovery in coronary heart disease (ENRICHD) clinical trial. *Psychosomatic Medicine, 66*, 475–483.

Schwaber, J., & Schneiderman, N. (1975). Aortic nerve activated cardioinhibitory neurons and interneurons. *American Journal of Physiology, 229*, 783–789.

Scientific Psychology

▶ Psychological Science

Screening

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

Screening refers to the process of surveying a population or sample of a population, in the attempt to identify people at risk for or with a given health condition. Screening is a crucial part of epidemiology, as it informs about the prevalence and risk factors of various health conditions in a population. Furthermore, screening is crucial for preventive medicine, since it enables to identify people who may benefit from primary, secondary, or tertiary interventions. Screening for primary prevention reflects identifying people without a risk factor (e.g., hypertension, depression), to prevent the risk factor and subsequent illnesses. Screening for secondary prevention could be among people with a risk factor, to prevent an illness. And screening for tertiary prevention would be done to prevent relapse or mortality in people already ill (e.g., after a first myocardial infarction). Screening could be in relation to psychosocial factors such as hostility or anxiety, behavioral risk factors of disease such as smoking or excessive alcohol drinking, and for genetic profiles. For implementing screening tests in clinical use to reliably predict disease risk, it is crucial to know the relative risk for a disease in people high and low on a screening risk factor as well as the correct value of "false positives" (Wald & Morris, 2011). It is of outmost importance to identify the criteria or cutoffs for screening in clear, precise, and operational manners (e.g., smoking more than 10 cigarettes/day, depression score above 10 on the Center for Epidemiological Studies Depression scale). Screening then enables either to study specific sub-populations at risk for health conditions, or for treating them. One important criterion for screening tests is their accuracy. A test is thought to be 95% accurate if in 95% of the times it predicts correctly who has a disease (sensitivity) and if 95% of the time it predicts correctly who does not have a disease (specificity). Screening also enables to increase one's therapeutic and statistical effects, since by excluding people below a certain cutoff, researchers can prevent a "floor effect" of therapeutic effectiveness. The cutoffs used to screen are a function of previously defined cutoffs from research or clinical studies, a function of how severe a risk the researchers aim to indentify, and the available therapeutic resources that can be allocated for treating the "screened in" subpopulation later. Furthermore, in randomized-controlled trials (RCT), the more strict screening criteria are, the longer could be the trial's duration as the sought patient profile becomes more rare. Thus, the screening criteria are a function of the research question and available resources for such screening and subsequent treatment, and this is a vital part of clinical epidemiology and research and of therapeutic interventions.

Cross-References

- ▶ Cancer Prevention
- ▶ Epidemiology
- ▶ Population-Based Study
- ▶ Population Health

References and Readings

Wald, N. J., & Morris, J. K. (2011). Assessing risk factors as potential screening tests. A simple assessment tool. *Archives of Internal Medicine, 171*, 286–291.

Screening, Cognitive

Richard Hoffman
Academic Health Center, School of Medicine-Duluth Campus University of Minnesota, Duluth, MN, USA

Synonyms

Cognitive impairment tests; Cognitive status tests; Dementia screening tests; Mental status examination

Definition

Cognitive screening is a brief, performance-based assessment of one or more domains of neurobehavioral or cognitive functioning. These assessments typically are completed using standardized cognitive screening tests that can be completed at bedside or in the clinic in 20–30 min or less, often accompanied by interview information elicited from family members or other informants who know the examinee well and can comment on their observations about the examinee's behaviors or changes in their behaviors.

Description

Cognitive screening tests are very commonly used in behavioral medicine, neuropsychiatry, and primary care medicine. Surveys indicate that cognitive screening instruments are used by over 50% of practitioners in neuropsychiatry and such tests have become a mainstay in the practice of medicine over the course of the last 35 years. Because cognitive screening tests are brief and require a minimum of specialized testing equipment, they can in most cases be administered at bedside, in a busy clinic, or in the emergency department and serve to identify those patients who might benefit from more extensive workups, including neuroimaging, metabolic assays, and blood work, or more extensive neuropsychological testing. Cognitive screening tests are used as one central component in the initial differential diagnosis of delirium versus dementia and are perhaps most frequently used in the initial screening for dementias and mild cognitive impairment (MCI), both of which are underdiagnosed in their earliest stages in primary care practice due to the subtlety of their initial presenting symptoms.

Changes in cognitive functioning are frequently seen as a consequence of a number of neurological and general medical diseases, prominently including dementias and degenerative diseases of the cerebral cortex and subcortical regions of the brain. In addition to central nervous system diseases, cognition may also be affected by other systemic diseases, including respiratory, cardiovascular, and renal diseases as well as some infectious diseases, diseases of the liver and pancreas, nutritional deficiencies, metabolic diseases and diabetes, adverse effects of medications, and exposure to toxic substances. Judicious use of cognitive screening instruments can provide evidence to suggest an underlying medical disorder heretofore undiagnosed and may help guide the use of medications and medication dosages, as well as provide information that may prompt the treatment of reversible conditions, such as reversible dementias and pseudo-dementias.

In neuropsychiatry, cognitive screening tests can help detect deficits associated with disorders

Screening, Cognitive, Table 1 Cognitive screening tests

AB Cognitive Screen (ABCS)
Abbreviated Mental Test (AMT)
Addenbrooke's Cognitive Examination – Revised (ACE-R)
Blessed Information-Memory-Concentration Test (BIMC)
Blessed Orientation-Memory-Concentration Test (OMC)
Brief Alzheimer Screen (BAS)
Brief Cognitive Scale (BCS)
Bowles-Langley Technology/Ashford Memory Test
Cambridge Cognitive Examination – Revised (CAMCOG-R)
Clock Drawing Test (CDT)
Cognitive Abilities Screening Instrument (CASI)
Cognitive Assessment Screening Test (CAST)
Cognistat (also known as the Neurobehavioral Cognitive Status Examination or NCSE)
Cognitive Capacity Screening Exam (CCSE)
Computer-Administered Screen for Mild Cognitive Impairment (CANS-MCI)
DemTect
General Practitioner Assessment of Cognition (GPCOG)
Geriatric Evaluation of Mental Status (GEMS)
High Sensitivity Cognitive Screen (HSCS)
Hopkins Verbal Learning Test (HVLT)
Kokmen Short Test of Mental Status (STMS)
Memory Impairment Screen (MIS)
Mental Alteration Test
Mental Status Questionnaire (MSQ)
Mini-Cog
Mini-Mental Status Examination (MMSE)
Modified Mini-Mental Status Examination (3MS)
Modified WORLD Test (WORLD)
Montpellier Screen (Mont)
Montreal Cognitive Assessment (MoCA)
Neurobehavioral Cognitive Status Examination (NCSE)
Rapid Dementia Screening Test (RDST)
Repeatable Battery for the Assessment of Neuropsychological Status (RBANS)
Revised Mattis Dementia Rating Scale (DRS-2)
Rowland Universal Dementia Assessment Scale
Seven-Minute Screen (7MS)
Short and Sweet Screening Instrument (SASSI)
Short Blessed Test (SBT)
Short Portable Mental Status Questionnaire (SPMSQ)
Short Test of Mental Status (STMS)
Six-item Cognitive Impairment Test (6CIT)
Test for the Early Detection of Dementia from Depression (TE4D-Cog)
Three Word Recall (3WR)
Time and Change Test (T&C)
Trail Making Test (TMT)
Verbal Fluency-Categories (VFC)
Verbal Fluency-Animals (VFA)
Cognitive Screening Tests for Specialized Patient Populations
High Sensitivity Cognitive Screen
HIV Dementia Scale
Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT)
Informant- or Proxy-Rated Screening Instruments
Blessed Dementia Rating Scale (BDRS)
Deterioration Cognitive Observee (DECO)
Informant Questionnaire for Cognitive Decline in the Elderly (IQCODE)
Telephone and Mail Screening Instruments
Dementia Questionnaire
Five-Minute Telephone Version of the Short Blessed Test (SBT)
Minnesota Cognitive Acuity Screen
Structured Telephone Interview for Dementia Assessment (STIDA)
Telephone Interview for Cognitive Status (TICS)

that are commonly missed in a standard psychiatric intake interview, especially in emergency room settings, including patients who present with mild disorientation or evidence of possible substance abuse. In addition, many primary psychiatric disorders have significant effects on cognition, such as affective disorders and schizophrenia, and some focal neurological disorders such as focal strokes, neoplasias, and seizure disorders may have combined cognitive and affective sequelae.

Among the most commonly used and well-researched brief cognitive screening tests are the Mini-Mental State Examination (MMSE), the Cognitive Capacities Screening Examination (CCSE), and the Short Portable Mental Status Questionnaire (SPMSQ), but there are numerous cognitive screening tests available to practitioners at the present time and these are listed in Table 1. Although there is considerable variability in the component sections of the cognitive

screening tests listed in Table 1, in general each contains some assessment of orientation (does the patient know who they are, where they are, and know the day and date), attention and concentration, language skills, memory and immediate recall of verbal information, and visuaospatial or drawing/copying skills. Most cognitive screening tests are designed to be completed within 10 min or less. The BIMC, the ACE-R, the CASI, the Cognistat, the RBANS, the HSCS, and the CAMCOG-R contain more extensive subtests and may require up to 30 min to complete. The Mattis Dementia Rating Scale requires 20–45 min to complete and provides assessment of attention, initiation perseveration, visuospatial construction, reasoning, and memory.

Since 1988, there have been several cognitive screening tests designed to be administered by phone, typically used in epidemiological studies as more extensive follow-up instruments after an initial administration face-to-face of a brief screening instrument such as the MMSE or the SASSI. Five such instruments are listed in Table 1.

Also listed in Table 1 are three guided interview or informant-based cognitive screening instruments which are designed to document information from family members or caregivers of patients regarding observed cognitive decline, changes in behavior, or – in the case of the Deterioration Cognitive Observee (DECO) instrument – changes in activity level, long-term memory, short-term memory, visuospatial processing, and new skill learning. Although these can be used as stand-alone measures, they are perhaps best used to complement the findings from cognitive screening tests directly administered to the patient in question.

There is now considerable interest in the development of cognitive screening tests for specific at-risk populations, and recent examples include two cognitive screening tests designed to detect the early signs of AIDS-related dementia in AIDS patients (the High Sensitivity Cognitive Screen test and the HIV Dementia Scale) and a recently developed test to screen for post-concussion cognitive changes, the Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT).

With the aging of the population has come an increased interest in cognitive screening in geriatric populations and the increased need to identify early signs of dementia and early signs of mild cognitive impairment, especially as new treatments are developed that are capable of modifying the progression of dementias. In primary care medicine, the standard of practice in the very near future may well include cognitive screening of all patients over the age of 75 in addition to screening of all younger patients when there is a reason to suspect cognitive impairment.

Cross-References

▶ Neuropsychology

References and Readings

Cullen, B., O'Neill, B., Evans, J. J., Coen, R. F., & Lawlor, B. A. (2007). A review of screening tests for cognitive impairment. *Journal of Neurology, Neurosurgery, and Psychiatry, 78*, 790–799.

Demakis, G. J., Mercury, M. G., & Sweet, J. J. (2000). Screening for cognitive impairments in primary care settings. In M. E. Maruish (Ed.), *Handbook of psychological assessment in primary care settings* (pp. 555–582). London: Lawrence Erlbaum.

Lonie, J. A., Tierney, K. M., & Ebmeier, K. P. (2009). Screening for mild cognitive impairment: A systematic review. *International Journal of Geriatric Psychiatry, 24*, 902–915.

Malloy, P. F., Cummings, J. L., Coffey, C. E., Duffy, J., Fink, M., Lauterbach, E. C., et al. (1997). Cognitive screening instruments in neuropsychiatry: A report of the Committee on Research of the American Neuropsychiatric Association. *Journal of Neuropsychiatry and Clinical Neurosciences, 9*, 189–197.

Mitchell, A. J., & Malladi, S. (2010). Screening and case finding tools for the detection of dementia. Part I: Evidence-based meta-analysis of multidomain tests. *American Journal of Geriatric Psychiatry, 18*, 759–782.

Mitrushina, M. (2009). Cognitive screening methods. In I. Grant & K. M. Adams (Eds.), *Neuropsychological assessment of neuropsychiatric and neuromedical disorders* (pp. 101–126). New York: Oxford University Press.

Tombaugh, T. N., & McIntyre, N. J. (1992). The mini-mental state examination: A comprehensive review. *Journal of the American Geriatrics Society, 40*, 922–935.

Seasonal Affective Disorder

Kathryn A. Roecklein and Patricia M. Wong
Department of Psychology, University of Pittsburgh, Pittsburgh, PA, USA

Synonyms

Bipolar disorder, with seasonal pattern; Major depressive disorder, with seasonal pattern

Definition

The most common presentation of seasonal affective disorder (SAD) is recurrent depressive episodes in winter followed by spring remission (Rosenthal et al., 1984). SAD is diagnosed according to the American Psychiatric Association not as a separate disorder, but rather as a course specifier to describe the pattern of depressive episodes in patients meeting criteria for major depressive disorder, or bipolar I or II disorder (American Psychiatric Association [DSM-IV-TR], 2000). Criteria for the seasonal specifier include (1) recurrence of major depressive episodes at a specific time of year; (2) full remission (or change to mania/hypomania) from depression also recurring at a specific time of year; (3) at least two major depressive episodes meeting criteria 1 and 2 within the last 2 years, with no occurrence of nonseasonal depression within the same period; and (4) experiencing a greater number of major depressive episodes meeting SAD criteria than that of nonseasonal depression throughout the individual's lifetime (American Psychiatric Association [DSM-IV-TR], 2000).

Description

Epidemiology. SAD is characterized by depressed mood, anhedonia, and fatigue, as well as higher rates of appetite increase, weight gain, and hypersomnia compared to nonseasonal major depression (Magnusson & Partonen, 2005). Ten to twenty percent of patients with depression seeking outpatient treatment have a seasonal pattern of recurrences, and SAD accounts for 10–22% of all unipolar and bipolar mood disorders (Roecklein, Rohan, & Postolache, 2010). A notable characteristic of sleep in SAD is that, in contrast with the predominance of insomnia in nonseasonal depression, a majority of individuals with SAD experience hypersomnia (68–80%; e.g., Rosenthal et al., 1985). Given that seasonality, the tendency to vary in mood and behavior across the seasons (Kasper, Wehr, Bartko, Gaist, & Rosenthal, 1989; Rosen et al., 1990), is normally distributed, a range of mild to severe seasonal changes are likely to occur in behavioral medicine research and practice.

Etiology. Etiological models propose that seasonal changes in the environment, being light levels or other conditioned cues, trigger onset in fall or winter (Rohan, Roecklein, & Haaga, 2009; Sohn & Lam, 2005). Lewy, Sack, Miller, and Hoban (1987) proposed that winter changes in day length lead to a delay in internal circadian rhythms relative to clock time or other rhythms like sleep and wake. Wehr et al. (2001) proposed that winter changes in day length are encoded by nocturnal melatonin release duration as a "circadian signal of change of season," leading to behavioral and physiological changes in humans similar to those of seasonally breeding mammals. Transforming environmental light levels to neural signals is mediated by a retinal pathway to the central clock, and this pathway could be differentially sensitive across individuals, leading some to be vulnerable to insufficient input in winter (Hebert, Dumont, & Lachapelle, 2002). These circadian and retinal hypotheses may interact with one another, as well as with the monoamine hypothesis (i.e., serotonin and dopamine) and cognitive behavioral mechanisms (Rohan et al., 2009).

Treatment. The recommended first-line treatment for SAD is light therapy, while antidepressants are also commonly used (Lam & Levitt, 1999). Light therapy typically requires daily exposure to 10,000-lux of white or full-spectrum fluorescent light for at least 30 min, although efforts to refine the wavelength and reduce

duration are being tested clinically. Among antidepressants, Bupropion XL is the first FDA-approved drug for the treatment of winter depression. A double-blind, placebo-controlled, multisite trial testing Bupropion XL on adults with a history of SAD demonstrated that the overall proportion of depression recurrences following treatment was lower for those taking Bupropion (16%) than for those using a placebo (28%; Modell et al., 2005), although the low rate of recurrence indicates a significant placebo response. In addition, cognitive behavioral therapy is as effective as light therapy for acute treatment during a depressive episode, and has prophylactic effects 1 year later in reducing the risk of a subsequent episode (Rohan et al., 2004). Given that multiple empirically validated treatments are available, detecting seasonal patterns in clinical settings can improve patient outcomes.

Implications for behavioral medicine. Seasonal variations in mood and behavior are relevant to Behavioral Medicine research and clinical practice. Such implications can be divided into specific biopsychosocial components including biological characteristics (e.g., genetic risk for seasonality, neurotransmitter and neurohormonal seasonal fluctuations), behaviors (e.g., seasonal changes in physical activity, sleep, substance use, and eating behavior), and social factors (e.g., seasonal changes in social activity rhythms). Candidate behavioral mechanisms in SAD include behavioral disengagement (i.e., lack of response-contingent positive reinforcement) as well as emotional and psychophysiological reactivity to light and seasonal visual stimuli. Several biological mechanisms in SAD have also been proposed (Rohan et al., 2009). The circadian phase shift hypothesis suggests that in the fall and winter months, the timing of different circadian rhythms (e.g., melatonin release, sleep-wake cycle) is out of phase, or desynchronized from other rhythms and/or environmental factors (e.g., dusk/dawn cycle). Another hypothesis is that individuals with SAD have retinas that are less sensitive to light; low environmental light levels in the winter then lead to subthreshold levels of light information transmitted to the brain. The photoperiodic hypothesis proposes that some individuals with SAD maintain biological mechanisms to track changes in photoperiod, a circadian signal of change between seasons, evidenced by individuals with SAD who demonstrate a longer duration of nocturnal melatonin release in the winter months. Rohan et al. (2009) proposed that behavioral and cognitive mechanisms contribute to a psychological vulnerability that, when integrated with biological vulnerabilities, may explain the onset, maintenance, or remission of SAD. Although these separate mechanisms have been shown to play a role in SAD, it is not yet clear if these factors are mechanistic in the cause or maintenance of the disease.

Cross-References

- ▶ Circadian Rhythm
- ▶ Depression: Symptoms
- ▶ Psychiatric Diagnosis
- ▶ Unipolar Depression

References and Readings

American Psychiatric Association. (2000). *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.). Washington, DC: Author.

Hebert, M., Dumont, M., & Lachapelle, P. (2002). Electrophysiological evidence suggesting a seasonal modulation of retinal sensitivity in subsyndromal winter depression. *Journal of Affective Disorders, 68*(2–3), 191–202.

Kasper, S., Wehr, T. A., Bartko, J. J., Gaist, P. A., & Rosenthal, N. E. (1989). Epidemiological findings of seasonal changes in mood and behavior. A telephone survey of Montgomery County, Maryland. *Archives of General Psychiatry, 46*(9), 823–833.

Lam, R. W., & Levitt, A. J. (Eds.). (1999). *Clinical guidelines for the treatment of seasonal affective disorder*. Vancouver, BC: Clinical & Academic.

Lewy, A. J., Sack, R. L., Miller, L. S., & Hoban, T. M. (1987). Antidepressant and circadian phase-shifting effects of light. *Science, 235*(4786), 352–354.

Magnusson, A., & Partonen, T. (2005). The diagnosis, symptomatology, and epidemiology of seasonal affective disorder. *CNS Spectrums, 10*(8), 625–634.

Modell, J. G., Rosenthal, N. E., Harriett, A. E., Krishen, A., Asgharian, A., Foster, V. J., et al. (2005). Seasonal affective disorder and its prevention by anticipatory

treatment with bupropion XL. *Biological Psychiatry, 58*(8), 658–667.
Roecklein, K. A., Rohan, K. J., & Postolache, T. T. (2010). SAD: Is seasonal affective disorder a bipolar variant? *Current Psychiatry, 9*(2), 42–54.
Rohan, K. J., Roecklein, K. A., & Haaga, D. A. F. (2009). Biological and psychological mechanisms of seasonal affective disorder: A review and integration. *Current Psychiatry Reviews, 5*(1), 37–47.
Rohan, K. J., Tierney Lindsey, K., Roecklein, K. A., & Lacy, T. J. (2004). Cognitive-behavioral therapy, light therapy, and their combination in treating seasonal affective disorder. *Journal of Affective Disorders, 80*, 273–283.
Rosen, L. N., Targum, S., Terman, M., Bryant, M., Hoffman, H., Kasper, S., et al. (1990). Prevalence of seasonal affective disorder at four latitudes. *Psychiatry Research, 31*(2), 131–144.
Rosenthal, N. E., Sack, D. A., Gillin, J. C., Lewy, A. J., Goodwin, F. K., & Davenport, Y. (1984). Seasonal affective disorder. A description of the syndrome and preliminary findings with light therapy. *Archives of General Psychiatry*, *41*(1), 72–80.
Rosenthal, N. E., Sack, D., James, S., Parry, B., Mendelson, W., Tamarkin, L., et al. (1985). Seasonal affective disorder and phototherapy. *Annals of the New York Academy of Sciences, 453*, 260–269.
Sohn, C. H., & Lam, R. W. (2005). Update on the biology of seasonal affective disorder. *CNS Spectrums, 10*(8), 635–646.
Wehr, T. A., Duncan, W. C., Jr., Sher, L., Aeschbach, D., Schwartz, P. J., Turner, E. H., et al. (2001). A circadian signal of change of season in patients with seasonal affective disorder. *Archives of General Psychiatry, 58*(12), 1108–1114.

Secondary Care

▶ Clinical Settings

Secondary Gain

▶ Symptom Magnification Syndrome

Secondary Parkinsonism

▶ Parkinson's Disease: Psychosocial Aspects

Secondary Prevention Programs

▶ Cardiac Rehabilitation

Secondhand Smoke

Susan J. Bondy
Dalla Lana School of Public Health, University of Toronto, Toronto, ON, Canada

Synonyms

Environmental tobacco smoke; Involuntary exposure to tobacco smoke; Passive smoking

Definition

The exposure to, and effects of, inhalation of cigarette smoke by an individual other than the active smoker. The term is also applied, more specifically, to smoke exhaled by an active smoker that remains in the environment.

Description

Secondhand smoke includes sidestream smoke from the end of a lit cigarette and exhaled smoke (United States Department of Health and Human Services, 2006, 2010; World Health Organization International Agency for Research on Cancer, 2004). Harmful components identified specifically in cigarette smoke measured in the air include gases (e.g., carbon monoxide), droplets, and respirable particles which result from the release, combustion, and partial combustion of the tobacco leaves and cigarette paper, as well as flavorants, additives, and other chemicals introduced at agricultural, manufacturing, or packaging stages (California Environmental Protection Agency, 2005a). Secondhand smoke, has been shown to contain elevated levels of a large number of known and probable human

carcinogens as well as many other toxins with proven causal links to human health conditions (California Environmental Protection Agency, 2005b; Institute of Medicine 2010; United States Department of Health and Human Services, 2010; United States Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment & U.S. EPA, 1992; World Health Organization International Agency for Research on Cancer, 2004). It has been estimated that, in 2004, one third of all children and nonsmoking adults worldwide were exposed to secondhand smoke, and that this avoidable exposure was responsible for over 600,000 premature deaths or 1% of all deaths (Oberg, Jaakkola, Woodward, Peruga, & Pruss-Ustun, 2010). As with active smoking, there is no confirmed risk-free level of exposure to secondhand cigarette smoke (United States Department of Health and Human Services, 2006, 2010).

Concentrations of secondhand smoke (and resulting levels of toxic exposure) vary widely and are influenced by: the number of cigarettes and rate of active smoking; the time elapsed since cigarettes were lit and extinguished; the volume of the affected air space; ventilation, air exchange rates, and direction of air flow; and the duration of exposure (California Environmental Protection Agency, 2005a). Air concentrations in occupational and private settings, where smoking is permitted, often exceed occupational safety standards for specific agents, and biomarker levels in heavily exposed nonsmokers can overlap levels observed in active smokers (California Environmental Protection Agency, 2005b; United States Department of Health and Human Services, 2006). Exposure levels in outdoor settings can vary from negligible to concentrations similar to indoor levels if the passive smoker is exposed to the stream of smoke (Institute of Medicine, 2010).

Measuring exposure for research, surveillance, and program evaluation may be achieved through air sampling or use of markers of human exposure in biological samples. Air monitoring assesses for concentrations for one or more specific component of tobacco smoke or respirable particulates of specific sizes (Institute of Medicine, 2010; United States Department of Health and Human Services, 2006). The most widely used biomarkers include exhaled carbon monoxide, and cotinine (a metabolite of nicotine) in saliva, urine, or blood samples. Other biomarkers used in research include, metabolites other than cotinine and concentrations of known carcinogens, as well as through use of other biological media (e.g., testing for accumulated cotinine and nicotine in hair samples from exposed individuals, even newborns as an indication of late prenatal exposure).

Over 40 years of evidence exists on the adverse health effects caused by passive smoke exposure. This evidence has been summarized in prominent reports by international health agencies including International Agency for Research on Cancer (IARC) (International Agency for Research on Cancer, 2004, 2009), the Office of the United States Surgeon General (United States Department of Health and Human Services, 2006, 2010), and others (California Environmental Protection Agency, 2005b; Institute of Medicine, 2010). The major classes of health effects linked to passive smoking include cancers, respiratory disorders, cardiovascular diseases, reproductive effects, and adverse effects on pre-and postnatal growth and development.

Carcinogenic effects of passive smoking can be expected to be consistent with those for active smoking (International Agency for Research on Cancer, 2004), and for both, the increased risk is dose-dependent. Secondhand smoke has a confirmed causal role in the development of lung cancer in exposed nonsmokers (California Environmental Protection Agency, 2005b; International Agency for Research on Cancer, 2004; United States Department of Health and Human Services, 2006) and is suggested to increase the risks of nasal and nasopharyngeal cancers in adults (California Environmental Protection Agency, 2005b; United States Department of Health and Human Services, 2006). There is also evidence to suggest secondhand smoke increases the risk of all childhood cancers, studied collectively, as well as specific childhood cancers (California Environmental Protection

Agency, 2005b; United States Department of Health and Human Services, 2006). For several cancers with definitive evidence linking them to active smoking (including various digestive, kidney, and bladder cancers), there is not sufficient quantitative data to show a causal association with secondhand smoke exposure in humans (International Agency for Research on Cancer, 2009). The role of secondhand smoke in breast cancer remains more controversial with The California EPA (California Environmental Protection Agency, 2005b) being the first health agency to draw the conclusion of a causal association, while other agencies have not concluded that there is a strong link between active or passive cigarette smoke exposure and breast cancer (International Agency for Research on Cancer, 2009; United States Department of Health and Human Services, 2010).

Conclusive evidence of noncancer respiratory effects of passive smoking include lower respiratory tract illness in children and adults, prevalence of asthma in children, and severity of asthma in children and adults (United States Department of Health and Human Services, 2006). Secondhand smoke also causes recurrent otitis media and middle ear effusion in children (United States Department of Health and Human Services). Passive smoke exposure has also been found to cause adverse and lasting effects on lung function and lung development in children associated with prenatal passive smoking and secondhand exposure in childhood (United States Department of Health and Human Services). Secondhand smoke has been identified as a risk factor for sudden infant death syndrome, with sufficient evidence to suggest a causal association, and associated with a small reduction in birth weight when nonsmoking mothers are exposed while pregnant (United States Department of Health and Human Services).

In terms of cardiovascular diseases, secondhand smoke exposure is accepted as a cause of coronary heart disease morbidity and mortality in adult women and men as well as acute cardiac events (Institute of Medicine, 2010; United States Department of Health and Human Services, 2006). Even brief exposure to environmental cigarette smoke can lead to vascular function changes and arrhythmic effects associated with acute cardiovascular events in susceptible individuals (Institute of Medicine, 2010; United States Department of Health and Human Services, 2010).

The World Health Organization Framework Convention on Tobacco Control (FCTC) requires that all signatory nations adopt and enforce measures to protect their populations from exposure to tobacco smoke (World Health Organization, 2003, 2011). Protecting the population from secondhand smoke is identified as a key, evidence-based, measure to reduce death, disease, and disability caused by tobacco (World Health Organization, 2003). FCTC guidelines (and others International Agency for Research on Cancer (2009); United States Department of Health and Human Services, 2006, 2010) recommend that this be achieved through making all environments 100% smoke free as opposed to reliance on ventilation or creation of designated smoking spaces, which have proven ineffective. Legislation and other measures should apply equally to outdoor spaces wherever there is evidence of exposure (United States Department of Health and Human Services, 2006, 2010; World Health Organization, 2011).

A number of educational, legislative, occupational, and clinical interventions to eliminate exposure to tobacco smoke have been implemented and evaluated. Evidence from several countries has shown that legislated bans in a variety of settings including workplaces, bars, and restaurants have been effective in terms of: achievement of compliance, improved air quality, reduced human biomarker levels of exposure, and a corollary effect of reducing smoking prevalence among individuals exposed to the restrictions (Institute of Medicine, 2010; International Agency for Research on Cancer, 2009; United States Department of Health and Human Services, 2006). There is also growing evidence that event rates for acute cardiac events have been reduced successfully by legislative and other interventions to eliminate smoking in workplaces and other settings and reduce secondhand smoke exposure (Institute of Medicine, 2010; United States Department of Health and Human Services, 2010).

Cross-References

- Cancer and Smoking
- Cigarette Smoking Behavior
- Heart Disease and Smoking
- Institute of Medicine
- Smoking and Health
- Smoking Behavior
- Smoking Cessation
- Tobacco Control
- Tobacco Use
- World Health Organization (WHO)

References and Readings

California Environmental Protection Agency. (2005a). *Proposed identification of environmental tobacco smoke as a toxic air contaminant. Part A: Exposure assessment*. Sacramento, CA: California Environmental Protection Agency, Office of Environmental Health Hazard Assessment.

California Environmental Protection Agency. (2005b). *Proposed identification of environmental tobacco smoke as a toxic air contaminant. Part B: Health effects*. Sacramento, CA: California Environmental Protection Agency, Office of Environmental Health Hazard Assessment.

Institute of Medicine. (2010). *Secondhand smoke exposure and cardiovascular effects: Making sense of the evidence*. Washington, DC: Committee on Secondhand Smoke Exposure and Acute Coronary Events, Board on Population Health and Public Health Practice, Institute of Medicine of the National Academies.

International Agency for Research on Cancer. (2004). *IARC monographs on the evaluation of carcinogenic risks to humans*. Volume 83: Tobacco smoke and involuntary smoking. Lyon: International Agency for Research on Cancer.

International Agency for Research on Cancer. (2009). *IARC handbook of cancer prevention. Volume 13: Evaluating the effectiveness of smoke-free policies*. Lyon: International Agency for Research on Cancer, World Health Organization.

Oberg, M., Jaakkola, M. S., Woodward, A., Peruga, A., & Pruss-Ustun, A. (2010). Worldwide burden of disease from exposure to second-hand smoke: A retrospective analysis of data from 192 countries. *Lancet, 377*(9760), 139–146.

United States Department of Health and Human Services. (2006). *The health consequences of involuntary exposure to tobacco smoke: A report of the Surgeon General*. Rockville, MD: United States Department of Health and Human Services, Public Health Service, Office of the Surgeon General.

United States Department of Health and Human Services. (2010). *How tobacco smoke causes disease: The biology and behavioral basis for smoking-attributable diseases. A report of the Surgeon General*. Rockville, MD: United States Department of Health and Human Services, Public Health Service, Office of the Surgeon General.

United States Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment & U.S. EPA. (1992). *Respiratory health effects of passive smoking (also known as exposure to secondhand smoke or environmental tobacco smoke ETS). EPA/600/6-90/006F*. Washington, DC: United States Environmental Protection Agency (US EPA).

World Health Organization. (2003). *WHO framework convention on tobacco control*. Geneva: Author.

World Health Organization. (2011). *WHO framework convention on tobacco control: Guidelines for implementation Article 5.3; Article 8; Articles 9 and 10; Article 11; Article 12; Article 13; Article 14–2011 edition*. Geneva: Author.

World Health Organization International Agency for Research on Cancer. (2004). *IARC monographs on the evaluation of carcinogenic risks to humans. Volume 83: Tobacco smoke and involuntary smoking*. Lyon: International Agency for Research on Cancer.

Sedentary Activity

- Lifestyle, Sedentary

Sedentary Behaviors

Yori Gidron
Faculty of Medicine and Pharmacy,
Free University of Brussels (VUB),
Jette, Belgium

Definition

Sedentary behaviors are an increasingly common problem worldwide, with important health consequences. These behaviors include long durations of sitting in front of the TV or the computer, playing computer or TV games, and a general lack of peripheral limb movements. These behaviors have risen due to a multitude of reasons including technological advancements,

greater dependence on transportation, urbanization and hence smaller distances to work or schools spent walking, the omnipresence of TV and computers, and our dependence on such means for information, work, leisure, and communication. Various measures and scales exist to assess sedentary behaviors, and these depend on the type of behaviors assessed, the time frame the questions refer to (days, weeks, etc.), and the response format (e.g., a Likert scale or hours). This variability in assessment and use of different cutoffs could of course impact on the prevalence of sedentary behaviors identified in various samples. The prevalence of sedentary behaviors was found to be 58% in a nationally representative sample of Americans aged between 20 and 59 years. When looking just at sitting, one in four Americans spends 70% of their waking time sitting. Furthermore, people in developed countries may spend 4 h a day watching TV and 1 h a day in their vehicle. Importantly, the metabolic and health consequences of sedentary behaviors are distinct from the effects of lack of physical exercise (Owen, Healy, Matthews and Dunstan 2010). In a 21-year follow-up study, number of hours riding in a car, alone, or in combination with hours in front of a TV, significantly predicted cardiovascular disease mortality, independent of confounders (Warren et al. 2010). In contrast, taking daily breaks from sedentary behaviors is related to reduced waist circumference and to improved metabolic outcomes, independent of total amount of sedentary behaviors and of physical exercise (Healy et al. 2008). Mental health problems such as anxiety and depression are also associated with more sedentary behaviors, independent of general physical activity level (de Wit, van Straten, Lamers, Cuijpers and Penninx 2011). Thus, sedentary behaviors are an important topic for research and intervention in behavior medicine.

Cross-References

- ▶ Cardiovascular Risk Factors
- ▶ Lifestyle, Sedentary
- ▶ Obesity: Causes and Consequences
- ▶ Physical Activity and Health

References and Readings

de Wit, L., van Straten, A., Lamers, F., Cuijpers, P., & Penninx, B. (2011). Are sedentary television watching and computer use behaviors associated with anxiety and depressive disorders? *Psychiatry Research, 186*, 239–243.

Healy, G. N., Dunstan, D. W., Salmon, J., Cerin, E., Shaw, J. E., Zimmet, P. Z., et al. (2008). Breaks in sedentary time: Beneficial associations with metabolic risk. *Diabetes Care, 31*, 661–666.

Owen, N., Healy, G. N., Matthews, C. E., & Dunstan, D. W. (2010). Too much sitting: The population health science of sedentary behavior. *Exercise and Sport Sciences Reviews, 38*, 105–113.

Warren, T. Y., Barry, V., Hooker, S. P., Sui, X., Church, T. S., & Blair, S. N. (2010). Sedentary behaviors increase risk of cardiovascular disease mortality in men. *Medicine and Science in Sports and Exercise, 42*, 879–885.

Seek Feedback

- ▶ Self-Monitoring

Selection Bias

- ▶ Bias

Selective Serotonin Reuptake Inhibitors (SSRIs)

Michael Kotlyar[1] and John P. Vuchetich[2]
[1]Department of Experimental and Clinical Pharmacology, College of Pharmacy, University of Minnesota, Minneapolis, MN, USA
[2]Department of Psychiatry, University of Minnesota School of Medicine, Minneapolis, MN, USA

Synonyms

Celexa®; Citalopram; Escitalopram; Fluoxetine; Fluvoxamine; Lexapro®; Luvox®; Paroxetine; Paxil®; Prozac®; Sertraline; Zoloft®

Definition

The SSRIs are a family of medications that act primarily (but not exclusively) by inhibiting serotonin reuptake pumps (Finley, 2009; Sussman, 2009). These medications are used to treat a variety of psychiatric disorders including depression, generalized anxiety disorder, obsessive-compulsive disorder (OCD), panic disorder, premenstrual dysphoric disorder, bulimia nervosa, social phobia, and posttraumatic stress disorder (Sussman). Clinicians should consult the product labeling for each individual medication to determine the indications that each drug is currently approved to treat, since not all of the SSRIs are indicated in the treatment of all of these disorders (although in some cases there is data in the literature suggesting efficacy for a given agent for the treatment of conditions for which it does not have an indication). In the treatment of most types of depression, the SSRIs have similar efficacy to older classes of medications such as the tricyclic antidepressants. However better tolerability at therapeutic doses and lower toxicity in overdose has led to an increased use of SSRIs and decreased use of these older agents over the past several decades (American Psychiatric Association [APA], 2010; Baldessarini, 2006; Finley, 2009). As with all antidepressants, full therapeutic effects are not observed for as long as 8 weeks in the treatment of depression or longer in the treatment of other disorders (such as OCD), however some symptoms may start to improve sooner (APA, 2010; Finley, 2009; Kirkwood, Makela, & Wells, 2008). In the treatment of depression, all of the SSRIs are thought to be approximately equally effective and therefore initial choice of therapy is often based on factors such as patient preference, prior response to the medication, cost, side effect profile of the individual agents, and the probability that a drug interaction will occur between the antidepressant being chosen and the other medications that the patient is on (APA, 2010; Finley, 2009). Although the mechanism of action of the SSRIs is similar, lack of efficacy following treatment with one of the drugs in this class does not necessarily predict lack of efficacy by another medication in this class (APA, 2010).

There are currently six medications classified as SSRIs that are approved for marketing in the United States (i.e., fluoxetine, paroxetine, sertraline, citalopram, escitalopram, and fluvoxamine) (APA, 2010). The SSRIs are similar in many respects; however, important differences between agents are present. Some of these differences are in the side effect profiles of the various drugs and in the likelihood of each drug contributing to a drug-drug interaction with other medications that a patient is taking.

All of the SSRIs have been commonly associated with side effects such as gastrointestinal complaints (e.g., nausea and diarrhea), disturbances in sleep and headache (APA, 2010; Finley, 2009). Although an individual patient could have any of the sides effects listed, fluoxetine is generally considered the most likely to cause insomnia while paroxetine is often considered to be the most sedating. In many patients, these side effects decrease after the first week of therapy. Additionally, all of the SSRIs have been associated with sexual dysfunction (in both men and women) with the most common symptom reported being delayed orgasm, although decreased interest in sex or erectile dysfunction can also occur (APA, 2010; Finley, 2009). Since many patients may not spontaneously report sexual side effects, clinicians should inform patients that these may occur and determine if these have been problematic. Serotonin syndrome which includes neurobehavioral (e.g., lethargy and mental status changes), autonomic (e.g., sweating, blood pressure, and heart rate changes), and neuromuscular (e.g., rigidity and tremor) signs and symptoms has been rarely reported with the use of an SSRI as mono-therapy (Chyka, 2008). However, the risk of serotonin syndrome increases when SSRIs are used in combination with other serotonergic agents, particularly with monoamine oxidase inhibitors (MAOIs) (APA, 2010; Chyka, 2008). SSRIs have also been associated with increased bleeding risk, likely due to the presence of serotonin transporters on blood platelets (Sussman, 2009). Other side effects such as increased sweating, osteoporosis, bruxism, akathisia, and hyponatremia have been reported occasionally as have a wide range of

other side effects (APA, 2010; Finley, 2009). As with all antidepressants, the SSRIs carry a warning regarding increased suicidality, particularly in children, adolescents, and young adults (under age 24) during the initial stages of therapy (Sussman, 2009).

Substantial differences between the SSRIs are present in the pharmacokinetic properties of the agents and in the likelihood that the drug can contribute to drug-drug interactions. For example, fluoxetine is notable for its long half-life (i.e., 4–6 days) and that of its active metabolite norfluoxetine (4–16 days) (Teter, Kando, Wells, & Hayes, 2008). A longer half-life means that any side effects will persist for a longer period of time after discontinuation of the medication. Discontinuation of SSRIs has been associated with withdrawal symptoms characterized by headache, anxiety, flu-like symptoms, and paresthesias (APA, 2010). Therefore, it is advisable to taper the medication when possible. The likelihood of a withdrawal syndrome is less likely in SSRIs with longer half-lives (such as fluoxetine) since the decline in the concentration of medications occurs more gradually.

Many of the SSRIs have been found to interact with other medications, some of which may be commonly co-administered with the SSRIs. The cytochrome P450 (CYP450) superfamily of enzymes is responsible for the metabolism of a large number of medications. There are numerous specific isoenzymes within the CYP450 superfamily of enzymes and the degree to which each is affected by an individual SSRI varies considerably. For example, paroxetine and fluoxetine are both strong inhibitors of CYP2D6 (with fluoxetine being a moderate inhibitor of several other CYP450 isoenzymes) (APA, 2010; Finley, 2009). Since antidepressants are frequently co-administered with other medications, care should be taken to identify and manage any potential drug-drug interactions. It is important to consider the impact of drug interactions both when initiating an enzyme inhibitor (since concentrations of affected medications can increase) and when discontinuing an enzyme inhibitor (since concentrations of affected medication can decrease).

Cross-References

▶ Anxiety Disorder
▶ Depression: Symptoms

References and Readings

American Psychiatric Association. (2010). *Practice guideline for the treatment of patients with major depressive disorder* (3rd ed.). Washington, DC: Author. *The American Journal of Psychiatry, 167* (Suppl. 10), 1–124.

Baldessarini, R. J. (2006). Drug therapy of depression and anxiety disorders. In L. S. Goodman, A. Gilman, L. L. Brunton, J. S. Lazo, & K. L. Parker (Eds.), *Goodman & Gilman's the pharmacological basis of therapeutics* (11th ed.). New York: McGraw-Hill, Medical.

Chyka, P. A. (2008). Clinical toxicology. In J. T. DiPiro, R. L. Talbert, G. C. Yee, G. R. Matzke, B. G. Wells, & L. M. Posey (Eds.), *Pharmacotherapy: A pathophysiologic approach* (7th ed.). New York: McGraw-Hill Medical.

Finley, P. R. (2009). Mood disorders: major depressive disorders. In M. A. Koda-Kimble, L. Y. Young, B. K. Alldredge, R. L. Corelli, B. J. Gugielmo, W. A. Kradjan, & B. R. Williams (Eds.), *Applied therapeutics: The clinical use of drugs* (9th ed.). Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins.

Kirkwood, C. K., Makela, E. H., & Wells, B. G. (2008). Anxiety disorders: Posttraumatic stress disorder and obsessive-compulsive disorder. In J. T. DiPiro, R. L. Talbert, G. C. Yee, G. R. Matzke, B. G. Wells, & L. M. Posey (Eds.), *Pharmacotherapy: A pathophysiologic approach* (7th ed.). New York: McGraw-Hill Medical.

Sussman, N. (2009). Selective serotonin reuptake inhibitors. In B. J. Sadock, V. A. Sadock, P. Ruiz, & H. I. Kaplan (Eds.), *Kaplan & Sadock's comprehensive textbook of psychiatry* (9th ed.). Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins.

Teter, C. J., Kando, J. C., Wells, B. G., & Hayes, P. E. (2008). Depressive disorders. In J. T. DiPiro, R. L. Talbert, G. C. Yee, G. R. Matzke, B. G. Wells, & L. M. Posey (Eds.), *Pharmacotherapy: A pathophysiologic approach* (7th ed.). New York: McGraw-Hill Medical.

Self, The

▶ Self-Identity

Self-Assessment

► Self-examination

Self-Attitude

► Self-Concept
► Self-image

Self-Blame

Stephanie Ann Hooker
Department of Psychology, University of Colorado, Denver, CO, USA

Synonyms

Responsibility

Definition

Self-blame is the attribution that the consequences one experiences are a direct result of one's actions or character. In the context of behavioral medicine, this may be either beneficial or harmful depending on if it leads to positive behavior change or increased negative affectivity and lack of behavior change.

Description

Self-blame is indirectly related to perceived control, where individuals who self-blame more often also are more likely to believe they have greater control over their lives. Because enhancements in perceived self-control are adaptive to psychological well-being, one may assume that self-blame may also be adaptive. However, this is not always the case.

Janoff-Bulman (1979) proposed two types of self-blame: (1) an adaptive, control-oriented response where the focus is on the individual's behavior and (2) a maladaptive, esteem-oriented response where the focus is on the individual's character. Self-blame is adaptive when individuals recognize that they had some control over the situation but failed to act appropriately. Thus, these individuals can modify their behavior for future events. On the other hand, self-blame is maladaptive when individuals blame their character flaws for the outcome; this is referred to as characterological self-blame. These flaws are generally seen as stable, so these individuals make no efforts to change. This can lead to recurrence of the same problems and feelings of helplessness and depression.

Although Janoff-Bulman's (1979) theory of self-blame seems plausible, there is little support for these notions in the literature. Two studies did not support the theory that behavioral self-blame is adaptive in breast cancer patients; rather, behavioral self-blame was positively associated with symptoms of anxiety and depression (Bennett, Compas, Beckjord, & Glinder, 2005; Glinder & Compas, 1999). Moreover, Bennett et al. found that both forms of self-blame were unrelated to perceptions of control, which directly contradicts the theory of self-blame.

However, one study of head and neck cancer patients supported Janoff-Bulman's (1979) theory. Low behavioral self-blame (smoking specific) was related to greater likelihood of continued smoking after the cancer diagnosis (Christensen et al. 1999). The relationship held for those patients with high and low perceived control over cancer recurrence, although those with low perceived control had almost a three times greater probability of continued smoking after a cancer diagnosis than those with higher perceived control over cancer recurrence. Furthermore, in this study, it appeared that specific behavior self-blame was more predictive of behavior than a general behavioral self-blame. The authors suggest this may be because of the patients' knowledge of how specific behaviors related to the likelihood of having cancer and that patients do not attribute their behavior in

general as the cause of cancer. This study illustrates the importance of understanding how patients' attributions of self-blame, perceived control, and knowledge of their condition may interact in predicting behavior change following a cancer diagnosis.

In the literature self-blame may be used interchangeably with responsibility. However, there are important differences between self-blame and responsibility that should be recognized. Self-blame differs from responsibility in that self-blame suggests that one intentionally brings about negative consequences, whereas responsibility is related more to perceived control over the event (Voth & Sirois, 2009). Indeed, Voth and Sirois demonstrated in their study of patients with inflammatory bowel disease that self-blame is related to poor psychological adjustment, whereas responsibility is related to better psychological adjustment. Self-blame was associated with increased use of avoidant coping strategies, whereas responsibility was associated with decreased use of avoidant coping strategies. Avoidant coping was related to poorer psychological adjustment.

Theories of self-blame in behavioral medicine suggest that self-blame has maladaptive and adaptive qualities. Self-blame can be adaptive when individuals recognize their past actions caused their negative consequences, and they also recognize that their behavior is modifiable. Thus, individuals can make positive behavior changes in these cases, which can improve health. However, self-blame is maladaptive when it is primarily characterological in nature. This may lead individuals to feel helpless and to have poorer psychological adjustment to disease. Thus, it is imperative for researchers to properly define the self-blame construct for their research in order to further understand self-blame's role in behavioral medicine.

References and Readings

Bennett, K. K., Compas, B. E., Beckjord, E., & Glinder, J. G. (2005). Self-blame and distress among women with newly diagnosed breast cancer. *Journal of Behavioral Medicine, 28*, 313–323.

Christensen, A. J., Moran, P. J., Ehlers, S. L., Raichle, K., Karnell, L., & Funk, G. (1999). Smoking and drinking behavior in patients with head and neck cancer: Effects of behavioral self-blame and perceived control. *Journal of Behavioral Medicine, 22*, 407–418.

Glinder, J. G., & Compas, B. E. (1999). Self-blame attributions in women with newly diagnosed breast cancer: A prospective study of psychological adjustment. *Health Psychology, 18*, 475–481.

Janoff-Bulman, R. (1979). Characterological versus behavioral self-blame: Inquiries into depression and rape. *Journal of Personality and Social Psychology, 37*, 1798–1809.

Voth, J., & Sirois, F. M. (2009). The role of self-blame and responsibility in adjustment to inflammatory bowel disease. *Rehabilitation Psychology, 54*, 99–108.

Self-care

Linda C. Baumann and Alyssa Karel
School of Nursing, University of Wisconsin-Madison, Madison, WI, USA

Synonyms

Self-management

Definition

Self-care as described by Orem (1995) is "action of persons who have developed or developing capabilities to use appropriate, reliable and valid measures to regulate their own functioning and development in stable or changing environments" (p. 43). Self-care is both caring "for" oneself and "by" oneself. Self-care promotes well-being and is a perceived condition of personal existence characterized by experiences of contentment, pleasure, and happiness. It is associated with health and with sufficiency of resources. This definition is consistent with Diener's (2009) concept of subjective well-being as an individual's global judgment of values and standards that are significant to life satisfaction.

A paradigm that is emerging in health care delivery for people with chronic conditions is that they are their own principal caregivers; health care professionals act as consultants and advisors in

supporting them in self-care and self-management of their condition. This paradigm of collaborative care and self-management education involves shared decision making between providers and patients. Self-management education includes providing patients with information, problem-solving skills, and behavioral strategies to enhance their lives.

Diabetes is an excellent example of a health condition that requires self-management skills to maintain optimal control through healthy eating, being active, taking medications, monitoring, problem solving, reducing risks, and healthy coping. http://www.diabeteseducator.org/DiabetesEducation/Patient_Resources/AADE7_PatientHandouts.html Self-management education can occur in group settings where peers can provide emotional support and practical information for problem solving. In addition to knowledge and skills, self-care behaviors are determined by attitudes and beliefs, social and environmental influences, and self-efficacy expectations.

References and Readings

Bodenheimer, T., Lorig, K., Holman, H., & Grumbach, K. (2002). Patient self-management of chronic disease in primary care. *Journal of the American Medical Association, 288*, 2469–2475.

Diener, E. (2009). *The science of well-being* (Social indicators book series, Vol. 37). New York: Springer.

Orem, D. E. (1995). *Nursing: Concepts and practice* (6th ed.). St. Louis, MO: Mosby.

Self-Concept

Tara McMullen
Doctoral Program in Gerontology, University of Maryland Baltimore and Baltimore County, Baltimore, MD, USA

Synonyms

Self-attitude; Self-identity; Self-image

Definition

Self-concept can be defined as one's beliefs about oneself.

Description

Self-concept is a difficult yet important terms to define as self-concept attempts to explain human behavior (micro). Defining self-concept is difficult as a large number of terms use the term 'self' to define some sort of individualistic behavior (Burns, 1980). However, in its simplest form, self-concept can be defined as one's beliefs about oneself. Carl Rogers (1951) suggested that oneself, or the "self," plays a role in the development of personality and behavior.

Self-concept can be seen as what an individual understands him or herself to be, cultivated by the appraisal of oneself (Epstein, 1973). Self-concept is an organized system of learned beliefs, perceptions, and feelings that aid in the understanding of oneself. Simply, self-concept is the perception an individual has of one's personal characteristics, formed and shaped by society and attitudes. The understanding of oneself is established by one's character, personality, traits, and appearance. Self-concept is developed from an individual's "I," "me," and/or "mine" experience (Burns, 1980).

As individuals age, their self-concept is organized and reorganized by their social and nonsocial experiences (Burns, 1980). Characteristics in an individual's social environment structure understanding of who the individual is (Epstein, 1973). The self-concept or the "who am I" assessed by the individual can be defined and then redefined as the individual encounters many life experiences. In this sense, self-concept can be designated as a multifaceted phenomenon that is dynamic and can change due to experiences, environments, and social affiliations (Markus & Wurf, 1987). Thus, self-concept is not fixed and is based individual on the context or situation. Moreover, Burns (1980) suggests that individuals may have many overlapping self-concepts that have been shaped and developed by various beliefs, experiences, and events.

Therefore, self-concept can be seen as a multifaceted and individualized process.

An individual can develop a positive or negative self-concept based entirely on the evaluations of oneself. Bailey (2003) states that self-concept is associated with individualistic qualities that can be assessed rather than measured. "Non-measurable" aspects to one's self can be seen as physical attributes, religious preferences, and/or personality traits (Bailey, 2003). Thus, self-concept is a learned trait. Rogers (1951) suggests that the self, through self-concept, must be maintained in order to avoid anxiety and stress. This ability to maintain one's self-concept can be achieved through the maintenance of self-esteem (Rosenberg, 1979). It has been suggested that self-concept consists of one's self-image and one's self-esteem (Burns, 1980). Self-image can be defined as the perception individuals have of themselves physically, psychologically, philosophically, and politically, developed through the agency of their societal experiences and development (Fisher, 1986; Statt, 1990). Self-esteem is operationally defined as an individual's orientation toward oneself (Rosenberg, 1965); self-worth, motivations, and perceptions encompass the conceptualization of an individual's understanding of who they are (Rosenberg, 1965). Thus, the process in which individuals view themselves constructed by the perceptions they have of their self-worth aids in the development of self-concept.

Self-concept can be measured in children and adults by means of various psychometric scales such as the Tennessee Self Concept Scale (Fitts, 1991) and the Piers-Harris Children's Self-Concept Scale (Piers, 1984).

Cross-References

- ▶ Self-Blame
- ▶ Self-esteem
- ▶ Self-examination

References and Readings

Bailey, J. A. (2003). Self-image, self-concept, and self-identity revisited. *Journal of the National Medical Association, 95*, 383–386.

Burns, R. B. (1980). *Psychology for the health professions*. Lancaster, England: MTP Press.

Epstein, S. (1973). The self-concept revisited – Or a theory of a theory. *The American Psychologist, 28*, 404–416.

Fisher, S. (1986). *Development and structure of the body image* (Vol. 1). Hillsdale, NJ: Erlbaum.

Fitts, W. H. (1991). Tennessee self-concept scale manual. Los Angeles: Western Psychological Services.

Harriman, P. L. (1947). *The new dictionary of psychology*. New York: The Philosophical Library.

Markus, H., & Nurius, P. (1986). Possible selves. *The American Psychologist, 41*, 954–969.

Markus, H., & Wurf, E. (1987). The dynamic self-concept: A social psychological perspective. *Annual Review of Psychology, 38*, 299–337.

Piers, E.V. (1984). Revised manual for the Piers-harris Children's Self-Concept Scale. Los Angeles: Western Psychological Services.

Piers, E. V. (1986). *The Piers-Harris children's self-concept scale, revised manual*. Los Angeles: Western Psychological Services.

Rogers, C. R. (1951) Client-Centered Counselling, Boston: Houghton-Mifflin.

Rosenberg, M. (1965). *Society and the adolescent self-image*. Princeton, NJ: Princeton University Press.

Rosenberg, M. (1979). *Conceiving the self*. New York: Basic Books.

Rosenberg, M. (1986). *Conceiving the self*. Malabar, FL: Krieger.

Rosenberg, M. (1989). *Society and the adolescent self-image*. Middeltown, CT: Wesleyan University Press.

Statt, D. (1990). *The concise dictionary of psychology*. New York: Routledge.

Self-Conception

- ▶ Self-image

Self-Consciousness

- ▶ Self-image

Self-Construal

- ▶ Self-Identity

Self-Control

▶ Behavioral Inhibition
▶ Self-Regulation Model

Self-Control Capacity

▶ Self-Regulatory Capacity

Self-Control Failure

▶ Self-Regulatory Fatigue

Self-determination Theory

Hannah G. Lawman and Dawn Wilson
Department of Psychology, University of South Carolina, Columbia, SC, USA

Synonyms

Cognitive evaluation theory

Definition

Self-determination theory is a theory of human motivation that describes two distinct types of motivation: autonomous (regulated through natural and internal processes such as inherent satisfaction) and controlled (regulated through externally held demands and expectations). Furthermore, autonomous motivation is more likely to contribute to long-term maintenance of a behavior compared to controlled motivation and can be facilitated through social contextual conditions (i.e., high autonomy, competence, and relatedness) that elicit and sustain intrinsic motivation versus conditions that undermine one's innate propensity for it.

Description

Self-determination theory (SDT) is a theory of human motivation that describes motivation in two distinct types: autonomous and controlled (Deci & Ryan, 2008). Autonomous motivation is regulated through natural and internal processes such as inherent satisfaction and can be thought of as an individual's innate desire to engage in healthy behaviors independent of external influences. For example, an individual may engage in autonomously motivated healthy eating because it is part of his/her self-concept and is enjoyable. On the other hand, controlled motivation is regulated through externally held demands and expectations that are contingent on rewards or punishments. An individual who engages in healthy eating because he/she has a high need for approval from others may not really enjoy eating healthy but is motivated by an external reinforcement. While both types of motivation represent an individual's intention to act, health behavior outcomes resulting from autonomous versus controlled motivation may be qualitatively different. For example, autonomous motivation is more likely to contribute to long-term maintenance of health behaviors compared to controlled motivation. Additionally, autonomous motivation can be facilitated through social contextual conditions that elicit and sustain intrinsic motivation in contrast to conditions that undermine one's innate propensity for it (Ryan & Deci, 2000).

Self-determination theory provides a motivational-theory-based framework for understanding influences on health behaviors, such as healthy diet, physical activity, safe sex practices, and substance use. The conceptualization of motivation on a continuum allows for distinctions to be made in the type and quality of motivation that may contribute to different outcomes. This can be compared to previous interpretations of motivation in which researchers' conceptualized motivation as unitary and, as such, universally concluded that more motivation would be better. SDT makes distinctions between autonomous and controlled motivation with autonomous motivation being more inherently enjoyable, long-lasting, and internally

regulated, while controlled motivation consists of motivation that is primarily driven by externally held demands, expectations and reinforcers (Deci & Ryan, 2008). For example, controlled motivation based on extrinsic values of fame and wealth has been shown to be related to higher risk for smoking (Williams, Cox, Hedberg, & Deci, 2000).

SDT originally conceptualized the motivation continuum as ranging from extrinsic to intrinsic motivation. On the left end, motivation was regulated extrinsically and controlled by rewards and punishments or other externally regulated processes. This was called extrinsic motivation. At the right end of the continuum was motivation that was regulated or controlled by an individual's inherent satisfaction, novelty, and drive called intrinsic motivation. Furthermore, extrinsic motivation was broken down into subcategories based on increasing levels of intrinsic regulation: extrinsic, introjected (i.e., somewhat external regulation or internal rewards and/or punishments), and identified (somewhat internal regulation and holds personal importance; Deci & Ryan, 1985; Ryan & Deci, 2000). Previous research has supported beneficial effects of intrinsic motivation compared to extrinsic motivation in sport (Vallerand & Losier, 1999). However, more recent research has combined extrinsic and introjected into controlled-type motivation and combined identified and intrinsic into autonomous-type motivation (Deci & Ryan, 2008). This has resulted in a shift of the primary motivation differentiation moving toward autonomous and controlled in conceptualizing intrinsic and extrinsic in a more dynamic fashion.

SDT emphasizes the role of social context in understanding health behavior motivation and suggests its influence on behavior is through affecting social contextual conditions that may help to elicit and sustain intrinsic motivation. These conditions are described as psychological needs that are inherent to being human and consist of the needs for competence, autonomy, and relatedness. Social relationships (e.g., social support), environmental characteristics (e.g., built environment, resources), and cultural practices and norms (e.g., gender roles) can influence these psychological needs and in turn facilitate or undermine one's sense of intrinsic motivation for engaging in healthy behaviors (Deci & Ryan, 1985, 2008; Ryan & Deci, 2000).For example, an intervention to increase physical activity may focus on increasing social support (relatedness), teaching behavioral skills (competence), and encouraging choice (autonomy) to provide conditions that facilitate the development of autonomous motivation to be physically active for a lifetime (Wilson et al., 2008). Indeed, researchers have begun to use SDT as a framework for large-scale health behavior interventions, such as the Active by Choice Today (ACT) trial to increase physical activity (Wilson et al.). Similarly, researchers and clinicians interested in reducing substance use may aim to teach strategies for resisting peer pressure (competence) to use substances while preserving those peer relationships (relatedness) and giving a variety of response options (autonomy) (Williams et al., 2000). In conclusion, SDT has been shown to be a promising theory for behavioral health change and emphasizes social context in understanding motivation as a dynamic construct.

Cross-References

- ▶ Health Behaviors
- ▶ Motivational Interviewing

References and Readings

Deci, E. L., & Ryan, R. M. (1985). *Intrinsic motivation and self-determination in human behavior*. New York: Plenum Press.

Deci, E. L., & Ryan, R. M. (2008). Facilitating optimal motivation and psychological well-being across life's domains. *Canadian Psychology/Psychologie canadienne, 49*(1), 14–23.

Ryan, R. M., & Deci, E. L. (2000). Self-determination theory and the facilitation of intrinsic motivation, social development, and well-being. *American Psychologist, 55*(1), 68–78.

Vallerand, R. J., & Losier, Gt. F. (1999). An integrative analysis of intrinsic and extrinsic motivation in sport. *Journal of Applied Sport Psychology, 11*(1), 142–169.

Williams, G. C., Cox, E. M., Hedberg, V. A., & Deci, E. L. (2000). Extrinsic life goals and health-risk behaviors in adolescents. *Journal of Applied Social Psychology, 30*(8), 1756–1771.

Wilson, D. K., Kitzman-Ulrich, H., Williams, J. E., Saunders, R., Griffin, S., Pate, R., et al. (2008). An overview of "The Active by Choice Today" (ACT) trial for increasing physical activity. *Contemporary Clinical Trials, 29*(1), 21–31.

Self-Directed Violence

► Suicide

Self-Efficacy

Jorie Butler
Department of Psychology, University of Utah, Salt Lake City, UT, USA

Definition

Self-efficacy: Self-efficacy is the belief in personal ability to successfully perform challenging life tasks. Self-efficacy plays an important role in a person's emotions, cognitions, motivational activities, and behaviors across a variety of activities.

Description

Self-efficacy is rooted within Social Cognitive Theory (Bandura, 1986) in which people are characterized as active agents of control within their own lives – dynamically influencing their personal environments by organizing responses to opportunities, reflecting on past performances, and self-regulating behavior. Self-efficacy influences response organization, develops in part from reflection, and contributes to self-regulation, particularly, by fostering approach-oriented behavior and persistence in the face of obstacles. Attribution Theory (Weiner, 1992), which includes the properties of locus, stability, controllability, also provides a framework for understanding self-efficacy. Locus reflects the cause of a situation as internal or external to a person. Stability reflects how changeable the situation is perceived to be. Controllability is an indicator of whether the person can willfully change a situation. A negative situation such as reaching an unhealthy weight could be interpreted as internal (*Being this heavy is my fault*), stable (*I've always been too heavy*), and uncontrollable (*It doesn't matter what I eat, I just get heavier*), resulting in poor self-efficacy for weight control in the future. In contrast, reaching an unhealthy weight could be interpreted as external (*Weight gain is common with age*), unstable (*This weight came on, it can come off!*), and controllable (*Now that I realize I'm eating too much and moving too little, I can change that behavior*), producing opportunities for improvements in future self-efficacy.

Social Learning Theory (Bandura, 1977b) explains development of self-efficacy via four principle pathways: mastery experiences, modeling, social persuasion, and physiology. Past experiences are most predictive of future experiences. People engage in tasks and actions, interpret the outcome, and develop perceptions of their competence within the task domain. Mastery experiences result from multiple successes and promote self-efficacy. Failures are detrimental to developing self-efficacy, although the negative impact of failures is diminished when failures occur attempting a task that has been successfully completed on multiple occasions. The primacy of mastery experiences in self-efficacy development speaks to the titration between efficacy development and actual performance. Self-efficacy develops in domains in which skills are acquired often through the combination of effortful practice and natural talent. Self-efficacy development can be fostered by modeling, particularly when successful completion of activities is modeled by someone viewed as admirable and possessing desired capabilities of the observer. Conversely, a model who fails to perform a desired activity can weaken an observer's sense of competence

for performing the activity. Social persuasions involve effective encouragement (not empty praise) that can be instrumental in fostering self-efficacy when the tasks are achievable or nearly achievable. Negative social persuasion deflates self-efficacy. Social persuasion is an important pathway by which parents, teachers, coaches, employment supervisors, and others can facilitate or damage developing self-efficacy. Physiological responses during task attempts influence self-efficacy development. Emotional states indicative of negative arousal such as stress and fear are indicators that the task is difficult and may indicate anticipated failure. In contrast, excitement, anticipation of fun, happiness, or a sense of work "flow" indicate positively developing efficacy. Individuals experiencing negative adjustment periods – such as depressive states or grief – will have difficulty developing self-efficacy. Self-efficacy can develop more freely when negative emotional states are resolved.

Self-efficacy influences behavior across many domains, including the choices of activities to become involved in effortful work to complete activities, persistence in the face of setbacks or failures, and resilience following adversity (Schunk & Pajares, 2005). Individuals tend to avoid activities for which they anticipate poor performance and approach tasks for which they anticipate success. Thus, individuals with low self-efficacy in a given domain may avoid it all together, thus contributing to narrowing of life skills. High self-efficacy contributes to task engagement. High self-efficacy is also associated with effortful engagement in tasks – particularly when intrinsic motivation to engage in the task is also present. Persistence in the face of setbacks is more likely when individuals have high self-efficacy for the task. With the expectation for eventual failure (a component of low self-efficacy), persistence is unlikely. In addition, when failures or enduring obstacles are encountered, low self-efficacy may contribute to a sense of failure and withdrawal from the situation whereas high self-efficacy may contribute to resilience.

Self-efficacy is an integral component of a number of models designed to explain health behavior – primarily because self-efficacy contributes to the willingness to try to change undesirable health behavior, successful implementation of health behavior change, and persistently maintaining health behavior change over time. The Theory of Reasoned Action was extended to form the Theory of Planned Behavior. The extension incorporated self-efficacy as a key factor in changing health behavior along with personal attitudes toward change and the attitudes of significant others toward the change (Ajzen, 1991). Self-efficacy was also incorporated into the Health Belief Model during the 1980s – reflecting understanding of the importance of the construct in promoting health (Rosenstock, Strecher & Becker, 1988). The Health Behavior Change Model (Prochaska & Velicer, 1997) incorporates self-efficacy as a contributor to stages of change. Self-efficacy can influence a person's thoughts about needing change (in the contemplation stage). A person high in self-efficacy will anticipate success and may progress more quickly to the active preparation stage – involving active planning for change behaviors. In addition, self-efficacy will influence effective active change (action stage) as persons high in self-efficacy may more effectively respond to setbacks with persistence and resilience. This quality of those high in self-efficacy also contributes to effective sustainment of the changed behavior (maintenance stage).

Self-efficacy is generally best understood in specific domains and the concept is well supported across multiple domains including school performance, athletic achievement, occupational arenas, and in health behaviors including health maintenance (such as diet and exercise), recovery from acute events such as surgery, and coping with chronic illness or dangerous diagnoses. There is some evidence for a generalized self-efficacy as individuals expect better competence for activities when they have demonstrated aptitude in other activities in the past (Smith, 1989). Self-efficacy contributes a theoretically grounded explanation for the myriad ways in which people shape their own environments by seeking out opportunities for success, persisting in the face of hard work and adversity, learning from past experiences, and responding with resilience to failure.

Cross-References

- ▶ Efficacy
- ▶ Efficacy Cognitions
- ▶ Hopelessness
- ▶ Locus of Control
- ▶ Salutogenesis
- ▶ Self-Concept
- ▶ Self-Image
- ▶ Theory of Planned Behavior
- ▶ Theory of Reasoned Action

References and Readings

Ajzen, I. (1991). The theory of planned behavior. *Organizational Behavior and Human Decision Processes, 50*, 179–211.

Ajzen, I., & Fishbein, M. (1980). *Understanding attitudes and predicting social behavior*. Englewood Cliffs: Prentice-Hall.

Bandura, A. (1977a). Self-efficacy: Toward a unifying theory of behavioral change. *Psychological Review, 84*, 191–215.

Bandura, A. (1977b). *Social learning theory*. Englewood Cliffs: Prentice-Hall.

Bandura, A. (1986). *Social foundations of thought and actions: A social cognitive theory*. Englewood Cliffs: Prenctice-Hall.

Fishbein, M., & Ajzen, I. (1975). *Belief, attitude, intention, and behavior: An introduction to theory and research*. Reading, MA: Addison-Wesley.

Prochaska, J. O., & Velicer, W. F. (1997). The transtheoretical model of health behavior change. *American Journal of Health Promotion, 12*, 38–48.

Rosenstock, I. M., Strecher, V. J., & Becker, M. H. (1988). Social learning theory and the health belief model. *Health Education Quarterly, 15*, 175–183.

Schunk, D. H., & Pajares, F. (2005). Competence perceptions and academic functioning. In A. J. Elliot & C. S. Dweck (Eds.), *Handbook of competence and motivation* (pp. 84–104). New York: Guilford Press.

Smith, R. E. (1989). Effects of coping skills training on generalized self-efficacy and locus of control. *Journal of Personality and Social Psychology, 56*, 228–233.

Walker, J. (2001). *Control and the psychology of health: Theory, measurement and applications*. Buckingham: Open University Press.

Weiner, B. (1986). *An attributional theory of motivation and emotion*. New York: Springer.

Weiner, B. (1992). *Human motivation: Metaphors, theories, and research*. Newbury Park: Sage.

Self-Esteem

Shin-ichi Suzuki[1] and Koseki Shunsuke[2]
[1]Faculty of Human Sciences, Graduate School of Human Sciences, Waseda University, Tokorozawa-shi, Saitama, Japan
[2]Department of School Education, Aichi University of Education, Kariya-shi, Aichi, Japan

Synonyms

Pride; Self-respect

Definition

Self-esteem can be defined as a positive self-evaluation or a concept broader than confidence. It refers to an individual's cognitive appraisal that is constant over time. A positive self-appraisal indicates higher self-esteem, and a negative self-appraisal indicates lower self-esteem. Self-esteem is not perceived anytime, but it essentially influences one's actions, consciousness, or attitude. One who is perceived to have high self-esteem pursues goals aggressively and actively. Further, they are perceived to be amiable by themselves or by others. In this sense, self-esteem becomes indispensable to mental health or social adaptation.

In the previous study concerning self-esteem, an individual's self-esteem was considered in terms of not only his or her tendency and degrees of appraisal, which could be positive or negative, but also its relationship with the individual's cognitive faculty. James (1890) propounded that "self-esteem is successes divided by desire." This formula suggests that just thinking that one could succeed in the desired field increases self-satisfaction. This formula is similar to the theory on the gap between ideal self and real self (Rogers, Dorfman, Gordon, & Hobbs, 1951).

An individual's self-esteem strongly correlates with the affection, unconditional acceptance, and nurturing attitude that the parents display. Self-esteem can be measured in various ways, the most representative being Rosenberg's (1965)

questionnaire and Coopersmith's (1967) scales. Because each scale may be different in terms of its dimensionalities or factors, it is necessary to consider the characteristics of the scales used or interpreted on a case-by-case basis.

Cross-References

- ▶ Attitudes
- ▶ Cognitive Appraisal
- ▶ Self-Concept

References and Readings

Coopersmith, S. (1967). *The antecedents of self-esteem*. San Francisco: WH Freeman.
James, W. (1890). *The principles of psychology*. New York: Holt.
Rogers, C. R., Dorfman, E., Gordon, T., & Hobbs, N. (1951). *Client-centered therapy: Its current practice, implications, and theory*. Boston: Houghton Mifflin.
Rosenberg, M. (1965). *Society and adolescent self-image*. New Jersey: Princeton University Press.

Self-Evaluate

- ▶ Self-Monitoring

Self-Evaluation

- ▶ Self-examination

Self-examination

Tara McMullen
Doctoral Program in Gerontology, University of Maryland Baltimore and Baltimore County, Baltimore, MD, USA

Synonyms

Self-assessment; Self-evaluation; Self-monitoring; Self-rating

Definition

Self-examination can be seen as a process of evaluation or appraisal of one's qualities, traits, and characteristics.

Description

Self-examination can be seen as a process of evaluation or appraisal of one's qualities, traits, and characteristics. The evaluation of one's qualities, traits, and characteristics helps develop one's self-image and aids in the development of one's self-awareness or self-concept. Self-examination may result in a positive or negative self-feeling, which may enhance or decrease individuals' ideas about themselves. The evaluation of oneself can be seen as the attempt to understand one's motivations and behaviors. In addition, the examination of oneself is likely to affect self-esteem, which may affect self-image. Individuals who deem themselves as worthy may have a greater self-esteem, which may maintain one's "self." Rogers (1951) suggests that the self must be maintained in order to avoid anxiety and stress. Further, theorists often define self-examination as the evaluation or rating of oneself. This evaluation or rating is often defined as the degree to which an individual is self-aware and may result in a negative or positive self-actualization. This formed self-awareness helps develop the basis of individual self-regulation or the ability of one to control one's behaviors and actions (Hull, 2002). Therefore, an individual defines one's self-concept from the degree to which the individual self-examines their personal traits, motivations, behaviors.

The act of self-examination may result in individuals self-monitoring themselves. Self-monitoring can be seen as the degree to which an individual manages or controls the image presented to others in social circumstances (Rawn, 2007). An individual who is a "high self-monitor" may be adept at self-monitoring and motivated to alter individual behavior in order to impact the responses of peers in social

situations (Rawn, 2007). An individual who is a "low self-monitor" remains constant in individual behavior and will not alter individual behavior in social situations (Rawn, 2007). Further, the act of self-examination may result in self-criticism, or the awareness that individual traits and/or characteristics do not compare to the ideal self-image one may have for oneself. This self-criticism may be a result of the self-examination of one's strengths and weaknesses and may increase the evaluation of one's image. One may examine and reexamine individual strengths and weaknesses in order to measure self-actualization, or the fulfillment of one's potential (Maslow, 1943, 1976). Therefore, the greater the self-actualization, the more defined one's self-concept may be, and possibly the greater the self-acceptance an individual may have for oneself. Self-acceptance is defined as the attitude toward one's self and ones individual qualities (English & English, 1958).

Research that explores how self-evaluation impacts everyday situations has become common. For example, Judge, Locke, and Durham (1997), in a study exploring workplace job satisfaction, found that individuals with greater self-evaluations were more likely to have a higher self-esteem, self-efficacy, personal control, and emotional stability and were motivated to perform well in the workplace. Judge et al. (1997) conceptually define self-esteem, self-efficacy, locus of control, and "neuroticism-stability" as core self-evaluations. Judge et al. suggest that core self-evaluations are the "fundamental, subconscious conclusions individuals reach about themselves, other people, and the world" (Judge, Locke, Durham, & Kluger, 1998).

Cross-References

- ▶ Self-blame
- ▶ Self-concept
- ▶ Self-esteem
- ▶ Self-identity
- ▶ Self-image

References and Readings

English, H. B., & English, A. C. (1958). A comprehensive dictionary of psychology and psychoanalytic terms. New York: Longmans, Green.

Hull, J.G. (2002). Modeling the structure of self-knowledge and the dynamics of self-regulation. In A. Tesser, D.A. Stapel, & J.V. Wood (Eds.). Self and Motivation: Emerging Psychological Perspectives (pp. 173–206). Washington, DC: American Psychological Association.

Judge, T. A., Locke, E. A., & Durham, C. C. (1997). The dispositional causes of job satisfaction: A core evaluations approach. *Research in Organizational Behavior, 19*, 151–188.

Judge, T. A., Locke, E. A., Durham, C. C., & Kluger, A. N. (1998). Dispositional effects on job and life satisfaction: The role of core evaluations. *Journal of Applied Psychology, 83*, 17–34.

Maslow, A. H. (1943). A theory of human motivation. *Psychological Review, 50*, 37.

Maslow, A. H. (1976). Self-actualization psychology. In J. Fadiman & R. Frager (Eds.), *Personality and personal growth*. New York: Harper Collins.

Rawn, C. D., Mead, N., Kerkhof, P. & Vohs, K. D. (2007). The influence of self-esteem and ego threat on decision making. In K. D. Vohs, R. F. Baumeister, & G. Loewenstein (Eds.), Do Emotions Help or Hurt Decision Making? A Hedgefoxian Perspective (pp. 157–182). New York : Russell Sage Foundation Press.

Rogers, C. (1951a). Perceptual reorganization in client-centered therapy. In R. R. Blake & G. V. Ramsey (Eds.), Perception: An approach to personality. New York: Ronald Press.

Rogers, C. (1951b). Client-centered therapy (pp. 13–71). Boston: Houghton Mifflin Company.

Self-Identity

Katherine T. Fortenberry[1], Kate L. Jansen[2] and Molly S. Clark[2]
[1]Department of Family and Preventative Medicine, The University of Utah, Salt Lake City, UT, USA
[2]Department of Family Medicine, University of Mississippi Medical Center, Jackson, MS, USA

Synonyms

Self-concept; Self-construal; Self-perspective; Self-schema; Self-system; Self, The; Sense of self

Definition

Self-identity can be conceptualized as a dynamic, contextually based system (Baumeister, 1998). It is a complex structure centered in memory and cognition that helps define who we are, how we relate to others, and our place in the world (Swann & Bosson, 2008). It is also considered a key motivating force that influences personality and behavior. Self-identity is thought to drive our interactions with others (Andersen & Chen, 2002), goals and future roles (Markus & Wurf, 1987), and experience of emotions (Higgins, 1989). Self-identity is also believed to regulate and motivate behavior by providing key self-regulation through a feedback system (Carver & Scheier, 2002).

Description

A person's self-views are considered fundamental to how he or she interprets events, experiences emotion, and behaves. Individuals have distinct identities in different social roles, and differentiation into multiple role-related selves (e.g., self as a student, self as an athlete) is a process of normal development that begins in adolescence or earlier (Oosterwegel & Oppenheimer, 2002). Self-identity differs in content and structure across individuals, and likely varies as a function of culture or gender (Cross & Madson, 1997).

Research has examined the importance of the organization of positive and negative attributes within self-identity, namely, compartmentalization (i.e., negative attributes enclosed within a single role) and integration (i.e., negative attributes spread across multiple roles; see Showers & Zeigler-Hill, 2007). The structure of self-identity is considered contextual and fluctuates between situations as different aspects of the self-structure are activated, strongly relating to mood and self-esteem. A person with a compartmentalized self-structure would experience more positive moods when a role containing predominantly positive attributes is activated frequently, but experience negative moods when a role containing negative attributes is activated. In contrast, both positive and negative attributes are frequently activated in an integrated self-structure, moderating the adverse emotional consequences of activating negative beliefs.

Self-identity is also conceptualized as dynamic. Andersen and Chen (2002) take an interpersonal developmental approach to understanding self-identity in different situations. In their theory of the relational self, they suggest that self-identity develops through interactions with significant others, who set *exemplars*, or cognitive templates stored in long-term memory. These exemplars are set in motion by environmental cues, so that behavior with different individuals varies based upon the active exemplar. Therefore, *who we are now* may differ when with different people.

In addition to describing *who we are now*, views of self-identity also describe *who we may become*. Higgins (1987) suggests that a driving force in self-identity is comparison of the *actual* self to the *ideal* self and *ought* self. A discrepancy between the selves is thought to cause negative psychological states, which initiate behavior that is designed to reduce the discrepancy. Similarly, Carver and Scheier (1998) describe that all individuals strive toward goals, which organize and motivate behavior. Comparisons between future goals and current behavior create emotions that drive future behavior. Positive emotions are experienced and behavior is reinforced when an action is judged to move us closer to a goal; negative emotions are experienced and behavior may change when an action is inconsistent with attaining a goal. Therefore, self-identity is part of a regulatory system that not only reflects, but also drives, who we are and who we will become.

Markus and Nurius (1986) suggest that current self-identity is strongly influenced by *possible selves,* or who we either want to become, or fear becoming, in the future. In this view, self-identity is fluid, continuously developing as possible selves are achieved, modified, or relinquished. Possible selves are thought to directly influence current behavior by providing movement toward or away from possible selves (i.e., approaching hoped-for selves or avoiding feared selves).

Exposure to possible selves has been shown to influence exercise behavior (Ouellette, Hessling, Gibbons, Reis-Bergan, & Gerrard, 2005) and school involvement in adolescents (Oyserman, Terry, & Bybee, 2002) over time. Possible selves are thus considered to play key roles in self-regulatory processes of motivation and behavior (Hoyle & Sherrill, 2006). By comparing current self-identity with future selves, possible selves provide a framework in which to interpret and contextualize *who we are now*.

Self-identity is important for Behavioral Medicine because one's self-conceptions can influence how one responds to chronic illness, and can be altered by the experience of chronic illness. As views of the future are highly impacted by circumstances, major life events are likely to lead to changes in these views and, likewise, to changes in self-identity (Tesser, Crepaz, Collins, Cornell, & Beach, 2002). The diagnosis of a chronic illness is an example of this type of event. Adverse outcomes are likely if an illness contains attributes that reflect negatively onto an individual's self-identity. For example, individuals with lung cancer are more likely than individuals with prostate or breast cancer to associate stigma and self-blame with their illness, leading to negative psychological outcomes (Else-Quest, LoConte, Schiller, & Hyde, 2009). However, a growing body of literature suggests that positive outcomes such as perceptions of personal growth, improvements in life priorities and important relationships, and positive changes in personality (i.e., increased patience, tolerance, and empathy) can also occur as a result of dealing with adverse circumstances such as a chronic illness (Pakenham, 2005; Tedeschi, & Calhoun, 2004).

Self-identity is also relevant to health promotion and illness prevention behaviors. Self-identity plays key roles in self-regulatory processes of motivation and behavior (Hoyle & Sherrill, 2006), with the potential to influence health behavior in both positive and negative ways. For example, contemplating an image of a future possible self as an exerciser or non-exerciser influenced exercise behavior at four-week follow-up (Ouellette et al., 2005). Additionally, individuals' self-identity related to both smoking and quitting smoking independently predicted future attempts to quit. In contrast, sexually active teens who viewed STD's as more stigmatized were significantly less likely to have received STD screening over the last year (Cunningham, Kerrigan, Jennings, & Ellen, 2009). Goals that individuals wish to achieve, and views of who they are as they achieve these goals, are thought to be continuously present in the self-concept, providing a feedback loop that regulates these behaviors.

Cross-References

- ▶ Benefit Finding
- ▶ Chronic Disease Management
- ▶ Posttraumatic Growth
- ▶ Self-Image
- ▶ Self-Regulation
- ▶ Stigma

References and Readings

Andersen, S. M., & Chen, S. (2002). The relational self: An interpersonal social-cognitive theory. *Psychological Review, 109*, 619–645.

Baumeister, R. R. (1998). The self. In D. T. Gilbert, S. T. Fiske, & G. Lindzey (Eds.), *The handbook of social psychology* (4th ed., Vol. 1, pp. 680–740). New York: McGraw-Hill.

Carver, C. S., & Scheier, M. F. (1998). *On the self-regulation of behavior*. New York: Cambridge University Press.

Carver, C. S., & Scheier, M. F. (2002). Coping processes and adjustment to chronic illness. In A. J. Christensen & M. H. Antoni (Eds.), *Chronic physical disorders: Behavioral medicine's perspective* (pp. 47–68). Malden, MA: Blackwell.

Cross, S. E., & Madson, L. (1997). Models of the self: Self-construals and gender. *Psychological Bulletin, 122*, 5–37.

Cunningham, S. D., Kerrigan, D. L., Jennings, J. M., & Ellen, J. M. (2009). Relationships between perceived STD-related stigma, STD-related shame and STD screening among a household sample of adolescents. *Perspectives on Sexual and Reproductive Health, 41*, 225–230.

Else-Quest, N. M., LoConte, N. K., Schiller, J. H., & Hyde, J. S. (2009). Perceived stigma, self-blame, and adjustment among lung, breast, and prostate cancer patients. *Psychology and Health, 24*, 949–964.

Higgins, E. T. (1987). Self-discrepancy: A theory related self and affect. *Psychological Review, 94*, 319–340.

Higgins, E. (1989). Continuities in self-regulatory and self-evaluative processes: A developmental theory relating self and affect. *Journal of Personality, 57*, 407–444.
Hoyle, R. H., & Sherrill, M. R. (2006). Future orientation in the self-system: Possible selves, self-regulation, and behavior. *Journal of Personality, 74*(6), 1674–1696.
Markus, H., & Nurius, P. (1986). Possible selves. *American Psychologist, 41*, 954–969.
Markus, H., & Wurf, E. (1987). The dynamic self-concept: A social psychological perspective. *Annual Review of Psychology, 38*, 299–337.
Oosterwegel, A., & Oppenheimer, L. (2002). Jumping to awareness of conflict between self-representations and its relation to psychological wellbeing. *International Journal of Behavioral Development, 26*, 548–555.
Ouellette, J. A., Hessling, R., Gibbons, F. X., Reis-Bergan, M., & Gerrard, M. (2005). Using images to increase exercise behavior: Prototypes versus possible selves. *Personality and Social Psychology Bulletin, 31*, 610–620.
Oyserman, D., Terry, K., & Bybee, D. (2002). A possible selves intervention to enhance school involvement. *Journal of Adolescence, 25*, 313–326.
Pakenham, K. I. (2005). Benefit finding in multiple sclerosis and associations with positive and negative outcomes. *Health Psychology, 24*, 123–132.
Showers, C. J., & Zeigler-Hill, V. (2007). Compartmentalization and integration: The evaluative organization of contextualized selves. *Journal of Personality, 75*(6), 1181–1204.
Swann, W. B., & Bosson, J. K. (2008). Identity negotiation: A theory of self and social interaction. In R. W. Robins & L. A. Pervin (Eds.), *Handbook of personality psychology: Theory and research* (3rd ed., pp. 448–471). New York: Guillford Press.
Tedeschi, R. G., & Calhoun, L G. (2004). Posttraumatic growth: Conceptual foundations and empirical evidence. *Psychological Inquiry, 15*, 1–18.
Tesser, A., Crepaz, N., Collins, J. C., Cornell, D., & Beach, S. R. H. (2002). Confluence of self-esteem regulation mechanisms: On integrating the self-zoo. *Personality and Social Psychology Bulletin, 26*, 1476–1489.

Self-image

Tara McMullen
Doctoral Program in Gerontology, University of Maryland Baltimore and Baltimore County, Baltimore, MD, USA

Synonyms

Self-attitude; Self-concept; Self-conception; Self-consciousness; Self-identity

Definition

Self-image is how an individual thinks they should be (English & English, 1958). An individual's self-image is comprised of many attitudes, opinions, and ideals.

Description

Like self-concept, self-image is conceptually a difficult term to define due to the large number of varied terms using "self" as a phrase to define some sort of behavior (Burns, 1980). However, self-image is important as it delineates how a self-aware individual views themselves or their image, which further establishes one's self-concept (Harriman, 1947). Self-image is how an individual thinks they should be (English & English, 1958). An individual's self-image is comprised of many attitudes, opinions, and ideals. Self-image develops at a young age and is a process which develops throughout the lifespan. Beginning at a young age, self-image can be seen as a physical process (Statt, 1990). However, self-image is developed not only from body image and physical aspects but also from concepts shaped by society and attitudes.

Self-image is a measurable assessment of one's characteristics. Measurable aspects belonging to an individual build and maintain an individual's self-image. Measurable aspects can be identified as achievements and appearance (Bailey, 2003). Self-image is how an individual perceives him/herself based on measurable traits developed at an early age (Bailey, 2003). Like self-concept, an individual's self-image may be a learned trait structured from an individual's attitude toward a group, idea, object, and so forth (Rosenberg, 1989). Thus, defined behavior may emerge from self-image; however, this behavior is not seen as fixed as it may deviate with the occurrence of different experiences and roles (Burns, 1980). In addition, self-image may be affected by an individual's self-esteem. Self-esteem is defined as an individual's orientation toward oneself (Rosenberg, 1965). Rosenberg (1989) suggested that the more uncertain an individual is in regard to who they perceive he/she is, the more likely the individual will have a lowered self-esteem. A lower

degree of self-esteem may render a negative self-image, diverging from the ideal self-image individuals may hold for themselves (Burns, 1980). The ideal self can be defined as who an individual aspires to be. The ideal self is one part of self-concept and helps individuals better evaluate who they are (Burns, 1980).

How individuals perceive who they are may depend on the individual's social environment and/or culture. Self-image can further be characterized as the perception an individual has of who he/she is physically, psychologically, philosophically, and politically, developed through the agency of individual societal experiences and development (Fisher, 1986; Statt, 1990). Individuals may develop a self-image that is associated with societal roles and norms (Markus & Kityama, 1991). Individuals in collectivist or individualistic cultures may establish who they are based on the culture in which they were raised. A collectivist culture accentuates individual's social roles and responsibilities within the context of social groups, while an individualistic culture accentuates individual identity and achievements (Nevid, 2009). Thus, experiences and culture aid in the development of one's self-image. In defining individual roles, culture imparts a strong effect on the individual self-image.

Self-image has been measured in many populations by means of various psychometric scales such as the Rosenberg Self-Esteem Scale (Rosenberg, 1989). Further, theory, such as the Social Identity Theory (Tajfel & Turner, 1979) and the Objective Self-Awareness Theory (Duval & Wicklund, 1972) have emerged from interpretations of self-image and self-consciousness.

References and Readings

Bailey, J. A. (2003). Self-image, self-concept, and self-identity revisited. *Journal of the National Medical Association, 95*, 383–386.

Burns, R. B. (1980). *Psychology for the health professions*. Lancaster, England: MTP Press.

Duval, T. S., & Wicklund, R. A. (1972). *A theory of objective self-awareness*. New York: Academic Press.

English, H. B., & English, A. C. (1958). *A comprehensive dictionary of psychology and psychoanalytic terms*. New York: Longmans, Green.

Fisher, S. (1986). *Development and structure of the body image* (Vol. 1). Hillsdale, NJ: Erlbaum.

Fitts, W. H. (1991). *Tennessee self concept scale, manual*. Los Angeles: Western Psychological Services.

Harriman, P. L. (1947). *The new dictionary of psychology*. New York: The Philosophical Library.

Markus, H., & Kitayama, S. (1991). Culture and the self: Implication for cognition, emotion, and motivation. *Psychology Review, 98*, 224–253.

Markus, H., & Nurius, P. (1986). Possible selves. *The American Psychologist, 41*, 954–969.

Markus, H., & Wurf, E. (1987). The dynamic self-concept: A social psychological perspective. *Annual Review of Psychology, 38*, 299–337.

Nevid, J. S. (1990). *Essentials of psychology: Concepts and applications* (2nd ed.). Boston: Houghton Mifflin Company.

Nevid, J. S. (2009). *Psychology: Concepts and applications* (3rd ed.) Belmont, CA: Cengage.

Rogers, C. (1951a). Perceptual reorganization in client-centered therapy. In R. R. Blake & G. V. Ramsey (Eds.), *Perception: An approach to personality*. New York: Ronald Press.

Rogers, C. (1951b). *Client-centered therapy* (pp. 13–71). Boston: Houghton Mifflin Company.

Rosenberg, M. (1965). *Society and the adolescent self-image*. Princeton, NJ: Princeton University Press.

Rosenberg, M. (1979). *Conceiving the self*. New York: Basic Books.

Rosenberg, M. (1986). *Conceiving the self*. Malabar, FL: Krieger.

Rosenberg, M. (1989). *Society and the adolescent self-image*. Middeltown, CT: Wesleyan University Press.

Statt, D. (1990). *The concise dictionary of psychology*. New York: Routledge.

Tajfel, H., & Turner, J. C. (1979). An integrative theory of intergroup conflict. In W. G. Austin & S. Worchel (Eds.), *The social psychology of intergroup relations* (pp. 33–47). Monterey, CA: Brooks/Cole.

Self-Inflicted Injurious Behavior

► Suicide

Self-management

Andrea Wallace
College of Nursing, University of Iowa, Iowa City, IA, USA

Synonyms

Self-care

Definition

The process of actively engaging in activities aimed at controlling the negative effects of an illness, particularly a chronic illness, on one's health.

Description

Self-management concerns the acquisition of knowledge, as well as application of skills, necessary to engage in a complex set of health-promoting behaviors in the context of daily living. This process of integrating a number of complex behaviors in an effort to maintain wellness often involves problem-solving, decision-making, resource utilization, and communicating with multiple health-care providers. The ability to adapt health behaviors based on physiological or psychological information is a key element of self-management. The importance of self-management is undergirded by its role in health outcomes: It has been demonstrated that those who successfully engage in self-management activities experience better health-related outcomes for a number of chronic conditions.

Based on early studies of those living with chronic conditions, it has been proposed that the tasks related to self-management generally fall within three primary categories addressing: (1) medical management, which includes activities such as taking medications or adhering to a special diet; (2) role management, which includes actions allowing one to adopt roles that accommodate for ones condition; and (3) emotional management, which includes actions aimed at coping with emotions associated with an illness, such as uncertainty, depression, and fear (Corbin & Strauss, 1988). It follows, then, that a wide array of psychosocial factors play a role in one's ability to successfully engage in self-management including, but not limited to, social support, motivation, confidence (self-efficacy), and depression. In addition, the ability to read and understand written information (literacy), understand and manipulate numerical information (numeracy), verbal memory, planning, and motor speed have all been associated with disease self-management behavior.

Although disease-specific self-management education is widely accepted as beneficial, its effect on health outcomes has been difficult to establish, primarily due to variability in the nature of the programs and populations tested. Because of the complexity associated with self-management, education and support aiming to train patients to manage their chronic disease attempt to address the many factors and tasks associated with self-management as well as tailor training to the individual needs of patients. However, some common elements among self-management education programs include general information about an illness (e.g., physiology), establishing and personalizing a treatment or self-care "plan" that addresses the behaviors necessary for improved disease outcomes and strategies to facilitate health-related behavior change and maintenance (Funnell et al., 2009; Lorig & Holman, 2003). Many programs are guided by a specific theoretical framework and plan training to target variables believed to be important facilitators and barriers to self-management such as means of promoting patients' perceived self-efficacy (confidence) in engaging in health-promoting activities (Bandura, 1997). A recent focus has been on the feasibility and broad dissemination of programs facilitating self-management and behavior change.

Driven by rising prevalence, costs, and poor outcomes associated with chronic illnesses, health-care settings have recently begun to focus on models of service delivery that best support patients' self-management needs. Although these efforts have been given a number of names, some common elements include easy access to health-care providers, providing disease-specific education, incorporating interdisciplinary teams of health-care providers (e.g., physicians, social workers, physical therapists), proactive reminders to both clinicians and patients about health maintenance needs (e.g., routine blood tests), and strategies for supporting the adoption and maintenance of health-promoting behaviors, such as behavioral goal setting between health-care providers and

patients (Bodenheimer, Wagner, & Grumbach, 2002a, 2002b; Patient-Centered Primary Care Collaborative, 2010; The MacColl Institute for Healthcare Innovation, 2010).

Cross-References

- ▶ Behavior Change
- ▶ Chronic Disease Management
- ▶ Disease Management
- ▶ Fatigue
- ▶ Health Behaviors
- ▶ Health Promotion
- ▶ Self-efficacy

References and Readings

Bandura, A. (1997). *Self-efficacy: The exercise of control*. New York: Freeman.

Bodenheimer, T., Wagner, E. H., & Grumbach, K. (2002a). Improving primary care for patients with chronic illness. *Journal of the American Medical Association, 288*(14), 1775–1779.

Bodenheimer, T., Wagner, E. H., & Grumbach, K. (2002b). Improving primary care for patients with chronic illness: The chronic care model, Part 2. *Journal of the American Medical Association, 288*(15), 1909–1914.

Corbin, J., & Strauss, A. (1988). *Unending work and care: Managing chronic illness at home*. San Francisco: Jossey-Bass.

Funnell, M. M., Brown, T. L., Childs, B. P., Haas, L. B., Hosey, G. M., & Jensen, B. (2009). National standards for diabetes self-management education. *Diabetes Care, 32*(Suppl. 1), S87–S94.

Lorig, K. R., & Holman, H. (2003). Self-management education: History, definition, outcomes, and mechanisms. *Annals of Behavioral Medicine, 26*(1), 1–7.

Patient-Centered Primary Care Collaborative. (2010). *Patient-centered medical Home*. Retrieved January 24, 2010, from http://www.pcpcc.net/patient-centered-medical-home

The MacColl Institute for Healthcare Innovation. (2010). *The chronic care model*. Retrieved January 24, 2010, from http://www.improvingchroniccare.org/index.php?p=The_Chronic_Care_Model&s=2

Self-Management Education

- ▶ Diabetes Education

Self-medication

Nicole Brandt
School of Pharmacy, University of Maryland, Baltimore, MD, USA

Synonyms

Self-treatment

Definition

Self-medication is the use of medications, treatments, and/or substances by an individual without a medical prescription. Self-medication is the most popular form of self-care, which is defined as the personal preservation of health through prevention and self-treatment of ailments (Ryan, Wilson, Taylor, & Greenfield, 2009). In regards to self-care, substances used to self-medicate include but are not limited to over-the-counter (OTC) medications, nutritional supplements, and other nonprescription medications. The number of OTC medications has increased significantly, allowing more individuals to practice self-medication. These nonprescription medications can be purchased at various locations such as pharmacies, supermarkets, and retail superstores (Wazaify, Shields, Hughes, & McElnay, 2005).

This increase in self-medicating practices entails both advantages and disadvantages. The benefits of self-medication include increased access to treatment, increased patient involvement in their own health care, economical choices, and evidence of cost-effectiveness compared to prescription treatments in some situations. The drawbacks of self-treatment are the threat of misuse and abuse of medications, incorrect self-diagnosis, a delay in appropriate treatment, and an increased risk of drug-drug interactions (Hughes, McElnay, & Fleming, 2001). Due to the many risks listed above, safety is a major concern with self-medication. To ensure safety with self-treatment, patient education about the

medication is a necessity (Bradley & Blenkinsopp, 1996). Pharmacists are in a pivotal position to help ensure appropriate and safe use of various medications by patients that are obtained without a prescription.

Cross-References

- ▶ Nutritional Supplements
- ▶ Self-care
- ▶ Self-management

References and Readings

Bradley, C., & Blenkinsopp, A. (1996). Over the counter drugs: The future for self medication. *British Medical Journal, 312*, 835–837.

Hughes, C. M., McElnay, J. C., & Fleming, G. F. (2001). Benefits and risks of self medication. *Drug Safety, 24*(14), 1027–1037.

Ryan, A., Wilson, S., Taylor, A., & Greenfield, S. (2009). Factors associated with self-care activities among adults in the united kingdom: A systematic review. *BMC Public Health, 9*, 96.

Wazaify, M., Shields, E., Hughes, C. M., & McElnay, J. C. (2005). Societal perspectives on over-the-counter (OTC) medicines. *Family Practice, 22*, 170–176.

Self-Monitor

- ▶ Self-Monitoring

Self-Monitoring

Thomas Webb
Department of Psychology, The University of Sheffield, Sheffield, UK

Synonyms

Seek feedback; Self-evaluate; Self-monitor

Definition

Self-monitoring can refer to:

1. Self-monitoring expressive behavior and self-presentation: the extent to which people observe and control their expressive behavior and self-presentation (Snyder, 1974).
2. Self-monitoring goal progress: periodically noting current state and comparing these perceptions with whichever goals are currently relevant (Carver & Scheier, 1990).

Description

Self-Monitoring Expressive Behavior and Self-Presentation

People differ in the extent to which they self-monitor (observe and control) their expressive behavior and self-presentation (Snyder, 1974, 1979). High self-monitors think about how they appear to others and take care to portray themselves in a socially appropriate manner. Thus, high self-monitors are likely to monitor their facial expression, content of speech, tone of voice, expressed emotionality, and so on. In contrast, low self-monitors do not monitor these things, either because they lack the ability to do so or because they are not motivated to do so (for a review, see Gangestad & Snyder, 2000).

The Self-Monitoring Scale (Snyder, 1974) was developed to measure these individual differences. There are 25 items including "I guess I put on a show to impress or entertain people", "in different situations and with different people, I often act like very different persons", and "even if I am not enjoying myself, I often pretend to be having a good time." The scale was revised by Lennox and Wolfe (1984) who proposed a shorter 13-item scale with 6 items measuring sensitivity to the expressive behavior of others (e.g., "I am often able to read people's true emotions correctly through their eyes") and 7 items measuring the ability to modify self-presentation (e.g., "once I know what the situation calls for, it's easy for me to regulate my actions accordingly"). Lennox and Wolfe also proposed a separate 20-item "Concern for Appropriateness" scale

measuring cross situational variability (e.g., "different people tend to have different impressions about the type of person I am") and attention to social comparison information (e.g., "I usually keep up with clothing style changes by watching what others wear"). Despite this revision (and others), there remains debate over exactly what the Self-Monitoring Scale measures (e.g., Gangestad & Snyder, 2000).

A number of studies have examined differences between high and low self-monitors. For example, low self-monitors tend to behave in ways that are more consistent with their attitudes (Ajzen, Timko, & White, 1982) and are better able to imagine how their own behavior relates to particular traits (e.g., the extent to which they are sociable; Snyder & Cantor, 1980). In contrast, individuals who score high on Self-Monitoring Scales are particularly sensitive to social cues (Snyder, 1974) and are better able to imagine the prototypic type of person that would be described as holding a particular trait (e.g., a sociable person; Snyder & Cantor) perhaps because they are keen to be able to tailor their own behavior so as to demonstrate certain traits (e.g., to appear sociable). High self-monitors are even more likely to choose friends who facilitate the construction of their own situationally appropriate appearances (Snyder, Gangestad, & Simpson, 1983) and romantic partners with an attractive physical appearance (Snyder, Berscheid, & Glick, 1985). The idea that people self-monitor their expressive behavior has been hugely influential, and these studies just only hint at the wealth of differences (for a more comprehensive review, see Gangestad & Snyder, 2000).

Self-Monitoring Goal Progress

A separate, but potentially overlapping, literature has examined whether and how people monitor their current standing in relation to their personal goals. For example, whether and how people assess if they are on track to reduce their energy bill. While monitoring goal progress can involve feedback from external sources (for a review of feedback interventions, see Kluger & DeNisi, 1996), people can, and do, self-monitor. Self-monitoring in this sense involves periodically noting current state and comparing these perceptions with whichever goals are currently relevant (Carver & Scheier, 1990). For example, a person with the goal to reduce their energy bills might monitor how long they are spending in the shower. Monitoring goal progress, therefore, involves a series of processes from deciding to seek information (e.g., I need to monitor how long I spend in the shower), becoming aware of and directing attention toward relevant information (e.g., looking at the bathroom clock), interpreting the information (e.g., 10 min is quite a long shower), and so on. Relevant goals provide both a comparative standard, but also a schema for making sense of the information available (Ashford & Cummings, 1983). The person can monitor behavior (e.g., number of showers taken per week) or the outcomes of behavior (e.g., weekly energy costs) (Abraham & Michie, 2008). Monitoring may also vary on a temporal dimension (e.g., hourly, daily, weekly, or monthly) and can occur with respect to goals represented at different levels of specificity. For example, monitoring progress toward relatively high-level values comprising principles (or "be" goals, e.g., to be eco-friendly), specific behavioral goals (or "do" goals, e.g., to take shorter showers), or even the performance of motor programs (e.g., turn off the tap).

Monitoring goal progress is central to a number of models of goal striving and self-regulation (e.g., Control Theory; Carver & Scheier, 1982), but only a few studies have examined the effect of manipulating the likelihood that people would or could self-monitor. Polivy, Herman, Hackett, and Kuleshnyk (1986, Study 1) investigated the effect of being able to monitor consumption on unhealthy eating. Participants were asked to taste some chocolates and to "eat as many as necessary to ensure accurate ratings." Unbeknown to participants, the researchers manipulated how easy it was for participants to monitor their consumption; some participants were asked to leave their chocolate wrappers on the table, others to place them in a wastebasket that was already half full of wrappers. The main finding was that participants asked to leave their wrappers on the table

(and so, presumably, found it easy to monitor how many chocolates they had eaten) ate less than those asked to put the wrappers in the waste-basket. Quinn, Pascoe, Wood, and Neal (2010) found that self-monitoring helped people to break bad habits. Across two studies, prompting participants to use vigilant monitoring (thinking "don't do it" and watching carefully for mistakes) proved more effective in helping participants to avoid habitual responses (e.g., staying up too late, eating too much) than prompting stimulus control (removing oneself from the situation or removing the opportunity to perform the behavior). Prompting or facilitating behavioral monitoring has also proved an influential technique for promoting behavior change (for reviews, see Kanfer, 1970; Michie, Abraham, Whittington, McAteer, & Gupta, 2009). In summary, people who self-monitor their current standing in relation to their goals tend to be better able to achieve their goals and make changes to their behavior than people who do not.

Cross-References

- ▶ Behavior Change
- ▶ Self-examination
- ▶ Self-Regulation

References and Readings

Abraham, C., & Michie, S. (2008). A taxonomy of behavior change techniques used in interventions. *Health Psychology, 27*, 379–387.

Ajzen, I., Timko, C., & White, J. B. (1982). Self-monitoring and the attitude behavior relation. *Journal of Personality and Social Psychology, 42*, 426–435.

Ashford, S. J., & Cummings, L. L. (1983). Feedback as an individual resource: Personal strategies of creating information. *Organizational Behavior and Human Performance, 32*, 370–398.

Carver, C. S., & Scheier, M. F. (1982). Control theory: A useful conceptual framework for personality, social, clinical, and health psychology. *Psychological Bulletin, 92*, 111–135.

Carver, C. S., & Scheier, M. F. (1990). Origins and functions of positive and negative affect: A control process view. *Psychological Review, 97*, 19–35.

Gangestad, S. W., & Snyder, M. (2000). Self-monitoring: Appraisal and reappraisal. *Psychological Bulletin, 126*, 530–555.

Kanfer, F. H. (1970). Self-monitoring: Methodological limitations and clinical applications. *Journal of Consulting and Clinical Psychology, 35*, 148–152.

Kluger, A. N., & DeNisi, A. (1996). The effects of feedback interventions on performance: A historical review, a meta-analysis, and a preliminary feedback intervention theory. *Psychological Bulletin, 119*, 254–284.

Lennox, R. D., & Wolfe, R. N. (1984). Revision of the self-monitoring scale. *Journal of Personality and Social Psychology, 46*, 1349–1364.

Michie, S., Abraham, C., Whittington, C., McAteer, J., & Gupta, S. (2009). Effective techniques in healthy eating and physical activity interventions: A meta-regression. *Health Psychology, 28*, 690–701.

Polivy, J., Herman, C. P., Hackett, R., & Kuleshnyk, I. (1986). The effects of self-attention and public attention on eating in restrained and unrestrained subjects. *Journal of Personality and Social Psychology, 50*, 1253–1260.

Quinn, J. M., Pascoe, A., Wood, W., & Neal, D. T. (2010). Can't help yourself? Monitor those bad habits. *Personality and Social Psychology Bulletin, 36*, 499–511.

Snyder, M. (1974). Self-monitoring expressive behavior. *Journal of Personality and Social Psychology, 30*, 526–537.

Snyder, M. (1979). Self-monitoring processes. *Advances in Experimental Social Psychology, 12*, 85–128.

Snyder, M., Berscheid, E., & Glick, P. (1985). Focusing on the exterior and the interior: Two investigations of the initiation of personal relationships. *Journal of Personality and Social Psychology, 48*, 1427–1439.

Snyder, M., & Cantor, N. (1980). Thinking about ourselves and others: Self-monitoring and social knowledge. *Journal of Personality and Social Psychology, 39*, 222–234.

Snyder, M., Gangestad, S., & Simpson, J. A. (1983). Choosing friends as activity partners: The role of self-monitoring. *Journal of Personality and Social Psychology, 45*, 1061–1072.

Self-Monitoring of Blood Glucose

- ▶ Glucose Meters and Strips

Self-Murder

- ▶ Suicide

Self-Perspective

► Self-Identity

Self-Rating

► Self-examination

Self-Regulation

► Self-Regulation Model

Self-Regulation Model

Pablo A. Mora[1] and Gozde Ozakinci[2]
[1]Department of Psychology, The University of Texas at Arlington, Arlington, TX, USA
[2]Lecturer in Health Psychology, School of Medicine, University of St Andrews, St Andrews, Scotland, UK

Synonyms

Model of self-regulation; Self-control; Self-Regulation

Definition

Self-regulation is a dynamic and systematic process that involves efforts to modify and modulate thoughts, emotions, and actions in order to attain goals.

Description

Self-regulation refers to the dynamic cognitive, affective, and behavioral processes that underlie goal attainment. It is important to note that not all types of situations involving goal attainment or problem solving constitute self-regulation. What makes self-regulation unique is that the target of problem solving is set by or focuses on the individual (i.e., self), its "machinery" (e.g., physical problems such as symptoms), and subjective feelings or affect.

Models of self-regulation are based on the idea of cybernetic control and propose that actions are regulated by a TOTE (Test, Operate, Test, Exit) feedback control loop. Central to the idea of feedback loop is the corrective actions that result from the detection and evaluation of discrepancies between input (internal or external) and a reference value. In the context of health, "feeling good" (i.e., not having any symptoms) can be considered a reference value or goal. Thus, when a person experiences a headache (i.e., discrepancy between current state and feeling good), he or she will engage in corrective actions to rid himself or herself of the headache such as taking a pain reliever or resting. If the headache subsides (i.e., test has determined that the discrepancy was eliminated), then the loop ends (i.e., exit). If not, then a new loop will begin.

These self-regulation principles have been widely applied to the study and explanation of multiple psychological phenomena. Psychological models of self-regulation diverge in terms of their foci of interest and emphases; however, they do share core features. Common features of psychological self-regulation models are: (1) goal setting, (2) developing and enacting strategies to achieve these goals, (3) developing criteria to determine proximity to the goal, and (4) determining, based on goal proximity, whether corrective actions are needed or whether the goal needs to be revised. Self-regulation is an iterative process that may require constant evaluation to ensure that the distance between the person's status and the goal is the desired one. One additional commonality shared by behavioral models is the importance they ascribe to affective experiences as integral to self-regulation. Affect, as a core component of the motivational system, can be the reference value that

triggers self-regulatory behaviors, be the product of progress toward a goal or lack thereof (i.e., negative affect resulting from not attaining a goal), and/or influence cognitions and behaviors involved in self-regulatory activities (e.g., symptom perception). Models that integrate affective experiences with self-regulation propose that problem-focused and emotion-focused goals and the behavioral processes used by individuals to attain such goals operate in a parallel yet interrelated fashion (see commonsense model of self-regulation, stress-behavior model advanced by Lazarus and Folkman, or work on self-regulation of affect conducted by Carver and Scheier).

Hierarchical Structure of Goals

Goals are usually differentiated and hierarchically organized in terms of their level of abstraction (Carver & Scheier, 1990a). At the highest level of abstraction, one can find goals related to self-concepts (e.g., ideal self and undesired self), self-assessments (e.g., self-rated health), and general affect (e.g., depressed or happy mood). Specific, concrete actions such as daily activities performed by an individual (e.g., buying low fat food) are at the lower level of the hierarchy. Attaining higher-order goals requires that lower-level goals are accomplished; that is, lower-level goals constitute routes to higher-order ones (e.g., buying and consuming low fat food in order to achieve better health). Abstract goals also provide internal consistency and coherence to lower-level goals and to the actions performed to achieve specific higher-order goals. The relationship between goals of different levels of abstraction is quite dynamic. Thus, while a single abstract goal can be attained by pursuing multiple lower-level, concrete goals, it is also possible to simultaneously attain multiple, distinct higher-order goals by setting and pursuing the same lower-level, concrete goals. For instance, a person can get closer to their ideal, healthy self and farther from their undesired, decrepit self by engaging in similar lower-level self-regulatory activities (e.g., engaging in regular exercises and eating a healthful diet).

Feedback Loops and Behavioral Strategies Involved in Goal Attainment

In self-regulation, goals provide the reference values for feedback loops and motivate and guide actions. Individuals may engage in two types of overall feedback loops depending on whether the reference value (i.e., goal) represents a desired state (i.e., approach) or whether it represents an undesired one (i.e., avoidance, Carver, 2006). Behavior directed toward desired goals is regulated by a negative feedback loop. In this case, a reduction of the discrepancy between the current state (i.e., input) and the goal (i.e., reference value) dominates the individual's actions. Behaviors involved in the avoidance of reference value, on the other hand, are controlled by a positive feedback loop. Thus, efforts are deployed to maintain or enlarge a discrepancy between the input and the reference value. Many times avoiding an undesired state may require that individuals set desired goals. For example, infirmity (undesired goal) can be avoided by establishing a regime of regular exercise and a healthy diet (desired goals). One can argue that positive feedback loops are part of larger negative feedback loops that motivate individuals to reduce discrepancy between their current status and the reference value. In the case discussed above, an individual for whom avoiding infirmity is critical will need to develop achievable, concrete goals in order to appraise progress. Thus, the reference value for "avoiding infirmity" can take the form of "being symptom-free," a goal that is regulated by a negative feedback loop.

The two strategies discussed above assume that goals set by an individual are attainable; however, there are situations in which, regardless of effort, goal attainment can become difficult or impossible. In such situations, abandoning activities directed at the pursuit of the goal will result in better adjustment than maintaining goal-directed efforts. Research has shown that goal disengagement can result in improved well-being if goal-directed efforts are focused on alternative goals when available. If alternative goals are unavailable, inability to disengage from goal pursuit can result in frustration, negative affect, and increased stress (Miller & Wrosch, 2007).

For older adults, goal disengagement and goal reengagement may be a key strategy for adjustment to physical changes and for successful aging.

Goal attainment and affect. As indicated above, affect can be an indicator of progress toward the attainment of a goal. Carver and Scheier (1990b) have proposed a second feedback process that monitors whether a person's efforts are being successful in attaining a goal. Success in closing the gap between one's status and a given goal results in positive affect. Slow progress or failure to attain a goal, on the other hand, results in negative affect. Individuals differ in terms of the reference values they use to determine what constitutes acceptable or unacceptable progress. Accordingly, similar rates of discrepancy reduction can result in two very different responses if the individuals use different criteria to appraise the effectiveness of their actions.

Research on self-discrepancies provides an interesting example of how proximity to a goal can elicit positive or negative moods (Mora, Musumeci-Szabo, Popan, Beamon, & Leventhal, in press). These data have shown that individuals who felt they were farther from being at their worst (i.e., undesired self) reported less anxiety and depression and more happiness than their counterparts who felt closer to their feared self. These affective experiences can, in turn, influence subsequent evaluations of progress and actions. Negative mood arisen from perceptions that progress toward a goal is slow can result in maladaptive behaviors especially if new actions are focused on reducing negative feelings. Depressive feelings may alter perceptions of control over their environment and future outcomes, and lead individuals to give up on their efforts to attain a goal. Despite its critical and multifaceted role in self-regulation, there is a dearth of research examining the multiple interactive ways in which cognitive/behavioral and affective self-regulation operates.

Organization of Goals and Actions: Goal Cognitions

Germane to the relationships among goals of different levels of abstraction are goal cognitions (Karoly, 1993) and action plans (Leventhal, 1970). Goal cognitions are mental models or schemas that organize domain-specific goals and actions or procedures to attain those goals (in health literature, goal cognitions are referred as illness representations). For instance, a schema for an abstract, higher-order goal of "being healthy" would involve several less abstract concrete goals such as engaging in exercise, eating healthful food, and improving coping skills. These goals, in turn, would be connected in the mental schema to more concrete, lower-level goals such as setting days to go the gym, eating more complex carbohydrates, and learning how to meditate to reduce stress. Action plans translate aspects of goal pursuit (e.g., beliefs, attitudes, evaluation criteria) into actual, concrete behaviors. These behaviors help link goals at different levels of abstraction and provide continuity to the pursuit of abstract goals. In the current example, being healthy requires the person to set and attain multiple concrete goals. Goal cognitions will specify the action plans required to attain the concrete goals in route to reach the higher-order, abstract goal. Thus, to attain the various goals on route to being healthy, the person will need to engage in actions such as joining a gym, building a routine of going to the gym, and choosing the specific workout routine (e.g., cardio and/or weightlifting).

Conscious and Nonconscious Aspects of Self-Regulation

Although the terms "goal setting" and "self-regulation" suggest conscious volition, processes involved in self-regulation are the result of both automatic and intentional behaviors. Awareness of every single process involved in self-regulation would unnecessarily tax the organism and result in maladaptation. Action plans required for the attainment of a goal can become automated behaviors if goal pursuit is an ongoing activity. For instance, the management of a chronic condition such as hypertension requires that people take medications every day. To ensure that doses are not missed, a person may decide to put pills in a case and place this case on top of the counter next to the coffee maker.

By doing this, taking the pill can become an automated behavior that imposes a minimal burden on the cognitive system of the individual. It is in consciousness, however, where automatic and intentional processes are integrated. Ensuring that doses have not been missed, the person needs to engage in a conscious decision-making process (e.g., pick up the pill case and count the remaining pills).

Recently, investigators interested in the volitional aspects of self-regulation have been devoting increased attention to the idea of self-control. Although sometimes self-control and self-regulation are used interchangeably, self-control refers to the capacity to consciously and effortfully regulate one's affect, cognitions, and behaviors (Hagger, Wood, Stiff, & Chatzisarantis, 2010). In attempting to explain the reasons individuals engage in maladaptive behaviors (e.g., smoking), researchers have argued that self-control is a limited resource (Ego Strength model: Baumeister, Vohs, & Tice, 2007). The Ego Strength model argues that self-control works as "a muscle" that can become tired after repeated use (i.e., ego depletion). In other words, a person's capacity to engage in self-control becomes reduced following previous acts of self-control. Although the idea of ego depletion has received support from experimental studies, a recent meta-analysis suggests that the strength model does not fully explain the mechanisms underlying self-control and additional theoretical perspectives need to be integrated into the strength model (Hagger et al., 2010). The results from the meta-analysis suggest that motivation, fatigue, self-efficacy, and affect are involved in self-control and ego-depletion processes. Further research in this area is needed to better understand how self-control and ego depletion operate in real world contexts in which long-term and unplanned attempts at self-control occur frequently.

Moderators of Self-Regulation

Self-regulation occurs within particular settings and is, thus, influenced by individual, social, and cultural factors. Personality, an individual factor, has been implicated in various processes of self-regulation such as appraisal, coping, and behaviors (Cervone, Shadel, Smith, & Fiori, 2006). For instance, research has shown that individuals high in neuroticism (one of the big five personality traits) consistently and reliably perceive and report more physical symptoms than their low neuroticism counterparts (Watson & Pennebaker, 1989). Because physical symptoms are a departure from normal functioning, increases in symptomatology may trigger prompt self-regulatory actions (e.g., care seeking) if symptoms are perceived to be indicators of imminent threat. Research on coping has revealed that individual differences such as optimism and pessimism influence goal pursuit. Specifically, individuals who have positive views about the future tend to engage in problem-focused coping, whereas individuals who perceive the future as uncertain tend to either engage in emotion-focused coping or else disengage from goal pursuit (Rasmussen, Wrosch, Scheier, & Carver, 2006). Personality can also have an impact on the selection of specific behaviors used by individuals to deal with threat. There is evidence that individuals high in optimism and conscientiousness who face stressful situations select coping procedures that improve well-being and adjustment (O'Connor, Conner, Jones, McMillan, & Ferguson, 2009).

Social factors and culture can also influence the various stages of self-regulation from goal setting to selection and performance of corrective actions through multiple pathways. Social comparisons can help individuals to determine the origin of their physical symptoms (e.g., food poisoning if everybody at dinner has the same symptoms or stomach flu if symptoms are unique to one person) and, thereby, select the necessary corrective actions to deal with such symptoms (Leventhal, Hudson, & Robitaille, 1997). Research has also demonstrated that social relations (i.e., social network size) can influence long-term self-regulatory activities such as participation in cardiac rehabilitation among patients with acute coronary syndrome (Molloy, Perkins-Porras, Strike, & Steptoe, 2008).

Culture has been shown to influence the type of stimuli that trigger self-regulation, the content

of goals, the specific corrective actions, and the appraisal criteria involved in goal attainment. Anthropological research has shown that the interpretation and attribution of bodily symptoms varies across culture. This body of evidence suggests that culturally determined illness models are powerful determinants of the ways people experience illness conditions. For example, work on depression has shown that among certain ethnic groups (e.g., Chinese), depression is usually experienced as a somatic disorder (Kleinman, 1977). This difference in experience can shape what actions are taken to remediate the problem (e.g., seeing a primary care physician instead of a mental health specialist) and the criteria to determine the success of the remedial actions (e.g., improvement in physical versus affective well-being). The impact of culture on the construction and development of the self has led some authors to question the idea that self-regulation is set by and focuses on the individual. In collectivistic cultures where the self is construed as closely interrelated to others, goal setting can be motivated by a need to please the person's social network (Trommsdorff, 2009). Future research needs to determine whether the mechanisms of self-regulation guided by interrelatedness are different from those underlying self-regulation of intraindividual processes.

Self-Regulation and Health

Although most models of self-regulation have originated outside the domain of behavioral medicine (see the Commonsense Model of Self-regulation for an exception), the use of self-regulatory principles and ideas to understand health-related behaviors has been relatively widespread. Models such as the Health Beliefs Model, Theory of Reasoned Action, and Social Cognitive Theory which focus on specific aspects of self-regulation such as social (e.g., norms) and psychological (e.g., self-efficacy) determinants have been utilized to understand self-regulation of health behaviors. In general, the use of these models has focused more on the description of predictors of goals rather than on the underlying mechanisms that explain self-regulatory behaviors. In addition, goal pursuit is usually examined by these models as a single-event rather than a continuous process (Maes & Karoly, 2005). Recent efforts, however, have been directed at the understanding and examination of self-regulation as a process by investigating how goal-directed behaviors in health domains are initiated and maintained. Evidence has provided convincing support that the various phases of self-regulation are controlled by different processes. Studies examining health behaviors such as smoking cessation and engagement in exercise activities have revealed that factors such as self-efficacy and attitudes toward the specific behavior are important determinants of initiation. Maintenance of health behaviors, on the other hand, is influenced by factors such as satisfaction with behavioral change. A recent study examining data from participants in a smoking cessation program provided further evidence of the complexity of self-regulation processes (Baldwin, Rothman, & Jeffery, 2009). In this study, the authors found that maintenance of health-related behaviors was a heterogeneous process and that the factors that influenced satisfaction with behavioral maintenance varied according to whether they had quit smoking recently or not.

Final Remarks

Literature and research on self-regulation has grown rapidly over the past 20 years. New research has expanded the understanding of self-regulation mechanisms but more research is needed. Some of the research questions that remained underexplored include changes in self-regulation over time, factors that contribute to long-term self-regulatory behaviors (e.g., adherence to medical treatment for chronic conditions), and the multiple and simultaneous pathways through which cognitive and affective self-regulation interact.

New opportunities provided by advances in technology, especially in brain imaging, are opening exciting areas of inquiry (e.g., cognitive neuroscience of self-regulation). Similarly, increased understanding of the psychophysiology of stress offers an invaluable opportunity to understand how self-regulatory processes get under the skin.

Cross-References

- ▶ Active Coping
- ▶ Behavioral Intervention
- ▶ Common-Sense Model of Self-regulation
- ▶ Optimism
- ▶ Self-Efficacy
- ▶ Self-Regulatory Capacity
- ▶ Self-Regulatory Fatigue

References and Readings

Baldwin, A. S., Rothman, A. J., & Jeffery, R. W. (2009). Satisfaction with weight loss: Examining the longitudinal covariation between people's weight-loss-related outcomes and experiences and their satisfaction. *Annals of Behavioral Medicine, 38*, 213–224.

Baumeister, R. F., & Vohs, K. D. (2004). *Handbook of self-regulation: research, theory, and applications*. New York: Guilford Press.

Baumeister, R. F., Vohs, K. D., & Tice, D. M. (2007). The strength model of self-control. *Current Directions in Psychological Science, 16*(6), 351–355. doi:10.1111/j.1467-8721.2007.00534.x.

Boekaerts, M., Zeidner, M., & Pintrich, P. R. (1999). *Handbook of self-regulation*. San Diego, CA\London: Academic Press.

Carver, C. (2006). Approach, avoidance, and the self-regulation of affect and action. *Motivation and Emotion, 30*(2), 105–110. doi:10.1007/s11031-006-9044-7.

Carver, C. S., & Scheier, M. (1990a). Principles of self-regulation: Action and emotion. In E. T. Higgins, R. M. Sorrentino, et al. (Eds.), *Handbook of motivation and cognition: Foundations of social behavior* (Vol. 2, pp. 3–52). New York: Guilford Press.

Carver, C. S., & Scheier, M. F. (1990b). Origins and functions of positive and negative affect: A control-process view. *Psychological Review, 97*(1), 19–35.

Carver, C. S., & Scheier, M. F. (1998). *On the self-regulation of behavior*. New York: Cambridge University Press.

Cervone, D., Shadel, W. G., Smith, R. E., & Fiori, M. (2006). Self-regulation: Reminders and suggestions from personality science. *Applied Psychology: An International Review, 55*(3), 333–385. doi:10.1111/j.1464-0597.2006.00261.x.

Folkman, S., & Lazarus, R. S. (1988). The relationship between coping and emotion: Implications for theory and research. *Social Science & Medicine, 26*(3), 309–317.

Hagger, M. S., Wood, C., Stiff, C., & Chatzisarantis, N. L. D. (2010). Ego depletion and the strength model of self-control: A meta-analysis. *Psychological Bulletin, 136*(4), 495–525. doi:10.1037/a0019486.

Karoly, P. (1993). Mechanisms of self-regulation: A systems view. *Annual Review of Psychology, 44*, 23–52.

Kleinman, A. M. (1977). Depression, somatization and the "new cross-cultural psychiatry". [Case reports comparative study]. *Social Science and Medicine, 11*(1), 3–10.

Leventhal, H. (1970). Findings and theory in the study of fear communications. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol. 5, pp. 120–186). New York: Academic.

Leventhal, H. (1983). Behavioral medicine: Psychology in health care. In D. Mechanic (Ed.), *Handbook of health, health care, and the health professions* (pp. 709–743). New York: The Free Press.

Leventhal, H., Hudson, S., & Robitaille, C. (1997). Social comparison and health: A process model. In B. P. Buunk & F. X. Gibbons (Eds.), *Health, coping, and well-being: Perspectives from social comparison theory* (pp. 411–432). Mahwah, NJ: Lawrence Erlbaum Associates.

Maes, S., & Karoly, P. (2005). Self-regulation assessment and intervention in physical health and illness: A review. *Applied Psychology, 54*(2), 267–299. doi:10.1111/j.1464-0597.2005.00210.x.

Miller, G. E., & Wrosch, C. (2007). You've gotta know when to fold 'em. *Psychological Science, 18*(9), 773–777. doi:10.1111/j.1467-9280.2007.01977.x.

Molloy, G. J., Perkins-Porras, L., Strike, P. C., & Steptoe, A. (2008). Social networks and partner stress as predictors of adherence to medication, rehabilitation attendance, and quality of life following acute coronary syndrome. *Health Psychology, 27*(1), 52–58. doi:10.1037/0278-6133.27.1.52.

Mora, P. A., Musumeci-Szabo, T. J., Popan, J., Beamon, T., & Leventhal, H. (in press). Me at my worst: Exploring the relationship between the undesired self, health, and mood among older adults. *Journal of Applied Social Psychology*.

O'Connor, D., Conner, M., Jones, F., McMillan, B., & Ferguson, E. (2009). Exploring the benefits of conscientiousness: An investigation of the role of daily stressors and health behaviors. *Annals of Behavioral Medicine, 37*(2), 184–196. doi:10.1007/s12160-009-9087-6.

Rasmussen, H. N., Wrosch, C., Scheier, M. F., & Carver, C. S. (2006). Self-regulation processes and health: The importance of optimism and goal adjustment. *Journal of Personality, 74*(6), 1721–1747. doi:10.1111/j.1467-6494.2006.00426.x.

Schunk, D. H., & Zimmerman, B. J. (1994). *Self-regulation of learning and performance: issues and educational applications*. Hillsdale, NJ: Lawrence Erlbaum Associates.

Trommsdorff, G. (2009). Culture and development of self-regulation. *Social and Personality Psychology Compass, 3*(5), 687–701. doi:10.1111/j.1751-9004.2009.00209.x.

Watson, D., & Pennebaker, J. W. (1989). Health complaints, stress, and distress: Exploring the central role of negative affectivity. *Psychological Review, 96*(2), 234–254.

Self-Regulatory Ability

► Self-Regulatory Capacity

Self-Regulatory Capacity

David Cameron and Thomas Webb
Department of Psychology, The University of Sheffield, Sheffield, UK

Synonyms

Self-control capacity; Self-regulatory ability; Strength model of self-control

Definition

Self-regulatory capacity refers to people's ability to exert control over their thoughts, feelings, and actions. For example, the capacity to inhibit prejudice, make oneself feel better, or select healthy food. Self-regulatory capacity is, however, thought to be a limited resource that (1) is temporarily depleted by exertions of self-control (an effect termed "ego-depletion") and (2) differs in strength from person to person.

Description

Limited Self-Regulatory Capacity

Self-regulatory capacity refers to an individual's ability to exert control over their behavior, thoughts, and feelings. The capacity for self-regulation differs between individuals and can depend on situational factors such as the experience of self-regulatory fatigue. Self-regulation is closely related to goal-driven behavior and is characterized by the process of attempting to work toward a desired held goal. For example, in terms of health behavior, an individual may regulate their diet in order to reach their goal of being a healthy weight or resist a personal temptation such as cigarettes to reach their goal of overcoming a smoking addiction. Self-regulatory capacity would influence how successfully an individual pursues such goals, alongside other factors such as the prepotency of the action that needs to be overcome. Persistence with self-regulation can lead to a temporary state of fatigue in which self-regulatory capacity is reduced and a state of depleted self-regulatory capacity is associated with lapses of self-regulation. In terms of the prior examples, this could include short-term gratifications such as deviations from a planned diet or the resumption of smoking.

Self-regulatory capacity can be measured using a wide variety of cognitive or behavioral tests. Examples include persistence at impossible (e.g., completing unsolvable anagrams) or aversive (e.g., consuming unpleasant drinks, squeezing a handgrip, cold pressor) tasks; response time or errors made in inhibition tasks (Stroop task, stop signal); resisting temptation (limiting alcohol consumption); and suppression of emotions. The key criterion for a task that measures self-regulatory capacity is the requirement for the person to override an otherwise dominant response (e.g., the desire to give up, to read the words in the Stroop task). Tasks that are simply difficult (e.g., complex math puzzles) or stressful (e.g., watching an emotional video) are unlikely to reflect the same ability. As self-control is considered to draw from a universal pool, putting two of these tasks together, one after the other, can provide a measure of self-control capacity – the extent to which a person's ability to perform a self-control task is impaired by the initial exertion of self-control.

Self-regulatory capacity has an important relation to health behavior. Many behaviors which are considered to be beneficial in the long term such as eating a healthy diet or maintaining an exercise regimen are notably absent from people's routines, while behaviors that are demonstrably harmful in the long term such as smoking, unhealthy diets, unprotected sexual intercourse, or substance abuse are common in society. The contrast between these behaviors can be seen in the temporal distance between

the costs and benefits: behaviors associated with improved health will generally only show a beneficial change over a longer term while the costs (such as the effort required to change behavior through self-regulation) are high in the short term, whereas behaviors that show a high cost of being harmful to health in the long term offer immediate benefits (such as the satiation of an immediate desire or need). In the context of temporal self-regulation theory (Hall & Fong, 2007), self-regulatory capacity acts as a moderator of the link between an individual's intentions for health behavior, which are shaped by the temporal differences between perceived costs and benefits for an action, and the individual's actual observed behavior. A greater capacity for self-regulation is predicted to be associated with the ability to overcome prepotent responses, such as habitual smoking, and follow intentions for behavior, such as intention to quit smoking. Limited capacity for regulation is predicted to be associated with lapses in self-control and the resumption of prepotent responses. Thus, despite the intention to quit smoking, this would not translate into actionable behavior.

Nature of Self-Regulatory Capacity

Despite many studies showing effects consistent with the idea that people have a finite capacity for self-regulation, the precise nature of self-regulatory capacity remains more elusive. Gailliot, Baumeister, DeWall, Maner, Plant, Tice, Brewer, and Schmeichel (2007) found that (1) depletions in self-control capacity (as evidenced by performance on self-control tasks) were correlated with blood glucose levels and (2) self-control performance could be restored by blood glucose supplements (namely, lemonade). On the basis of this evidence, they argue that self-regulatory capacity reflects the available supply of glucose. However, it seems unlikely that the body would struggle to quickly provide sufficient glucose to the brain following single or repeated attempts at self-control, and further research is needed to interrogate the glucose hypothesis in more detail (Beedie & Lane, 2012).

Self-regulatory capacity's apparent dependence on a glucose supply suggests a similarity with other broader processes in executive function which are known to be affected by available glucose levels, for example, that of the attentional system. Self-regulation is considered to exist alongside this diverse array of higher cognitive processes and may work in concert with others, particularly that of planning and error detection. Self-regulation can be considered as a goal-driven process because it is a process used by an individual who seeks to override one state of behavior, thoughts, or feelings with another goal state. As such, an individual is required to recruit multiple executive processes to reach goal states, representations of one's current state and goal state much be held alongside a plan for how to achieve the goal state, detection of discrepancy (or error) between these two states must occur, and self-regulation to change the current experienced state toward the goal is required. Apparent failures in the wider system of self-regulation may not be restricted to a depleted self-regulatory capacity; related systems such as that of error detection may impair self-regulation, if there is an incorrect perception of error between the current state and desired state.

Like other aspects of executive function, self-regulatory capacity is associated with the frontal lobes. Both neuroimaging and lesion studies indicate that self-regulatory actions such as the control of behavioral and emotional output is closely associated with the ventromedial prefrontal cortex. Alongside this, the ventromedial-orbitofrontal cortex has been particularly associated with the self-regulatory process of suppressing responses that have previously been considered rewarding. Damage to this area often results in a diminished or absent ability to inhibit immediately rewarding actions, even though the semantic knowledge of an action's inappropriateness may still be intact. These regions associated with self-regulatory capacity show high connectivity with executive areas related to the processes involved in self-monitoring, which support the process of self-regulation. The anterior cingulate cortex is associated with the degree and nature of conflict between competing responses to situations such as the short-term desire to satiate an immediate need against

the longer-term goal for a healthier lifestyle. The dorsolateral prefrontal cortex has been associated with the resolution of detected response conflict alongside attentional and planning behavior. These regions working in cohort are considered to form the neural circuitry underpinning self-regulation.

Individual Differences in Self-Regulatory Capacity

In addition to situational demands on self-regulatory capacity, there is also a body of research suggesting that there may be stable individual differences in self-regulatory capacity. Some people simply seem better than others at, for example, selecting healthy food, cheering themselves up, and acting in an egalitarian manner. In support of this idea, Hofmann, Gschwendner, Friese, Wiers, and Schmitt (2008) found that individual differences in working memory capacity moderated participants' ability to self-regulate their sexual interest, consumption of tempting food, and anger expression. In a similar vein, a number of authors have proposed measures of self-regulatory capacity such as the Self-Control Behavior Inventory (Fagen, Long, & Stevens, 1975), the Self-Control Questionnaire (Brandon, Oescher, & Loftin, 1990), and the Brief Self-Control Scale (BSCS; Tangney, Baumeister, & Boone, 2004). In each case, differences have been found between people that reliably map onto various self-control outcomes such as task performance, impulse control, interpersonal relations, and so on.

Building Self-Regulatory Capacity

Finally, there is some evidence that people can build self-regulatory capacity through regular training. For example, Muraven (2010) found that participants who practiced self-control by cutting back on sweets or squeezing a handgrip over a 2-week period showed significant improvement in self-control capacity relative to those who practiced tasks that were comparably effortful, but did not require self-control. This improvement was demonstrated in a stop signal task, unrelated to that of the training. In support of these findings, the meta-analysis conducted by Hagger, Wood, Stiff, and Chatzisarantis (2010) reported large and significant effect sizes across seven further tests of the training hypothesis.

Cross-References

- ▶ Cold Pressor Task
- ▶ Ego-Depletion
- ▶ Emotion
- ▶ Limited Resource
- ▶ Prejudice
- ▶ Self-Control

References and Readings

Beedie, C., & Lane A. M. (2012). The role of glucose in self-control: another look at the evidence and an alternative conceptualization. *Personality and Social Psychology review, 16*, 143–153.

Brandon, J. E., Oescher, J., & Loftin, J. M. (1990). The self-control questionnaire: An assessment. *Health Values, 14*, 3–9.

Fagen, S. A., Long, N. J., & Stevens, D. J. (1975). *Teaching children self-control: Preventing emotional and learning problems in the elementary school*. Columbus, OH: Charles E. Merrill.

Gailliot, M. T., Baumeister, R. F., DeWall, C. N., Maner, J. K., Plant, E. A., Tice, D. M., et al. (2007). Self-control relies on glucose as a limited energy source: Willpower is more than a metaphor. *Journal of Personality and Social Psychology, 92*, 325–336.

Hagger, M. S., Wood, C., Stiff, C., & Chatzisarantis, N. L. D. (2010). Ego depletion and the strength model of self-control: A meta-analysis. *Psychological Bulletin, 136*, 495–525.

Hall, P. A., & Fong, G. T. (2007). Temporal self-regulation theory: A model for individual health behavior. *Health Psychology review, 1*, 6–52.

Hofmann, W., Friese, M., Gschwendner, T., Wiers, R. W., & Schmit, M. (2008). Working memory capacity and self-regulatory behavior: Toward an individual differences perspective on behavior determination by automatic versus controlled processes. *Journal of Personality and Social Psychology, 95*, 962–977.

Muraven, M. (2010). Building self-control strength: Practicing self-control leads to improved self-control performance. *Journal of Experimental Social Psychology, 46*, 465–468.

Tangney, J. P., Baumeister, R. F., & Boone, A. L. (2004). High self-control predicts good adjustment, less pathology, better grades, and interpersonal success. *Journal of Personality, 72*, 271–324.

Self-Regulatory Fatigue

David Cameron and Thomas Webb
Department of Psychology, The University of Sheffield, Sheffield, UK

Synonyms

Ego-depletion; Limited resource; Self-control failure

Definition

Self-regulatory fatigue refers to the temporary depletion of individuals' capacity for self-control. In a state of self-regulatory fatigue, individuals find it harder to resist making impulsive purchases, inhibit prejudice, or regulate their own emotions (an effect often termed "ego-depletion"). Self-regulatory fatigue arises from the extended use of self-regulation, which is thought to be a limited resource.

Description

Limited Self-Regulatory Capacity

Self-regulatory fatigue describes the temporary impairment in self-control performance after prior exertions of self-control (an effect also referred to as "ego-depletion"). The strength model of self-regulation, first suggested by Baumeister, Heatherton, and Tice in 1994, draws the analogy between muscular fatigue and self-regulatory fatigue. Muscular fatigue occurs after repeated or prolonged acts of physical exertion, where longer or more effortful exertions lead to a greater experience of fatigue. Similarly, persistent acts of self-regulation, such as the overriding of impulses, result in a mental fatigue, limiting the effectiveness of further regulatory efforts. Again, longer or more effortful exertions of self-regulation lead to a greater experience of self-regulatory fatigue. Like muscular fatigue, rest and recuperation are hypothesized to restore the temporary depletion in self-regulatory strength.

It is important to distinguish between subjective and actual self-regulatory fatigue, although the two can coincide. Self-regulatory persistence and ego-depletion is associated with the physiological indications and subjective feelings of fatigue such as somnolence and decreased attention. Subjective fatigue is thought to mediate between exertions of self-regulation and subsequent decrements in self-regulatory ability. However, subjective fatigue – or the belief that one is fatigued – can have effects that are independent of actual fatigue. Despite experiencing prior effortful self-regulation, individuals perceiving themselves to be low in fatigue can engage in subsequent regulation more effectively than those perceiving themselves to be high in fatigue. A second contributing factor to the state of self-regulatory fatigue is motivation. Individuals may cease persistence at self-regulation if they believe that the effort exerted during self-regulation costs more than the outcome of self-regulation is worth. Self-regulatory fatigue is, therefore, not merely a state of exhaustion in which the individual is willing to engage a task and yet unable to persist but can also lead to acquiescence – the person willfully consents to relax self-control because they are not motivated to do otherwise.

Measuring Self-Regulatory Fatigue

The state of the resource required for self-regulation (assuming that one subscribes to a limited resource model of self-regulation) has proven difficult to objectively measure. At present, the primary means of measuring self-regulatory fatigue is a comparison of performance at a self-regulatory task between individuals in a depleted state (those having exerted prior self-regulation) against individuals in a nondepleted state (those having completed a similar task that does not require self-regulation). Because experimental studies of ego-depletion rely on a comparison of self-regulatory task performance pre- and postdepletion, they only indirectly measure the limited resource. As a result, the relationship between subjective fatigue, motivation, and self-regulatory fatigue is

still unclear. Blood glucose levels have been reported to show a correlation with self-regulatory fatigue, suggesting a positive link between available caloric energy to the brain and available mental resource (Gailliot, 2008). This development offers an alternative avenue to exploring self-regulatory fatigue and the dynamics of ego-depletion through physiological measures rather than behavioral outcomes. However, suggestions of a direct, causal link have been criticized because recorded changes in blood glucose are too small to account for differences between depleted and nondepleted states (Beedie & Lane, 2012). Alternative physiological measures, such as glycogen store levels and heart rate variability, have been suggested as means of measuring resource availability and self-regulatory fatigue.

Overcoming Self-Regulatory Fatigue

The effects of self-regulatory fatigue may be moderated or counteracted by a number of means. The induction of a positive mood by others including humor and laughter, observation of others undergoing successful self-regulation, salient social goals, primed ideas of success, a broadened mindset through self-affirmation, external attribution of the causes of depletion, perception of a low state of fatigue, simultaneous tensing or firming of muscles, and monetary incentives all serve to encourage individuals to persist at self-regulation. However, in many cases, this increased persistence is believed to be associated with a decreased motivation to conserve remaining resources, rather than a restoration of resource levels. Poor performance in an unannounced third self-regulatory task after the standard two-task design often indicates that depleted participants have persisted beyond their typical point of self-regulatory fatigue rather than restored resource. Further interventions, such as the prior practice of self-regulation tasks or the formulation of implementation intentions ("if... then..." plans), serve to encourage persistence at self-regulation through moderating the effort required to expend during successful self-regulation (Webb & Sheeran, 2003). Self-regulatory fatigue may only be overcome on a longer-term basis through the replenishment of available regulatory resource, which is thought to only occur with caloric intake, or rest and relaxation. Sufficient sleep is closely connected with the reduction of subjective fatigue and with performance on cognitive tasks; while it has been suggested to reduce self-regulatory fatigue, it is yet to be formally integrated into the strength model.

Wider Implications of Self-Regulatory Fatigue

Self-regulatory fatigue has been implicated in a number of problems for both the individual and society. While warnings about substance abuse, risky sexual practice, and unhealthy diets are extensively promoted, there equally exists a widespread failure of individuals to adhere to these guidelines or rules. Experimental studies such as those examining alcohol or unhealthy food consumption demonstrate that these behaviors are affected by both self-regulatory fatigue and individuals' chronic tendencies. A chronic tendency that is typically suppressed, such as a high temptation to drink alcohol, becomes more likely to shape behavior when an individual is depleted. In contrast, an individual with low trait temptation to drink alcohol might show no change in alcohol consumption when depleted because it is not necessary to engage in self-regulation. Further examples of behaviors affected by self-regulatory fatigue include impulsive or overspending, emotional regulation such as anger management, interpersonal interaction, self-presentation or impression formation, and stereotype suppression.

Cross-References

▶ Self-Regulatory Capacity

References and Readings

Baddeley, A. (2007). *Working memory, thought and action. Chapter 13*. New York: Oxford University Press.

Baumeister, R. F., Gailliot, M., DeWall, N., & Oaten, M. (2006). Self-regulation and personality: How interventions increase regulatory success, and how

depletion moderates the effects of traits on behavior. *Journal of Personality, 74*, 1773–1802.
Baumeister, R. F., Heatherton, T., & Tice, D. M. (1994). *Losing control: How and why people fail at self-regulation*. London: Academic.
Baumeister, R. F., & Vohs, K. D. (2004). *Handbook of self-regulation: Research theory and applications*. New York: Guildford Press.
Baumeister, R. F., Vohs, K. D., & Tice, D. M. (2007). The strength model of self-control. *Current Directions in Psychological Science, 16*, 351–355.
Beedie, C., & Lane, A. M. (2012). The role of glucose in self-control: another look at the evidence and an alternative conceptualization. *Personality and Social Psychology Review*, *16*, 143–153.
Gailliot, M. (2008). Unlocking the energy dynamics of executive functioning: Linking executive functioning to brain glycogen. *Perspectives on Psychological Science, 3*, 245–263.
Hagger, M. S., Wood, C., Stiff, C., & Chatzisarantis, N. L. D. (2010). Ego-depletion and the strength model of self-control: A meta-analysis. *Psychological Bulletin, 136*, 495–525.
Webb, T. L., & Sheeran, P. (2003). Can implementation intentions overcome ego-depletion? *Journal of Experimental Social Psychology*, *39*, 279–286.

Self-report

Jane Upton
School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Self-report inventory

Definition

Self-report includes an individual's reports about what they are feeling, what they are doing, and what they recall happening in the past (Stone et al., 2009).

These are captured by validated self-report questionnaires, of which there are many. Indeed, one of the challenges facing behavioral medicine is the bewildering variety of measurement instruments (Dekker, 2009). Although validated, the limitations of self-report questionnaires are that the researcher is dependent on the research participant to be completely truthful and unbiased and to be able to accurately remember details.

References and Readings

Dekker, J. (2009). Measurement instruments in behavioral medicine. *International Journal of Behavioural Medicine, 16*, 89–90. doi:10.1007/s12529-009-9049-1.
Stone, A., Turkkan, J., Bachrach, C., Jobe, J., Kuftzman, H., Cain, V., et al. (2009). The science of self-report. *Taylor & Francis e-library*. Retrieved from http://books.google.co.uk/books

Self-Report Inventory

► Self-report

Self-Reported Patient Outcome Measure

► Health Assessment Questionnaire

Self-Respect

► Self-Esteem

Self-Schema

► Self-Identity

Self-System

► Self-Identity

Self-Treatment

► Self-medication

Seligman, Martin

Stephanie Ann Hooker
Department of Psychology, University of Colorado, Denver, CO, USA

Biographical Information

Dr. Martin Seligman

Martin E. P. Seligman was born August 12, 1942, in Albany, NY. He received his A.B. from Princeton University in 1964 and his Ph.D. in Psychology from the University of Pennsylvania in 1967 (Shah, n.d.). After receiving his doctorate, he began his career as an assistant professor at Cornell University in Ithaca, NY. He soon returned to the University of Pennsylvania where he was promoted to associate professor and to professor of Psychology and then to director of the Clinical Training Program. He is currently the Zellerbach Family Professor of Psychology in the Department of Psychology at the University of Pennsylvania and the director of the Positive Psychology Center (University of Pennsylvania, 2007). Early in his career, Seligman studied depression and defined the theory of learned helplessness and pessimism. His worked progressed from a focus on pessimism to optimism, and hence from depression to happiness. He believed that psychology focused too much on mental illness and not enough on health and flourishing; thus, he pioneered the field of positive psychology in 2000 (Seligman & Csikzentmihalyi, 2000).

Major Accomplishments

In 1996, Seligman was elected president of the American Psychological Association (APA) by the largest vote in history. As president, he chose the theme of positive psychology and called for an integration of human flourishing, strengths, and virtues into the science and practice of psychology. He noted that psychology and psychiatry had focused primarily on mental illness (e.g., depression, suffering, victimization, anger, anxiety), but had forgotten positive forms of mental health (e.g., positive emotion, engagement, positive relationships, purpose, accomplishment) (Seligman, 2008). His mission in the APA paralleled his personal mission to promote positive psychology.

In 2008, he began his next mission: to promote a new movement in psychology, positive health (Seligman, 2008). He argued that people desire more than just the absence of suffering and pain; they desire well-being and flourishing that in itself can be protective against mental illness and disease. Thus, he defined positive health as the subjective, biological, and functional realms that can predict positive aspects of mental health, e.g., longevity, positive emotion, prognosis, and suggested areas for which positive health could be incorporated into studies of well-being and illness.

Seligman has published 20 books and over 200 articles on motivation and personality, including best sellers such as *Learned Optimism*, *Authentic Happiness*, and *The Optimistic Child* (Shah, n.d.). He is one of the most often-cited psychologists and the thirteenth most likely name to appear in a general psychology textbook (TED Conferences LLC, 2008). Seligman also created the Masters of Applied Positive Psychology program at the University of Pennsylvania. Many institutions, including the National Institute on Aging, the National Science Foundation, and the

National Institute of Mental Health, have supported his research. He has received numerous awards, including two Distinguished Scientific Contribution awards from the APA, the William James Fellow Award, and the James McKeen Cattell Fellow Award from the Association for Psychological Science, the MERIT Award from the National Institute of Mental Health, the Laurel Award of the American Association for Applied Psychology and Prevention, and the Lifetime Achievement Award of the Society for Research in Psychopathology.

Cross-References

- ▶ Learned Helplessness
- ▶ Optimism
- ▶ Positive Psychology

References and Readings

Seligman, M. E. P. (1975). *Helplessness: On depression, development, and death*. San Francisco: W. H. Freeman.

Seligman, M. E. P. (1990). *Learned optimism*. New York: Knopf.

Seligman, M. E. P. (1993). *What you can change and what you can't: The complete guide to successful self-improvement*. New York: Knopf.

Seligman, M. E. P. (1996). *The optimistic child: Proven program to safeguard children from depression and build lifelong resilience* (p. 1996). New York: Houghton Mifflin.

Seligman, M. E. P. (2002). *Authentic happiness: Using the new positive psychology to realize your potential for lasting fulfillment*. New York: Free Press.

Seligman, M. (2008). Positive health. *Applied Psychology: An International Review, 57*, 3–18.

Seligman, M., & Csikzentmihalyi, M. (2000). Positive psychology: An introduction. *American Psychologist, 55*, 5–14.

Shah, N. (n.d.). *Seligman, Martin E. P.* Retrieved July 15, 2011 from http://www.pabook.libraries.psu.edu/palitmap/bios/Seligman_Martin.html

TED Conferences, LLC. (2008). *Speakers: Martin Seligman: Psychologist*. Retrieved July 15, 2011 from http://www.ted.com/index.php/speakers/martin_seligman.html

The Trustees of the University of Pennsylvania. (2006). *Authentic happiness*. http://www.authentichappiennes.sas.upenn.edu/seligman.aspx

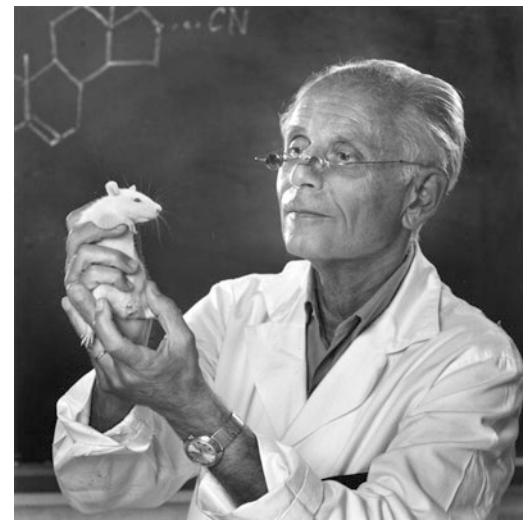
University of Pennsylvania. (2007). *Positive psychology center: Seligman bio*. Retrieved July 15, 2011 from http://www.ppc.sas.upenn.edu/bio.htm

Selye, Hans

Adrienne Stauder
Institute of Behavioural Sciences, Semmelweis University Budapest, Budapest, Hungary

Biographical Information

Dr. Hans Selye 1973
Photo by Yousuf Karsh



Hans Selye was born on January 26, 1907, in Vienna, Austria. His mother Maria Felicitas Langbank was an Austrian, his father Hugo Selye was a Hungarian military surgeon, and his grandfather and great grandfather were family doctors. Selye completed his elementary and secondary school education in Komarno (Slovakia). Since 1924 he studied medicine in Prague, Rome, and Paris, and obtained his diploma at the German University of Prague in 1929. He started his research at the Institute of Experimental Pathology in Prague, and got his doctorate in biochemistry in 1931.

A Rockefeller Research Fellowship allowed Selye to continue his scientific career at the Department of Biochemical Hygiene of the Johns Hopkins University in Baltimore in 1931,

and then from 1932 at the Department of Biochemistry of the McGill University in Montreal. He became lecturer then associate professor in biochemistry, and also in histology. He received Canadian citizenship in 1939, and became Doctor of Sciences in 1942. From 1945 to 1976 he directed the Institute of Experimental Medicine and Surgery (IMCE) at the University of Montreal, which gained international reputation under his leadership. After his retirement he remained active as founding president of the International Institute of Stress in Montreal (1976), and as co-founder of the Hans Selye Foundation (1979) until his death on October 15, 1982.

Selye held honorary doctorates from 18 universities, was a member of the Academy of Science of the Royal Society of Canada and 43 scientific societies, was an honorary citizen of many cities and countries, and received numerous high-ranking awards and distinctions. He was nominated for the Nobel Prize in physiology or medicine for 10 consecutive years (first in 1949), but he never received it. In 2006 he was inducted as a member of the Canadian Medical Hall of Fame.

Major Accomplishments

Selye is one of the most well-known, most productive scientists of the twentieth century. He wrote his name in the history of science by introducing the concept of stress. As a result of his work the word "stress" previously used in other contexts gained a physiological meaning and has been adopted in all languages. His research work had great impact in the field of endocrinology, physiology, biochemistry, epidemiology of chronic diseases, and behavioral medicine, not only on scientific thinking but also among lay people all around the world. Selye authored or coauthored 39 books and over 1,600 scientific articles, and his work has been estimated to have been cited in more than 300,000 scientific papers, although Somorjai (2007) could compile a list of only 800 of all his publications.

Selye's most important finding, the discovery of the stress syndrome, was accidental during his attempts to isolate a special female hormone from the placenta (Selye, 1964, 1967b). The originality of his thinking was that he started to research the significance of the nonspecific reactions that he observed. His first short note describing "the nonspecific response to nocuous agents" was published in Nature in 1936, followed in the same year by a longer article in The British Journal of Experimental Pathology describing the General Adaptation Syndrome. In the following years, he systematically developed his comprehensive theory on stress (Selye, 1950) and envisaged the existence of diseases of adaptation (Selye, 1946).

His researches on steroids turned his interest to the inflammatory process (Selye, 1943, 1949, 1965), then to the phenomena of calciphylaxis (Selye, 1962) and of thrombohemorrhagy (Selye, 1967a). Through these experiments he confirmed his hypothesis that diseases such as heart disease, rheumatoid arthritis, anaphylaxis, depression, autoimmune diseases, and Alzheimer disease are in fact all diseases of adaptation. Although his experiments were always on animals, over the years Selye became more and more interested in how his research results could be applied to medicine and to society. While his original definition of stress – "nonspecific response of the living organism to any stimuli, for example, effort, focused attention, pain, illness, failure, joy, success, that cause changes," – implied that the stressor can be either pleasant or unpleasant, since similar physiological/biochemical changes are produced, he later made the distinction between good stress (eustress) and bad stress (distress) (Selye, 1974).

Selye expanded his model to include Perception, Conditioning Factors, and Coping Mechanisms. This so-called *Selye – Smith Conceptual Model of Stress Variables* served as theoretical basis to the comprehensive course "Stress Management for Optimal Health" offered by Selye's Institute first to health professionals and then to the lay public (Smith & Selye, 1979). Selye proposed that stress education and stress management should be important elements of preventive medicine.

Research was a passion for Selye. He was very much interested in great discoveries, the history

and psychology of science, and in personal characteristics of scientists. These topics appeared in his lectures, books, and also in his every day discussions. He emphasized that original ideas were the most important elements in research that must be tested and proved by well-designed experiments. He also gave special attention to research methodology. On one hand, he was reluctant to adapt complicated technological methods, and he emphasized in all forums that one always should try to view the entire organism in its complexity and not to get lost with tiny little details without considering how they relate to the whole organism (Selye, 1967b). On the other hand he introduced new methods in experimental surgery (Selye, Bajusz, Grasso, & Mendell, 1960). He also carefully selected his laboratory assistants based on their skillfulness.

Effective information processing was another key element of Selye's exceptional productivity. He systematically developed his library that became world famous. He worked out his own "Symbolic Shorthand System for Medicine and Physiology" (SSS) that was subsequently published and used until the start of the computer era (Selye & Mishra, 1957). He also followed a very structured daily schedule at his Institute as well as in his private life (Selye, 1964). Selye was also a charismatic teacher. He shared his knowledge and his devotion to science with his fellow workers and students, exerting a deep influence on their lives and careers. He invited talented young researchers from all over the world to work in his Institute. He also invited many world renowned professors (the so-called Claude Bernard Professors). Thus, young researchers had the opportunity to meet famous personalities. They not only delivered one or two lectures, but participated in daily routine of the Institute and at informal dinners and discussions at Selye's house. Selye also traveled over the world and very often he obtained the recognition of his audience not only by his research results and presentation style, but also by giving his lecture in the language of the respective country. He not only shared his experiences with the scientific community, but could transmit his knowledge about stress, the process of scientific research, and related subjects to the general lay population by writing several popular books such as The Stress of Life (1956), From Dream to Discovery (1964), In Vivo: The Case for Supramolecular Biology (1967b), and Stress without Distress (1974) that have been translated into many languages.

Selye's scientific heritage is formally maintained by the Hans Selye Foundation, Montreal, Canada, www.stresscanada.org.

Cross-References

- ▶ Coping
- ▶ Stress
- ▶ Stress: Appraisal and Coping

References and Readings

Berczi, I. Stress and Disease: The contribution of Hans Selye to: Neuroimmune Biology. A personal reminiscence. Retrieved 30 Mar 2012 from http://home.cc.umanitoba.ca/~berczii/page2.htm.

Selye, H. (1936a). A syndrome produced by diverse nocuous agents. *Nature, 138*, 32.

Selye, H. (1936b). Thymus and adrenals in the response of the organism to injuries and intoxications. *British Journal of Experimental Pathology, 17*, 234–248.

Selye, H. (1943). Morphological changes in the fowl following chronic overdosage with various steroids. *Journal of Morphology, 73*, 401.

Selye, H. (1946). The general adaptation syndrome and the diseases of adaptation. *Journal of Clinical Endocrinology, 6*, 117–230.

Selye, H. (1949). Effect of ACTH and cortisone upon an anaphylactoid reaction. *Canadian Medical Association Journal, 61*, 553–556.

Selye, H. (1950). *Stress*. Montreal, QC: Acta.

Selye, H. (1956). *The stress of life*. New York: McGraw Hill.

Selye, H. (1962). *Calciphylaxis*. Chicago: University of Chicago Press.

Selye, H. (1964). *From dream to discovery*. New York: McGraw Hill.

Selye, H. (1965). *The mast cells*. Washington, DC: Butterworths.

Selye, H. (1967a). Experimental thrombohemorrhagic phenomena. *The American Journal of Cardiology, 20*(2), 153–160.

Selye, H. (1967b). *In vivo: The case for supramolecular biology*. New York: Livesight.

Selye, H. (1974). *Stress without distress* (p. 364). Philadelphia: Lippincott.

Selye, H. (1979). *The stress of my life: A scientist's memory*. New York: Van Nostrand Reinhold.
Selye, H., Bajusz, E., Grasso, S., & Mendell, P. (1960). Simple techniques for the surgical occlusion of coronary vessels in the rat. *Angiology, 11*, 398–407.
Selye, H., & Mishra, R. K. (1957). Symbolic shorthand system for medicine and physiology. *Federation Proceedings, 16*(3), 704–706.
Smith, M. J. T., & Selye, H. (1979). Stress: Reducing the negative effects of stress. *The American Journal of Nursing, 11*, 1953–1955.
Somorjai, N. (2007). Bibliography Hans Selye. Retrieved 30 Mar 2012 from http://www.selyesociety.hu/pdf/Selye_bibliography_2011.pdf.
Stauder, A., & Kovács, P. B. (Eds.). (2007). *Stress. A memorial book on the birth centenary of Hans Selye*. Budapest: Downtown Artists' Society.
The American Institute of Stress: Hans Selye and The Birth of Stress. Retrieved 30 Mar 2012 from http://www.stress.org/hans.htm
Université de Montréal: Hans Selye: Une vie en images. Retrieved 30 Mar 2012 from http://www.archiv.umontreal.ca/exposition/Hans_Selye/index.html.

SEM

▶ Structural Equation Modeling (SEM)

Semenogelase

▶ Prostate-Specific Antigen (PSA)

Seminin

▶ Prostate-Specific Antigen (PSA)

Seminoma

▶ Cancer, Testicular

Senior

▶ Elderly

Sense of Coherence

▶ Salutogenesis

Sense of Coherence – Measurement

▶ Resilience: Measurement

Sense of Self

▶ Self-Identity

Separation

▶ Divorce and Health

SEPs

▶ Needle Exchange Programs

Sera

▶ Serum

Serostatus: Seronegative and Seropositive

Angela White
Department of Psychology, University of Connecticut, Storrs, CT, USA

Synonyms

HIV status

Definition

Serostatus refers to the extent to which HIV antibodies can be detected in an individual's serum. This detection is an indicator of HIV infection.

Description

An individual is considered to be seronegative when the amount of HIV antibodies are not sufficient enough to be detected using an antibody test, although indication of HIV infection can be determined through the use of more sensitive culture, antigen, and viral gene detection techniques (Fultz, 1989; Kaslow & Francis, 1989; O'Malley, 1988). Individuals who are seronegative may not produce HIV antibodies for months or year after HIV infection, or they may stop producing these antibodies after some unknown time interval. There is a long incubation period associated with being seronegative; individuals may not show physical symptoms associated with HIV/AIDS for several months or years after the initial HIV infection. As such, it is difficult to determine the ability of seronegative individuals to spread HIV (Kaslow & Francis, 1989).

An individual is considered to be seropositive when HIV antibodies are detected on a HIV antibody test (Fultz, 1989; O'Malley, 1988). Individuals who have detectable HIV antibodies are considered to be "HIV-positive." The period of time between HIV infection and seroconversion (the presence of detectable antibodies in the serum) may range from 2 weeks to 3 months; this period of time is referred to as the "window period" (Stine, 2003).

Some individuals who are seropositive, or "HIV-positive," exhibit symptoms associated with an acute mononucleosis-like syndrome immediately after being infected with HIV. This syndrome has been known as acute HIV syndrome or acute retroviral syndrome; its symptoms include sweats, lethargy, headaches, muscle aches, fever, and sore throat (Fultz, 1989; Stine, 2003). Not all seropositive individuals experience this syndrome. Other seropositive individuals remain asymptomatic for several weeks. All seropositive individuals, whether symptomatic or asymptomatic, are infected with HIV and can infect others through blood and genital secretions and by transmission from mother to fetus (Fultz, 1989; O'Malley, 1988). In 2001, the Center for Disease Control and Prevention proposed the Serostatus Approach to Fighting the HIV Epidemic (SAFE) program to encourage awareness that individuals may be HIV-positive even if they appear to be outwardly healthy. This program attempted to reduce the spread of HIV by extending prevention services and improving treatment adherence for individuals with a seropositive status and by providing training to individuals who give these services (Normand, Vlahov, & Moses, 1995; Stine, 2003).

Being notified of a seropositive status is associated with behavioral and psychological changes. For instance, individuals who receive a positive test result may reduce their high-risk sexual activity. However, they also may experience increased stress and depression. Also, these individuals may feel compelled to disclose their HIV status to family, friends, and potential sexual parents. Some barriers associated with the disclosing HIV status include the disclosure of a stigmatized identity (such as being gay or an intravenous drug user), the fear of losing health insurance or employment, and the concern of being stigmatized by family and friends (Stall, Coates, Mandel, Morales, & Sorensen, 1989).

References and Readings

Black, P. H., & Levy, E. M. (1988). The HIV seropositive state and progression to AIDS: An overview of factors promoting progression. In P. O'Malley (Ed.), *The AIDS epidemic: Private rights and the public interest* (pp. 97–107). Boston: Beacon.

Fultz, P. (1989). The biology of human immunodeficiency viruses. In R. A. Kaslow & D. P. Francis (Eds.), *The epidemiology of AIDS: Expression, occurrence, and control of human immunodeficiency virus type 1 infection* (pp. 3–17). New York: Oxford University Press.

Kaslow, R. A., & Francis, D. P. (1989). Epidemiology: General considerations. In R. A. Kaslow & D. P. Francis (Eds.), *The epidemiology of AIDS: Expression, occurrence, and control of human immunodeficiency virus type 1 infection* (pp. 117–135). New York: Oxford University Press.

Normand, J., Vlahov, D., & Moses, L. E. (1995). *Preventing HIV transmission: The role of sterile needles and bleach*. Washington, DC: National Academy Press.

O'Malley, P. (1988). *The AIDS epidemic: Private rights and the public interest*. Boston: Beacon.

Stall, R., Coates, T. J., Mandel, J. S., Morales, E. S., & Sorensen, J. L. (1989). Behavioral factors and intervention. In R. A. Kaslow & D. P. Francis (Eds.), *The epidemiology of AIDS: Expression, occurrence, and control of human immunodeficiency virus type 1 infection* (pp. 266–281). New York: Oxford University Press.

Stine, G. J. (2003). *AIDS update2003: An annual overview of acquired immune deficiency syndrome*. Upper Saddle River, NJ: Prentice Hall.

Serotonin

Marc D. Gellman
Behavioral Medicine Research Center, Department of Psychology, University of Miami, Miami, FL, USA

Definition

Serotonin (5-hydroxytryptamine or 5-HT) is a neurotransmitter that is particularly important in central nervous system modulation. It is involved in the regulation of mood, appetite, and sleep, and in the cognitive functions of learning and memory.

Modulation of serotonin at synapses is thought to be a major action of several classes of pharmacological antidepressants, including selective serotonin reuptake inhibitors (SSRIs).

Cross-References

► Antidepressant Medications
► Central Nervous System
► Depression: Treatment
► Sleep

Serotonin Transporter Gene

Anett Mueller[1] and Turhan Canli[2]
[1]Department of Psychology, State University of New York at Stony Brook, Stony Brook, NY, USA
[2]Department of Psychology, Stony Brook University Psychology B-214, Stony Brook, NY, USA

Synonyms

SERT; SLC6A4 (solute carrier family 6, member 4)

Definition

In humans, the gene that encodes for the serotonin transporter is called the serotonin transporter gene which is modulated by the functional serotonin-transporter-linked polymorphism (5-HTTLPR), a variable number of tandem repeats in the 5' promoter region.

Description

The neurotransmitter *serotonin* (5-hydroxytryptamine, 5-HT) is probably best known for its modulation of neural activity and the modulation of various neuropsychological processes such as mood, perception, emotion, and cognition. Serotonin is also implicated in the pathogenesis of many psychiatric and neurological disorders and furthermore, it is involved in brain development and plasticity of brain areas related to cognitive and emotional processes (Berger, Gray, & Roth, 2009; Trevor, Katzung, & Masters, 2010). Additionally, the serotonin transporter is considered to be the initial site of action of broadly used antidepressant drugs, such as selective serotonin uptake inhibitors (SSRIs), and several potentially neurotoxic compounds.

Following neuronal stimulation, serotonin is transmitted into the synaptic cleft and then binds to receptors on the membrane of the postsynaptic

neuron. Serotonin is then removed from the synaptic cleft via special proteins, called *transporters*. Serotonin transporters are located in the serotonin neuron; they transport serotonin from the synaptic cleft back into the presynaptic neuron in both the brain and many peripheral tissues terminals. In humans, the gene that codes for the serotonin transporter contains a number of common variants (polymorphisms), including the serotonin-transporter-linked promoter region (*5-HTTLPR)* of the serotonin transporter gene (SLC6A4). This polymorphism is located upstream of the transcription start site on the long arm of the 17th chromosome (17q11.1-q12). The majority of alleles are composed of either fourteen ("short" or "*S*" allele) or sixteen repeated ("long" or "*L*" for allele) units, which differentially modulate on the expression and function of 5-HTT. The short form of *5-HTTLPR* has been associated with a reduced transcription of the 5-HTT gene, which leads to a decreased 5-HTT expression and availability and also a reduced 5-HT uptake (Lesch et al., 1996). In addition to the *S* and *L* alleles, there is an A > G single nucleotide polymorphism (SNP), a single nucleotide variation in a genetic sequence, upstream of the repetitive region that comprises the *5-HTTLPR*. The derived L_G allele has been associated with decreased 5-HTT transcription relative to the L_A allele. The frequency of *5-HTTLPR* alleles can vary substantially across ethnic groups, thus, showing population stratification.

The short allele variant of *5-HTTLPR* was first reported to be associated with personality traits such as neuroticism and harm avoidance (Lesch et al.,). Subsequent work has reported *5-HTTLPR* allelic variation to a wide range of phenotypes including aggression, anxiety, and affective disorders. Most studies have implied that *5-HTTLPR* has only a moderate impact on these behavioral predispositions of 3–4% or less of the total variance. The less active *S* allele has been associated, either by itself or in interaction with adverse life events, with abnormal levels of anxiety, fear, and depression. Despite the association between presence of the *S* allele and psychopathology, studies of patient responsiveness to SSRIs suggest no strong link between *5-HTTLPR* genotype and drug effectiveness.

However, if the *S* allele produced only deleterious consequences, evolutionary pressures should have led to its removal from the gene pool. Thus, more recent studies have begun to accrue evidence for favorable phenotypes associated with the *S* allele. For example, studies revealed an improved performance in (social) cognition in individuals with the *S* allele (Homberg & Lesch, 2010). On the other hand, the *L* allele, originally viewed as the protective allele, also has negative associations with at-risk phenotypes, such as cardiovascular health (e.g., increased cardiovascular reactivity and greater probability of myocardial infarction) (Fumeron et al., 2002; Williams et al., 2001) or certain psychiatric diseases (e.g., psychosis or posttraumatic stress disorder, PTSD) (Goldberg et al., 2009; Grabe et al., 2009). In addition to allelic association on observed phenotypes, there is growing evidence for gene-by-environment (G x E) interactions, suggesting that individuals possessing the *S* allele are predisposed to an increased risk for major depression or suicidal ideation as a function of early life stress. The first evidence for this G x E interaction in humans was presented by Caspi et al., (2003) who investigated more than 800 individuals over 23 years and found that life stress and depression was moderated by the *5-HTTLPR* genotype. Individuals with the *S* allele showed a higher probability of depressive symptoms, diagnosis of depression and suicidal attempts when exposed to stressful life events. However, replication studies have shown somewhat inconsistent results and thus, these findings are still a matter of debate. In addition to behavioral studies, noninvasive functional MRI (fMRI) studies have also shown that structural and functional characteristics of neural circuits involved in emotion and cognition can be moderated by the interaction of life stress and 5-HTTLPR genotype.

Most recently, investigators have come to recognize the potential gene regulatory role of epigenetic mechanisms in mediating environmental effects on brain function and on behavior

(Rutter, Moffitt, & Caspi, 2006). For example, it has been argued that environmental influences bear the potential to persistently modify neuronal units during early development by epigenetic programming of emotionality (Weaver et al., 2004; Weaver, 2007). This has first been shown with respect to the glucocorticoid receptor gene and individual differences in rodents' stress reactivity: variations in maternal care have been shown to modify the expression of genes that regulate behavioral and endocrine responses to stress and hippocampus synaptic development. Thus, alterations of particular genomic regions within the 5-HTT in response to varying environmental conditions might serve well as a major source of variation in biological and behavioral phenotypes. Indeed, there is now emerging evidence linking *5-HTTLPR* genotype to individual differences in epigenetic methylation (Philibert et al., 2007, 2008).

Studies that use biological endophenotypes, such as stress-induced HPA activation, might be more strongly associated with a specific polymorphism than a psychiatric disorder (Uher & McGuffin, 2010). However, given the fact that brain serotonin and more specifically *5-HTTLPR* shows pleiotropic behavioral effects, we need to learn and understand more about the biological function and how *5-HTTLPR* becomes associated with various different phenotypes. The modulation of these multiple behavioral processes might very likely be regulated by multiple serotonin receptors that are expressed in multiple brain regions. In summary, *5-HTTLPR* seems to play an important, though not yet fully understood, role in behavioral medicine. We will likely gain new serotonergic drugs and disease treatments as well as a more thorough understanding of complexity of human biology from this research.

Cross-References

- ▶ Depression
- ▶ Gene-Environment Interaction
- ▶ Serotonin

References and Readings

Berger, M., Gray, J. A., & Roth, B. L. (2009). The expanded biology of serotonin. *Annual Review of Medicine, 60*, 355–366.

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., et al. (2003). Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. *Science, 301*(5631), 386–389.

Fumeron, F., Betoulle, D., Nicaud, V., Evans, A., Kee, F., Ruidavets, J. B., et al. (2002). Serotonin transporter gene polymorphism and myocardial infarction: Etude Cas-Temoins de l'Infarctus du Myocarde (ECTIM). *Circulation, 105*(25), 2943–2945.

Goldberg, T. E., Kotov, R., Lee, A. T., Gregersen, P. K., Lencz, T., Bromet, E., et al. (2009). The serotonin transporter gene and disease modification in psychosis: Evidence for systematic differences in allelic directionality at the 5-HTTLPR locus. *Schizophrenia Research, 111*(1–3), 103–108.

Grabe, H. J., Spitzer, C., Schwahn, C., Marcinek, A., Frahnow, A., Barnow, S., et al. (2009). Serotonin transporter gene (SLC6A4) promoter polymorphisms and the susceptibility to posttraumatic stress disorder in the general population. *The American Journal of Psychiatry, 166*(8), 926–933.

Homberg, J. R., & Lesch, K. P. (2010). Looking on the bright side of serotonin transporter gene variation. *Biological Psychiatry, 69*, 513–519.

Lesch, K. P., Bengel, D., Heils, A., Sabol, S. Z., Greenberg, B. D., Petri, S., et al. (1996). Association of anxiety-related traits with a polymorphism in the serotonin transporter gene regulatory region. *Science, 274*(5292), 1527–1531.

Philibert, R., Madan, A., Andersen, A., Cadoret, R., Packer, H., & Sandhu, H. (2007). Serotonin transporter mRNA levels are associated with the methylation of an upstream CpG island. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics, 144B*(1), 101–105.

Philibert, R. A., Sandhu, H., Hollenbeck, N., Gunter, T., Adams, W., & Madan, A. (2008). The relationship of 5HTT (SLC6A4) methylation and genotype on mRNA expression and liability to major depression and alcohol dependence in subjects from the Iowa Adoption Studies. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics, 147B*(5), 543–549.

Rutter, M., Moffitt, T. E., & Caspi, A. (2006). Gene-environment interplay and psychopathology: Multiple varieties but real effects. *Journal of Child Psychology and Psychiatry, 47*(3–4), 226–261.

Trevor, A. J., Katzung, B. G., & Masters, S. B. (2010). *Katzung and Trevor's pharmacology examination and board review* (9th ed.). New York: McGraw-Hill Medical.

Uher, R., & McGuffin, P. (2010). The moderation by the serotonin transporter gene of environmental adversity in the etiology of depression: 2009 update. *Molecular Psychiatry, 15*(1), 18–22.

Weaver, I. C. (2007). Epigenetic programming by maternal behavior and pharmacological intervention. Nature versus nurture: Let's call the whole thing off. *Epigenetics, 2*(1), 22–28.
Weaver, I. C., Cervoni, N., Champagne, F. A., D'Alessio, A. C., Sharma, S., Seckl, J. R., et al. (2004). Epigenetic programming by maternal behavior. *Nature Neuroscience, 7*(8), 847–854.
Williams, R. B., Marchuk, D. A., Gadde, K. M., Barefoot, J. C., Grichnik, K., Helms, M. J., et al. (2001). Central nervous system serotonin function and cardiovascular responses to stress. *Psychosomatic Medicine, 63*(2), 300–305.

SERT

▶ Serotonin Transporter Gene

Sertraline

▶ Selective Serotonin Reuptake Inhibitors (SSRIs)

Serum

Briain O. Hartaigh
School of Sport and Exercise Sciences,
The University of Birmingham,
Edgbaston, Birmingham, UK

Synonyms

Antiserum; Sera

Definition

Serum is blood plasma with the coagulatory proteins removed. It is a clear, pale-yellow, thin, and sticky fluid that moistens the surface of serous membranes or that is secreted by such membranes when they become inflamed. In blood, serum is obtained after coagulation, upon separating whole blood into its solid and liquid components. This is achieved whereby blood is drawn from the subject and is allowed to naturally form a blood clot. After blood is allowed to clot and stand, a centrifuge is used to extract the red blood cells and the blood clot, which contains platelets and fibrinogens. In practice, blood serum is used in numerous diagnostic tests as well as blood typing.

Cross-References

▶ Antibodies
▶ Antigens

References and Readings

Abbas, A. K., & Lichtman, A. H. (2004). *Basic immunology: Functions and disorders of the immune system* (2nd ed.). Philadelphia: Saunders.

Service Attendance

▶ Religious Ritual

Sex

▶ Sexual Behavior

Sex Differences

▶ Gender Differences

Sex Hormones

▶ Estrogen

Sexual Activity

► Sexual Behavior

Sexual Behavior

Jennifer L. Brown
Department of Behavioral Sciences and Health Education, Emory University School of Public Health, Atlanta, GA, USA

Synonyms

Sex; Sexual activity

Definition

There is a diverse array of activities that can be classified as sexual behavior: masturbation, oral-genital stimulation (oral sex), penile-vaginal intercourse (vaginal sex), and anal stimulation or anal intercourse. Sexual behaviors may also include activities to arouse the sexual interest of others or attract partners. Individuals engage in sexual behaviors for a variety of reasons, differ in their acceptability based on societal norms, and change across the lifespan.

Description

What is Sexual Behavior: Types of Sexual Behaviors

Sexual behavior includes a wide variety of activities individuals engage in to express their sexuality (Crooks & Baur, 2008). Abstinence and celibacy are terms used for individuals who do not engage in certain or any sexual behaviors. Kissing and touching are sexual behaviors that stimulate the erogenous zones of one's partner. Masturbation is a sexual behavior referring to stimulation of one's genitals to create sexual pleasure. Individuals may also engage in oral stimulation of a partner's genitals; terms used to describe oral-genital stimulation include: oral sex (referring broadly to oral-genital stimulation), cunnilingus (oral stimulation of the vulva), and fellatio (oral stimulation of the penis). Anal stimulation includes either touching around the anus or penile insertion in the anus (often referred to as anal sex). Penile-vaginal intercourse involves insertion of the penis into a female's vagina; this behavior too has a variety of other synonymous terms (e.g., vaginal sex, coitus). The frequency that these and other sexual behaviors are engaged in has enormous individual variability and may differ based upon many factors (e.g., social acceptability, age).

Sexual Behavior: The Role of Societal Norms

Societal norms for acceptable sexual behaviors differ across cultures. Paraphilia refers to less common sexual behaviors within a given society or culture; an example of such behavior is a fetish. In some cultures, the nature of the relationship affects which behaviors are deemed acceptable. For instance, sexual behavior may be deemed appropriate only within the context of marriage. Similarly, societal perspectives on sexual orientation may influence whether sexual behaviors are viewed as socially acceptable.

Reasons for Sexual Behavior Engagement

Individuals engage in sexual behaviors for a multitude of reasons. Sexual behavior may be engaged in to experience sexual pleasure, sexual arousal, or orgasm. Procreation or a desire for children may motivate sexual behavior. Sexual behaviors may also be used to earn money or acquire other goods or services; prostitution refers to the exchange of a sexual behavior for monetary or other compensation. Additionally, pornography may motivate engagement in sexual behaviors. Unfortunately, sexual behaviors also occur in nonconsensual or coerced contexts (e.g., rape) and in the form of abuse (e.g., child sexual abuse) or sexual exploitation (e.g., pedophilia). Sexual behavior engagement

may also have unintended consequences (e.g., unplanned pregnancy) or pose health risks associated with the acquisition of sexually transmitted diseases, including human immunodeficiency virus (HIV).

Developmental Perspectives on Sexual Behavior Engagement

Engagement in sexual activity changes across one's lifespan, and there is considerable variation in sexual development (Crooks & Baur, 2008). During childhood, sexual behaviors may include self-stimulation of the genitals or engagement in play that may be viewed as sexual in nature (e.g., "playing doctor" with a peer). Puberty typically occurs during adolescence and results in dramatic physical changes including the development of secondary sex characteristics. Adolescence is typically linked to increases in sexual activity, both self-stimulation behaviors and sexual behavior with partners. During adulthood, and as people age, there is considerable individual variation of sexual behavior engagement.

Cross-References

- ▶ Condom Use
- ▶ HIV Infection
- ▶ HIV Prevention
- ▶ Sexual Functioning
- ▶ Sexual Hookup
- ▶ Sexual Risk Behavior

References and Readings

Crooks, R., & Baur, K. (2008). *Our sexuality* (10th ed.). Pacific Grove, CA: Thomson.

Sexual Dysfunction

- ▶ Sexual Functioning

Sexual Functioning

Robyn Fielder
Center for Health and Behavior, Syracuse University, Syracuse, NY, USA

Synonyms

Sexual dysfunction

Definition

Sexual functioning is characterized by absence of difficulty moving through the stages of sexual desire, arousal, and orgasm, as well as subjective satisfaction with the frequency and outcome of individual and partnered sexual behavior.

Description

Sexual functioning is an important aspect of quality of life. Our understanding of sexual functioning is influenced by not only the current state of medical knowledge but also the social values upheld in our culture. Healthy sexual functioning is characterized by a lack of pain or discomfort during sexual activity and a lack of physiological difficulty moving through the three-phase sexual response cycle of desire, arousal, and orgasm. In addition, sexual functioning is indicated by subjective feelings of satisfaction with the frequency of sexual desire and sexual behavior, as well as subjective pleasure during individual and partnered sexual activity.

Kaplan's three-phase model is the basis for current models of healthy sexual response. The desire phase consists of sexual fantasies and desire to engage in sexual behavior. The arousal phase involves subjective feelings of pleasure along with physiological changes conducive to sexual intercourse. Males experience penile tumescence and erection, and females experience pelvic vasocongestion and vaginal lubrication. The orgasm phase consists of peak feelings of

sexual pleasure and a release of sexual tension. Males ejaculate semen, whereas females experience contractions of the outer vaginal wall; additionally, in both males and females, the anal sphincter contracts. Individuals may experience physiological and/or psychological difficulties at any or all of the three phases of sexual response. A resolution period, characterized by relaxation and, for males, a refractory period, follows orgasm.

Etiology of Sexual Dysfunction

The etiology of sexual problems is often a complex combination of biological/medical, psychological, and social factors. For example, the sexual dysfunction may be secondary to a chronic health condition or psychotropic medication. In other cases, performance anxiety, low mood, or previous traumatic experiences may impair sexual functioning. Moreover, conflicts within a relationship as well as within the larger sociocultural context may affect an individual's sexual functioning. Due to the variety of potential predisposing, precipitating, and maintaining factors, clinicians are encouraged to take a biopsychosocial approach to assessment and treatment of problems in sexual functioning.

Sexual Dysfunction Disorders

Consistent with the medical model of disease, most research and scholarship focuses on sexual dysfunction rather than healthy sexual functioning. The American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (2000) describes nine main disorders of sexual dysfunction, which are grouped into four categories: desire, arousal, orgasm, and pain. All nine disorders share some common diagnostic criteria: the dysfunction is persistent and recurrent; the dysfunction is not substance-induced, due to a general medical condition, or part of another Axis I mental disorder; and the dysfunction causes clinically significant distress or interpersonal difficulty. Sexual dysfunctions are also classified according to their onset (lifelong or acquired), context (generalized or situational), and etiology (due to psychological factors or due to combined psychological and medical factors). Additional diagnostic options include sexual dysfunction due to a general medical condition, substance-induced sexual dysfunction, and sexual dysfunction not otherwise specified.

Desire disorders are characterized by lack of interest in sex, absence of sexual fantasies and sexual behavior, or fear of sexual contact. In hypoactive sexual desire disorder, there is a low level of sexual fantasy and desire for sex. In sexual aversion disorder, genital sexual contact is feared and actively avoided.

Arousal disorders are characterized by difficulty attaining or maintaining sexual arousal. Male erectile disorder is the most researched type of sexual dysfunction and receives the most media attention, particularly since the advent of Sildenafil (Viagra) in 1998. Erectile dysfunction is the inability to maintain an erection adequate for sexual penetration until completion of sexual activity. Female sexual arousal disorder is the inability to attain or maintain vaginal lubrication until completion of sexual activity.

Orgasm disorders are characterized by delay in or absence of orgasm on one extreme, or, on the other extreme, by occurrence of orgasm before the individual wants. In female orgasmic disorder and male orgasmic disorder, orgasm is delayed or absent despite normal sexual arousal and sufficient sexual stimulation. Premature ejaculation describes the occurrence of ejaculation with minimal stimulation before, upon, or soon after penetration and before the individual wants to orgasm.

Sexual pain disorders are characterized by genital pain that is not due to a general medical condition. In dyspareunia, which affects both males and females, genital pain occurs during sex. In vaginismus, involuntary muscle spasms of the vagina prevent penetration or may cause pain if it is attempted.

Prevalence of Sexual Dysfunction

Epidemiological surveys suggest problems with sexual functioning are common among the general population. For example, prevalence rates of premature ejaculation, erectile dysfunction, and female orgasmic disorder are 5%, 5%,

and 10%, respectively, among community samples (Wincze & Carey, 2001). Symptoms that do not meet full diagnostic criteria for a sexual dysfunction disorder are likely much more common. Patients struggling with sexual health problems may be reluctant to seek medical consultation due to embarrassment or privacy concerns. Many health care providers are also uncomfortable discussing sexuality, so patients' sex-related questions and concerns may be neglected in clinical settings.

Assessment of Sexual Functioning

Clinicians are advised to employ multimethod assessment of sexual functioning by including medical, psychosocial, and psychophysiological components. All three perspectives provide valuable information that aids in diagnosing sexual dysfunction, hypothesizing the etiology of the problem, and developing an appropriate treatment plan.

A medical evaluation is an essential piece of the sexual functioning assessment. A general physical examination allows for biological causes (e.g., general medical conditions, such as diabetes or cancer, as well as other vascular, neurologic, or hormonal conditions) to be ruled out. A gynecological or urological exam ensures no anatomical complications. Physical symptoms, such as bleeding or pain, can also be addressed. Medical providers should attend to any notable medical history (e.g., surgeries) as well as any prescription medications or substance use that may affect sexual functioning.

For the psychosocial evaluation, an interview is essential to learn about the onset, frequency, intensity, and duration of the presenting complaint(s). In addition, clinicians should assess pertinent areas including family history, adolescence, significant relationships in adulthood, sexual history, and sexual abuse or trauma. Although an individual patient may present with sexual complaints, difficulties with sexual functioning are often better understood in the context of the individual's sexual relationship. In many cases (e.g., when working with a patient who is married or in a committed relationship), involving the patient's sexual partner in the psychosocial evaluation (with the patient's permission) facilitates a better resolution. It is often advisable to interview the patient's sexual partner separately to find out more about the presenting complaint. A joint interview with the patient and his or her partner also provides additional insight into the couple's interaction style, relationship quality, and non-sex-related problems that may cause interpersonal tension. For some patients, self-administered questionnaires may be used to supplement the psychosocial interview. Questionnaires may provide an easier method whereby to disclose sensitive information compared to a face-to-face interview.

The third potential component of the evaluation is psychophysiological assessment. Depending on the presenting complaint, psychophysiological measures can be quite helpful in differential diagnosis. For example, nocturnal penile tumescence is the gold standard for differential diagnosis for male patients with erectile dysfunction. Inability to obtain erections during sleep indicates a medical cause, whereas ability to maintain erections during sleep suggests a psychosocial cause.

Treatment of Sexual Dysfunction

Often the first therapeutic intervention occurs during the comprehensive assessment. During the psychosocial and medical evaluations, clinicians normalize the patient's problem, provide information, and correct misunderstandings. Formal treatment plans will depend on the presumed cause of the sexual dysfunction. Medical treatments include options such as pharmacotherapy (e.g., Viagra for erectile dysfunction), gels and creams (e.g., lubricating gels to compensate for problems with female arousal), hormone replacement therapy (e.g., testosterone to increase sexual desire), and surgery (e.g., penile implants to treat organic erectile dysfunction). Psychological treatments include psychoeducation about healthy sexual functioning, behaviorally focused sex therapy with the patient or the patient and his or her partner (e.g., using the stop-start technique to treat premature ejaculation), interpersonally focused couples therapy with the patient and his or her partner (e.g., working on trust or

communication), or more traditional individual therapy with the patient (e.g., treating mood, anxiety, or trauma symptoms).

Cross-References

► Sexual Behavior

References and Readings

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.

Balon, R., & Segraves, R. T. (Eds.). (2005). *Handbook of sexual dysfunction* (Medical psychiatry series, Vol. 30). Boca Raton, FL: Taylor & Francis.

Balon, R., & Segraves, R. T. (Eds.). (2009). *Clinical manual of sexual disorders*. Arlington, VA: American Psychiatric Publishing.

Carey, M. P., & Gordon, C. M. (1995). Sexual dysfunction among heterosexual adults: Description, epidemiology, assessment, and treatment. In L. Diamant & R. McAnulty (Eds.), *The psychology of sexual orientation, behavior, and identity: A handbook* (pp. 165–196). Westport, CT: Greenwood.

Fagan, P. J. (2004). *Sexual disorders: Perspectives on diagnosis and treatment*. Baltimore, MD: The Johns Hopkins University Press.

IsHak, W. W. (Ed.). (2008). *The guidebook of sexual medicine*. Beverly Hills, CA: A & W Publishing.

Maurice, W. L. (1999). *Sexual medicine in primary care*. St. Louis, MO: Mosby.

Nusbaum, M., & Rosenfeld, J. A. (2005). *Sexual health across the lifecycle: A practical guide for clinicians*. Cambridge: Cambridge University Press.

Rowland, D. L., & Incrocci, L. (Eds.). (2008). *Handbook of sexual and gender identity disorders*. Hoboken, NJ: Wiley.

Wincze, J. P., & Carey, M. P. (2001). *Sexual dysfunction: Guide for assessment and treatment* (2nd ed.). New York: Guilford.

Sexual Hookup

Robyn Fielder
Center for Health and Behavior, Syracuse University, Syracuse, NY, USA

Synonyms

Casual sex; Hooking up

Definition

A sexual hookup is a sexual interaction between partners who are not dating or in a committed romantic relationship. There is no universal definition of sexual hookups, but qualitative research has begun to converge on the most common interpretation of hookup, which has three main components. First, hookups may involve a range of sexual behaviors, from kissing to sex. Kissing and sexual touching occur more frequently, but oral and vaginal sex occur during a significant minority of hookups. Anal sex during a hookup is rare. The variety of sexual behaviors that can occur during hookups causes ambiguity. From a research or public health perspective, behavioral specificity is needed to distinguish among different levels of sexual risk behavior. Condom use is rare during oral sex hookups, suggesting a potential risk for sexually transmitted diseases.

Second, hookup partners are not dating or in a committed romantic relationship. They may be friends, acquaintances, or strangers, or they may have been in a romantic relationship in the past. The most common connection between partners is friendship. Third, hookups do not signify an impending romantic commitment, so partners typically do not expect a relationship to result from the encounter. Instead, hookups are expected to serve a utilitarian function of sexual pleasure. However, individuals may desire a relationship with their hookup partner, and some engage in hookups with the hope that a relationship will eventually develop. Besides these three main criteria, hookups are also understood in terms of what they lack (emotional attachment and commitment). Emerging adults' descriptions of typical hookups are highly consistent, even between those who have and have not hooked up.

Several biomedical, sociocultural, and college environment changes occurring over the past 50 years have contributed to emergence of the hookup culture. Notably, emerging adults increasingly choose to postpone serious committed relationships to focus on self-development. Hookups offer a convenient way to obtain sexual intimacy without the commitment or time

investment required by a relationship. Accordingly, hooking up has become very common among adolescents and emerging adults. A minority of middle and high school students and the majority of college students report hookup experience. Hooking up has replaced traditional dating as the main way to explore relationships and sexual behavior on college campuses. Hookup behavior among similarly aged noncollege attending youth is rarely studied.

Research has investigated several characteristics of sexual hookups. Hookups frequently co-occur with alcohol use, and alcohol use is a strong predictor of hookup behavior. Hookups are often spontaneous, but some individuals plan to hook up (either with a particular partner or with anyone). A variety of sexual, emotional, and social motives may lead individuals to hook up, such as sexual desire, intoxication, excitement, and desire to feel attractive. Some hookup partners interact only once, but some hook up multiple times, which is sometimes known as "friends with benefits." Hookups are also related to casual sex, as both lack emotional attachment. The main differences are the greater variety of sexual behaviors and partner types involved in hookups compared to casual sex and the extent to which hooking up has become a normative experience for youth.

Cross-References

- ▶ Condom Use
- ▶ Sexual Behavior
- ▶ Sexual Risk Behavior

References and Readings

Bogle, K. A. (2008). *Hooking up: Sex, dating, and relationships on campus*. New York: New York University Press.

Owen, J. J., Rhoades, G. K., Stanley, S. M., & Fincham, F. D. (2010). "Hooking up" among college students: Demographic and psychosocial correlates. *Archives of Sexual Behavior, 39*, 653–663.

Stinson, R. D. (2010). Hooking up in young adulthood: A review of factors influencing the sexual behavior of college students. *Journal of College Student Psychotherapy, 24*, 98–115.

Sexual Maturation

▶ Puberty

Sexual Orientation

Jason W. Mitchell
Center for AIDS Intervention Research, Medical College of Wisconsin, Milwaukee, WI, USA

Definition

Sexual orientation is the sexual attraction, emotional, and/or romantic state that a person endures toward women, men, or both sexes. Sexual orientation also pertains to a person's sense of identity that is tied to these attractions, behaviors, and membership into a community with similar individuals (American Psychological Association [APA], 2011). Therefore, sexual orientation is the compilation of a person's sexual behavior and sense of sexual identity.

These two core components of sexual orientation are developed across a person's life span, yet many believe it to be an innate and fixed state (APA, 2011). Nonetheless, an individual's sexual orientation is often characterized with a label, such as heterosexuality, homosexuality, or bisexuality. Sometimes asexuality is also considered as a separate entity of sexual orientation. Those labels normally, but not always, incorporate and include the individuals' sexual identity and sexual behavior of her or his sexual orientation. Because sexuality may be viewed as a fluid construct, an individual's sexual behavior may change over time while maintaining the same sexual identity. An example of this phenomenon would be when a heterosexual male experiments sexually with another male. Another exception to this generalization is when a self-identified lesbian woman has sex with a male. As such, these categories exist on a continuum of sexuality.

Scientists and psychologists have created measures of sexual orientation to better assess

an individuals' sexuality. Typically, these measures will include a range of "solely heterosexual" to "solely homosexual" with "bisexuality" falling somewhere in between these two categories and "asexuality" not being included. A variety of measurements exist to assess sexual orientation. However, an individual's sexual orientation may change over time, and as such, measuring this construct at a single point in time (i.e., cross-sectional study) does have its limitations. Nonetheless, more studies are including a variety of measures that assess the sexual behavior and identity dimensions of sexual orientation.

The most well-recognized measurement of sexual orientation for males and females is the Kinsey scale (Kinsey, Pomeroy, & Martin, 1948; Kinsey, Pomeroy, Martin, & Gebhard, 1953). The scale ranges from 0 for "exclusively heterosexual with no homosexual" to 6 for "exclusively homosexual." The original scale does not include an "asexual" rating or category nor does it take into account any changes of sexual orientation over a period of time. Since then, other measurements of sexual orientation have been created and reviewed. For more information and references, please refer to the further readings section.

Cross-References

▶ Sexual Behavior

References and Readings

American Psychological Association. (2011). *Sexual orientation and homosexuality*. Retrieved February 4, 2011, from http://www.apa.org/topics/sexuality/orientation.aspx

Chung, Y. B., & Katayama, M. (1996). Assessment of sexual orientation in lesbian/gay/bisexual studies. *Journal of Homosexuality, 30*(4), 49–62.

Kinsey, A., Pomeroy, W., & Martin, C. (1948). *Sexual behavior in the human male*. Philadelphia: Saunders. ISBN 978-0253334121.

Kinsey, A., Pomeroy, W., Martin, C., & Gebhard, P. (1953). *Sexual behavior in the human female*. Philadelphia: Saunders. ISBN 978-0253334114.

Sell, R. L. (1996). The sell assessment of sexual orientation: Background and scoring. *Journal of Gay, Lesbian, and Bisexual Identity, 1*(4), 295–310.

Sexual Risk

▶ Sexual Risk Behavior

Sexual Risk Behavior

Theresa Senn
Center for Health and Behavior,
Syracuse University, Syracuse, NY, USA

Synonyms

Sexual risk; Unprotected sex

Definition

Sexual risk behavior is any sexual behavior (typically condom-unprotected oral, vaginal, or anal intercourse) that puts one at risk for an adverse health outcome. Adverse health outcomes may include an unwanted pregnancy or contracting a sexually transmitted disease (STD), including human immunodeficiency virus (HIV).

Vaginal intercourse is the only sexual behavior that puts an individual at risk for unwanted pregnancy. There are many methods for reducing the risk of unwanted pregnancy, including hormonal contraceptives, correct and consistent condom use, surgical methods such as a vasectomy or tubal ligation, and other methods such as an intrauterine device or a diaphragm.

Sexual behavior falls on a continuum from no risk to low risk to high risk for contracting an STD. The risk level of the sexual behavior depends on the STD under consideration. For example, with respect to HIV, masturbation incurs virtually no risk, oral sex is low risk, and vaginal and anal intercourse are high-risk behaviors. However, oral sex is a high-risk behavior for contracting some STDs such as gonorrhea.

The risk level of a particular sexual behavior also depends on with whom the behavior occurs.

Any sexual behavior that occurs when an individual is alone is generally no risk because the individual is not at risk of contracting an STD from him- or herself. In addition, any sexual behavior that occurs within the context of a mutually monogamous relationship, in which both individuals are not infected with any STDs (especially when confirmed by testing), confers no risk of contracting an STD for either individual. Sexual behavior puts an individual at risk for contracting an STD only when his or her sexual partner is infected with an STD.

The risk of a particular sexual behavior also depends on the individual's sexual network. Sexual risk increases with an increasing number of sexual partners, and/or an increasing number of a sex partner's partners, because there is an increasing likelihood that one of these individuals is infected with an STD. In other words, the larger the sexual network, the greater the sexual risk.

STDs can have serious consequences, including epididymitis in men, pelvic inflammatory disease and pregnancy complications in women, infertility, and cancer (Centers for Disease Control and Prevention, 2010d). In addition, the presence of another STD facilitates the transmission of HIV through sexual exposure (Centers for Disease Control and Prevention, 2007). HIV weakens the immune system, ultimately leading to death when the immune system is so weakened it is unable to fight off infections or cancers (Centers for Disease Control and Prevention, 2010a). STDs can also have negative relationship and social consequences due to the stigma surrounding some STDs.

The majority of STDs are either bacterial or viral (Holmes et al., 2008). In general, bacterial STDs can be cured with antibiotics, although some STDs are becoming resistant to antibiotics that had previously successfully treated the disease (Centers for Disease Control and Prevention, 2010b). Viral STDs have no cure, although there are medications that can help to manage outbreaks or viral load. STDs may sometimes be cleared from the body with no treatment (Centers for Disease Control and Prevention, 2010c).

Condoms are an effective way to reduce the risk of contracting an STD when they are used consistently and correctly. Although the value of condoms for the prevention of some STDs that can be transmitted through genital-to-genital contact, such as human papillomavirus and herpes simplex virus, has been debated, recent evidence suggests that condoms reduce the risk of STD infection from these pathogens as well (Holmes et al., 2008).

Numerous factors influence sexual risk behavior. These factors can be broadly categorized into individual-level factors, partner- or relationship-level factors, and social or structural factors. Researchers have typically focused on only one level of influence at a time, although the integration of individual-level, partner or relationship-level, and social or structural factors has recently been attempted in the Network-Individual-Resource Model for HIV prevention (Johnson et al., 2010).

The relation between individual-level factors and sexual risk behavior has been extensively researched. Numerous health behavior theories, including the health belief model, social-cognitive theory, the theory of planned behavior, and the information-motivation-behavioral skills model, have been used to explain why individuals engage in sexual risk behavior (Fisher & Fisher, 2000). Constructs from these models such as perceived risk, benefits of and barriers to risk reduction, self-efficacy, social norms, attitudes, intentions, and skills have been associated with sexual risk behavior (Fisher & Fisher, 2000).

Because sexual risk behavior usually occurs within a dyad, researchers have begun to consider partnership-level influences on sexual behavior. At the partnership level, variables such as intimate partner violence and the balance of power in a relationship may influence sexual risk behavior. There are few existing theories that incorporate partner influences on sexual risk behavior; one exception is a framework recently proposed by Karney et al. (2010), which posits that sexual risk behavior is influenced by the ability to communicate about and coordinate sexual behavior, which, in turn, is influenced by the individual beliefs and motivations of each partner as well as by the nature of the relationship.

Although it is commonly accepted that social and structural factors influence sexual risk behavior, because of the complexity and breadth of

these factors, it is difficult to develop a general model that predicts how these factors influence sexual risk behavior. Several broad frameworks have been suggested that specify different levels of structural influence (Gupta, Parkhurst, Ogden, Aggleton, & Mahal, 2008). Structural factors that influence sexual risk behavior vary depending on the social, cultural, and economic conditions faced by the population under study. Some structural factors associated with sexual risk behavior include gender inequality and poverty (Gupta et al., 2008). In the United States, one factor that has received considerable recent attention is the male-to-female ratio. Social and structural factors such as the high mortality rate among African American males due to disease and violence, high rate of incarceration among African American males, and high rates of poverty and unemployment among African American males (making them less desirable as potential husbands) have led to an unbalanced ratio of available African American men to women. This shortage of men relative to women may reduce women's power in relationships and their ability to insist on monogamy, ultimately leading to high rates of partner concurrency (Adimora & Schoenbach, 2002).

Behavioral medicine researchers and practitioners have played an important role in the design and evaluation of interventions to reduce sexual risk behavior. Numerous interventions have been developed to target the individual-level determinants of sexual risk behavior. These interventions, particularly those that include motivational and skills elements, are effective in reducing sexual risk behavior (Crepaz, Horn et al., 2007; Crepaz, Marshall et al., 2009; Johnson, Carey, Chaudoir, & Reid, 2006). Few interventions have been developed to target the partnership-level determinants of sexual risk behavior (Karney et al., 2010); this is an important area for future research. Although there are challenges to implementing and evaluating structural interventions, some programs, such as microcredit programs for women and policies requiring condoms be used for sex work, have successfully reduced sexual risk behavior in some settings (Gupta et al., 2008). Additional research on structural-level sexual risk reduction interventions is needed, as is research on interventions that target multiple levels of influence.

References and Readings

Adimora, A. A., & Schoenbach, V. J. (2002). Contextual factors and the black–white disparity in heterosexual HIV transmission. *Epidemiology, 2002*, 707–712.

Centers for Disease Control and Prevention. (2007). *CDC fact sheet: The role of STD prevention and treatment in HIV prevention*. Retrieved 6 Jan 2011 from http://cdc.gov/std/HIV/stds-and-hiv-fact-sheet.pdf

Centers for Disease Control and Prevention. (2010a). *Basic information about HIV and AIDS*. Retrieved 6 Jan 2011 from http://www.cdc.gov/hiv/topics/basic/index.htm

Centers for Disease Control and Prevention. (2010b). *CDC fact sheet: Basic information about antibiotic-resistant gonorrhea (ARG)*. Retrieved 6 Jan 2011 from http://cdc.gov/std/Gonorrhea/arg/basic.htm

Centers for Disease Control and Prevention. (2010c). *CDC fact sheet: Genital HPV*. Retrieved 6 Jan 2011 from http://cdc.gov/std/hpv/hpv-fact-sheet-press.pdf

Centers for Disease Control and Prevention. (2010d). *CDC fact sheets: Sexually transmitted diseases*. Retrieved 6 Jan 2011 from http://cdc.gov/std/healthcomm/fact_sheets.htm

Crepaz, N., Horn, A. K., Rama, S. M., Griffin, T., Deluca, J. B., Mullins, M. M., et al. (2007). The efficacy of behavioral interventions in reducing HIV risk sex behaviors and incident sexually transmitted disease in Black and Hispanic sexually transmitted disease clinic patients in the United States: A meta-analytic review. *Sexually Transmitted Diseases, 34*, 319–332.

Crepaz, N., Marshall, K. J., Aupont, L. W., Jacobs, E. D., Mizuno, Y., Kay, L. S., et al. (2009). The efficacy of HIV/STI behavioral interventions for African American females in the United States: A meta-analysis. *American Journal of Public Health, 99*, 2069–2078.

Fisher, J. D., & Fisher, W. A. (2000). Theoretical approaches to individual-level change in HIV risk behavior. In J. L. Peterson & R. J. DiClemente (Eds.), *Handbook of HIV prevention* (pp. 3–55). New York: Kluwer Academic/Plenum.

Gupta, G. R., Parkhurst, J. O., Ogden, J. A., Aggleton, P., & Mahal, A. (2008). Structural approaches to HIV prevention. *The Lancet, 372*, 764–775.

Holmes, K. K., Sparling, P. F., Stamm, W. E., Piot, P., Wasserheit, J. N., et al. (2008). Introduction and overview. In K. K. Holmes, P. F. Sparling, W. E. Stamm, P. Piot, J. N. Wasserheit, L. Corey, et al. (Eds.), *Sexually transmitted diseases* (4th ed., pp. xvii–xxv). New York: McGraw Hill.

Johnson, B. T., Carey, M. P., Chaudoir, S. R., & Reid, A. E. (2006). Sexual risk reduction for persons living

with HIV: Research synthesis of randomized controlled trials, 1993 to 2004. *Journal of Acquired Immune Deficiency Syndromes, 41*, 642–650.
Johnson, B. T., Redding, C. A., DiClemente, R. J., Mustanski, B. S., Dodge, B., Sheeran, P., et al. (2010). A network-individual-resource model for HIV prevention. *AIDS and Behavior, 14*, S204–S221.
Karney, B. R., Hops, H., Redding, C. A., Reis, H. T., Rothman, A. J., & Simpson, J. A. (2010). A framework for incorporating dyads in models of HIV-prevention. *AIDS and Behavior, 14*, S189–S203.

Sexually Transmitted Disease/ Infection (STD/STI)

▶ AIDS: Acquired Immunodeficiency Syndrome
▶ HIV Infection

Sexually Transmitted Diseases (STDs)

Theresa Senn
Center for Health and Behavior,
Syracuse University, Syracuse, NY, USA

Synonyms

Sexually transmitted infections; Venereal diseases

Definition

A sexually transmitted disease is a disease that is transmitted through sexual contact (World Health Organization).

Our knowledge of sexually transmitted diseases (STDs) is still evolving. Currently, at least 35 pathogens that can be transmitted sexually have been identified (Holmes et al., 2008). Although some sexually transmissible pathogens can be transmitted through other routes besides sexual contact, generally a disease is classified as an STD when the primary method of transmission in a population is sexual contact (Holmes et al.).

There are five different types of pathogens that can be transmitted sexually: (a) bacteria, such as gonorrhea or chlamydia; (b) viruses, such as human immunodeficiency virus (HIV), herpes simplex virus, or human papillomavirus (HPV); (c) protozoa, such as trichomoniasis; (d) fungi, such as *Candida albicans* (although the primary mode of transmission for this pathogen is not sexual); and (e) ectoparasites, such as pubic lice (Holmes et al., 2008). Depending on the pathogen, STDs can be transmitted through bodily fluids (e.g., blood, semen, cervicovaginal fluid), feces, or skin-to-skin contact. Thus, the transmission of an STD usually involves vaginal or anal intercourse, oral sex (including oral-genital contact and oral-anal contact), or genital-to-genital contact.

STD symptoms vary depending on the pathogen involved, as well as the sex of the infected person and the site of infection. Symptoms associated with some of the more common STDs include blisters or ulcers, pain or burning during urination, discharge, abdominal pain, and pain during intercourse. However, many individuals who are infected with an STD do not have any symptoms (Centers for Disease Control and Prevention, 2010d). Such "asymptomatic" individuals may unknowingly infect a sexual partner with the STD.

Testing for STDs is generally conducted in medical facilities (although community- and home-based testing protocols are now available). Depending on the STD, testing may involve urethral or cervical swabs, swabs taken from the site of an ulcer, urine testing, blood testing, and clinical examination (Centers for Disease Control and Prevention, 2010d). In the United States, some STDs must be reported (by health-care providers) to county, state, or federal health authorities; for example, positive test results for chlamydia, gonorrhea, and syphilis must be reported to the Centers for Disease Control and Prevention for disease surveillance and monitoring (Centers for Disease Control and Prevention, 2009).

STDs can have serious consequences, including epididymitis in men, pelvic inflammatory disease and pregnancy complications in women, infertility, and cancer (Centers for Disease Control and Prevention, 2010d). In addition, individuals who are co-infected with HIV and another STD are more likely to transmit HIV through sexual exposure, and individuals who are infected with an STD are more likely to acquire HIV through sexual exposure from an HIV-positive partner (Centers for Disease Control and Prevention, 2007). HIV weakens the immune system, ultimately leading to death when the immune system is so weakened it is unable to fight off infections and cancers (Centers for Disease Control and Prevention, 2010a). STDs can also have negative interpersonal and social consequences due to the stigma surrounding some STDs.

The majority of STDs are either bacterial or viral (Holmes et al., 2008). In general, bacterial STDs can be cured with antibiotics, although some STDs are becoming resistant to many classes of antibiotics that had previously successfully treated the disease (Centers for Disease Control and Prevention, 2010b). Viral STDs have no cure, although there are medications that can help to manage outbreaks or viral load. STDs such as HPV may sometimes be cleared from the body with no treatment (Centers for Disease Control and Prevention, 2010c).

An individual's likelihood of acquiring an STD is based on his or her sexual behavior and the risk of transmission per sexual act with an infected partner. Sexual behaviors that affect the likelihood of acquiring an STD include the number of sexual partners, the number of unprotected sexual acts, and the types of unprotected sexual acts. The risk of transmission per sexual act depends in part on the pathogen as well as the individual's biology. Different pathogens are associated with different risks of transmission per sexual act. Biological factors associated with transmission risk include the individual's immune response and the mucosal surface area exposed during the sexual act; women, for example, are more likely than men to be infected with an STD through vaginal intercourse because women have a larger mucosal surface area that is exposed during vaginal intercourse, and young women are more likely to acquire an STD because they have an immature cervix. STD risk is also affected by the STD prevalence in an individual's sexual network, which is influenced by the rate of sexual partner change, partner concurrency (i.e., multiple, overlapping sexual partnerships), and degree of disassortative mixing (i.e., sexual partners who are dissimilar in certain characteristics, such as age or sexual activity Garnett, 2008).

To date, some types of HPV and some types of hepatitis are the only STDs that can be prevented through vaccination. Other medical strategies are currently being developed for STD prevention, such as vaginal microbicides and preexposure prophylaxis. STDs can also be prevented through behavioral change. Abstaining from sexual contact is the only certain way to prevent most STDs. However, other behavioral strategies including correct and consistent condom use, engaging in sexual activity only with one partner who is not infected with any STD and who has no other sexual partners, and having fewer sexual partners will reduce the likelihood of acquiring an STD.

Behavioral medicine can play a large role in STD prevention. Behavioral interventions can promote sexual risk reduction behavior, by encouraging individuals to use condoms consistently and correctly for all sexual activity, be in a mutually monogamous sexual relationship with an uninfected partner, or adopt other strategies that will reduce the risk of contracting an STD. Behavioral interventions have been shown to be effective in reducing sexual risk behavior and STDs in a variety of populations (Crepaz, Horn et al., 2007; Crepaz, Marshall et al., 2009; Johnson, Carey, Chaudoir, & Reid, 2006). Behavioral medicine can also play a role in encouraging the adoption of biomedical strategies. For example, behavioral medicine strategies could be used to encourage STD and HIV testing, vaccine acceptance, the completion of medications for curable STDs and adherence to medications for viral STDs, and male circumcision, which may reduce the spread of STDs and HIV.

Cross-References

▶ Sexual Risk Behavior

References and Readings

Centers for Disease Control and Prevention. (2007). *CDC fact sheet: The role of STD prevention and treatment in HIV prevention*. Retrieved January 6, 2011 from http://cdc.gov/std/HIV/stds-and-hiv-fact-sheet.pdf

Centers for Disease Control and Prevention. (2009). *Sexually transmitted disease surveillance, 2008*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for HIV/AIDS, Viral Hepatitis, STD, and TB Prevention, Division of STD Prevention.

Centers for Disease Control and Prevention. (2010a). *Basic information about HIV and AIDS*. Retrieved January 6, 2011 from http://www.cdc.gov/hiv/topics/basic/index.html

Centers for Disease Control and Prevention. (2010b). *CDC fact sheet: Basic information about antibiotic-resistant gonorrhea (ARG)*. Retrieved January 6, 2011 from http://cdc.gov/std/Gonorrhea/arg/basic.htm

Centers for Disease Control and Prevention. (2010c). *CDC fact sheet: Genital HPV*. Retrieved January 6, 2011 from http://cdc.gov/std/hpv/hpv-fact-sheet-press.pdf

Centers for Disease Control and Prevention. (2010d). *CDC fact sheets: Sexually transmitted diseases*. Retrieved January 6, 2011 from http://cdc.gov/std/healthcomm/fact_sheets.htm

Crepaz, N., Horn, A. K., Rama, S. M., Griffin, T., Deluca, J. B., Mullins, M. M., et al. (2007). The efficacy of behavioral interventions in reducing HIV risk sex behaviors and incident sexually transmitted disease in Black and Hispanic sexually transmitted disease clinic patients in the United States: A meta-analytic review. *Sexually Transmitted Diseases, 34*, 319–332.

Crepaz, N., Marshall, K. J., Aupont, L. W., Jacobs, E. D., Mizuno, Y., Kay, L. S., et al. (2009). The efficacy of HIV/STI behavioral interventions for African American females in the United States: A meta-analysis. *American Journal of Public Health, 99*, 2069–2078.

Garnett, G. P. (2008). The transmission dynamics of sexually transmitted infections. In K. K. Holmes, P. F. Sparling, W. E. Stamm, P. Piot, J. N. Wasserheit, L. Corey, et al. (Eds.), *Sexually transmitted diseases* (4th ed., pp. 27–40). New York: McGraw Hill.

Holmes, K. K., Sparling, P. F., Stamm, W. E., Piot, P., Wasserheit, J. N., et al. (2008). Introduction and overview. In K. K. Holmes, P. F. Sparling, W. E. Stamm, P. Piot, J. N. Wasserheit, L. Corey, et al. (Eds.), *Sexually transmitted diseases* (4th ed., pp. xvii–xxv). New York: McGraw Hill.

Johnson, B. T., Carey, M. P., Chaudoir, S. R., & Reid, A. E. (2006). Sexual risk reduction for persons living with HIV: Research synthesis of randomized controlled trials, 1993 to 2004. *Journal of Acquired Immune Deficiency Syndromes, 41*, 642–650.

World Health Organization. *Health topics: Sexually transmitted infections*. Retrieved January 6, 2011 from http://www.who.int/topics/sexually_transmitted_infections/en/

Sexually Transmitted Infections

▶ Sexually Transmitted Diseases (STDs)

SF-36

Stephanie Ann Hooker
Department of Psychology, University of Colorado, Denver, CO, USA

Synonyms

Short form 36

Definition

The SF-36 is a 36-item self-report measure of health-related quality of life. It has eight subscales measuring different domains of health-related quality of life: physical functioning (PF), role-physical (RP), bodily pain (BP), general health (GH), vitality (VT), social functioning (SF), role-emotional (RE), and mental health (MH). Two component scores are derived from the eight subscales: a physical health component score and a mental health component score. The SF-36 also includes a single item that assesses perceived change in health status over the past year. Higher scores on all subscales represent better health and functioning. From its development to 2011, more than 16,000 articles were published using the SF-36. SF-36 is also known as the Short Form 36 Health Survey Questionnaire.

Description

Development

Ware and colleagues (Stewart & Ware, 1992; Ware, 1988, 1990) developed the SF-36 from the Medical Outcomes Study, a study of the health, well-being, and functioning of randomly selected patients seen by randomly selected physicians and other medical providers in three large metropolitan areas. Items were chosen for the SF-36 because they were items commonly used in other health surveys and the domains were ones that seemed to be commonly affected by differing health and disease states. After 10 years of use, the SF-36 was revised to address wording and response choice categories. The SF-36 version 2 (SF-36v2) is the current version (Ware et al., 2007).

Health Domain Scales

Physical Functioning (PF). The PF scale is a 10-item measure of physical limitation in a range of activities from vigorous exercise to performing self-care activities.

Role-Physical (RP). The RP scale contains four items and measures limitations in various roles, including work and daily activities.

Bodily Pain (BP). The BP scale has two items that measure body pain intensity and the extent to which pain interferes with daily activities.

General Health (GH). The five-item GH scale measures overall self-rated health.

Vitality (VT). The VT scale has four items that measure vitality, energy level, and fatigue and is meant to be a measure of subjective well-being.

Social Functioning (SF). The SF scale includes two items that measure the impact of physical and mental health on social functioning.

Role-Emotional (RE). The RE scale measures role limitations due to mental health difficulties with three items, including amount of time spent on work or other activities, amount of work accomplished, and the care with which work is performed.

Mental Health (MH). The MH scale has five items that measure anxiety, depression, loss of behavioral/emotional control, and psychological well-being.

Component Summary Scales

Component summaries were developed to reduce the number of scores derived from this measure from 8 to 2. They also have the advantages of having smaller confidence intervals than the health domain scales and limiting floor and ceiling effects. The Physical Component Summary (PCS) combines items from the PF, RP, BP, and GH scales, and the Mental Health Component Summary (MCS) combines items from the VT, SF, RE, and MH scales. Each provides one score to assess physical and mental health, respectively.

Reliability

Estimates for internal consistency reliability are very good for all subscales. The two component summary scores show evidence for very high internal consistency ($\alpha = .95$ and $\alpha = .93$ for the PCS and MCS, respectively). Internal consistency estimates for the health domain scales are also high, ranging from $\alpha = .83$ (GH) to $\alpha = .95$ (RP) (Ware et al., 2007). Evidence suggests that test-retest reliability for the SF-36 over a 3-week interval is very good, with estimates of .94 and .81 for the PCS and MCS scales, respectively (Ware, Kosinski, DeBrota, Andrejasich, & Bradt, 1995).

Validity

The SF-36 has been widely used in health research, and the user manual (Ware et al., 2007) provides a comprehensive list of studies offering evidence for the scales' construct validity. The content of the SF-36 survey was compared with other well-known health surveys to establish content validity (cf., Ware, Gandek, & the IQOLA Project Group, 1994, for a list of references).

Options

Along with the 36-item SF-36, the shorter 12-item (SF-12) and 8-item (SF-8) versions of the SF are also available. Both shorter versions offer scores on all eight health domains and the two component summary scores. Versions 1 and 2 of both the SF-36 and the SF-12 are available for use (there is only one version of the SF-8). All forms are available in the standard 4-week

recall and the acute 1-week recall versions. Additionally, the SF-8 is available in a 24-h recall version.

Administration

The survey is designed for adults 18 and over and can be given in a self-report paper/pencil format or in an interview format. The SF-36 and its other forms are available for licensure from QualityMetric Incorporated (www.qualitymetric.com).

Cross-References

► Health-Related Quality of Life

References and Readings

Quality Metric Incorporated. (2011). http://www.qualitymetric.com/

Stewart, A. L., & Ware, J. E., Jr. (Eds.). (1992). *Measuring functioning and well-being: The medical outcomes study approach*. Durham, NC: Duke University Press.

Ware, J. E., Jr. (1988). *How to score the revised MOS short-form health scales*. Boston: Institute for the Improvement of Medical Care and Health, New England Medical Center.

Ware, J. E., Jr. (1990). Measuring patient function and well-being: Some lessons from the medical outcomes study. In K. A. Heitgoff & K. N. Lohr (Eds.), *Effectiveness and outcomes in health care: Proceedings of an invitational conference by the Institute of Medicine Division of Health Care Services* (pp. 107–119). Washington, DC: National Academy Press.

Ware, J. E., Jr., Kosinski, M., DeBrota, D. J., Andrejasich, C. M., & Bradt, E. W. (1995, October). *Comparison of patient responses to SF-36 Health Surveys that are self-administered, interview administered by telephone, and computer-administered by telephone*. Paper presented at the Eastern Regional Meeting of the American Federation for Clinical Research, New York, NY.

Ware, J. E., Jr., Gandek, B., & The IQOLA Project Group. (1994). The SF-36 health survey: Development and use in mental health research and the IQOLA project. *International Journal of Mental Health, 23*, 49–73.

Ware, J. E., Jr., Kosinski, M., Bjorner, J. B., Turner-Bowker, D. M., Gandek, B., & Meruish, M. E. (2007). *User's manual for the SF-36v2 health survey* (2nd ed.). Lincoln, RI: Quality Metric Incorporated.

Short Form 36

► SF-36

Short Form 36 Health Survey Questionnaire (SF-36)

► SF-36

Sick Headache

► Migraine Headache

Sickness Behavior

Aric A. Prather
Center for Health and Community, University of California, San Francisco, CA, USA

Synonyms

Cytokine-induced depression; Inflammation-associated depression

Definition

Sickness behavior is a coordinated set of adaptive behavioral changes that occur in physically ill animals and humans during the course of infection. These behaviors include lethargy, depressed mood, reduced social exploration, loss of appetite, sleepiness, hyperalgesia, and, at times, confusion. This set of behaviors often accompanies fever and is considered a motivational state responsible for reorganizing an ill individual's perceptions and actions to enable better coping with infection (Dantzer, O'Connor, Freund, Johnson, & Kelley, 2008).

Description

Sickness behavior is a normal response to infection and is characterized by endocrine, autonomic, and behavioral changes triggered by soluble proteins produced at the site of infection. Activated immune cells, such as macrophages and dendritic cells, release biochemical mediators called pro-inflammatory cytokines, such as interleukin (IL)-1, IL-6, and tumor necrosis factor (TNF)-alpha, which coordinate the local and systemic inflammatory response during active infection. These inflammatory mediators, in turn, act on the brain facilitating behavioral changes associated with sickness.

Much of the evidence supporting a link between pro-inflammatory cytokines and sickness behaviors comes from experimental studies in animals and humans. Peripheral and central administration of IL-1 and TNF-alpha in healthy laboratory animals induces fever and behavioral symptoms of sickness, including depressed activity, decreased food intake, and a curled posture. Sexual behavior is similarly reduced, particularly among females. IL-1 receptor antagonist (IL-1RA) blocks the biological effects of IL-1 when co-administered at 100- to 1,000-fold excess dose with IL-1. Treatment with IL-1RA abrogates the depressing effect on social behavior but not food-motivated behavior, suggesting that IL-1 is a key mechanism in social function (Bluthe, Dantzer, & Kelley, 1992). Similar effects are seen when IL-1RA is injected directly into the brain. Time course studies of the behavioral effects of IL-1 in animals show changes in social exploration gradually develop within 2 h of peripheral administration whereas changes in food-motivated behavior reach a maximum by 1 h following treatment. Interestingly, IL-6 administered systemically or centrally has no behavioral effects despite inducing a fever response. That said, IL-6 does have the capacity to potentiate the effects of subthreshold dose of IL-1 administration suggesting that IL-6 may be behaviorally active only in the context of other pro-inflammatory mediators.

In humans, administration of endotoxin, a component of the outer membrane of Gram-negative bacteria, leads to systemic elevations in pro-inflammatory cytokines. This stimulus has been shown to cause participants to experience flu-like symptoms (e.g., fever, chills) as well as fatigue and depressed affect (reviewed in DellaGioia & Hannestad, 2010). Moreover, a recent study demonstrated that subjects exposed experimentally to endotoxin led to increased self-reported levels of depressed mood and reduced activity in the ventral striatum in response to reward cues (Eisenberger et al., 2010), which is consistent with anhedonia.

There is substantial overlap between the behavioral components of sickness behavior and major depression in humans. As such, pro-inflammatory cytokines are proposed to participate in the pathophysiology of depression and potentially account for the high prevalence of depression among the medically ill (Smith, 1991; Raison, Capuron, & Miller, 2006; Dantzer et al., 2008). Patients treated with immune-activating medications, such as IFN-a therapy prescribed for patients suffering with Hepatitis C or certain cancers, show elevated rates of depression compared to patients undergoing alternative therapies (Raison et al., 2006). Indeed, patients undergoing IFN-a therapy tend to experience depressive symptoms coupled with anxiety and irritability over a background of neurovegetative sickness–like symptoms, including sleep disorders, fatigue, and decreased appetite. While the mood disturbances generally occur between 4 and 12 weeks of treatment, the neurovegetative symptoms occur more rapidly, within the first 2 weeks of treatment.

Significant research efforts have focused on the neurochemical effects of inflammation that underlie sickness behavior and related depressive symptoms. Experimental animal data demonstrates that pro-inflammatory cytokines enhance indoleamine 2,3 dioxygenase (IDO) that peaks 24-h after endotoxin administration. This increase in IDO leads to a decrease in tryptophan (TRP), an essential amino acid that is actively transported into the brain for the synthesis of serotonin. IDO also leads to a decrease in kynurenine (KYN) and other tryptophan-related metabolites. Animals pretreated with a potent anti-inflammatory agent that blocks

pro-inflammatory cytokines in the periphery and in the brain show significant reductions in both sickness and depressive behaviors. In contrast, animals treated with an inhibitor of IDO show a reduction in depressive behaviors but not neurovegetative symptoms, providing important evidence for the role of tryptophan metabolism in cytokine-induced depression (Dantzer et al., 2008). It is anticipated that this research will have important implications for effective treatment of inflammation-related depression in humans.

Cross-References

- ▶ Depression: Symptoms
- ▶ Illness Behavior
- ▶ Inflammation
- ▶ Psychoneuroimmunology

References and Readings

Bluthe, R. M., Dantzer, R., & Kelley, K. W. (1992). Effects of interleukin-1 receptor antagonist on the behavioral effects of lipopolysaccharide in rat. *Brain Research, 573*, 318–320.

Dantzer, R., Bluthe, R. M., Castanon, N., Kelley, K. W., Konsman, J. P., Laye, S., et al. (2007). Cytokines, sickness behavior, and depression. In R. Ader (Ed.), *Psychoneuroimmunology* (4th ed., pp. 281–318). New York: Academic Press.

Dantzer, R., O'Connor, J. C., Freund, G. G., Johnson, R. W., & Kelley, K. W. (2008). From inflammation to sickness and depression: When the immune system subjugates the brain. *Nature Neuroscience Reviews, 9*, 46–56.

DellaGioia, N., & Hannestad, J. (2010). A critical review of human endotoxin administration as an experimental paradigm of depression. *Neuroscience and Biobehavioral Reviews, 34*, 130–143.

Eisenberger, N. I., Berkman, E. T., Inagaki, T. K., Rameson, L. T., Mashal, N. M., & Irwin, M. R. (2010). Inflammation-induced anhedonia: Endotoxin reduces ventral striatum responses to reward. *Biological Psychiatry, 15*, 748–754.

Miller, A. H., Maletic, V., & Raison, C. L. (2009). Inflammation and its discontents: The role of cytokines in the pathophysiology of major depression. *Biological Psychiatry, 65*, 732–741.

Raison, C. L., Capuron, L., & Miller, A. H. (2006). Cytokines sing the blues: Inflammation and pathogenesis of depression. *Trends in Immunology, 27*, 24–31.

Smith, R. S. (1991). The macrophage theory of depression. *Medical Hypotheses, 35*, 298–306.

Siegrist, Johannes

Johannes Siegrist
Department of Medical Sociology, University of Duesseldorf, Düsseldorf, Germany

Biographical Information

Johannes Siegrist was born in Zofingen, Switzerland, on August 6, 1943. His nationality is Swiss, and he is married to Karin and has two daughters. Siegrist studied Sociology, Social Psychology, Philosophy, and History at the Universities of Basel (Switzerland) and Freiburg i.Br. (Germany). He received his M.A. (1967) and his Ph.D. (1969) in Sociology at the University of Freiburg. After postdoctoral training at the Universities of Ulm and Freiburg, he accomplished his habilitation in Sociology at the University of Freiburg (1973). In 1973, he was appointed as Professor of Medical Sociology at the Faculty of Medicine, University of Marburg (Germany), where he served until 1992, interrupted by Visiting Professorships at the Institute of Advanced Studies in Vienna (Austria) and at the Johns Hopkins University School of Public Health in Baltimore, USA. In 1992, Siegrist was appointed as Professor of Medical Sociology and Director of the Department of Medical Sociology at the Faculty of Medicine, Heinrich Heine-University of Duesseldorf, Germany, and as Director of the Postgraduate

Training Program of Public Health at the same university. While officially retired since 2011, he continues his research activities at Duesseldorf University and as a Visiting Professor at the University of Bern (Switzerland).

Siegrist has been President of the International Society of Behavioral Medicine (ISBM; 1996–1998), President of the European Society of Health and Medical Sociology (1990–1992), and Director of the European Science Foundation Program on Social Variations in Health Expectancy in Europe (1999–2003). He is Chair of the Section "Behavioral Sciences" of Academia Europaea (since 2004), member of the Expert Panel of the German Research Foundation (since 2006), and member of the Scientific Committee on Demographic Change of the German Academy of Sciences Leopoldina (since 2011).

He has been a Task Group Leader to the Marmot Review (Strategic Review of Health Inequalities in England post-2010) for the British Government, with a focus on work and health. In this same function, he has coordinated and edited a Report on Employment and Working Conditions in the context of the "Review of Social Determinants of Health and the Health Divide in the WHO European Region," commissioned by the WHO European Office in 2011.

Siegrist served and continues to serve as Associate Editor of several international journals, in particular International Journal of Behavioral Medicine, *Social Science & Medicine*, *Social Psychiatry and Psychiatric Epidemiology, Work & Stress, European Journal of Public Health*, and *Scandinavian Journal of Work, Environment and Health*. The awards he received include Honorary Member of the European Society of Health and Medical Sociology where he also received the Research Award, Member of Academia Europaea (London), and Corresponding Member of the Heidelberg Academy of Sciences. He received the Salomon Neumann Award of the German Society of Social Medicine and Prevention, the Hans Roemer Award of the German College of Psychosomatic Medicine, and the Belle van Zuylen Chair at the University of Utrecht, the Netherlands.

Major Accomplishments

Siegrist's major contribution to scientific research concerns the development and test of a theoretical model of an adverse psychosocial work environment with the aim of explaining stress-related disorders, termed "effort-reward imbalance" (ERI). The model posits that failed reciprocity of effort spent and rewards received at work ("high cost-low gain") elicits strong negative emotions and psychobiological stress responses with adverse long-term effects on health. Starting from cross-sectional and longitudinal epidemiological research in the late 1970s and early 1980s, together with collaborators Ingbert Weber, Karin Siegrist, Richard Peter, and others at Marburg University, he systematically elaborated and expanded research on the ERI-model in a network of national and international scientific collaboration. This model has been incorporated in many epidemiological studies, most importantly the British Whitehall II-Study and the French GAZEL Study. More recently, the model was successfully applied in other sociocultural contexts (e.g., Japan) and in rapidly developing societies (e.g., China, Brazil).

Evidence from prospective cohort studies indicates that continued experience of failed reciprocity in terms of the ERI model is associated with an almost twofold elevated risk of coronary heart disease as well as depressive disorder. Single studies additionally observed elevated risks of alcohol dependence, type 2 diabetes, reduced health functioning, sickness absence, and disability. This epidemiological evidence was supplemented by experiments and "naturalistic" studies (e.g., ambulatory blood pressure and heart rate monitoring), where reduced immune function, enhanced autonomic activity, and altered release patterns of stress hormones were linked to ERI, often in a dose-response relationship. Siegrist was also involved in some intervention studies where measures of reducing ERI at work were followed by improvements of well-being and mental health.

Siegrist applied the ERI model to other types of contractual social exchange, e.g., voluntary

work, marital, or parent-child relations. Available results support the notion that failed reciprocity in core social roles exerts negative effects on health and well-being, suggesting a basic link between perceived injustice of effortful exchange and the development of stress-related disorders in humans. Siegrist has expanded this research with a focus on retirement, volunteering, and healthy aging, together with Morten Wahrendorf and other colleagues, in the frame of the Survey of Health, Ageing and Retirement in Europe (SHARE) and additional longitudinal investigations on ageing populations. As a cross-cutting topic of his long-lasting research career, Siegrist has put special emphasis on explaining and reducing avoidable social inequalities in health, both as a scientist and as an advocate for different stakeholders where he has proposed evidence-based policy recommendations for improving quality of work and employment and for reducing the burden of disease.

Cross-References

- ▶ International Society of Behavioral Medicine
- ▶ Occupational Health

References and Readings

Bosma, H., Peter, R., Siegrist, J., & Marmot, M. (1998). Two alternative job stress models and the risk of coronary heart disease. *American Journal of Public Health, 88*, 68–74.

Siegrist, J., & Marmot, M. (Eds.). (2006). *Social inequalities in health: New evidence and policy implications*. Oxford: Oxford University Press.

Siegrist, J., Siegrist, K., & Weber, I. (1986). Sociological concepts in the etiology of chronic disease: The case of ischemic heart ischemic disease. *Social Science & Medicine, 22*, 247–253.

Siegrist, J., Starke, D., Chandola, T., Godin, I., Marmot, M., Niedhammer, I., & Peter, R. (2004). The measurement of effort-reward imbalance at Work. European comparisons. *Social Science & Medicine, 58*, 1483–1499.

Siegrist, J., & Wahrendorf, M. (2009). Quality of work, health, and retirement. *Lancet, 374*, 1872–1873.

Single Nucleotide Polymorphism (SNP)

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

SNP (pronounced "snip")

Definition

The term "single nucleotide polymorphism" contains two defining criteria. First, it refers to a single nucleotide, i.e., an individual base pair, that can differ between individuals. Second, the word polymorphism indicates that a particular nucleotide change of interest is shared by at least 1% of the population.

SNPs occur when one base pair replaces another base pair in a point mutation (see ▶ DNA entry for discussion of bases). For example, an A-T pairing may be replaced by a G-C pairing. Such a mutation does not typically harm the organism.

Cross-References

- ▶ DNA
- ▶ Polymorphism

References and Readings

Britannica. (2009). *The Britannica guide to genetics* (Introduction by Steve Jones). Philadelphia: Running Press.

Single Subject

▶ Outcome for the Single Case: Random Control Index, Single Subject Experimental Design, and Goal Attainment Scale

Single-Case Experimental, or N of 1 Clinical Trials

▶ Outcome for the Single Case: Random Control Index, Single Subject Experimental Design, and Goal Attainment Scale

Situational Responsiveness

▶ Job Performance

Skeletal Muscle Atrophy

▶ Sarcopenia

Skin Cancer Prevention: Sun Protection, Sun Safety, Sunscreen Use

Karen Glanz
Schools of Medicine and Nursing, University of Pennsylvania, Philadelphia, PA, USA

Definition

Skin cancer is the most commonly diagnosed cancer in the United States, with more than one million Americans diagnosed with skin cancer each year. The incidence of skin cancer has increased dramatically worldwide in the last decade. Both main types of skin cancer – malignant melanoma and non-melanoma skin cancer (NMSC) – are now significant public health concerns. While skin cancer rates are increasing, it is considered one of the most preventable types of cancer.

The greatest risk factor for skin cancer is exposure to ultraviolet radiation, or UV radiation, which comes mainly from the sun. Behavioral recommendations for primary prevention of skin cancer include: limit time spent in the sun, avoid the sun during peak hours (10 a.m. to 4 p.m.), use sunscreen with a sun protection factor (SPF) of 15 or higher when outside, wear protective clothing (hats, shirts, pants) and sunglasses, seek shade when outdoors, and avoid sunburn. These behaviors, if consistently practiced, can help prevent all forms of skin cancer. There is some concern that using sunscreen will lead people who are trying to get a suntan to stay in the sun for a longer time, so another recommendation for prevention is not to intentionally bake in the sun or seek a tan.

Additional, important recommendations for behaviors to prevent skin cancer and related morbidity and mortality include performing regular skin self-examination and seeking professional evaluation of suspicious skin changes. Further, avoidance of indoor tanning and the use of tanning salons and tanning beds (also called "solaria") are strongly recommended.

An understanding of patterns of behavior can help to guide efforts to prevent skin cancer. More people take precautions at the beach or on vacation than when taking outdoor recreation. Parents are more likely to protect their children than themselves. Children are more often protected from UV radiation if their parents also protect themselves. Adolescents seem especially resistant to advice about skin cancer prevention and minimizing sun exposure.

Most skin cancer prevention interventions reported in the literature are directed at the general population through school-based curricula, multicomponent community programs, or media campaigns, and some recent trials have targeted people with high sun exposure at work or during outdoor recreation. Children, adolescents, and adults at high risk are important audiences for skin cancer prevention.

This chapter provides an overview of skin cancer prevention for the general population and groups at increased risk due to genetic or environmental exposures. The reference sources include evidence reviews, key research articles reporting on well-designed studies, and works addressing issues in measurement and methodology for skin cancer prevention research and evaluation.

Description

Evidence Reviews

An extensive evidence review of strategies to prevent skin cancer was undertaken by the Task Force on Community Preventive Services, and the results and recommendations were published in Saraiya et al. (2004). This report presents the results of systematic reviews of the effectiveness of interventions to prevent skin cancer by reducing exposure to ultraviolet radiation (UVR). The Task Force on Community Preventive Services found that education and policy approaches were effective when implemented in primary schools and in recreational or tourism settings but found insufficient evidence to determine effectiveness in other settings. This evidence review is currently being updated to reflect the continuing growth of the scientific literature on behavioral interventions to prevent skin cancer during the past decade.

Comprehensive Community Programs Including Mass Media

There is a long history of comprehensive, multicomponent community skin cancer prevention programs, especially in Australia, where skin cancer is highly prevalent. These programs include mass media and communication campaigns as an integral part of these community programs.

Two related sun protection programs have been conducted in Australia for more than 20 years: Slip! Slop! Slap! from 1980 to 1988 and SunSmart from 1988 to the present (Montague, Borland, & Sinclair, 2001). These programs have played an important role in changing the whole society's approach to the sun and have resulted in marked reductions in sun exposure. An examination of trends in behavioral risk factors for skin cancer over 15 years was examined in an Australian population exposed to the SunSmart program including SunSmart television advertising. Higher exposure to SunSmart advertising in the weeks before the interview increased preferences for not tanning, hat and sunscreen use, and greater clothing protection. These results indicate that sustained multicomponent programs with media campaigns can both prompt and reinforce skin cancer preventive behaviors.

Interventions in Schools

The most often studied settings for skin cancer prevention programs are schools, and there is good evidence that educational and policy interventions can be effective in primary schools (Saraiya et al., 2004). Of the many reported studies, a few are particularly well designed, carefully described, have long follow-up periods, and/or use objective outcome measures. The Kidskin intervention trial in Western Australia is particularly noteworthy and had a 6-year follow-up period. The "Kidskin" study involved three groups: control, "moderate," and "high" intervention. Results showed that children in the intervention groups – especially the "high" group – reported less sun exposure and spent less time outdoors in the middle of the day. There was little difference between groups in the wearing of hats or sunscreen (Milne et al., 2001). Children in the intervention groups – especially the high group – were less tanned at the end of the summer; this effect was greater for the back than for the forearms. There was also a smaller increase in the number of nevi (or moles) on the backs of children in the intervention groups (English et al., 2005). Further, the program had a positive effect on hat wearing on the playground, especially in the "high" intervention groups, but did not change children's use of shade at lunchtime (Giles-Corti et al., 2004).

Outdoor Workers

Outdoor workers are at high risk for skin cancer because they receive regular and significant solar UVR exposure. In a well-designed study to reduce UVR exposure among ski instructors, greater program implementation was associated with less sunburn. In an intervention for US Postal Service workers, regular sunscreen and hat use were higher among the intervention group than among the control group after 3 months and at 3-year follow-up (Mayer et al., 2007, 2009).

Recreation Settings

Intense and prolonged sun exposure often occurs during outdoor recreation activities. High UVR exposure, often with minimal clothing, tends to occur at beach and swimming pool settings. Other outdoor recreation settings include camps,

zoos, and parks. Large and well-designed studies of skin cancer prevention in these locations have been reported. Effective skin cancer prevention programs for children have been evaluated in swimming pool settings (Glanz, Geller, Shigaki, Maddock, & Isnec, 2002; Glanz, Steffen, Elliott, & O'Riordan, 2005) and for beachgoers at Northeastern (Weinstock, Rossi, Redding, & Maddock, 2002) and Midwestern (Pagoto, McChargue, & Fuqua, 2003) beaches as well as at zoos (Mayer et al., 2001).

High-Risk Groups

Targeting skin cancer prevention to people at high risk may result in greater effects of preventive strategies and an efficient public health strategy. Risk factors for skin cancer include age, sun-sensitive phenotypes, excess sun exposure, family history, personal history of skin cancer or precancerous lesions, and some other medical conditions. There is a need to develop low-cost, effective interventions to improve skin cancer prevention and early detection behaviors among a broader population of persons at moderate and high risk. (Geller, Emmons, Brooks, Powers, Zhang, Koh, Heeren, Sober, Li, & Gilchrest, 2006) and Glanz, Schoenfeld, and Steffen (2010) describe studies of effective tailored interventions that specifically target individuals at high risk, either siblings of melanoma patients or adults determined to be at moderate or high risk for skin cancer. These studies focused on both prevention and skin examinations. A study of a group of people who have tested positive in genetic testing for skin cancer-related mutations and found that positive genetic test results led to greater intentions to obtain total body skin examinations and adhere to skin self-examination recommendations (Aspinwall, Leaf, Dola, Kohlmann, & Leachman, 2008).

Screening and Early Detection

Screening for skin cancer through health-care-provider skin exams and skin self-examination has the potential to help detect skin cancers at an earlier stage (i.e., when they are thinner) so that they are more curable and less serious. Although there has not been a large randomized trial of skin screening in the United States, an Australian trial reported by Aitken et al. (2006) provides promising evidence of the impact of skin screening and how it can be successfully implemented. A randomized trial was conducted to determine whether a multicomponent intervention can increase total skin self-examination (TSSE) performance. Participants received instructional materials, a video, and a brief counseling session and a brief follow-up phone call and tailored feedback letters. Results showed that the intervention group increased TSSE performance in the intervention group compared to the control group (Weinstock et al., 2007). A follow-up article aimed to identify the most important Check-It-Out intervention components for promoting TSSE. Results showed that watching the video, using the hand mirror, shower card, American Cancer Society brochure, sample photographs, and finding the health educator helpful were associated with performing TSSE at 2 months, 12 months, or both. The studies of high-risk groups reported by Geller et al. (2006) and Glanz et al. (2010) also targeted behavioral outcomes of skin self-examination and thorough examination of all moles.

Measurement and Methodology

Advances in skin cancer prevention depend on good quality research and ideally different intervention studies that can be compared to understand the impact of various strategies. Most skin cancer prevention studies uses verbal reports, or self-report, to measure habitual sun exposure and solar protection behaviors. Despite the well-known limitations of verbal reports of behavior, these measures are the most practical for use in both population surveillance and descriptive and intervention research (Glanz & Mayer, 2005). Therefore, the comparability of assessments across population-based surveys and outcome measures used in intervention research is important, and a core set of measures was recently published by a diverse group of investigators in the field (Glanz et al., 2008). In addition, because it is important to continue to build a research tool kit for measures other than surveys, including

objective biological measures and observational measures, recent research to validate self-reports of sunscreen use (Glanz et al., 2009) and other behaviors is of particular importance to the field.

Conclusion

Skin cancer prevention interventions have demonstrated modest success, with the majority of programs being conducted in school settings. It is believed that the ideal intervention strategies for reducing exposure to ultraviolet radiation (UVR) exposure are coordinated, sustained, community-wide approaches that combine education, mass media, and environmental and structural changes. Interventions within specific organizational settings such as schools, health care, recreation programs, and workplaces provide useful ways to reach important audiences like children and are suitable venues for structural supports such as environmental and policy change that complement educational efforts. It is generally agreed that environmental and structural changes also need to be part of successful skin cancer prevention efforts. Advances in measurement and methods in skin cancer prevention research will contribute to future efforts to address this important and widespread health and behavior problem.

Cross-References

► Cancer Prevention

References and Readings

Aitken, J. F., Janda, M., Elwood, M., Youl, P. H., Ring, I. T., & Lowe, J. B. (2006). Clinical outcomes from skin screening clinics within a community-based melanoma screening program. *Journal of the American Academy of Dermatology, 54*, 105–114.

Aspinwall, L. G., Leaf, S. L., Dola, E. R., Kohlmann, W., & Leachman, S. A. (2008). CDKN2A/p16 genetic test reporting improves early detection intentions and practices in high-risk melanoma families. *Cancer Epidemiol Biomarkers Prev., 17*,1510–1519.

English, D. R., Milne, E., Jacoby, P., Giles-Corti, B., Cross, D., & Johnston, R. (2005). The effect of a school-based sun protection intervention on the development of melanocytic nevi in children: 6-year follow-up. *Cancer Epidem Biomarkers Prevention, 14*, 977–980.

Geller, A. C., Emmons, K. M., Brooks, D. R., Powers, C., Zhang, Z., Koh, H. K., Heeren, T., Sober, A. J., Li, F., & Gilchrest, B. A. (2006). A randomized trial to improve early detection and prevention practices among siblings of melanoma patients. *Cancer, 107*, 806–814.

Giles-Corti, B., English, D., Costa, C., Milne, E., Cross, D., & Johnston, R. (2004). Creating SunSmart schools. *Health Education Research, 19*, 98–109.

Glanz, K., Geller, A. C., Shigaki, D., Maddock, J., & Isnec, M. R. (2002). A randomized trial of skin cancer prevention in aquatic settings: The Pool Cool program. *Health Psychology, 21*, 579–587.

Glanz, K., & Mayer, J. A. (2005). Reducing UVR exposure to prevent skin cancer: Methodology and measurement. *American Journal of Preventive Medicine, 29*, 131–142.

Glanz, K., McCarty, F., Nehl, E. J., O'Riordan, D. L., Gies, P., Bundy, L., et al. (2009). Validity of self-reported sunscreen use by parents, children and lifeguards. *American Journal of Preventive Medicine, 36*, 63–69.

Glanz, K., Schoenfeld, E. R., & Steffen, A. (2010). Randomized trial of tailored skin cancer prevention messages for adults: Project SCAPE. *American Journal of Public Health, 100*, 735–741.

Glanz, K., Steffen, A., Elliott, T., & O'Riordan, D. (2005). Diffusion of an effective skin cancer prevention program: Design, theoretical foundations, and first-year implementation. *Health Psychology, 24*, 477–487.

Glanz, K., Yaroch, A. L., Dancel, M., Saraiya, M., Crane, L. A., Buller, D. B., et al. (2008). Measures of sun exposure and sun protection practices for behavioral and epidemiologic research. *Archives of Dermatology, 144*, 217–222.

Mayer, J. A., Lewis, E. C., Eckhardt, L., Slymen, D., Belch, G., Elder, J., et al. (2001). Promoting sun safety among zoo visitors. *Preventive Medicine, 33*, 162–169.

Mayer, J. A., Slymen, D. J., Clapp, E. J., Pichon, L. C., Eckhardt, L., Eichenfield, L. F., et al. (2007). Promoting sun safety among US postal service letter carriers: Impact of a 2-year intervention. *American Journal of Public Health, 97*, 559–565.

Mayer, J. A., Slymen, D. J., Clapp, E. J., Pichon, L. C., Elder, J. P., Sallis, J. F., et al. (2009). Long-term maintenance of a successful occupational sun safety intervention. *Arch Dermatol, 145*, 88–89.

Milne, E., English, D. R., Johnston, R., Cross, D., Borland, R., Giles-Corti, B., et al. (2001). Reduced sun exposure and tanning in children after 2 years of a school-based intervention (Australia). *Cancer Causes and Control, 12*, 387–393.

Montague, M., Borland, R., & Sinclair, C. (2001). Slip! Slop! Slap! and SunSmart, 1980-2000: Skin cancer control and 20 years of population-based campaigning. *Health Education & Behavior, 28*, 290–305.

Pagoto, S., McChargue, D., & Fuqua, R. (2003). Effects of a multicomponent intervention on motivation and sun protection behaviors among midwestern beachgoers. *Health Psychology, 22*, 429–433.

Saraiya, M., Glanz, K., Briss, P. A., Nichols, P., White, C., Das, D., et al. (2004). Interventions to prevent skin cancer by reducing exposure to ultraviolet radiation – a systematic review. *American Journal of Preventive Medicine, 27*, 422–466.

Weinstock, M. A., Risica, P. M., Martin, R. A., Rakowski, W., Dubé, C., Berwick, M., et al. (2007). Melanoma early detection with thorough skin self-examination: The "check it out" randomized trial. *American Journal of Preventive Medicine, 32*, 517–524.

Weinstock, M. A., Rossi, J. S., Redding, C. A., & Maddock, J. E. (2002). Randomized controlled community trial of the efficacy of a multicomponent stage-matched intervention to increase sun protection among beachgoers. *Preventive Medicine, 35*, 584–592.

SLC6A4 (Solute Carrier Family 6, Member 4)

► Serotonin Transporter Gene

Sleep

Martica H. Hall
Department of Psychiatry, University of Pittsburgh, Pittsburgh, PA, USA

Definition

Sleep is a complex reversible neurobiological state characterized by closed eyes, behavioral quiescence, and perceptual disengagement from one's surroundings.

Description

Healthy adults cycle between two types of sleep during the typical nocturnal sleep period: non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep. When healthy adults fall asleep, they enter NREM sleep and usually move from lighter stages of sleep (e.g., Stages N1 and N2) to deeper sleep (e.g., Stage N3) before entering their first REM sleep period. The terms "light" and "deep" sleep refer to the ease with which one can be awakened from sleep and become fully oriented to one's surroundings. The descent from light into deep NREM sleep is characterized by decreasing inputs from external stimuli, a slowing of catabolic processes, and an increase in parasympathetic nervous system activity. In contrast, REM sleep is characterized by autonomic instability and active mental activity. In healthy adults, individual NREM-REM cycles generally last approximately 90 min, although the duration of sleep cycles varies across individuals. During the first third of the night, NREM sleep is more prevalent, whereas REM sleep becomes more prevalent during the last third of the night.

Sleep can be characterized along multiple dimensions. Here we focus on four dimensions of sleep that have been most widely evaluated in relation to health and functioning; these include sleep *duration*, *continuity*, *architecture*, and *quality*. It is important to recognize that each of these dimensions of sleep changes across the life span, from infancy through old age and may, additionally, be moderated by sex, race/ethnicity, and mental and physical health conditions (Carrier, Land, Buysse, Kupfer, & Monk, 2001; Carskadon & Dement, 2005; Hall et al., 2009; Ohayon, Carskadon, Guilleminault, & Vitiello, 2004).

Sleep Duration

The two most commonly assessed indices of sleep duration include "time in bed" and "total sleep time." Operationally, time in bed (TIB) may be defined as total hours elapsed between getting into bed to go to sleep at night ("good night time") and waking up in the morning ("good morning time"). Total sleep time (TST) may be operationalized as time in bed minus the amount of time needed to fall asleep ("sleep latency") and amount of time spent awake during the night ("wakefulness after sleep onset").

Sleep duration is one of the most widely studied dimensions of sleep in relation to health and

functioning (see entry on "▶ Sleep Duration, ▶ Sleep and Health"). For the most part, this literature has documented robust associations among sleep duration extremes (generally, <6 h or >8 h) and indices of morbidity and mortality. One meta-analysis of 23 studies reported pooled relative risk (RR) values of 1.10 (95% CI = 1.06–1.15) and 1.23 (95% CI = 1.17–1.30) for all-cause mortality and short and long sleep duration, respectively (Gallicchio & Kalesan, 2009).

It must be noted, however, that studies using objective measures (actigraphy, PSG) of sleep duration and health outcomes are lacking. This issue is especially important given discrepancies between self-reported and objective indices of sleep duration, which may be confounded by other risk factors for morbidity and mortality such as age, sex, race, BMI, and comorbidities. Nor do measures of sleep duration differentiate between individuals with or without primary sleep disorders such as sleep apnea and insomnia, which have been widely linked to health and functioning (Boivin, 2000; Somers, 2005).

Sleep Continuity

Measures of sleep continuity focus on one's ability to initiate and maintain sleep (see "▶ Sleep Continuity, ▶ Sleep Fragmentation" entries). *Sleep latency* refers to the amount of time it takes to fall asleep (e.g., minutes from "good night time" to onset of sleep), whereas *wakefulness after sleep onset* (WASO) refers to the total amount of wakefulness during the sleep period (e.g., minutes of wakefulness between sleep onset and "good morning time"). *Sleep efficiency* is a proportional sleep continuity measure which refers to the percentage of time in bed spent asleep. Although operational definitions may differ across laboratories, sleep efficiency is commonly calculated as follows: (time spent asleep/time in bed) × 100.

Compared to sleep duration, fewer population-based studies have evaluated relationships among sleep continuity and indices of health and functioning. Several studies have linked self-reported sleep continuity disturbances with incident Type 2 diabetes and cardiovascular disease (as reviewed by Mezick et al. under review). Although few in number, other studies have reported significant cross-sectional associations between objectively assessed sleep continuity disturbances and health outcomes including obesity, increased blood pressure, increased inflammation, decreased circulating natural killer cell numbers, and the metabolic syndrome (Hall et al., 1998; Knutson et al., 2009; Mills et al., 2007). In their longitudinal study of sleep and all-cause mortality in healthy older adults, Dew and colleagues reported that participants with PSG-assessed sleep latencies of greater than 30 min were at 2.14 times greater risk of death (95% CI = 1.25–3.6) compared to those who fell asleep in less than 30 min, after adjusting for age, medical burden, and other relevant covariates (Dew et al., 2003).

Emerging evidence based on experimental models of sleep fragmentation suggests that endocrine, immune, metabolic, and autonomic mechanisms may be important pathways through which sleep continuity disturbances influence health and functioning (Bonnet & Arand 2003; Janackova & Sforza, 2008; Redwine Dang, Hall, & Irwin, 2003; Tartar et al., 2009). In terms of its relevance to behavioral medicine and health, sleep continuity appears to be exquisitely sensitive to psychological and social factors such as stress, loneliness, relationship quality, and socioeconomic status Akerstedt et al., 2002; Cacioppo et al., 2002; Cartwright & Wood, 1991; Friedman et al., 2005; Hall, Buysse, Nofzinger, Reynolds, & Monk, 2008).

Sleep Architecture

Sleep architecture refers to the pattern or distribution of visually scored NREM and REM sleep stages as well as quantitative measures derived from power spectral analysis of the EEG (see "▶ Sleep Architecture" entry). Within NREM sleep, measures of sleep architecture include stages N1-N3. Lighter stages of sleep are characterized by low-amplitude, fast-frequency EEG activity whereas deeper stages of sleep are characterized by high-amplitude, low-frequency EEG activity generated by rhythmic oscillations of thalamic and cortical neurons (see Jones, 2005).

Patients with medical disorders including cardiovascular and kidney disease, diabetes, and cancer exhibit lighter sleep architecture profiles compared to healthy individuals (e.g., Jauch-Chara, Schmid, Hallschmid, Born, & Schultes 2008; Ranjbaran, Keefer, Stepanski, Farhadi, & Keshavarzian, 2007). Yet, these studies do not indicate whether sleep architecture profiles were a contributing cause or consequence of disease. Both possibilities are plausible given experimental evidence of bidirectional relationships among components of sleep architecture and physiological processes important to health and functioning including metabolic, endocrine, autonomic, and immune mechanisms (e.g., Hall et al., 2004; Opp, 2006; Rasch, Dodt, Moelle, & Born, 2007). The longitudinal study of sleep and mortality by Dew and colleagues (2003) is the only published study, to date, that has evaluated relationships among measures of sleep architecture and indices of morbidity or mortality. In this study, risk for mortality was significantly higher in individuals with extreme amounts of REM sleep (upper and lower 15th percentile of the sample distribution); the visually scored slow-wave sleep percentage was also modestly associated with survival time.

Experimental manipulation of sleep architecture, although technically complex, may be an especially promising approach to disentangling cause and effect and evaluating cellular and molecular mechanisms through which sleep architecture affects and is affected by health. Quantitative analysis of the EEG, which shows trait-like characteristics, may hold promise for identifying sleep phenotypes that confer vulnerability to or resilience against disease (e.g., Tucker, Dinges, & Van Dongen, 2007). This latter point may be especially relevant to behavioral medicine models of disease given that decreased slow-wave sleep and increased EEG spectral power in the fast-frequency beta band have been linked with symptoms of stress and a variety of chronic stressors including job strain, marital dissolution, and bereavement (Cartwright & Wood, 1991; Hall et al., 1997; Kecklund & Akerstedt, 2004).

Sleep Quality

Sleep quality generally refers to subjective perceptions about one's sleep. The Pittsburgh Sleep Quality Index (PSQI), which is the most widely used self-report sleep instrument and has been translated into over 30 languages, is an example of a "multiple-indicator" measure of sleep quality (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The PSQI includes 18 retrospective questions about one's sleep over the past month. These questions are used to derive seven subscales (sleep duration, sleep latency, sleep efficiency, sleep disturbance, daytime dysfunction, use of medications for sleep, and overall sleep quality), each of which has a range of 0–3. These subscales may be summed to generate a global measure of subjective sleep quality with a range of 0–21; higher values reflect greater subjective sleep complaints.

In a community-based study of midlife adults without clinical cardiovascular disease, Jennings and colleagues reported that higher PSQI-assessed sleep quality complaints were associated with increased prevalence of the metabolic syndrome (Jennings, Muldoon, Hall, Buysse, & Manuck, 2007). Other cross-sectional studies have reported greater subjective sleep quality complaints in patients with hypertension, diabetes, kidney disease, polycystic ovary syndrome, and cancer compared to age- and sex-matched healthy controls (e.g., Liu et al., 2009; Sabbatini et al., 2008; Tasali, Van Cauter, & Ehrmann, 2006). Subjective sleep quality complaints may be a consequence of disease. It may also indirectly impact health via health behavior pathways. For instance, subjective perceptions that one's sleep is not sound or restorative may lead to increased daytime caffeine use and increased use of alcohol prior to sleep which, in turn, may negatively impact health and functioning.

Summary

Converging evidence suggests numerous links between specific dimensions of sleep and important indices of health and functioning. The two most prevalent sleep disorders, insomnia and sleep apnea, too have been prospectively linked to adverse health outcomes (see entries for

"► Insomnia, ► Sleep Apnea"). Yet, little is understood about *how* specific dimensions of sleep or sleep disorders may confer vulnerability or resilience to disease. Identification of the cellular and molecular pathways through which sleep affects and is affected by health is critical to advancing our understanding of the sleep-health relationship in the context of behavioral medicine.

Cross-References

- ► Coffee Drinking, Effects of Caffeine
- ► Sleep Architecture
- ► Sleep Continuity
- ► Sleep Duration
- ► Sleep Quality

References and Readings

Akerstedt, T., Knutsson, A., Westerholm, P., Theorell, T., Alfredsson, L., Kecklund, G., et al. (2002). Sleep disturbances, work stress and work hours: A cross-sectional study. *Journal of Psychosomatic Research*, *53*, 741–748.

Boivin, D. B. (2000). Influence of sleep-wake and circadian rhythm disturbances in psychiatric disorders. *Journal of Psychiatry & Neuroscience*, *25*, 446–458.

Bonnet, M. H., & Arand, D. L. (2003) Clinical effects of sleep fragmentation versus sleep deprivation. *Sleep Medicine Reviews*, *7*, 297–310.

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., Kupfer, D. J. (1989). The Pittsburgh sleep quality index: A new instrument for psychiatric practice and research. *Psychiatry Research*, *28*, 193–213.

Cacioppo, J. T., Hawkley, L. C., Berntson, G. G., Ernst, J. M., Gibbs, A. C., Stickgold. R., et al. (2002). Do lonely days invade the nights? Potential social modulation of sleep efficiency. *Psychological Science*, *13*, 384–387.

Carrier. J., Land, S., Buysse, D. J., Kupfer, D. J., Monk, T. H. (2001). The effects of age and gender on sleep EEG power spectral density in the middle years of life (aged 20–60 years old). *Psychophysiology*, *38*, 232–242.

Carskadon, M. A., Dement, W. C. (2005). Normal human sleep: an overview. In: M. H. Kryger, T. Roth, T. Dement (eds) Principles and practice of sleep medicine. Elsevier/Saunders, Philadelphia, PA, pp 13–23.

Cartwright, R. D., Wood, E. (1991). Adjustment disorders of sleep: The sleep effects of a major stressful event and its resolution. *Psychiatry Research*, *39*, 199–209.

Dew, M. A., Hoch, C. C., Buysse, D. J., Monk, T. H., Begley, A. E., Houck, P. R., et al. (2003). Healthy older adults' sleep predicts all-cause mortality at 4 to 19 years of follow-up. *Psychosomatic Medicine*, *65*, 63–73.

Friedman, E. M., Hayney, M. S., Love, G. D., Urry, H. L., Rosenkranz, M. A., Davidson, R. J., et al. (2005). Social relationships, sleep quality, and interleukin-6 in aging women. *Proceedings of the National Academy of Sciences U S A*, *102*,18757–18762.

Gallicchio, L., Kalesan, B. (2009). Sleep duration and mortality: A systematic review and meta-analysis. *Journal of Sleep Research*, *18*, 148–158.

Hall, M., Baum, A., Buysse, D. J., Prigerson, H. G., Kupfer, D. J., Reynolds, C. F., et al. (1998). Sleep as a mediator of the stress-immune relationship. *Psychosomatic Medicine*, *60*, 48–51.

Hall, M., Buysse, D. J., Dew, M. A., Prigerson, H. G., Kupfer, D. J., & Reynolds, C. F. (1997). Intrusive thoughts and avoidance behaviors are associated with sleep disturbances in bereavement-related depression. *Depression and Anxiety*, *6*, 106–112.

Hall, M., Buysse, D. J., Nofzinger, E. A., Reynolds, C. F., Monk, T. H. (2008). Financial strain is a significant correlate of sleep continuity disturbances in late-life. *Biological Psychology*, *77*, 217–222.

Hall, M., Matthews, K. A., Kravitz, H. K., Gold, E. B., Buysse, D. J., Bromberger, J. T., et al. (2009). Race and financial strain are independent correlates of sleep in mid-life women: The SWAN sleep study. *Sleep*, *32*, 73–82.

Hall, M., Vasko, R., Buysse, D. J., Ombao, H., Chen, Q., Cashmere, J. D., et al. (2004). Acute stress affects heart rate variability during sleep. *Psychosomatic Medicine*, *66*, 56–62.

Janackova, S., & Sforza, E. (2008). Neurobiology of sleep fragmentation: Cortical and autonomic markers of sleep disorders. *Current Pharmaceutical Design*, *14*, 3474–3480.

Jauch-Chara K., Schmid S. M., Hallschmid, M., Born, J., Schultes, B. (2008). Altered neuroendocrine sleep architecture in patients with type 1 diabetes. *Diabetes Care*, *31*, 1183–1188.

Jennings, J. R., Muldoon, M., Hall, M., Buysse, D. J., Manuck, S. B. (2007). Self-reported sleep quality is associated with the metabolic syndrome. *Sleep*, *30*, 219–223.

Jones, B. E. (2005). Basic mechanisms of sleep-wake states. In: M. H. Kryger, T. Roth, W. C. Dement (eds) Principles and practice of sleep medicine. Elsevier/Saunders, Philadelphia, PA, pp 136–153.

Kecklund, G., Akerstedt, T. (2004). Apprehension of the subsequent working day is associated with a low amount of slow wave sleep. *Biological Psychology*, *66*, 169–176.

Knutson, K. L., Van Cauter, E., Rathouz, P. J., Yan, L. L., Hulley, S. B., Liu, K., et al. (2009). Association between sleep and blood pressure in midlife: The CARDIA sleep study. *Archives of Internal Medicine 169*, 1055–1061.

Liu, L., Fiorentino, L., Natarajan, L., Parker, B. A., Mills, P. J., Sadler, G. R., et al. (2009). Pre-treatment symptom cluster in breast cancer patients is associated

with worse sleep, fatigue and depression during chemotherapy. *Psycho-Oncology*, *18*, 187–194.
Mills, P. J., von Kanel, R., Norman, D., Natarajan, L., Ziegler, M. G., Dimsdale, J. E., et al. (2007). Inflammation and sleep in healthy individuals. *Sleep*, *30*, 729–735.
Ohayon, M. M., Carskadon, M.A., Guilleminault, C., Vitiello, M. V. (2004). Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: Developing normative sleep values across the human lifespan. *Sleep*, *27*, 1255–1273.
Opp, M. R. (2006). Sleep and psychoneuroimmunology. *Neurologic Clinics*, *24*, 493–506.
Ranjbaran, Z., Keefer, L., Stepanski, E., Farhadi, A., Keshavarzian, A. (2007). The relevance of sleep abnormalities to chronic inflammatory conditions. *Inflammation Research*, *56*, 1–7.
Rasch, B., Dodt, C., Moelle, M., Born, J. (2007). Sleep-stage-specific regulation of plasma catecholamine concentration. *Psychoneuroendocrinology*, *32*, 884–891.
Redwine, L., Dang, J., Hall, M., Irwin. M. (2003). Disordered sleep, nocturnal cytokines, and immunity in alcoholics. *Psychosomatic Medicine*, *65*, 75–85.
Sabbatini, M., Pisani, A., Crispo, A., Nappi, R., Gallo, R., Cianciaruso, B., et al. (2008). Renal transplantation and sleep: A new life is not enough. *Journal of Nephrology*, *21*(Suppl 13), S97–S101.
Somers, V. K. (2005). Sleep: A new cardiovascular frontier. *The New England Journal of Medicine*, *353*, 2070–2073.
Tartar, J. L., Ward, C. P., Cordeira, J. W., Legare, S. L., Blanchette, A. J., McCarley, R. W., & Strecker, R. E. (2009). Experimental sleep fragmentation and sleep deprivation in rats increases exploration in an open field test of anxiety while increasing plasma corticosterone levels. *Behavioural Brain Research*, *197*, 450–453.
Tasali, E., Van Cauter, E., Ehrmann, D. A. (2006). Relationships between sleep disordered breathing and glucose metabolism in polycystic ovary syndrome. *The Journal of Clinical Endocrinology and Metabolism*, *91*, 36–42.
Tucker, A. M., Dinges. D. F., Van Dongen, H. P. (2007). Trait interindividual differences in the sleep physiology of healthy young adults. *Journal of Sleep Research*, *16*, 170–180.

Sleep and Health

Faith S. Luyster
School of Nursing, University of Pittsburgh, Pittsburgh, PA, USA

Synonyms

Sleep deprivation

Definition

Sleep is defined as a reversible state of perceptual disengagement and unresponsiveness to the external environment. Sleep is a complex physiological and behavioral process that is part of every individual's life and a critical determinant of physical and mental health.

Description

It is generally accepted that 7–8 h is the optimal amount of sleep needed per night for adequate daytime functioning and to reduce the risk of developing serious medical conditions. However, many Americans sleep less than 7 h per night and many report sleep difficulties. The percentage of men and women reporting sleeping less than 6 h per night has increased significantly over the last 20 years. Broad societal changes, including longer work hours, shift work, later night life, increased dependence on technology, and a current mindset of "if you snooze, you lose" have contributed to the increases in sleep loss among adults. Sleep loss increases the risk and incidence of diseases that may ultimately result in death. Many of the studies examining sleep duration and adverse health outcomes have found a U-shaped relationship suggesting that too little sleep and too much sleep is detrimental to health. And, between 7 and 8 h of sleep appears to be associated with reduced health risk.

Sleep and Mortality

Growing evidence over the last few decades suggests that progressively shorter (<7 h per night) or longer (>8 h per night) sleep duration is associated with a greater risk of mortality (Cappuccio, D'Elia, Strazzullo, & Miller, 2010; Kripke, Garfinkel, Wingard, Klauber, & Marler, 2002). The mechanisms that underlie these associations are not fully understood. Potential adverse physiologic effects of short sleep duration may contribute to negative health outcomes like cardiovascular disease, diabetes, and obesity, all of which are associated with increased mortality risk. Sleep restriction has been shown to impair

glucose tolerance, increase evening cortisol levels, alter sympathetic nervous system activity, reduce leptin levels and increase levels of ghrelin, and increase inflammatory markers. The mechanisms linking long sleep duration with mortality is unknown and may be explained by underlying confounders such as depression, low socioeconomic status, undiagnosed medical disease, poor physical health, and less physical activity.

Sleep and Cardiovascular Disease

Sleeping less than 7 h per night has been found to increase the risk of developing high blood pressure (i.e., hypertension) and elevate blood pressure in those with existing hypertension (Calhoun & Harding, 2010). Long sleep duration (≥9 h per night) may also increase the risk of hypertension. The effect of insufficient sleep on blood pressure may help to explain the relationship between poor sleep and cardiovascular disease and stroke. Researchers have found both short (<7 h per night) and long (>8 h per night) sleep durations are associated with a greater risk of developing or dying from coronary heart disease and stroke (Cappuccio, Cooper, D'Elia, Strazzullo, & Miller, 2011).

There is also growing evidence of a connection between "► Sleep Apnea" and cardiovascular disease. People with sleep apnea have frequent awakenings at night as a result of repetitive pauses in breathing. This sleep fragmentation and reoccurring drops in oxygen levels in the blood called hypoxemia cause increases in blood pressure during the night that can persist during the daytime and over time can lead to hypertension. People with sleep apnea have an increased risk of developing coronary heart disease, stroke, and heart failure. In a 10-year study, severe sleep apnea was associated with an increased risk of fatal and nonfatal cardiovascular events.

Sleep and Obesity

Numerous studies have reported a link between sleep duration and obesity. For example, researchers have shown that people who sleep less than 6 h per night or more than 8 h per night are more likely to have a higher ► body mass index and that people who sleep 8 h have the lowest BMI (Cappuccio, Taggart, Kandala, & Currie, 2008). Several pathways have been identified that could mediate the relationship between short sleep and increased risk of obesity: alterations in glucose metabolism, appetite control, and energy expenditure. Sleep loss impacts hormones that regulate glucose processing and appetite (Taheri, Lin, Austin, Young, & Mignot, 2004). After sleep loss, the body's tissues are less responsive to insulin (i.e., insulin resistance), a hormone secreted by the pancreas that regulates the level of glucose in the body. As a result, glucose levels in the blood remain high, making it more difficult for the body to use stored fat for energy. Sleep loss also impacts hormones involved in appetite regulation causing people to eat even when they have had an adequate number of calories. Specifically, short sleep lowers levels of leptin, a "full signal" hormone, and increases levels of ghrelin, an appetite stimulant hormone. One night of sleep loss can decrease energy expenditure (i.e., calories burned) during rest.

Sleep and Diabetes

Numerous epidemiological studies have found that short (≤6 h per night) and long (≥9 h per night) sleep durations are associated with an increased risk of developing type 2 diabetes and impaired glucose tolerance, a precursor to diabetes (Knutson & Van Cauter, 2008). Insulin resistance associated with sleep loss can over time compromise the ability of β-cells in the pancreas to release insulin, causing higher than normal levels of glucose in the blood which can lead to type 2 diabetes. In a study of healthy adults, restricting sleep to 4 h per night for six nights led to impaired glucose tolerance.

In addition, sleep apnea is a risk factor for developing type 2 diabetes. Several potential mechanisms explaining how sleep apnea may alter glucose metabolism have been proposed. Sleep fragmentation and hypoxemia associated with sleep apnea can alter autonomic and neuroendocrine function, increase release of inflammatory cytokines, and induce adipokines and thus play a role in altering glucose metabolism.

Sleep and Cancer

Emerging evidence suggests that sleep duration may increase risks of several types of cancer. The first investigation of the association between sleep and breast cancer found women with longer sleep durations (≥9 h per night) had a decreased risk of breast cancer. Subsequent studies have found mixed results with some studies finding an inverse relationship between short sleep duration and risk of breast cancer and one study found no association. Short sleep duration was associated with an increased risk of colorectal cancer in patients undergoing colonoscopy screening and an increased risk of prostate cancer. Epidemiological studies have reported a significantly increased risk of developing a number of malignancies including breast, colon, prostate, and endometrial cancer in night-shift workers. Nocturnal melatonin suppression due to decreased sleep duration or light exposure at night in the case of shift workers may alter the oncostic action of melatonin (Blask, 2009).

Sleep and the Immune System

The relationship between sleep and immunity is reciprocal such that infections increase sleep duration and sleep deprivation negatively impacts immune function. Experimental studies have shown that sleep deprivation results in suppression in natural killer cell activity, reductions in interleukin-2 production, increased circulation of pro-inflammatory cytokines such as IL-6, tumor necrosis factor (TNF) α, and reduction of anti-inflammatory cytokines (Bryant, Trinder, & Curtis, 2004). Sleep is needed for optimal resistance to infection. Sleep restriction in healthy adults has been shown to reduce antibody production response to the influenza vaccination. One night of sleep deprivation reduced the formation of specific antibodies to hepatitis A antigens after vaccination. Short sleep duration in the weeks preceding exposure to rhinovirus increased the susceptibility to developing a cold.

Sleep and Neurocognitive Function

It has been established that sleep deprivation impairs cognitive and motor performance (Durmer & Dinges, 2005). However, some people are more vulnerable to the effects of sleep loss than others. Sleep deprivation studies have found a wide range of effects on cognitive function including decrements in attention especially vigilance, working and long-term memory, decision making, response inhibition, processing speed, and reasoning (Lim & Dinges, 2010). Sleep deprivation results in impairments in psychomotor performance that are comparable to those induced by alcohol consumption at or above the legal limit (Williamson & Feyer, 2000). Sleep loss increases the risk of traffic accidents as demonstrated by poor performance on driving simulators after sleep deprivation. In addition to an increased risk for motor vehicle crashes, sleep deprivation and related neurocognitive impairments are associated with work-related injuries and fatal accidents. Sleep apnea is also associated with deficits in cognitive function and accounts for a significant proportion of motor vehicle accidents.

Sleep and Mental Health

Numerous studies have shown a high rate of comorbidity between sleep complaints (e.g., insomnia) and psychiatric disorders, especially mood and anxiety disorders (Staner, 2010). This relationship goes beyond mere co-occurrence and is bidirectional since insomnia contributes to the development or exacerbation of depression and anxiety disorders, and affective disorders and their treatments contribute to insomnia (Neckelmann, Mykletun, & Dahl, 2007; Sateia, 2009). Nondepressed individuals with insomnia have a two times greater risk for developing depression than individuals without sleep difficulties. Sleep problems affect outcomes for patients with depression. Studies report that depressed patients who continue to experience insomnia are at greater risk of relapse and recurrence of depression and risk of suicide.

Conclusions

Sleep loss is a growing public health problem worldwide. The health consequences of a sleepy society are enormous and have a significant

economic impact with billions of dollars spent on direct medical costs associated with morbidities and sleep-related injuries and accidents. Sleep deprivation can alter biological processes underlying cardiovascular, metabolic, and immune function. Cumulative long-term effects of sleep loss have been associated with a number of serious health consequences, including cardiovascular disease, obesity, cancer, and type 2 diabetes. Sleep is not a luxury and is as important for health as other health-promoting behaviors like diet and exercise. Public awareness is needed to emphasize and reinforce the essentialness of sleep for health.

Cross-References

- ▶ Cardiovascular Disease
- ▶ Glucose: Levels, Control, Intolerance, and Metabolism
- ▶ Hypertension
- ▶ Immune Function
- ▶ Insomnia
- ▶ Insulin
- ▶ Sleep
- ▶ Sleep Apnea
- ▶ Sleep Duration
- ▶ Type 2 Diabetes

References and Readings

Blask, D. E. (2009). Melatonin, sleep disturbance and cancer risk. *Sleep Medicine Reviews, 13*(4), 257–264.

Bryant, P. A., Trinder, J., & Curtis, N. (2004). Sick and tired: Does sleep have a vital role in the immune system? *Nature Reviews Immunology, 4*, 457–467.

Calhoun, D. A., & Harding, S. M. (2010). Sleep and hypertension. *Chest, 138*(2), 434–443.

Cappuccio, F. P., Cooper, D., D'Elia, L., Strazzullo, P., & Miller, M. A. (2011). Sleep duration predicts cardiovascular outcomes: A systematic review and meta-analysis of prospective studies. *European Heart Journal, 32*(12), 1484–1492.

Cappuccio, F. P., D'Elia, L., Strazzullo, P., & Miller, M. A. (2010). Sleep duration and all-cause mortality: A systematic review and meta-analysis of prospective studies. *Sleep, 33*(5), 585–592.

Cappuccio, F. P., Taggart, F. M., Kandala, N. B., & Currie, A. (2008). Meta-analysis of short sleep duration and obesity in children and adults. *Sleep, 31*(5), 619–626.

Durmer, J. S., & Dinges, D. F. (2005). Neurocognitive consequences of sleep deprivation. *Seminars in Neurology, 25*(2), 117–129.

Knutson, K. L., & Van Cauter, E. (2008). Associations between sleep loss and increased risk of obesity and diabetes. *Annals of the New York Academy of Sciences, 1129*(1), 287–304.

Kripke, D. F., Garfinkel, L., Wingard, D. L., Klauber, M. R., & Marler, M. R. (2002). Mortality associated with sleep duration and insomnia. *Archives of General Psychiatry, 59*(2), 131–136.

Lim, J., & Dinges, D. F. (2010). A meta-analysis of the impact of short-term sleep deprivation on cognitive variables. *Psychological Bulletin, 136*(3), 375–389.

Neckelmann, D., Mykletun, A., & Dahl, A. A. (2007). Chronic insomnia as a risk factor for developing anxiety and depression. *Sleep, 30*(7), 873–880.

Sateia, M. J. (2009). Update on sleep and psychiatric disorders. *Chest, 135*(5), 1370–1379.

Staner, L. (2010). Comorbidity of insomnia and depression. *Sleep Medicine Reviews, 14*(1), 35–46.

Taheri, S., Lin, L., Austin, D., Young, T., & Mignot, E. (2004). Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. *PLoS Medicine, 1*(3), e62.

Williamson, A. M., & Feyer, A. M. (2000). Moderate sleep deprivation produces impairments in cognitive and motor performance equivalent to legally prescribed levels of alcohol intoxication. *Occupational and Environmental Medicine, 57*(10), 649–655.

Sleep Apnea

Faith S. Luyster
School of Nursing, University of Pittsburgh, Pittsburgh, PA, USA

Synonyms

Obstructive sleep apnea; Sleep-disordered breathing

Definition

Sleep apnea is a common sleep disorder characterized by repetitive pauses in breathing or very shallow breaths during sleep (Strollo & Rogers, 1996; Young, Peppard, & Gottlieb, 2002). Pauses in breathing, known as apneas, and shallow breathing events, called hypopneas, can last

a few seconds to minutes and can occur multiple times during the night. The termination of apneas and hypopneas is associated with a transient arousal from sleep. Sleep disruption due to frequent arousals may lead to excessive daytime sleepiness or fatigue. Loud snoring, witnessed breathing interruptions, and excessive daytime sleepiness are the most common signs of sleep apnea. Obstructive sleep apnea (OSA) occurs when the airway collapses or is blocked during sleep. Central sleep apnea results from temporary loss of ventilatory effort lasting at least 10 seconds and can co-occur with OSA. An evaluation for sleep apnea entails an assessment for signs and symptoms and a detailed craniofacial examination followed by a full night of in-laboratory or portable polysomnography to confirm diagnosis (Epstein et al., 2009). Sleep apnea requires long-term management. Positive airway pressure therapy, oral appliances, and surgery are the most common treatments for sleep apnea, but other behavioral interventions such as weight loss, smoking cessation, body position, and alcohol and sedative cessation, can be useful adjuncts to conventional therapies (Epstein et al., 2009). Untreated sleep apnea can result in a number of negative consequences, including excessive daytime sleepiness and fatigue, psychological symptoms, cognitive and performance impairments, and increased risk for cardiovascular and cerebrovascular disease (Bradley & Floras, 2009; Sateia, 2003). Decreased vigilance or falling asleep at the wheel increases the risk of motor vehicle crashes in individuals with sleep apnea (Ellen et al., 2006).

Cross-References

- ▶ Polysomnography
- ▶ Sleep Fragmentation

References and Readings

Bradley, T. D., & Floras, J. S. (2009). Obstructive sleep apnoea and its cardiovascular consequences. *The Lancet, 373*(9657), 82–93.

Ellen, R. L. B., Marshall, S. C., Palayew, M., Molnar, F. J., Wilson, K. G., & Man-Son-Hing, M. (2006). Systematic review of motor vehicle crash risk in persons with sleep apnea. *Journal of Clinical Sleep Medicine, 2*(2), 193–200.

Epstein, L. J., Kristo, D., Strollo, P. J., Friedman, N., Malhotra, A., Patil, S. P., et al. (2009). Clinical guideline for the evaluation, management, and long-term care of obstructive sleep apnea in adults. *Journal of Clinical Sleep Medicine, 5*(3), 263–276.

Sateia, M. J. (2003). Neuropsychological impairment and quality of life in obstructive sleep apnea. *Clinics in Chest Medicine, 24*(4), 249–259.

Strollo, P. J., & Rogers, R. M. (1996). Obstructive sleep apnea. *The New England Journal of Medicine, 334*(2), 99–104.

Young, T., Peppard, P. E., & Gottlieb, D. J. (2002). Epidemiology of obstructive sleep apnea: A population health perspective. *American Journal of Respiratory and Critical Care Medicine, 165*(9), 1217–1239.

Sleep Architecture

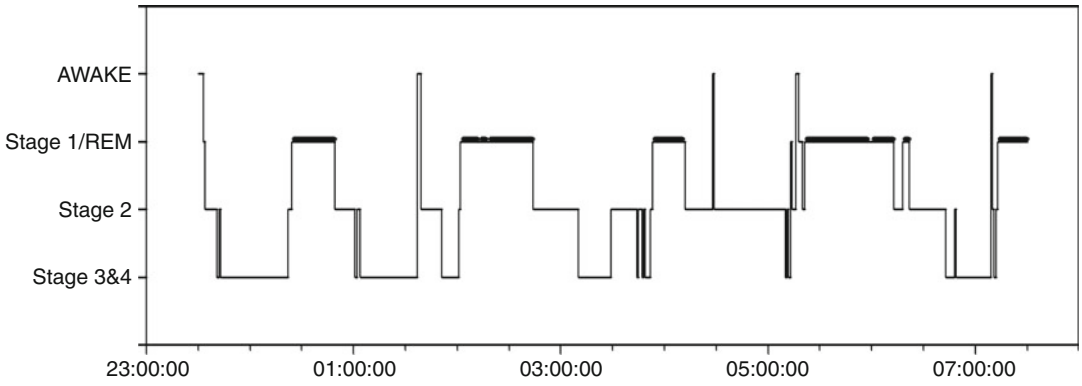
Salvatore Insana
Western Psychiatric Institute and Clinic, Pittsburgh, PA, USA

Definition

Sleep Architecture is the visual representation of the way sleep stages are organized throughout a polysomnographically recorded sleep interval. Sleep Architecture is displayed on a hypnogram plot.

Description

Sleep can be measured with polysomnography (PSG), which consists of multiple measures that include, but are not limited to, electroencephalography (EEG), electrooculography (EOG), and electromyography (EMG) (see for a review Keenan & Hirshkowitz, 2011). PSG literally translates to "many" (poly) "sleep" (somno) "writings" (graphy). When sleep is measured with PSG, the measured signals are typically sectioned into 30-s intervals consecutively throughout the entire PSG recording period. These 30-s intervals are termed "epochs." Within



Sleep Architecture, Fig. 1 Sleep hypnogram and sleep architecture during a 20-year-old female's overnight sleep monitoring period

each epoch, the PSG-measured signals (from EEG, EOG, and EMG) are cumulatively used to differentiate sleep from wake, and to further classify sleep into different categories that are known as sleep stages.

According to the 2007 American Academy of Sleep Medicine standard practice parameters, sleep can be classified into four stages that include N1, N2, N3, and Rapid Eye Movement (REM) sleep (Iber, Ancoli-Israel, Chesson, & Quan, 2007). The PSG signals are used to identify each individual epoch as a particular sleep stage. The act of using PSG signals to identify each epoch as a particular sleep stage is known as sleep stage scoring. Sleep stage scoring is completed by a trained technician who visually interprets the PSG signals in accordance with standard practice parameters.

Once the entire PSG sleep study is scored, the study is summarized into a clinical report. The clinical report describes the sleep parameters measured (e.g., EEG, EOG, EMG, airflow parameters), sleep scoring data (e.g., lights out clock time, lights on clock time, total sleep time), arousal events (e.g., number of arousals, arousal index), respiratory events (e.g., apnea index, hypopnea index), cardiac events (e.g., average heart rate during sleep), movement events (e.g., number of periodic limb movements during sleep), and summary statements (e.g., findings related to sleep diagnoses, behavioral observations, sleep hypnogram). Of particular relevance to sleep architecture is the sleep hypnogram.

The sleep hypnogram is a summary figure that visually displays the scored wake and sleep stages as they occurred throughout the entire PSG recording period. The sleep hypnogram is formatted with time throughout the entire PSG sleep study as a continuous variable on the X-axis, and the visually scored wake and sleep stages as categorical variables on the Y-axis. The categorical wake and sleep stages are positioned with REM closest to the intersection with the X-axis, followed by N3, N2, N1, and wake in an upward ascending order; however, the order and format of these stages can vary per laboratory (e.g., Fig. 1).

As time progresses throughout the sleep monitoring period, the person being monitored naturally enters and exits the wake and sleep stages that are indicated on the Y-axis. The time spent in a particular stage is represented by a horizontal line adjacent to the respective stage, and the length of that horizontal line reflects the time spent in that particular stage as it corresponds to the "real time" for which the stage occurred – indicated on the X-axis. During a stage transition, the horizontal line turns 90° (right angle) positive or negative to become vertical and adjacent to the newly entered stage – as indicated on the Y-axis; then the line turns 90° (right angle) once again to reach a horizontal position with a length that represents

the time spent in that particular stage – indicated on the X-axis. As the different wake and sleep stages fluctuate throughout the night, the hypnogram line appears at different horizontal levels (stage-dependent [Y-axis]), at different lengths on each level (time-dependent [X-axis]); and as the levels shift (stage shift), the horizontal line is connected to the previous and subsequent horizontal level by a vertical line (e.g., elbow connector).

Consequently, the wake and sleep stages represented on a sleep hypnogram resemble the back drop of a city skyline, with continuous geometric cubes and rectangles of different heights and widths that appear to continuously penetrate and retreat from the skyline. The figurative reference to a city skyline has been generally accepted, and has literally generated the term "sleep architecture." Thus, sleep architecture is the visual representation of wake and sleep stages that occurred throughout a polysomnographically recorded sleep interval, and is displayed on a hypnogram plot.

The visual structure of sleep architecture is dependent upon the sleep stage, and the time in the particular sleep stage. Typically, sleep is entered through N1, followed by N2, followed by N3, and then followed by REM – this progressive series is termed a "sleep cycle." The typical sleep cycle occurs throughout sleep at approximately 50-min intervals among infants (Grigg-Damberger et al., 2007) and approximately 90–110-min intervals among adults (Iber et al., 2007). The time spent in the different stages changes throughout the night. The first portion of the night primarily consists of non-REM sleep (i.e., N1, N2, and N3), and the second portion of the night primarily consists of REM sleep.

The sleep architecture displayed on the hypnogram from a normative adult PSG sleep study will display cumulatively longer horizontal lines adjacent to N1, N2, and N3 during the first portion of the night relative to the second portion, and will conversely display cumulatively longer horizontal lines adjacent to REM during the second portion of the night relative to the first portion. A sleep hypnogram will display the sleep stage cyclicity, as well as changes in time spent in particular stages as they occur throughout the recording period – this differs across nights. Since the time spent in different sleep stages generally changes throughout the lifespan (Ohayon, Carskadon, Guilleminault, & Vitiello, 2004), the percentage of time spent in each sleep stage, as reflected in the histogram, can differ by to the age of the person assessed.

Cross-References

- ▶ Non-REM Sleep
- ▶ Polysomnography
- ▶ REM Sleep
- ▶ Sleep

References and Readings

Grigg-Damberger, M., Gozal, D., Marcus, C. L., Quan, S. F., Rosen, C. L., Chervin, R. D., et al. (2007). The visual scoring of sleep and arousals in infants and children. *Journal of Clinical Sleep Medicine, 3*, 201–240.

Iber, C., Ancoli-Israel, S., Chesson, A., Quan, S. F., & American Academy of Sleep Medicine. (2007). *The AASM manual for the scoring of sleep and associated events: Rules, terminology and technical specifications* (1st ed.). Westchester, IL: American Academy of Sleep Medicine.

Keenan, S., & Hirshkowitz, M. (2011). Monitoring and staging human sleep. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5th ed., pp. 1602–1609). St. Louis, MO: Elsevier.

Ohayon, M. M., Carskadon, M. A., Guilleminault, C., & Vitiello, M. V. (2004). Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: Developing normative sleep values across the human lifespan. *Sleep, 27*, 1255–1273.

Sleep Continuity

Elizabeth Mezick
Department of Psychology, University of Pittsburgh, Pittsburgh, PA, USA

Synonyms

Sleep efficiency; Sleep fragmentation; Sleep maintenance

Definition

Sleep continuity refers to the amount and distribution of sleep versus wakefulness in a given sleep period; it includes both sleep initiation and sleep maintenance (Hall, Greeson, & Mezick, in press). Specific indices of sleep continuity may include latency to sleep onset, number of awakenings after sleep onset, total time of wakefulness after sleep onset, and overall sleep efficiency. Sleep continuity is most often assessed using self-report questionnaires (i.e., retrospective reports, morning diaries or logs), wrist actigraphy, or polysomnography.

Sleep continuity declines as part of the normal aging process (Ohayon, Carskadon, Guilleminault, & Vitiello, 2004). Disruptions in sleep continuity are typical among individuals with insomnia and are also commonly reported by or observed in those with mood disorders, anxiety disorders, and substance disorders. Many medical conditions and treatments are also related to disrupted sleep continuity; some of the most common examples include sleep apnea, chronic pain, asthma and respiratory conditions, chronic renal disease, infectious diseases, and cancer. Decreased sleep continuity has been associated with increased inflammatory markers, susceptibility to infection, elevated blood pressure, obesity, presence of metabolic syndrome, diabetes, and cardiovascular disease. Several studies have reported a link between decreased sleep continuity and incident diabetes or incident cardiovascular disease.

References and Readings

Hall, M., Greeson, J., & Mezick, E. (in press). Sleep as a biobehavioral risk factor for cardiovascular disease. In: S. R. Waldstein, W. J. Kop, & L. I. Katzel (Eds.), *Handbook of cardiovascular behavioral medicine*. New York: Springer.

Ohayon, M. M., Carkadon, M. A., Guilleminault, C., & Vitiello, M. V. (2004). Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: Developing normative sleep values across the human lifespan. *Sleep, 27*, 1255–1273.

Sleep Curtailment

▶ Sleep Restriction

Sleep Debt

▶ Sleep Restriction

Sleep Deprivation

Martica H. Hall
Department of Psychiatry, University of Pittsburgh, Pittsburgh, PA, USA

Synonyms

Sleep deprived

Definition

Sleep deprivation generally refers to the total loss of sleep due to experimental manipulations or circumstance.

Description

Sleep deprivation refers to the total loss of sleep. The term is generally used in the context of experimental manipulations which keep individuals (or experimental animals) awake throughout their usual sleep period. Sleep deprivation also occurs in naturalistic conditions such as when a student stays awake all night to study for an exam. Experimental and observational sleep deprivation studies have been used to evaluate the cognitive, behavioral, emotional, and physiological consequences of sleep loss.

Experimental sleep deprivation studies in humans generally deprive participants of

1–3 nights of sleep, resulting in 24–72 h of wakefulness. Experimental animal models can extend sleep deprivation for much longer periods of time (e.g., 2 weeks). In both kinds of studies, electroencephalographic (EEG) monitoring is used to ensure wakefulness, usually through interactions with study staff or procedural manipulations. For example, the classical "disk-over-water" method pioneered by Dr. Allan Rechtschaffen at the University of Chicago involved placing two animals on a rotating disk suspended above water (Rechtschaffen & Bergmann, 1995). A barrier divided the disk into half, with the experimental animal on one side of the disk and the yoked animal on the other. When the experimental animal showed EEG signs of sleep, the disk would rotate. If the animal did not wake up, they would fall into the water when they reached the barrier. Through conditioning, the experimental animal would learn to wake up as soon as the disk started to rotate. The yoked animal, on the other hand, would learn to sleep when the disk was *not* rotating. This elegant design allowed experimenters to tease apart the effects of movement and stress associated with older forms of sleep deprivation (e.g., placing the animal on a continuous running wheel with no option for escape) from the effects of sleep deprivation. In human studies, the constant routine protocol, which involves keeping participants in a recumbent position for the duration of the protocol, is used to control for the influence of extraneous factors such as increased movement on study outcomes.

Although individuals may feel that sleep deprivation has no adverse effects on them, experimental evidence suggests that perceptions of resiliency to sleep loss are unfounded. The "disconnect" between subjective perceptions of sleepiness and objective indices of health and functioning in humans has been systematically documented by Dr. David Dinges and his colleagues at the University of California (see Lim & Dinges, 2008). More recently, others have demonstrated that one night of total sleep deprivation in healthy young adults is associated with increased blood pressure and amydgala reactivity to negative emotional stimuli (Franzen et al., 2011; Yoo, Gujar, Hu, Jolesz, & Walker, 2007). As a complement to studies in humans, animal models, which allow exquisite control over sleep and wakefulness, have begun to elucidate the influence of sleep deprivation on gene expression, molecular signaling, and synaptic plasticity (e.g., Seugnet, Suzuki, Donlea, Gottschalk, & Shaw, 2011; Wang, Liu, Briesemann, Yan, 2010). Taken as a whole, these studies demonstrate that sleep is essential to health and functioning across species.

Cross-References

- ▶ Gene Expression
- ▶ Sleep
- ▶ Sleep and Health

References and Readings

Franzen, P. L., Gianaros, P. J., Marsland, A. L., Hall, M., Siegle, G. J., Dahl, R. E., et al. (2011). Cardiovascular reactivity to acute psychological stress following sleep deprivation. *Psychosomatic Medicine, 73*(8), 679–82.

Lim, J., & Dinges, D. F. (2008). Sleep deprivation and vigilant attention. *Annals of the New York Academy of Sciences, 1129*, 305–22.

Rechtschaffen, A., & Bergmann, B. M. (1995). Sleep deprivation in the rat by the disk-over-water method. *Behavioural Brain Research, 69*(1–2), 55–63.

Seugnet, L., Suzuki, Y., Donlea, J. M., Gottschalk, L., & Shaw, P. J. (2011). Sleep deprivation during early-adult development results in long-lasting learning deficits in adult *Drosophila*. *Sleep, 34*(2), 137–46.

Wang, H., Liu, Y., Briesemann, M., & Yan, J. (2010). Computational analysis of gene regulation in animal sleep deprivation. *Physiological Genomics, 42*(3), 427–36.

Yoo, S. S., Gujar, N., Hu, P., Jolesz, F. A., & Walker, M. P. (2007). The human emotional brain without sleep: A prefrontal amygdala disconnect. *Current Biology, 17*, R877–R878.

Sleep Deprived

- ▶ Sleep Deprivation

Sleep Duration

Christopher Kline
Department of Psychiatry, School of Medicine, University of Pittsburgh, Pittsburgh, PA, USA

Synonyms

Total sleep time

Definition

Sleep duration typically refers to the total amount of sleep obtained, either during the nocturnal sleep episode or across the 24-h period.

Description

Measurement

Sleep duration can be measured via questionnaire, diary, actigraphy, or polysomnography. In population-based epidemiologic studies, single-item questionnaire or self-report measures of sleep duration have often been utilized (e.g., *How many hours of sleep do you obtain on a typical night?*). In clinical and research settings, sleep diaries, actigraphy, and polysomnography provide assessments of sleep duration. Sleep diaries involve the subjective report of sleep duration, typically daily for a minimum of 1 week. Actigraphy provides an objective estimate of sleep/wake status from the detection of bodily movement, whereas polysomnography measures sleep duration through the assessment of multiple physiological signals, including brain, eye, and muscle activity. Although typically correlated, large discrepancies are often noted between subjective and objective measures of sleep duration; in most populations, self-reported sleep duration is overestimated compared to objective measurement (Silva et al., 2007).

Sleep Duration Across the Life span

Sleep is regulated by a complex interaction of homeostatic and circadian factors. The homeostatic process reflects the need for sleep, accumulating during sustained wakefulness and dissipating during sleep. The circadian process, driven by outputs of the circadian pacemaker, promotes wakefulness during the day and evening, and promotes sleep during the night, with the peak sleep-promoting signal during the early morning. Thus, circadian signals oppose the rise of homeostatic sleep pressure during the day, allowing for uninterrupted daytime wakefulness. Both circadian and homeostatic processes promote sleep onset, with circadian sleep-promoting signals facilitating a continuation of consolidated sleep despite the gradual dissipation of homeostatic sleep pressure over the course of the night. Aging results in changes in the homeostatic and circadian regulation of sleep, with a phase advance of the circadian wake-promoting signal (i.e., increased signal for wakefulness in the morning, but decreased in the evening) and reduced homeostatic drive for sleep with increasing age (Dijk, Duffy, Riel, Shanahan, & Czeisler, 1999). As a result, sleep duration in older adults is commonly phase-advanced, of shorter duration, and with greater fragmentation compared to younger adults.

In general, sleep duration decreases from infancy through old age. Sleep duration (per 24 h) averages 14 h during infancy, gradually declining to approximately 8 h during late adolescence (Iglowstein, Jenni, Molinari, & Largo, 2003). Normative sleep duration then declines throughout adulthood to approximately 6.5 h at age 60, at which point sleep duration tends to stabilize (Ohayon, Carskadon, Guilleminault, & Vitiello, 2004). It is important to note that these average values do not indicate whether actual sleep need is being met, particularly during development. For instance, although weekday sleep duration decreases throughout childhood and adolescence, weekend sleep duration remains similar from age 5 to 16. This pattern suggests a greater influence of environmental factors (e.g., school schedules) than biological or maturational influences on sleep duration during childhood and adolescence.

Whether sleep duration has changed dramatically over the latter half of the past century is

controversial. Whereas some studies have documented remarkable (i.e., >1 h) decreases in sleep duration and significantly increased prevalence of short sleep, others have presented evidence that longitudinal changes in sleep duration have been minimal (i.e., <20 min decrease over a >20-year span) (Rowshan Ravan, Bengtsson, Lissner, Lapidus, & Bjorkelund, 2010). Additional studies estimate that, although average sleep duration has changed from 8 to 7 h, the prevalence of extreme short and long sleep have not changed in the past 30 years (Kronholm et al., 2008). Moreover, longitudinal studies have documented that increased mortality risk is associated with an increase and decrease in sleep duration over time (Ferrie et al., 2007). The reduction in sleep duration in industrialized societies is likely due to both lifestyle (e.g., late bedtime due to television and/or computer diversions) and biological factors (e.g., reduced sleep need due to more sedentary lifestyles) (Horne, 2011).

Sleep Duration and Health

Sleep is an essential behavior for memory consolidation, development, and restoration of nervous, immune, skeletal, and muscular systems. Consequently, the amount of sleep obtained has a significant influence on one's health and functioning. Sleep duration is at least partly determined by genetic influences, so interindividual differences in sleep duration are to be expected. Nevertheless, a significant association between sleep duration and health has been consistently documented in epidemiologic research, highlighting the potential modulating influence of sleep on health (Bixler, 2009).

The association between sleep duration and health typically has a U-shaped distribution, with lowest risk associated with a sleep duration of 7–8 h (Cappuccio, D'Elia, Strazzullo, & Miller, 2010). Short and long sleep have been associated with increased risk of mortality and numerous morbidities, including cardiovascular disease (e.g., hypertension, atherosclerosis), metabolic dysfunction (e.g., type 2 diabetes, obesity), and cognitive impairment. However, the most marked morbidity and mortality risks have been associated with extreme short and long sleep (<5 and >9 h, respectively).

The possible mechanisms by which sleep duration affects health are likely different between short and long sleep duration (Krueger & Friedman, 2009). Most research has focused on how short sleep may adversely affect health, with studies finding alterations in the hormonal control of appetite, increased inflammatory levels, increases in hypothalamic-pituitary-adrenal axis and sympatho-adrenal activity, and blunted immunity to pathogens following short-term experimental sleep restriction (Grandner, Hale, Moore, & Patel, 2010). Plausible mechanisms linking long sleep duration to excess morbidity and mortality are less clear, though fatigue, impaired immunity, reduced photoperiod length, and underlying disease have been postulated (Youngstedt & Kripke, 2004). However, short and long sleep do share some common possible mechanisms. Both short and long sleep duration have been associated with low socioeconomic status, and sleep complaints are more prevalent in short and long sleepers compared to normal sleep duration. Moreover, poor health behaviors (e.g., smoking, alcohol, and physical inactivity) are more prevalent in short and long sleep in comparison to normal sleep duration.

Cross-References

- ▶ Polysomnography
- ▶ Sleep
- ▶ Sleep and Health
- ▶ Sleep Deprivation
- ▶ Sleep Quality
- ▶ Sleep Restriction

References and Readings

Bixler, E. (2009). Sleep and society: An epidemiological perspective. *Sleep Medicine, 10*, S3–S6.

Cappuccio, F. P., D'Elia, L., Strazzullo, P., & Miller, M. A. (2010). Sleep duration and all-cause mortality: A systematic review and meta-analysis of prospective studies. *Sleep, 33*, 585–592.

Dijk, D. J., Duffy, J. F., Riel, E., Shanahan, T. L., & Czeisler, C. A. (1999). Ageing and the circadian

and homeostatic regulation of human sleep during forced desynchrony of rest, melatonin and temperature rhythms. *The Journal of Physiology, 516*, 611–627.
Ferrie, J. E., Shipley, M. J., Cappuccio, F. P., Brunner, E., Miller, M. A., Kumari, M., et al. (2007). A prospective study of change in sleep duration: Associations with mortality in the Whitehall II cohort. *Sleep, 30*, 1659–1666.
Grandner, M. A., Hale, L., Moore, M., & Patel, N. P. (2010). Mortality associated with short sleep duration: The evidence, the possible mechanisms, and the future. *Sleep Medicine Reviews, 14*, 191–203.
Horne, J. (2011). The end of sleep: 'Sleep debt' versus biological adaptation of human sleep to waking needs. *Biological Psychology, 87*, 1–14.
Iglowstein, I., Jenni, O. G., Molinari, L., & Largo, R. H. (2003). Sleep duration from infancy to adolescence: Reference values and generational trends. *Pediatrics, 111*, 302–307.
Kronholm, E., Partonen, T., Laatikainen, T., Peltonen, M., Harma, M., Hublin, C., et al. (2008). Trends in self-reported sleep duration and insomnia-related symptoms in Finland from 1972 to 2005: A comparative review and re-analysis of Finnish population samples. *Journal of Sleep Research, 17*, 54–62.
Krueger, P. M., & Friedman, E. M. (2009). Sleep duration in the United States: A cross-sectional population-based study. *American Journal of Epidemiology, 169*, 1052–1063.
Ohayon, M. M., Carskadon, M. A., Guilleminault, C., & Vitiello, M. V. (2004). Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: Developing normative sleep values across the human lifespan. *Sleep, 27*, 1255–1273.
Rowshan Ravan, A., Bengtsson, C., Lissner, L., Lapidus, L., & Bjorkelund, C. (2010). Thirty-six-year secular trends in sleep duration and sleep satisfaction, and associations with mental stress and socioeconomic factors-results of the population study of women in Gothenburg, Sweden. *Journal of Sleep Research, 19*, 496–503.
Silva, G. E., Goodwin, J. L., Sherrill, D. L., Arnold, J. L., Bootzin, R. R., Smith, T., et al. (2007). Relationship between reported and measured sleep times; The sleep heart health study (SHHS). *Journal of Clinical Sleep Medicine, 3*, 622–630.
Youngstedt, S. D., & Kripke, D. F. (2004). Long sleep and mortality: Rationale for sleep restriction. *Sleep Medicine Reviews, 8*, 159–174.

Sleep Efficiency

► Sleep Continuity
► Sleep Fragmentation

Sleep Fragmentation

Elizabeth Mezick
Department of Psychology, University of Pittsburgh, Pittsburgh, PA, USA

Synonyms

Sleep continuity; Sleep efficiency; Sleep maintenance

Definition

Sleep fragmentation typically refers to brief arousals that occur during a sleep period. The American Sleep Disorders Association (1992) defines an arousal as an abrupt shift in electroencephalographic (EEG) frequency (suggestive of an awake state) which is 3 s or greater in duration and which occurs after at least 10 consecutive seconds of sleep. A number of other definitions of arousal have been published or suggested since that time, with some recommending that elements or physiological responses other than EEG frequency be taken into account (e.g., autonomic activation without cortical involvement) (Janackova and Sforza, 2008). When assessed with actigraphy, sleep fragmentation may refer to the amount of movement or restlessness in a sleep period. For example, actigraph software programs use algorithms to calculate a sleep fragmentation index, based on the number or proportion of total sleep epochs characterized by movement. When used in a more general sense, sleep fragmentation may also refer to the overall amount and distribution of wakefulness in a sleep period and can be considered the inverse of sleep continuity. For example, some authors have used the term "sleep fragmentation" to describe parameters such as wakefulness after sleep onset and sleep efficiency.

Elevated sleep fragmentation occurs in those with sleep apnea and may correlate with daytime sleepiness. Increased sleep fragmentation as assessed by actigraphy has been associated with

a number of physical and psychiatric health outcomes, as well as deficits in neurobehavioral performance. Experimental models of sleep fragmentation, which typically disrupt sleep briefly using auditory, mechanical, or other stimuli, have been used to examine the neurophysiologic, cognitive, and behavioral consequences of fragmented sleep.

Cross-References

▶ Sleep Apnea

References and Readings

American Sleep Disorders Association Atlas Task Force. (1992). EEG arousals: Scoring rules and examples. *Sleep, 15*, 173–184.

Bonnet, M. H., & Arand, D. L. (2003). Clinical effects of sleep fragmentation versus sleep deprivation. *Sleep Medicine Reviews, 7*, 297–310.

Janackova, S., & Sforza, E. (2008). Neurobiology of sleep fragmentation: Cortical and autonomic markers of sleep disorders. *Current Pharmaceutical Design, 14*, 3474–3480.

Sleep Maintenance

▶ Sleep Continuity
▶ Sleep Fragmentation

Sleep Quality

Christopher Kline
Department of Psychiatry, School of Medicine, University of Pittsburgh, Pittsburgh, PA, USA

Synonyms

Sleep refreshment; Sleep satisfaction

Definition

Sleep quality is defined as one's satisfaction of the sleep experience, integrating aspects of sleep initiation, sleep maintenance, sleep quantity, and refreshment upon awakening.

Description

Sleep quality is a vital construct to clinicians and researchers due to the high prevalence of disturbed sleep and insomnia, and the clear relevance of sleep quality to optimal health and functioning. Yet, despite its common usage, "sleep quality" is a term without a clear definition (Krystal & Edinger, 2008). In fact, sleep quality is likely to have different meanings from one person to the next. For someone with problems initiating sleep, the sleep onset period may be the strongest determinant of sleep quality. In contrast, the relative difficulty of going to sleep may be of trivial importance to someone whose sleep is restless and rife with frequent awakenings.

Measurement

Measurement of sleep quality is difficult due to its imprecise definition. The construct of sleep quality likely incorporates aspects of sleep quantity, wakefulness (both prior to and following sleep onset), and feeling of refreshment upon awakening, as well as daytime sleepiness (Harvey, Stinson, Whitaker, Moskovitz, & Virk, 2008). Many of these aspects cannot be easily measured in an objective fashion. Nevertheless, assessment of sleep quality has been attempted through the use of diary-based measures, subjective and objective sleep parameters, and self-report questionnaires.

Sleep diaries employ perhaps the most appropriate measure of sleep quality. Either a Likert-type rating scale (e.g., 1: *very poor sleep quality*; 5: *very good sleep quality*) or a visual analogue scale (with anchors of *very poor* and *very good* sleep quality) are traditionally incorporated into sleep diaries, providing a distinction from subjective sleep parameters.

Sleep quality is sometimes defined by subjective or objective sleep parameters related to the magnitude of wakefulness during the night, such as sleep onset latency, total wakefulness, sleep efficiency, number of awakenings, or arousals. Moreover, measures of sleep "depth" derived from polysomnography, such as the amount of N1 sleep or N3 sleep (sometimes referred to as "light" and "deep" sleep, respectively) has sometimes been used to characterize sleep quality, with increased "light" sleep linked to poor sleep quality and increased "deep" sleep linked to good sleep quality. However, these parameters often fail to completely capture the essence of sleep quality. In particular, sleep quality should not be considered to be synonymous with the amount of sleep obtained. For example, in comparison to those who report a sleep duration of 7–8 h, poor sleep quality is more prevalent with short and long sleep.

Self-report questionnaires attempt to assess sleep quality through the measurement of different domains of the sleep experience. The most widely used questionnaire, the Pittsburgh Sleep Quality Index, is an 18-item instrument with seven components: sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, sleeping medication use, and daytime dysfunction (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). Another common measure, the Medical Outcomes Study Sleep Scale, assesses six different aspects of sleep: sleep disturbance, sleep adequacy, daytime sleepiness, snoring, awakening with shortness of breath or headache, and sleep quantity (Hays, Martin, Sesti, & Spritzer, 2005). Scores on both of these measures have been shown to identify individuals who characterize their sleep as being poor in quality.

Correlates with Sleep Parameters

Sleep quality is often only weakly associated with subjective or objective sleep parameters, with wakefulness after sleep onset, sleep onset latency, and total sleep time typically showing the strongest, yet still modest, correlations ($r < 0.50$). The lack of strong concordance between common sleep parameters and sleep quality is perhaps best exemplified by the sleep of insomniacs, who by definition report poor sleep quality. For instance, only about one-half of insomniacs show differences in objective sleep parameters compared to normal sleepers. However, among those insomniacs whose objective sleep parameters were comparable to normal sleepers, sleep quality was significantly associated with spectral electroencephalographic (EEG) content, specifically lower delta-frequency EEG and higher beta-frequency EEG (Krystal & Edinger, 2008).

Sleep Quality and Health

Poor sleep quality is believed to be widespread in modern society. Approximately one third of adults complain of poor sleep quality, though in most studies prevalence estimates are based upon insomnia-related symptoms. Poor sleep quality has been associated with increasing age, low socioeconomic status, poor general health, psychological distress, and poor lifestyle behaviors (e.g., high caffeine use, sedentary lifestyle, smoking, etc.).

How poor sleep quality may influence future morbidity or mortality risk is less clear. Epidemiologic studies have found that poor sleep quality is predictive of increased risk of metabolic dysfunction and mortality risk (Cappuccio, D'Elia, Strazzullo, & Miller, 2010; Kojima et al., 2000). However, most studies have focused on sleep duration rather than sleep quality, perhaps due to the difficulty in concisely defining sleep quality. Interestingly, in recent studies that have concurrently assessed sleep duration and sleep quality, stronger associations with health risk have been found for poor sleep quality than sleep duration (Chandola, Ferrie, Perski, Akbaraly, & Marmot, 2010).

Cross-References

- ▶ Insomnia
- ▶ Polysomnography
- ▶ Sleep
- ▶ Sleep and Health

References and Readings

Buysse, D. J., Reynolds, C. F., III, Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh sleep quality index: A new instrument for psychiatric practice and research. *Psychiatry Research, 28*, 193–213.

Cappuccio, F. P., D'Elia, L., Strazzullo, P., & Miller, M. A. (2010). Quantity and quality of sleep and incidence of type 2 diabetes: A systematic review and meta-analysis. *Diabetes Care, 33*, 414–420.

Chandola, T., Ferrie, J. E., Perski, A., Akbaraly, T., Marmot, G. (2010). The effect of short sleep duration on coronary heart disease is greatest among those with sleep disturbance: a prospective study from the Whitehall II cohort. *Sleep, 33*, 739–744.

Harvey, A. G., Stinson, K., Whitaker, K. L., Moskovitz, D., & Virk, H. (2008). The subjective meaning of sleep quality: A comparison of individuals with and without insomnia. *Sleep, 31*, 383–393.

Hays, R. D., Martin, S. A., Sesti, A. M., & Spritzer, K. L. (2005). Psychometric properties of the medical outcomes study sleep measure. *Sleep Medicine, 6*, 41–44.

Kojima, M., Wakai, K., Kawamura, T., Tamakoshi, A., Aoki, R., Lin, Y., et al. (2000). Sleep patterns and total mortality: A 12-year follow-up study in Japan. *Journal of Epidemiology, 10*, 87–93.

Krystal, A. D., & Edinger, J. D. (2008). Measuring sleep quality. *Sleep Medicine, 9*, S10–S17.

Sleep Refreshment

► Sleep Quality

Sleep Restriction

Martica H. Hall
Department of Psychiatry, University of Pittsburgh, Pittsburgh, PA, USA

Synonyms

Partial sleep deprivation; Sleep curtailment; Sleep debt

Definition

Sleep restriction generally refers to situational or experimentally induced reductions in overall sleep duration.

Description

Sleep restriction refers to the partial loss of sleep. The term is generally used in the context of experimental manipulations which keep individuals (or experimental animals) awake for some portion of their usual sleep period. Sleep restriction also occurs in naturalistic conditions such as when new parents have to wake repeatedly to care for their newborn child or when an individual purposefully reduces their sleep time in order to meet the competing demands of work, family, or other obligations. Importantly, sleep restriction differs from insomnia in that sleep-restricted individuals do not have an adequate opportunity to sleep whereas individuals with insomnia have sleep difficulties despite adequate opportunity to sleep (see "► Insomnia" entry). Experimental and observational sleep restriction studies have been used to evaluate the cognitive, behavioral, emotional, and physiological consequences of sleep restriction, including the loss of specific components of sleep (e.g., slow-wave sleep).

Dr. Eve Van Cauter and her colleagues at the University of Chicago have been instrumental in highlighting the public health implications of partial sleep restriction, or sleep "curtailment" across the life span (Hanlon & Van Cauter, 2011). In a series of carefully controlled studies in healthy young adults, Van Cauter and colleagues demonstrated that sleep restriction is associated with metabolic and endocrine alterations that underlie glucose tolerance, insulin sensitivity, and weight gain. These results are mirrored in epidemiologic studies of obesity and diabetes risk in children and adults (Hanlon & Van Cauter, 2011). Other consequences of experimental sleep restriction include decrements in working memory, alterations in inflammatory markers, and decreased testosterone levels in males (Casement, Broussard, Mullington, & Press, 2006; Irwin, Wang, Campomayor, Collado-Hidalgo, & Cole, 2006; Leproult & Van Cauter, 2011; Prather et al., 2009). Experimental laboratory studies have also begun to selectively deprive experimental subjects of specific sleep stages to better understand their function. For instance, animal models have been

used to identify the neurophysiological mechanisms through which REM sleep regulates learning and memory (Poe, Walsh, & Bjorness, 2010). In the only study of its kind conducted to date, Tasali, Leproult, Ehrmann, & Van Cauter (2008) demonstrated that selective suppression of slow-wave sleep in healthy, lean adults resulted in marked decreases in insulin sensitivity. Importantly, these effects were independent of sleep duration.

Societal trends suggest that large numbers of adults are chronically sleep-restricted. During the work week, they may build up a sleep "debt" and perceive that this debt may be "paid" on non-work nights. Epidemiologic evidence that documents the buildup of sleep debt during the work week and its payment on non-work nights is lacking. Moreover, experimental evidence suggests that multiple, long recovery nights may be necessary to "repay" sleep debt induced by chronic partial sleep restriction (Banks, Van Dongen, Maislin, & Dinges, 2010). Although the systematic evaluation of the impact of sleep restriction on indices of health and functioning is in its infancy, the epidemiological and experimental data amassed thus far supports the belief that this is an important public health concern.

References and Readings

Banks, S., Van Dongen, H. P., Maislin, G., & Dinges D. F. (2010). Neurobehavioral dynamics following chronic sleep restriction: dose-response effects of one night for recovery. *Sleep, 33*, 1013–1026.

Casement, M. D., Broussard, J. L., Mullington, J. M., & Press, D. Z. (2006). The contribution of sleep to improvements in working memory scanning speed: A study of prolonged sleep restriction. *Biological Psychology, 72*(2), 208–212.

Hanlon, E. C., & Van Cauter, E. (2011). Quantification of sleep behavior and of its impact on the cross-talk between the brain and peripheral metabolism. *Proceedings of the National Academy of Sciences of the United States of America, 108*(Suppl 3), 15609–15616.

Irwin, M. R., Wang, M., Campomayor, C. O., Collado-Hidalgo, A., & Cole, S. (2006). Sleep deprivation and activation of morning levels of cellular and genomic markers of inflammation. *Archives of Internal Medicine, 166*(16), 1756–1762.

Leproult, R., & Van Cauter, E. (2011). Effect of 1 week of sleep restriction on testosterone levels in young healthy men. *JAMA: The Journal of the American Medical Association, 305*(21), 2173–2174.

Poe, G. R., Walsh, C. M., & Bjorness, T. E. (2010). Cognitive neuroscience of sleep. *Progress in Brain Research, 185*, 1–19.

Prather, A. A., Marsland, A. L., Hall, M., Neumann, S. A., Muldoon, M. F., & Manuck, S. B. (2009). Normative variation in self-reported sleep quality and sleep debt is associated with stimulated pro-inflammatory cytokine production. *Biological Psychology, 82*(1), 12–17.

Spiegel, K., Tasali, E., Penev, P., & Van Cauter, E. (2004). Brief communication: Sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite. *Annals of Internal Medicine, 141*(11), 846–850.

Tasali, E., Leproult, R., Ehrmann, D. A., & Van Cauter, E. (2008). Slow-wave sleep and the risk of type 2 diabetes in humans. *Proceedings of the National Academy of Sciences of the United States of America, 105*(3), 1044–1049.

Sleep Satisfaction

► Sleep Quality

Sleep Stages 1, 2, 3, and 4

► Non-REM Sleep

Sleep Stages 3 and 4

► Slow-Wave Sleep

Sleep Study

► Polysomnography

Sleep-Disordered Breathing

► Sleep Apnea

Slim Disease

► Cachexia (Wasting Syndrome)

Slow-Wave Sleep

Salvatore Insana
Western Psychiatric Institute and Clinic, Pittsburgh, PA, USA

Synonyms

Deep sleep; Delta sleep; N3; Sleep stages 3 and 4

Definition

Sleep can be measured with polysomnography (PSG) (see review, Keenan & Hirshkowitz, 2011). Polysomnographically measured sleep behaviors can be classified into distinct categories. Slow-wave sleep (SWS) is a distinct sleep behavior classification. SWS is also known as stage N3 sleep as defined by the 2007 American Academy of Sleep Medicine sleep scoring manual (Iber, Ancoli-Israel, Chesson, & Quan, 2007). SWS is otherwise known as the combination of sleep stages 3 and 4 according to the Rechtschaffen and Kales "classic criteria" (Rechtschaffen & Kales, 1968). Other common terms used to describe SWS are "delta sleep" and "deep sleep."

A characteristic component of SWS is the organized pattern of neurological activity that is emitted from the brain as measured by electroencephalography. A component of the organized pattern of neurological activity is the presence of slow waves, otherwise known as delta waves. Slow waves have high amplitude (>75 V) and low frequency (0.5–2 Hz). When slow waves occur during sleep, and present within 20% or more of an epoch, that epoch is classified as SWS, or N3.

Although the true function of sleep and particularly SWS is unknown (e.g., Rector, Schie, Van Dongen, Belenky, & Krueger, 2009; Siegel, 2009), to date the primary explanation is that SWS reflects the homeostatic sleep drive, or one's escalating need for sleep with increasing time awake. This relation is exemplified when One's time spent in SWS increases relative to their time previously spent awake (Bersagliere & Achermann, 2010). Thus, SWS is described as the "restorative component" of sleep. Common parasomnias that can occur during SWS are sleep walking and night terrors.

Human Development: Due to developmental changes in infant sleep patterns, typically SWS can be scored by 4–4.5 months post-term (Grigg-Damberger et al., 2007). The percentage of SWS obtained is highest during early-life and decreases from early childhood through old age (Ohayon, Carskadon, Guilleminault, & Vitiello, 2004).

Cross-References

► Non-REM Sleep
► Polysomnography
► REM Sleep
► Sleep
► Sleep Architecture

References and Readings

Bersagliere, A., & Achermann, P. (2010). Slow oscillations in human non-rapid eye movement sleep electroencephalogram: Effects of increased sleep pressure. *Journal of Sleep Research, 19*, 228–237.

Grigg-Damberger, M., Gozal, D., Marcus, C. L., Quan, S. F., Rosen, C. L., Chervin, R. D., et al. (2007). The visual scoring of sleep and arousals in infants and children. *Journal of Clinical Sleep Medicine, 3*, 201–240.

Iber, C., Ancoli-Israel, S., Chesson, A., & Quan, S. F., for the American Academy of Sleep Medicine. (2007). *The AASM manual for the scoring of sleep and associated events: Rules, terminology and technical specifications* (1st ed.). Westchester, IL: American Academy of Sleep Medicine.

Keenan, S., & Hirshkowitz, M. (2011). Monitoring and staging human sleep. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5th ed., pp. 1602–1609). St. Louis, MO: Elsevier.

Ohayon, M. M., Carskadon, M. A., Guilleminault, C., & Vitiello, M. V. (2004). Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: Developing normative sleep values across the human lifespan. *Sleep, 27*, 1255–1273.
Rechtschaffen, A., & Kales, A. (1968). *A manual of standardized, techniques and scoring system for sleep stages in human subjects* (NIH Publication No. 204). Washington DC: US Government Printing Office.
Rector, D. M., Schei, J. L., Van Dongen, H. P., Belenky, G., & Krueger, J. M. (2009). Physiological markers of local sleep. *The European Journal of Neuroscience, 29*, 1771–1778.
Siegel, J. M. (2009). Sleep viewed as a state of adaptive inactivity. *Nature Reviews: Neuroscience, 10*, 747–753.

Small-N

▶ Outcome for the Single Case: Random Control Index, Single Subject Experimental Design, and Goal Attainment Scale

Smokeless Tobacco

▶ Tobacco Control

Smoking

▶ Lifestyle Changes
▶ Nicotine
▶ Tobacco Control

Smoking and Health

Elizabeth Baker and Monica Webb Hooper
Department of Psychology, University of Miami, Coral Gables, FL, USA

Synonyms

Cigarette smoking and health; Health consequences of smoking; Smoking and health effects; Tobacco smoking and health

Definition

Health represents a physical, mental, and emotional state of well-being. Health is not simply the absence of disease or sickness but can be defined as a state of optimal wellness.

Cigarette smoking is the act of inhaling smoke from burning tobacco. Cigarette smoking is directly related to a decline in health and various associated health outcomes. There is no safe level of smoking; therefore, for optimal health, tobacco use should be avoided.

Description

The adverse health effects of smoking have been well documented. Each year, smoking causes more deaths than murders, suicides, HIV, drug and alcohol use, and auto accidents combined (Centers for Disease Control and Prevention [CDC] (2009)). In the United States, cigarette smoking is responsible for over 443,000 deaths, $96 billion in medical expenditures, and 5.1 million years of potential life lost annually (CDC, 2009). In fact, one out of five Americans will die prematurely from the effects of smoking. Tobacco use is responsible for five to six million deaths per year worldwide (Jha, 2009). This makes cigarette smoking the single largest preventable cause of disease and death.

Cigarette smoking causes much of its damage to the body through the inhaled smoke (U.S. Department of Health and Human Services et al. (2010)). Cigarettes contain over 7,000 chemical compounds, many of which are toxic and/or carcinogenic. It is the inhalation of these chemicals that leads to increased risks for heart disease, cancer, and stroke (USDHHS, 2004). Heart disease is the leading cause of death in the USA and particularly among smokers. Smoking cigarettes causes the heart to work harder by raising heart rate and blood pressure (Erhardt, 2009). Additionally, poisonous gases such as carbon monoxide limit the amount of oxygen carried in the blood. This results in a two- to four-fold increased risk of heart attack and stroke (USDHHS, 2004). Smoking also

causes lung disease such as chronic obstructive pulmonary disease (COPD), which includes emphysema and chronic bronchitis (U.S. Department of Health & Human Services et al., 2010). Indeed, smoking causes about 90% of all deaths from chronic airway obstruction (Forey, Thornton, & Lee, 2011). There is a well-established link between smoking and multiple cancers including the lung, mouth, throat, stomach, uterus, esophagus, cervix, bladder, and acute myeloid leukemia (USDHHS, 2004). Nearly one third of all cancer deaths can be linked directly to cigarette smoking (CDC, 2010). People who suffer from chronic diseases such as diabetes and HIV are especially vulnerable to the negative effects of tobacco use. Indeed, smokers with a chronic disease may experience increased complications, longer hospitalizations, interactions with medications, and increased risk of death (CDC, 2010).

Smoking also takes a toll on mental health and overall well-being. There is a robust relationship between cigarette smoking and perceived stress (Kassel, Stroud, & Parnois, 2003). Smokers tend to report greater stress levels compared to nonsmokers (Cohen & Williamson, 1988). And although smokers believe that cigarette smoking provides stress relief, research suggests that smoking might cause additional stress (Heishman, 1999). Smoking is also associated with increased risk of affective disorders, such as anxiety and depression. Smokers with mental health disorders report substantially greater symptom burden and functional disability compared to nonsmokers (Covey, 1998; McCloughen, 2003; Morissette, 2007). Smoking also reduces the effectiveness of a number of medications used to help manage the symptoms of depression and schizophrenia (Goff, 1992).

Smoking also affects health in other ways. Compared to nonsmokers, smokers have increased risks of blindness, periodontal disease, deafness, sexual dysfunction, sleeping difficulties, headaches, and premature aging, including wrinkles and damage to the skin (USDHHS, 2010). Among men, smoking causes an increased risk of erectile dysfunction (Tengs, 2001). Women who smoke have increased risk of hip fractures, infertility, and premature menopause (USDHHS, 2001).

Smoking not only affects the health of smokers but also impacts individuals around them (USDHHS, 2006). Nonsmokers who are exposed to tobacco smoke inhale many of the same toxins and cancer-causing substances as smokers. Secondhand smoke (or passive smoking) is responsible for numerous health problems among adults, including an increased risk of heart disease and cancer (USDHHS, 2006). Women who smoke during pregnancy risk passing on the toxins from cigarettes to their babies. Such exposure leads to increased risk for premature labor, low birth weight, and birth defects (USDHHS, 2006). Children and infants also suffer from secondhand smoke exposure including asthma attacks, ear infections, respiratory illness, and sudden infant death syndrome (USDHHS, 2006).

There are many immediate and long-term health benefits of smoking cessation (USDHHS, 2004). After the last cigarette, a person's heart rate and blood carbon monoxide level drop to normal within hours. Lung function and capacity improves within days. The excess risk of coronary heart disease, cancers of the lung and mouth, and stroke reduce to that of a nonsmoker 1–15 years after quitting smoking (USDHHS, 2004). Quitting smoking at any age and despite any existing medical conditions can help improve overall health.

Cross-References

▶ Smoking Cessation

References and Readings

Centers for Disease Control and Prevention. (2009). Annual smoking-attributable mortality, years of potential life lost, and productivity losses-United States, 2000–2004. *Morbidity and Mortality Weekly Report, 58*(02), 29–33. Retrieved from http://www.cdc.gov/mmwr/

Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion. (2010). *Cancer Statistics 2010*. Retrieved from http://www.cdc.gov/Features/

Cohen, S., & Williamson, G. (1988). Perceived stress in a probability sample of the United States. In S. Spacapan, & S. Oskamp (Eds.), *The social psychology of health: Claremont Symposium on applied social psychology* (pp. 31–68). Newbury Park, CA: Sage

Covey, L. L. S. C. (1998). Cigarette smoking and major depression. *Journal of Addictive Diseases, 17*(1), 35–46.

Erhardt, L. (2009). Cigarette smoking: An undertreated risk factor for cardiovascular disease. *Atherosclerosis, 205*(1), 23–32. doi:10.1016/j.atherosclerosis.2009.01.007.

Forey, B., Thornton, A., & Lee, P. (2011). Systematic review with meta-analysis of the epidemiological evidence relating smoking to COPD, chronic bronchitis and emphysema. *BMC Pulmonary Medicine, 11*(1), 36.

Goff, D. C. (1992). Cigarette smoking in schizophrenia: Relationship to psychopathology and medication side effects. *The American Journal of Psychiatry, 149*(9), 1189–1194.

Heishman, S. J. (1999). Behavioral and cognitive effects of smoking: Relationship to nicotine addiction. *Nicotine & Tobacco Research, 1*(2), S143–S147. doi:10.1080/14622299050011971.

Jha, P. (2009). Avoidable global cancer deaths and total deaths from smoking. *Nature Reviews. Cancer, 9*(9), 655–664. doi:10.1038/nrc2703.

Kassel, J. D., Stroud, L. R., & Paronis, C. A. (2003). Smoking, stress, and negative affect: Correlation, causation, and context across stages of smoking. *Psychological Bulletin, 129*, 270–304. doi:10.1037/0033-2909.129.2.270.

McCloughen, A. A. (2003). The association between schizophrenia and cigarette smoking: A review of the literature and implications for mental health nursing practice. *International Journal of Mental Health Nursing, 12*(2), 119–129.

Morissette, S. S. B. (2007). Anxiety, anxiety disorders, tobacco use, and nicotine: A critical review of interrelationships. *Psychological Bulletin, 133*(2), 245–272.

Tengs, T. O. (2001). The link between smoking and impotence: Two decades of evidence. *Preventive Medicine, 32*(6), 447.

U.S. Department of Health and Human Services, Office on Smoking and Health, National Center for Chronic Disease Prevention and Health Promotion. (2010). *How tobacco smoke causes disease: The biology and behavioral basis for smoking-attributable disease: A report of the surgeon general*. Atlanta, GA: Author.

U.S. Department of Health and Human Services, Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. (2004). *The health consequences of smoking: A report of the surgeon general*. Atlanta, GA: Author.

U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. (2006). *The health consequences of involuntary exposure to tobacco smoke: A report of the surgeon general*. Atlanta, GA: Author.

U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General. (2001). *Women and smoking: A report of the surgeon general*. Rockville, MD: Author.

Smoking and Health Effects

▶ Smoking and Health

Smoking Behavior

Elizabeth Baker and Monica Webb Hooper
Department of Psychology, University of Miami, Coral Gables, FL, USA

Synonyms

Cigarette smoking; Smoking habits; Smoking topography; Tobacco use

Definition

Smoking behaviors are actions taken by a person that are associated with the burning and inhalation of a substance. Smoking behavior is multifaceted and includes the actual act of smoking, puffing style, depth of inhalation, and rate and frequency of smoking.

Description

The act of smoking consists of several behaviors and is usually applied to tobacco/cigarettes. A smoker is defined as a person who has a lifetime history of smoking 100 cigarettes or more with current smoking on some days or every day.

Most people experiment with smoking during adolescence and do not intend to become regular, addicted, or dependent smokers. Environmental or social factors (e.g., peer pressure) often play a role in smoking initiation. Over time, smoking behavior can become a pattern (i.e., habit) and tolerance develops. The next step is the development of dependence, which is indicated by both tolerance and withdrawal symptoms during periods of abstinence from smoking. Finally, people maintain the compulsive (e.g., addictive) behavior largely because of nicotine dependence.

Smoking behavior is based on individual differences. Smoking topography is defined as the unique manner in which an individual smokes a cigarette. In particular, topography includes the quantity of puffs per cigarette, puff volume, velocity, and duration (Scherer, 1999). Each of these behaviors is a component of how a cigarette is smoked. On average, smokers ingest less than 1.5 mg of nicotine per cigarette (Jarvis, Boreham, Primatesta, Feyerabend, & Bryant, 2001; United States Federal Trade Committee, 2000). This amount varies depending on cigarette brand and how an individual smokes a cigarette (Djordjevic, 2000). Dependent smokers seek to regulate the amount of nicotine they receive to maintain the desired physical and emotional state and to avoid withdrawal. In general, a smoker takes about 10 puffs per cigarette (Scherer, 1999). A smoker can consciously or unconsciously control nicotine intake by the time taken in between puffs (referred to as puff frequency or puff interval). Smokers may also block the ventilation holes in filtered cigarettes to increase nicotine delivery. The depths of inhalation, duration of the puff, and the amount of smoke in a puff (volume) are all characteristics of smoking behavior that impact exposure to the chemicals and toxins in cigarettes and the subsequent damage to the body (Scherer, 1999).

The frequency of daily smoking is another aspect of smoking behavior. The Centers for Disease Control [CDC] (2005) reported that 83% of all smokers are daily users. Daily smokers average 20 cigarettes per day (CDC, 2005). Intermittent or light smokers do not smoke daily and are thought to be less nicotine dependent. The time to the first cigarette of the day and the frequency of daily smoking are indicators of nicotine dependence. Smoking immediately after waking is one of the best predictors of nicotine dependence. In addition, smoking most of one's cigarettes during the first 2 hour of the day is suggestive of dependence (Heatherton, 1989). A greater frequency of daily smoking may be related to difficulty quitting smoking.

Smoking behavior is variable and can change depending on the circumstance or day. For instance, puffs per cigarette may decrease while watching television or may increase while listening to another person talking. The number or duration of puffs may also change according to emotional state. Research has found that increased smoking often occurs during times of personal crisis and stressors (Shiffman, Paty, Gnys, Kassel, & Hickcox, 1996). Many smokers report increased smoking in social situations such as being in the company of other smokers or while attending parties. Smokers also vary in their handling of cigarettes; some may hold the cigarette in their mouth, while others allow them to burn in ashtrays or between their fingers.

Smoking behavior is complex. Learning models suggest that smoking behavior is maintained by operant conditioning, including positive and negative reinforcement, and classical conditioning, through the repeated pairing of smoking to various physical and emotional states (Wilker, 1973). Such learning makes smoking cessation a formidable challenge for most smokers (Patten & Martin, 1996).

Cross-References

- ▶ Behavior Change
- ▶ Smoking Cessation

References and Readings

Centers for Disease Control and Prevention. (2005). Cigarette smoking among adults – United States, 2004. *Morbidity and Mortality Weekly Report*, *54*(44), 1121–1124. Retrieved 23 November 2011, from http://www.cdc.gov/mmwr/

Djordjevic, M. M. V. (2000). Doses of nicotine and lung carcinogens delivered to cigarette smokers. *Journal of the National Cancer Institute, 92*(2), 106–111.
Heatherton, T. T. F. (1989). Measuring the heaviness of smoking: Using self-reported time to the first cigarette of the day and number of cigarettes smoked per day. *British Journal of Addiction, 84*(7), 791–800.
Jarvis, M. J., Boreham, R., Primatesta, P., Feyerabend, C., & Bryant, A. (2001). Nicotine yield from machine-smoked cigarettes and nicotine intakes in smokers: Evidence from a representative population survey. *Journal of the National Cancer Institute, 93*(2), 134–138. doi:10.1093/jnci/93.2.134.
Patten, C., & Martin, J. (1996). Does nicotine withdrawal affect smoking cessation? Clinical and theoretical issues. *Annals of Behavioral Medicine, 18*(3), 190–200. doi:10.1007/BF02883397.
Scherer, G. G. (1999). Smoking behaviour and compensation: A review of the literature. *Psychopharmacology, 145*(1), 1–20.
Shiffman, S., Paty, J., Gnys, M., Kassel, J., & Hickcox, M. (1996). First lapses to smoking: Within-subjects analysis of real-time reports. *Journal of Consulting and Clinical Psychology, 64*(2), 366–379. doi:10.1037/0022-006X.64.2.366.
United States Federal Trade Committee. (2000). *"Tar," nicotine, and carbon monoxide of the smoke of 1294 varieties of domestic cigarettes for the year 1998*. Retrieved 23 November 2011, from http://www.ftc.gov/reports/tobacco/1998tar&nicotinereport.pdf
Wilker, A. (1973). Dynamics of drug dependence: Implications of a conditioning theory for research and treatment. *Archives of General Psychiatry, 28*, 611.

Smoking Cessation

Denise de Ybarra Rodríguez and
Monica Webb Hooper
Department of Psychology, University of Miami, Coral Gables, FL, USA

Synonyms

Cigarette smoking cessation; Quit smoking; Stop smoking; Tobacco cessation; Tobacco smoking cessation

Definition

Tobacco smoking is defined as the practice of burning and inhaling tobacco. The combustion from the burning allows the nicotine, tar, and other chemicals and toxins to be absorbed through the lungs. Cigarette smoking is the most prevalent form of consuming tobacco. Most national surveys define a current smoker as having smoked at least 100 (5 packs) cigarettes in their lifetime and currently smokes on at least some days.

Cessation refers to a halting or stopping. Smoking cessation refers to the stopping of cigarette use. Smoking cessation may refer to choosing to stop smoking deliberately or become abstinent due to external and/or environmental factors leading to stopping or quitting smoking.

Description

Smoking cessation is the single most important health behavior change a person can make. Since the 1964 Surgeon General's Report concluded that smoking causes cancer, about 50 million people have successfully quit smoking. Approximately 69% of current smokers state that they want to stop, and 52% have made an attempt to quit smoking in the past year (Centers for Disease Control & Prevention [CDC], 2011).

Smoking cessation has major health benefits. The most common causes of death in the United States are cardiovascular disease, cancer, cerebrovascular accidents, and chronic lower respiratory diseases (Kochanek, Xu, Murphy, Miniño, & Kung, 2011); smoking cessation has been associated with a reduced risk of dying from these diseases (U.S. Department of Health & Human Services [USDHHS], 1990). The benefits of smoking cessation are greater the earlier one quits, though the benefits of quitting can be experienced even after an extended smoking history. For example, after 15 years of smoking cessation, the excess risk of heart disease for a former smoker is equivalent to a never-smoker. With the increasing duration of cessation, the overall rate of cancer mortality approaches that of nonsmokers (USDHHS).

There are several evidence-based smoking cessation methods. Behavioral interventions include brief physician advice to quit, self-help

materials, telephone-based interventions, internet-based counseling and support groups, and group and individual counseling (Fiore et al., 2008). Brief physician advice to quit smoking has been demonstrated to increase the likelihood of cessation by 30%. Self-help cessation interventions increase the likelihood of cessation by 20%. Telephone-based interventions increase the likelihood of cessation by 20%, though tobacco quitlines have increased the odds of cessation by up to 60%. Group and individual counseling increase the likelihood of cessation by 30% and 70%, respectively. The U.S. Food and Drug Administration (FDA) has approved seven smoking cessation pharmacotherapies. These include varenicline (marketed as Chantix in the United States and Champix in Canada and Europe), bupropion (marketed as Zyban), transdermal nicotine patches, nicotine gum, nicotine lozenges, nicotine nasal spray, and the nicotine inhaler. Nicotine patches, gum, and lozenges are available in the USA over the counter; varenicline, bupropion, nicotine nasal spray, and the nicotine inhaler require a physician's prescription. Use of nicotine replacement therapies or bupropion doubles the chances of smoking cessation, while recent evidence suggests that varenicline can triple the likelihood of cessation. Using more than one method when trying to quit (e.g., combination of nicotine replacement with counseling or a telephone quitline) further increases the likelihood of cessation, and the likelihood of cessation increases with the number of formats utilized.

There are also smoking cessation methods that are not based on empirical evidence. Indeed, the most common method of attempting to quit is unassisted quitting (going "cold turkey"). However, approximately 90% of unassisted quit attempts fail. Complementary and alternative methods such as acupuncture, acupressure, laser therapy, and hypnotherapy have been evaluated as smoking cessation methods. However, these methods are not more effective than a placebo (Barnes et al., 2010; White et al., 2011).

Public health policy and campaigns have been instrumental in decreasing the prevalence of smoking in the USA. These policies have restricted indoor smoking via clear indoor air acts (e.g., restaurants, airplanes, and workplaces) in most states and have increased taxes on cigarettes. Public health campaigns have utilized mostly media formats to increase knowledge about the dangers of smoking.

Smoking cessation can result in temporary discomfort, known as nicotine withdrawal. When a person quits smoking, they can enter into nicotine withdrawal within 30 min. The peak of withdrawal occurs within 48–72 h for dependent smokers and usually lasts 1–2 weeks if cessation is maintained. Symptoms of nicotine withdrawal include changes in mood (e.g., irritability, anger, depression, sadness, anxiety, and nervousness), desire or craving to smoke, difficulty concentrating, increased appetite, weight gain, insomnia, restlessness, impatience, constipation, dizziness, coughing, and dreaming or nightmares. The use of FDA-approved cessation aids can minimize withdrawal symptoms.

Smoking cessation is greatly encouraged because of the multiple health benefits that follow. Since the dangers of smoking have been identified, the prevalence has declined and leveled off at 20%. Cigarette smoking is the leading cause of preventable death, disease, and disability in the USA. Multiple methods to achieve smoking cessation exist, though some of these methods have a greater likelihood of success.

Cross-References

- ▶ Cancer and Smoking
- ▶ Lifestyle Changes
- ▶ Smoking and Health
- ▶ Tobacco Control

References and Readings

Barnes, J., Dong, C. Y., McRobbie, H., Walker, N., Mehta, M., & Stead, L. F. (2010). Hypnotherapy for smoking cessation. *Cochrane Database of Systematic Reviews*. doi:10.1002/14651858.CD001008.pub2.

Centers for Disease Control and Prevention. (2011). Quitting smoking among adults-United States, 2001–2010. *Morbidity and Mortality Weekly Report, 60*(44), 1513–1519.

Fiore, M. C., Jaén, C. R., Baker, T. B., Bailey, W. C., Benowitz, N. L., Curry, S. J., et al. (2008). *Clinical practice guideline: Treating tobacco use and dependence*. Rockville: U.S. Department of Health and Human Services. Public Health Service.

Kochanek, K. D., Xu, J., Murphy, S. L., Miniño, A. M., & Kung, H.-C. (2011). Deaths: Preliminary data for 2009. *National Vital Statistics Reports, 59*(4), 1–51.

U.S. Department of Health and Human Services. (1990). *The health benefits of smoking cessation: A report of the surgeon general*. Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health.

United States Department of Health and Human Services (USDHHS). (1982). *The health consequences of smoking: Cancer: A report of the surgeon general*. Rockville: USDHHS.

White, A. R., Rampes, H., Liu, J. P., Stead, L. F., & Campbell, J. (2011). Acupuncture and related interventions for smoking cessation. *Cochrane Database of Systematic Reviews, 1* Art. No.: CD000009. doi: 10.1002/14651858.CD000009.pub3

Smoking Habits

► Smoking Behavior

Smoking Prevention

► Tobacco Control

Smoking Prevention Policies and Programs

Andrea C. Villanti and David B. Abrams
Johns Hopkins Bloomberg School of Public Health, The Schroeder Institute for Tobacco Research and Policy Studies at Legacy, Washington, DC, USA

Synonyms

Tobacco control

Definition

Since up to 80% of smoking initiation has been shown to occur in adolescence, individual and group-level smoking prevention interventions have focused on youth to avert addiction and the long-term health consequences of smoking. A focus of policy efforts for youth prevention has been restricting youth access to tobacco products at the point of purchase. Effective interventions with broader reach to reduce tobacco product initiation among adolescents and adults include policies that increase the unit price of tobacco products and mass media campaigns when combined and coordinated with other interventions.

Description

From 2002 to 2010, the prevalence of past month tobacco use in the United States among those aged 12 and above declined from 30.4% to 27.4%, with stalled reductions in prevalence in the later years. Currently, 23.0% of the population aged 12 or older smokes cigarettes. Patterns in current tobacco and cigarette use and experimentation with smoking cigarettes were similar for middle school and high school students; while overall prevalence has reached a historic low, no overall declines were noted in youth tobacco use for the 2006–2009 period. Among youth, cigarette smoking is more prevalent among whites than blacks and slightly higher among males than females.

Young people are particularly vulnerable to tobacco addiction due to the effect of nicotine on the reward pathways in the developing brain (from in utero to young adulthood). Additionally, some people are at much greater risk of tobacco use due to genetics, poverty, abuse, neglect, trauma, and other comorbid psychological or cognitive factors such as mood disorders, behavioral, conduct, and attention problems. Beyond the individual, there are numerous variables that affect tobacco use, including adult, household and peer behavior, advertising and promotion, availability and

price of tobacco products, poverty, unemployment, neighborhood, community, and cultural norms.

Comprehensive national tobacco strategies use a combination of methods to achieve four main goals: (1) to prevent initiation among youth and young adults, (2) to promote quitting among adults and youth, (3) to eliminate exposure to secondhand smoke, and (4) to identify and eliminate tobacco-related disparities among population groups. Since up to 80% of smoking initiation has been shown to occur in adolescence, individual and group-level smoking prevention interventions have focused on youth to avert addiction and the long-term health consequences of smoking. A focus of policy efforts for youth prevention has been restricting youth access to tobacco products at the point of purchase. Recent data indicates increasing initiation in young adults in the USA, possibly due to targeted marketing efforts by the tobacco industry, and signals the need for extended prevention efforts beyond youth. Effective interventions with broader reach to reduce tobacco product initiation among adolescents and adults include policies that increase the unit price of tobacco products and mass media campaigns when combined and coordinated with other interventions.

Individual and School-Based Programs for Youth

For many years, smoking prevention efforts for adolescents were conducted primarily through school-based programs, which have been shown to have positive short-term effects, but little effect on long-term prevention. Early school-based tobacco curricula focused on social influences, training youth to resist social pressures to use tobacco; studies of these interventions have shown that social influences programming alone is not effective in reducing long-term initiation of smoking. More recent school-based programs have achieved greater success by addressing multiple determinants of tobacco use, including communication skills, coping, personal and social competence, and physical consequences of smoking.

Restricting Youth Access to Tobacco Products

Youth access laws prohibit retailers from selling tobacco products to youth under the age of 18. These laws require enforcement to ensure compliance by the many types of retailers selling tobacco products – from street vendors to convenience stores and online distributors. Adolescent access to cigarette vending machines, low enforcement of these laws, and difficulties in confirming age at purchase in online transactions are all barriers to the success of these laws. Studies indicate that youth access laws can slow increases in adolescent smoking and reduce both smoking prevalence and cigarette consumption, but a very high level of retailer compliance is necessary before these changes occur.

Interventions to Increase the Price of Tobacco Products

Tobacco taxation has been hailed as the most effective intervention to reduce demand for and consumption of tobacco products. Increases in tobacco taxes result in increased cigarette prices, and price has been shown to be a key factor in determining both smoking initiation and cigarette consumption among adults and adolescents.

Price elasticity of demand is defined as the percentage change in consumption of a product following a 1% increase in price. Among US adults, estimates of the price elasticity of cigarette demand typically fall between -0.3 and -0.5, relating to a 3% or 5% decrease in consumption, respectively, for a 10% increase in price. Adolescents have been shown to be almost three times more sensitive to cigarette price increases than adults for several reasons, including the following: First, adolescents have been posited to be less addicted to nicotine and more able to reduce or quit smoking following cigarette price increases. Second, adolescents typically have a lower income and spend a larger fraction of their disposable income on cigarettes than adults. Third, adolescents are also likely to be present-oriented and may not be willing to spend the additional money on cigarettes at the expense of other activities. Reductions in adolescent smoking following tobacco tax interventions may also result from fewer smoking peers.

Mass Media Campaigns to Prevent Tobacco Use

Tobacco marketing includes all efforts of the industry to promote tobacco products, and industry marketing has been linked to a variety of smoking behaviors among both adults and youth, notably youth tobacco initiation. Countermarketing is a strategy used by public health agencies to protect individuals who may be susceptible to the influence of tobacco industry marketing – particularly youth – by responding directly to that marketing. Prior to the end of 1999, major statewide comprehensive tobacco control programs in California, Massachusetts, Arizona, Oregon, and Florida included countermarketing media campaigns which were shown to reduce adult and youth smoking; results from evaluations of these interventions indicated that these campaigns had more influence on smoking behavior in younger compared to older adolescents. The 1998 Tobacco Master Settlement Agreement (MSA) in the United States included provisions for reducing youth access to tobacco products and restricted marketing in venues or media attended by youth. Funds were also dedicated from the MSA to create a foundation to develop and deliver national anti-tobacco messages. The truth® campaign, a national tobacco countermarketing effort launched in 2000 by the American Legacy Foundation, has repeatedly been shown to be effective in reducing smoking among adolescents, specifically younger adolescents. Around the same time, tobacco companies developed youth smoking prevention media campaigns which have been shown to be ineffective, and in the case of parent-targeted advertising, to reduce perceptions of smoking-related harm and to increase approval of smoking and intention to smoke among older adolescents.

Future Directions in Smoking Prevention

Passage of the Family Smoking Prevention and Tobacco Control Act in 2009 gave the Food and Drug Administration (FDA) authority to regulate tobacco products and their marketing to protect the public health. One of FDA's first actions was to ban candy flavorings in cigarettes shown to be appealing to youth in order to reduce youth smoking initiation. Other possible regulatory actions aimed at tobacco use prevention include banning menthol in cigarettes, reducing the amount of nicotine in cigarettes to a non-addictive level, and introducing large, graphic warning labels on health effects of tobacco use to cigarette and smokeless tobacco packaging. The national scale of regulatory action through the FDA has the potential to dramatically influence population-level tobacco use and must be complemented by tobacco control efforts at the community, local, and state levels to achieve the maximum impact on prevention.

Summary

Comprehensive tobacco control programs have taken a community-based approach to smoking prevention, using a combination of school-based, policy, educational, and mass media interventions to change the social environment and conditions related to smoking behavior with positive results. Reviews of community-based programs show that coordinated multicomponent interventions reduce smoking among young people more than single strategies alone and that the level of program funding and implementation is critical to the success of these interventions. A population-based health promotion approach that includes media, education, screening, interventions in community settings and policy can provide avenues to address tobacco use more comprehensively. Coordination of policy and program efforts from the local to the national level is needed to enhance the effectiveness of tobacco use prevention interventions.

Cross-References

- ▶ Smoking and Health
- ▶ Smoking Behavior
- ▶ Smoking Cessation
- ▶ Tobacco Cessation
- ▶ Tobacco Use

References and Readings

American Legacy Foundation. *Youth smoking prevention mass media campaign*. Retrieved from http://www.thetruth.com/

Center for Tobacco Products, Food and Drug Administration. Retrieved from http://www.fda.gov/tobaccoproducts/default.htm

Centers for Disease Control and Prevention. (2007). *Best practices for comprehensive tobacco control programs-2007*. Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. Retrieved from http://www.cdc.gov/tobacco/stateandcommunity/best_practices/index.htm

Farrelly, M. C., Davis, K. C., Haviland, M. L., Messeri, P., & Healton, C. G. (2005). Evidence of a dose–response relationship between "truth" antismoking ads and youth smoking prevalence. *American Journal of Public Health, 95*(3), 425–431.

Institute of Medicine. (2007). *Ending the tobacco problem: A blueprint for the nation*. Washington, DC: National Academies Press. Retrieved from http://www.iom.edu/Reports/2007/Ending-the-Tobacco-Problem-A-Blueprint-for-the-Nation.aspx

Substance Abuse and Mental Health Services Administration. (2011). *Results from the 2010 National Survey on Drug Use and Health: Summary of National Findings*, NSDUH Series H-41, HHS Publication No. (SMA) 11-4658. Rockville, MD: Substance Abuse and Mental Health Services Administration. Retrieved from http://www.samhsa.gov/data/NSDUH/2k10Results/Web/PDFW/2k10Results.pdf

Task Force on Community Preventive Services. (2005). Tobacco. In : S. Zaza, P. A. Briss, K. W. Harris, (Eds.), The guide to community preventive services: What works to promote health? (pp. 3–79). Atlanta, GA: Oxford University Press. Retrieved from http://www.thecommunityguide.org/tobacco/index.html

Smoking Topography

▶ Smoking Behavior

SNP (Pronounced "Snip")

▶ Single Nucleotide Polymorphism (SNP)

Social Behavior

▶ Interpersonal Circumplex

Social Capital and Health

Martin Lindström
Department of Clinical Sciences in Malmö, Lund University, Malmö, Sweden

Social capital has no direct synonyms. Social cohesion is a broader concept which is *not* directly synonymous with social capital. Social cohesion is a combination of absence of latent social conflict, e.g., in the form of social and economic inequality, ethnic tensions or other forms of polarization, and the presence of strong social bonds measured by levels of trust and reciprocity (i.e., social capital), a strong "civil society" which bridge social divisions, and institutions of conflict management (e.g., a responsive democracy and an independent judiciary) (Kawachi & Berkman, 2000).

Definition

The definition of social capital varies by author and academic tradition. Robert Putnam (political science) defines social capital as "features of social organization, such as trust, norms, and networks that can improve the efficiency of society by facilitating coordinated actions" (Putnam, 1993). According to James Coleman (sociology), social capital is ". . .a variety of different entities (that) facilitate certain actions of individuals who are within a structure" (Coleman, 1990), while Pierre Bourdieu (sociology) defines it as ". . .the sum of the resources, actual or virtual, that accrue to an individual or a group by virtue of possessing a durable network" (Bourdieu & Wacquant, 1992). The definitions of Putnam, pertaining to the population level of social

capital, and Coleman, pertaining to social networks of individuals, have been more commonly used in behavioral medicine and public health than that of Bourdieu, which is closer to the individual level, is less distinct in relation to social support, and also concerns the power structure within social groups and structures. Health as a concept is defined elsewhere.

Description

The social capital concept was first used in 1916 but originates in its modern context in the fields of sociology and political science. Social capital concerns the characteristics of interaction between actors such as individuals, social groups, and organizations rather than the characteristics of actors per se. Social capital includes interpersonal trust, trust in institutions, reciprocity, civic participation, social participation, and social networks which increase cooperation and decrease transaction costs in society.

The first articles concerning social capital and health appeared in international medical and public health journals in the mid-1990s with a fast increase in the number of publications after 1996–1998. From approximately 40 international journal publications in 2001 (Macinko & Starfield, 2001), the number of journal publications has increased to 1,179 in 2011 in PubMed only (December 19, 2011). Already in 2001, Macinko and Starfield identified four levels of analysis of social capital and health: the macro (countries, regions), meso (neighborhoods, municipalities), micro (social networks), and psychological (trust) levels (Macinko & Starfield, 2001). Putnam's definition of social capital, which includes trust despite Putnam's macro and meso perspectives because the level of trust may be regarded as a trait of populations and countries just as well as a cognitive trait of the individual, has, together with Coleman's definition, had the strongest impact in the behavioral medicine and public health literature, although other authors such as Bourdieu and Portes are also referred to and their definitions also used in empirical studies.

Although Putnam had earlier stated that the area of health and public health was probably a research area not particularly strongly associated with and affected by social capital (Putnam, 1993), he 7 years later contended, based on the dramatic increase of results of empirical studies, that health and public health was probably one of the most important areas associated with and affected by social capital (Putnam, 2000). Kawachi, Kennedy, and Wilkinson (1999) suggested four main causal pathways by which social capital may affect health: direct psychological and psychosocial stress pathways, social norms and values which foster health-related behaviors, access to health care and amenities through social networks and contacts, and crime, particularly violent crime (Kawachi et al., 1999).

Kawachi, Kennedy, Lochner, and Prothrow-Stith (1997) found in an early ecological study that low levels of social capital were associated with a higher total mortality rate and higher rates of a wide range of major causes of death, including coronary heart disease (CHD), cerebrovascular disease, unintentional injury, and infant mortality (Kawachi et al., 1997). Later epidemiological, ecological, and individual level studies have, just to give some examples, revealed significant associations between low levels of social capital and higher age-adjusted mortality rates, shorter life expectancy, higher mortality and violent crime rates, higher coronary heart disease morbidity and mortality, low birth weight rates, higher incidence rates of a variety of sexually transmitted diseases, low life satisfaction, less happiness, poor self-rated health, poor mental health (measured with the GHQ12 index), poor physical and mental health measured by SF-12, depression, psychiatric morbidity, higher suicide rates, worse chronic conditions, functional limitations (Islam, Merlo, Kawachi, Lindström, & Gerdtham, 2006), and health-related behaviors such as lower levels of leisure time physical activity (Lindström, Hanson, & Östergren, 2001). Some studies have investigated associations between vertical social capital, i.e., trust and participation across a well-defined power gradient, and health (Sundquist, Johansson, Yang, & Sundquist, 2006). Multilevel studies, which mostly include the individual

level and one or more contextual levels of analysis, have e.g. shown that low social capital is associated with poor self-rated health and violent crime (Islam et al., 2006). Multilevel analyses have also demonstrated significant associations between social capital and access to health care (Lindström et al., 2006). Multilevel analyses have increasingly been conducted in order to disentangle the associations between contextual level social capital and individual level health in order to avoid the "ecological fallacy," which denotes the risk of drawing erroneous conclusions concerning individual health from observations of ecological data, and the "individualist fallacy," which denotes the risk of missing associations and effects of neighborhood or other forms of contextual social capital on individual health.

Since social capital is a characteristic of social relations, trust, and cooperation between individuals, groups, and organizations rather than of individuals, one important issue related to social capital and health is to define relevant social contexts for the analysis of the association and impact of contextual social capital on health and health-related phenomena such as health-related behaviors and access to health care and amenities. Most multilevel studies include neighborhoods or other geographic entities as second, third, and so forth levels of analysis. However, one question concerns whether geographic entities are the most relevant social contexts. This discussion is highly relevant because many multilevel studies have shown small or moderate associations between neighborhood and geographic social capital and health of individuals within single countries, while the statistical associations between social capital and health have been stronger and more consistent in individual level studies. Recently, some prospective cohort studies have investigated workplace social capital and found significant effects on, e.g., depression (Kouvanonen et al., 2008). In studies of social capital and school children and adolescents, the family and schools are relevant social contexts in addition to neighborhoods. Putnam suggested already in 2000 that the Internet would become a relevant area for studies of social capital and social networks (Putnam, 2000).

Most studies of social capital and health are cross-sectional, i.e., they measure all factors at the same point in time which makes causal inference formally impossible. In many instances, causality may go in both directions. Social capital may, e.g., cause poor mental health, but mental health may also affect social capital in the forms of social participation in social networks, civic participation, and feelings of trust and reciprocity. A few longitudinal studies with panel data including three waves of observations or more have been conducted in recent years (Giordano & Lindström, 2011).

Social capital is not always associated with better health. Szreter and Woolcock developed the concepts of bonding, bridging, and linking social capital. Bonding social capital denotes "trusting and cooperative relations between members of a network who see themselves as being similar in terms of their shared social identity," while bridging social capital refers to "relations of respect and mutuality between people who know they are not alike in some sociodemographic (or social identity) sense (differing by age, ethnic group, class, etc.)" (Szreter & Woolcock, 2004). While many social networks, associations, and organizations, e.g., youth organizations, sports clubs, and labor unions, manage to combine bonding and bridging social capital, some other, e.g., criminal networks, do not. This phenomenon of exclusion of outsiders and the rest of society is sometimes referred to as "the dark side of social capital," and its effects on health may be detrimental. Linking social capital refers to "norms of respect and networks of trusting relationships between people who are interacting across explicit, formal, or institutionalized power or authority gradients in society" (Szreter & Woolcock, 2004).

Critique against the research concerning social capital and health has been expressed by the so-called neo-materialists, who claim that it obscures underlying ideological, political, administrative, and economic determinants of health inequalities and other public health issues. The neo-materialists emphasize the importance of active governments, active welfare politics, and economic preconditions for the realization of public health programs (Navarro, 2004).

Cross-References

- ▶ Cross-Sectional Study
- ▶ Longitudinal Study
- ▶ Multi-level Analysis
- ▶ Social Cohesion
- ▶ Social Support

References and Readings

Bourdieu, P., & Wacquant, L. (1992). *Invitation to reflexive sociology*. Chicago: University of Chicago Press.

Coleman, J. S. (1990). *Foundations of Social Theory*. Princeton: Harvard University Press.

Giordano, G. N., & Lindström, M. (2011). Social capital and change in psychological health over time. *Social Science and Medicine, 72*, 1219–1227.

Islam, K., Merlo, J., Kawachi, I., Lindström, M., & Gerdtham, U. (2006). Social capital and health: Does egalitarianism matter? A literature review. *International Journal for Equity in Health, 5*, 3.

Kawachi, I., & Berkman, L. (2000). Social cohesion, social capital, and health. In L. Berkman & I. Kawachi (Eds.), *Social epidemiology* (pp. 174–190). Oxford: Oxford University Press.

Kawachi, I., Kennedy, B. P., Lochner, K., & Prothrow-Stith, D. (1997). Social capital, income inequality, and mortality. *American Journal of Public Health, 87*, 1491–1498.

Kawachi, I., Kennedy, B. P., & Wilkinson, R. G. (1999). Social capital and self-rated health: A contextual analysis. *American Journal of Public Health, 89*, 1187–1193.

Kouvanonen, A., Oksanen, T., Vahtera, J., Stafford, M., Wilkinson, R., Schneider, J., et al. (2008). Low workplace social capital as a predictor of depression. The Finnish public sector study. *American Journal of Epidemiology, 167*(10), 1143–1151.

Lindström, M., Axén, E., Lindström, C., Beckman, A., Moghaddassi, M., & Merlo, J. (2006). Social capital and access to a regular doctor: A multilevel analysis in southern Sweden. *Health Policy, 79*, 153–164.

Lindström, M., Hanson, B. S., & Östergren, P. O. (2001). Socioeconomic differences in leisure-time physical activity: The role of social participation and social capital in shaping health related behaviour. *Social Science and Medicine, 52*, 441–451.

Macinko, S., & Starfield, B. (2001). The utility of social capital in research on health determinants. *The Milbank Quarterly, 79*(3), 387–427.

Navarro, V. (2004). Commentary: Is social capital the solution or the problem? *International Journal of Epidemiology, 33*, 672–674.

Putnam, R. D. (1993). *Making democracy work. Civic traditions in modern Italy*. Princeton: Princeton University Press.

Putnam, R. D. (2000). *Bowling alone. The collapse and revival of American community*. New York/London: Simon and Schuster.

Sundquist, J., Johansson, S. E., Yang, M., Sundquist, J., Johansson, S. E., Yang, M., et al. (2006). Low linking social capital as a predictor of coronary heart disease in Sweden: A cohort study of 2.6 million people. *Social Science and Medicine, 62*, 954–963.

Szreter, S., & Woolcock, M. (2004). Health by association? Social capital, social theory, and the political economy of public health. *International Epidemiology, 33*, 650–667.

Social Circumstance

- ▶ Sociocultural

Social Class

G. David Batty
Department of Epidemiology and Public Health, University College London, London, UK

Synonyms

Socioeconomic position

Definition

Social class refers to particular social strata in society.

Social class can be measured at a personal or geographical level. For an individual, indicators include occupational social class, occupational prestige, educational attainment, household income, housing tenure, household amenities, and car ownership. For a geographical area, composite measures are often derived from the characteristics of residents in a defined location (Galobardes, Lynch, & Smith, 2007).

In the context of epidemiology, socioeconomic status has been most commonly related to

disease (particularly cardiovascular disease) and disease risk factors (particularly health behaviors such as smoking). Socioeconomic inequalities (variations) in health are essentially universal: with the exception of very few outcomes, poorer health is more common in poorer people. As such, reducing these differentials is a priority for many governments and health agencies.

References and Readings

Galobardes, B., Lynch, J., & Smith, G. D. (2007). Measuring socioeconomic position in health research. *British Medical Bulletin, 81–82*, 21–37.

Social Cohesion

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

Social cohesion (SC) refers to the degree to which links between a society's members are strong and the degree to which people in a community share values and goals and are interdependent. Durkehiem conceptualized SC as a major protective variable against adversities including suicide. In his seminal work, Durkheim found that Catholics have lower suicide rates than Protestants, and he attributed this to greater social control and cohesion in the former than among the latter (Pickering & Walford, 2000). Kelleher and Daly (1990) found that reduced SC (indexed by reduced marriage rates and increased separations) was among the variables possibly contributing to increased suicide rates in Ireland, between 1970 and 1985. The Dutch sociologist Geert Hofstede conducted the first empirically based quantification of international cultural dimensions in the 1970s in IBM plants around the world. Among his four main cultural dimensions, individualism was conceptualized, which can be seen as the opposite of SC. Individualism does predict on a global level present suicide rates. Recent studies have also tested the relationship between SC and various health outcomes, pertinent to behavioral medicine. Chaix, Lindström, Rosvall, and Merlo (2008) found in Sweden that low SC predicted myocardial infarctions, independent of important demographic variables. More recently, Clark et al. (2011) found that each one point increase in SC predicted a reduction of 53% in deaths attributed to stroke, independent of confounders. SC was assessed individually by six items reflecting social interactions and contacts with neighbors. One mechanism could be that more cohesive societies (such as religious kibbutzim in Israel vs. secular ones) also promote a healthier psychosocial profile – higher sense of coherence and lower hostility levels (Kark, Carmel, Sinnreich, Goldberger, & Friedlander, 1996). The concept of SC is thus one domain where concepts important in behavioral medicine and sociology may influence health at the more macro level of societies. This could have important implications for preventative medicine.

Cross-References

▶ Social Support
▶ Suicide Risk, Suicide Risk Factors

References and Readings

Chaix, B., Lindström, M., Rosvall, M., & Merlo, J. (2008). Neighborhood social interactions and risk of acute myocardial infarction. *Journal of Epidemiology and Community Health, 62*, 62–68.
Clark, C. J., Guo, H., Lunos, S., Aggarwal, N. T., Beck, T., Evans, D. A., et al. (2011). Neighborhood cohesion is associated with reduced risk of stroke mortality. *Stroke, 42*, 1212–1217.
Kark, J. D., Carmel, S., Sinnreich, R., Goldberger, N., & Friedlander, Y. (1996). Psychosocial factors among members of religious and secular kibbutzim. *Israel Journal of Medical Sciences, 32*, 185–194.
Kelleher, M. F., Daly, M. (1990). Suicide in Cork and Ireland. *British Medical Journal*, 157:533–538.
Pickering, W. S. F., & Walford, G. (Eds.) (2000). *Durkheim's suicide: a century of research and debate*. Psychology Press, London: Routledge.

Social Conflict

Orit Birnbaum-Weitzman
Department of Psychology, University of Miami, Miami, FL, USA

Synonyms

Interpersonal conflict; Negative social interaction; Relational distress; Relationship conflict; Social stress

Definition

Social conflict refers to the various types of negative social interactions that may occur within social relationships including, but not limited to, arguments, rejection, criticism, hostility, insensitivity, unwanted demands, and ridicule (Seeman, 2001). Social conflict can also escalate to include physical violence. Conflict refers to overt behavior rather than subjective states. It typically results from purposeful interaction among two parties in a competitive setting in which the parties are aware of the incompatibility of positions. Social conflict can extend to different social relationships including those with significant others such as spouses, family members, and friends, as well as less intimate relationships.

Description

Studies on social conflict and interpersonal stress suggest that negative aspects of social interactions including social conflict and social control are inversely related to emotional well-being and health (Miller, Chen, & Cole, 2009). There is substantial evidence that interactions marked by acute conflict and negative emotions have direct physiological consequences. Epidemiological studies have linked social isolation, low social support, and high levels of social conflict with morbidity and mortality (Miller et al., 2009). Changes in immune function and elevated inflammation have been suggested as key pathways underlying the association between social conflict and health (Kiecolt-Glaser, Gouin, & Hantsoo, 2010). Inflammation is a key pathogenic mechanism in many infections and cardiovascular and neoplastic diseases. Recent studies have also linked stressful interpersonal relationships to alterations in gene expression and intracellular signaling mechanisms (Miller et al., 2009).

Consistent evidence also points to the strong relationship between social conflict and psychological distress. Specifically, interpersonal stress and conflict with family and friends has been reliably associated with negative affect, depression, and emotional stress responses (Graham, Christian, & Kiecolt-Glaser, 2007). Depression has been proposed as a particularly important psychological mechanism by which social conflict in close relationships affect immune function (Miller et al., 2009). The literature indicates that social conflict is associated with a range of physiological and psychological mechanisms that are in turn associated with concomitant alterations in the cardiovascular, endocrine, and immune systems. Family conflict and discord and domestic violence have also been linked to suicidal behavior (Van Orden et al., 2010). In drug abusers, interpersonal conflict has been also associated with an increased probability of a history of suicide attempt and ideation (Van Orden et al., 2010).

Social conflict has been assessed using self-report questionnaires about relationship quality and quantity as well as in experimental studies of laboratory-induced marital conflict (Kiecolt-Glaser et al., 2010). To date, there is not one standard measure to assess social conflict. Different types of study designs have been used to assess the impact of social conflict on physical and emotional well-being including correlational studies, experimental studies of couples, and animal studies (see Kiecolt-Glaser et al., 2010). Correlational studies suggest that social isolation, lack of social support, and interpersonal conflict are associated with biological markers of inflammation (i.e., C-reactive protein and interleukin-6). More recent studies have also suggested

a plausible link between distressed pair-bond relationships and plasma levels of oxytocin in females and vasopressin in males. However, this research is still preliminary. In other correlation studies, men and women who had recently undergone a marital separation or divorce had poorer immune function than demographically matched married individuals (see Kiecolt-Glaser et al., 2010).

According to Kiecolt-Glaser, Glaser, Cacioppo, and Malarkey (1998), being married is not always protective, especially if there is frequent interpersonal conflict in the couple. In experimental studies with married couples, this group of researchers has shown that conflictive social interactions consistently result in heightened blood pressure and heart rate, especially for those with high trait hostility. Conflict discussion tasks are also widely used in marital research by this group and others. Discussion of marital problems has been associated with both immediate and longer term physiological changes related to the degree of negativity or hostility displayed during conflict (see Kiecolt-Glaser et al., 2010). Gender differences have been observed in these studies, with women evidencing greater sensitivity to negative marital interactions than men. Similarly, marital conflict shows a greater impact on health and physiological functioning in older adults compared to young couples. In general, this research suggests that relationships that are stable and long lasting but marked by social conflict have the potential to function as both an acute and chronic stressor that may impact health over an extensive period of time (Kiecolt-Glaser et al., 2010). Relationship conflict and termination can also provoke detrimental health behaviors including disturbed sleep, unhealthy diets, less physical activity, smoking, and greater use of alcohol and other drugs. Thus, relationships characterized by hostility and conflict could have negative health consequences.

The most common social conflict models involving laboratory animals are variations of the resident-intruder model wherein one animal is placed in the home cage of another. Animal studies have shown that social conflict and disruption of social relationships have important immunological and endocrine consequences (Huhman, 2006). In rodents, an aggressive social encounter is typically accompanied by elevated levels of stress hormones and changes in cellular and humoral immunity. Exposure to social conflict appears to have long-lasting behavioral and physiological effects not just in defeated/subordinate animals but also in dominant ones that appear to be moderated by developmental level (Huhman, 2006). Data from experimental primate models also show that stressful social relationships can exacerbate viral infections by altering gene expression responses to infection. Social conflict models in animals may be useful for studying the physiological concomitants of a number of psychiatric disorders including major depression (see Huhman, 2006).

Cross-References

- ▶ Family Stress
- ▶ Interpersonal Relationships
- ▶ Marriage and Health

References and Readings

Graham, J. E., Christian, L. M., & Kiecolt-Glaser, J. K. (2007). Close relationships and immunity. In R. Ader (Ed.), *Psychoneuroimmunology* (pp. 781–798). Burlington, MA: Elsevier Academic Press.

Huhman, K. L. (2006). Social conflict models: Can they inform us about human psychopathology? *Hormones and Behavior, 50*, 640–646.

Kiecolt-Glaser, J. K., Glaser, R., Cacioppo, J. T., & Malarkey, W. B. (1998). Marital stress: Immunologic, neuroendocrine and autonomic correlates. *Annuals of New York Academic Sciences, 840*, 656–663.

Kiecolt-Glaser, J. K., Gouin, J. P., & Hantsoo, L. (2010). Close relationships, inflammation, and health. *Neuroscience and Biobehavioral Reviews, 35*, 33–38.

Miller, G., Chen, E., & Cole, S. W. (2009). Health psychology: Developing biologically plausible models linking the social world and physical health. *Annual Review of Psychology, 60*, 501–524.

Seeman T. (2001). How do others get under our skin: Social relationships and health. In C.D. Ryff and Singer (Eds.), Emotion, Social Relationships and Health (pp. 189–209).

Van Orden, K. A., Witte, T. K., Cukrowicz, K. C., Braithwaite, S. R., Selby, E. A., & Joiner, T. E. (2010). The interpersonal theory of suicide. *Psychological Review, 117*, 575–600.

Social Determinants of Health

▶ Social Factors

Social Ecological Framework

▶ Ecological Models: Application to Physical Activity

Social Ecological Model

▶ Ecological Models: Application to Physical Activity

Social Epidemiology

G. David Batty
Department of Epidemiology and Public Health, University College London, London, UK

Definition

While epidemiology is defined as the study of the distribution and determinants of disease and typically treats social determinants as background to biomedical phenomena, social epidemiology is a sphere of enquiry in its own right. It is distinguished by explicitly investigating social determinants of population distributions of health, disease, and well-being. Social epidemiology was perhaps first coined as a discipline in the 1950s; post-graduate programmes in social epidemiology are now offered.

Cross-References

▶ Epidemiology
▶ Social Class
▶ Socioeconomic Status (SES)

References and Readings

Berkman, L., & Kawachi, I. (Eds.). (2000). *Social epidemiology*. New York: Oxford University Press.
Krieger, N. (2001). A glossary for social epidemiology. *Journal of Epidemiology & Community Health, 55*(10), 693–700.

Social Factors

Emily Kothe
School of Psychology, University of Sydney, Sydney, NSW, Australia

Synonyms

Social determinants of health; Socioeconomic status (SES)

Definition

The conditions under which people are born, live, work, and age are collectively known as the social determinants of health. These social factors include both economic and social conditions and are may be responsible for the health inequities both within and between countries.

Description

Research into the social factors underpinning health arose from the dual observations of health disparities both within and between countries. These disparities can be easily (although crudely) demonstrated by way of differences in life expectancy for different groups.

Between-country differences in adult mortality are growing rapidly. Whereas mortality rose in Africa, Central and Eastern Europe between 1970 and 2002, global mortality fell overall during the same period (World Health Organization, 2003). These differences in mortality rates are especially stark when comparing life expectancy by country. For example, while the average life

expectancy at birth is 89.78 years in Monaco, it is just 29.3 years in Haiti (CIA, 2011).

Large differences in morbidity and mortality are also apparent within the same country. For example, in Australia, the life expectancy for Aboriginal and Torres Strait Islanders is approximately 10 years below the national average (Australian Bureau of Statistics, 2010).

Study of the social factors that underlie health provides possible mechanisms by which this large health disparity can be understood. The major social factors which influence health include both structural determinants of health, and the conditions of an individual's daily life. Research linking social factors to health outcome has identified a number of key social determinants of health (e.g., Wilkinson & Marmot, 2003). These include:

- The social gradient
- Stress
- Early life
- Social exclusion
- Work and unemployment

These factors are thought to influence health both indirectly and directly. In many cases, social factors act in combination to have a cumulative impact on health. For example, while unemployment is known to have a direct influence on health, it is also likely to have an indirect influence on health through its influence on other social factors (e.g., stress).

The Social Gradient

It is widely recognized that poverty is related to high levels of illness and disease. Data consistently shows that the health status of individuals in the poorest countries leads to significantly lower life expectancy and significantly higher risk of disease than that of individuals in richer countries.

The influence of socioeconomic status is also apparent within countries and appears to have an influence on health across the socioeconomic spectrum. This effect, known as the social gradient, shows that even within industrialized countries, both mortality and morbidity are linearly distributed across different levels of socioeconomic advantage and disadvantage. This research suggests that even moderate differences in wealth, and associated social factors, can have an important impact on health status.

For more information, see ▶ Social Class.

Early Life

Maternal deprivation and ill-health during pregnancy have both been linked to poor fetal development. Factors such as malnutrition, maternal stress, and inadequate prenatal care all increase the risk of childhood mortality and have profound impacts on health throughout later life.

Research has also linked circumstances during infancy to both physical and mental health in adult life. Social functioning, physical health, and the performance of health-protective behaviors have all been linked to experiences in early life.

For more information, see ▶ Maternal Stress; ▶ Birth Weight; ▶ Child Neglect; ▶ Child Development; ▶ Stress, Early Life.

Stress

Social and psychological circumstance can have a profound impact on the number of stressors an individual is exposed to and the level of stress that they experience. Increased levels of stress have been linked to increased risk of both morbidity and mortality.

Research suggests that exposure to stressors can have both direct and indirect influences on health. Chronic stress can directly increase risk of disease through its influence on physiological processes such as immune function. However, both acute and chronic stress can also lead to high rates of health-damaging behaviors such as alcohol consumption and smoking.

For more information, see ▶ Stress; ▶ Stressor.

Social Exclusion

Social exclusion relates to the isolation of certain member within a society and is associated with high rates of disease and mortality. It is most often related to relative poverty, low educational attainment, unemployment, and experiences of stigma and discrimination.

Individuals who experience social exclusion are subject to a pattern of multiple deprivations

that prevent them from participating fully in society. This is often characterized by poor access to services, including health care, housing, education, and transport.

For more information, see ▶ Social Capital and Health; ▶ Stigma.

Work and Unemployment

Research suggests that work and unemployment have separate – but at times overlapping – influences on health.

In general, being unemployed places individuals at increased risk of a number of diseases (e.g., depression and heart disease). The effects of unemployment on health are linked to both material deprivation that may occur due to a lack of income, and to the psychological consequences of being unemployed. The influence of unemployment on health appears to manifest itself even before individuals become unemployed, such that experiencing high levels of job insecurity (in many cases, a precursor of unemployment) can be as harmful to health as being unemployed.

In addition to the role of perceived job security, it appears that there are a range of factors that determine the extent to which employment is health protective. These include:

- Level of job control
- Level of job demand
- Adequacy of rewards for job performance

In particular it appears that professions that are characterized by low control but high demand place workers at increased risk of ill-health. Importantly social support and recognition of job performance, either through higher wages or increased social status, both appear to be health protective.

For more information, see ▶ Work-Related Stress.

Social Causation and Social Drift: Explanation of Possible Causal Relationships Between Social Factors and Health

When attempting to understand the influence of social factors on health, it is important to consider the possible causal relationships between these social factors and health. Broadly speaking, there are two possible ways of understanding the causal relationship between these factors. These explanations are described below using the example of the social gradient (Morrison & Bennett, 2008).

The first explanation – called social causation – would suggest that low socioeconomic status is causally related to health problems (i.e., that there is something about low socioeconomic status that "causes" ill-health). For example, an individual who experiences being socioeconomically disadvantaged may be exposed to poor living conditions which cause later health problems.

The second explanation – called social drift – would suggest that ill-health is causally related to low socioeconomic status (i.e., that experiencing illness "causes" an individual to lose their socioeconomic position). For example, an individual who has to leave work due to illness may find that without a steady income, they are now socioeconomically disadvantaged.

Longitudinal studies provide evidence for both explanations. For example, studies have consistently shown that low socioeconomic status baseline is a predictor of heart disease at follow-up and that individuals who become unemployed are more likely to suffer from health complaints than individuals who stay employed. On the basis of this evidence, it would appear most likely that social factors and health have a bidirectional relationship (Morrison & Bennett, 2008).

Cross-References

▶ Birth Weight
▶ Child Development
▶ Child Neglect
▶ Health Care Access
▶ Maternal Stress
▶ Social Capital and Health
▶ Social Class
▶ Stigma
▶ Stress
▶ Stress, Early Life
▶ Stressor
▶ Work-Related Stress

References and Readings

Australian Bureau of Statistics. (2010). *Experimental life tables for Aboriginal and Torres Strait Islander Australians, 2005–2007 (Cat. No. 3302.0.55.003)*. Canberra: Author.

CIA. (2011). *The world factbook: Life expectancy at birth*. Retrieved November 18, 2010 from https://www.cia.gov/library/publications/the-world-factbook/rankorder/2102rank.html#

Marmot, M. (2005). Social determinants of health inequalities. *The Lancet, 365*(9464), 1099–1104.

Morrison, V., & Bennett, P. (2008). *An introduction to health psychology*. New York: Prentice Hall.

Wilkinson, R. G., & Marmot, M. G. (2003). *Social determinants of health: The solid facts*. Copenhagen: World Health Organization.

World Health Organization. (2003). *World health report 2003: Shaping the future*. Geneva: Author.

Social Health

▶ Racial Inequality in Economic and Social Well-Being

Social Inhibition

Johan Denollet
CoRPS – Center of Research on Psychology in Somatic diseases, Tilburg University, Tilburg, The Netherlands

Synonyms

Behavioral inhibition

Definition

Social inhibition (SI) is a broad personality trait that refers to *the stable tendency to inhibit the expression of emotions and behaviors in social interaction* (Asendorpf, 1993). Individuals who are high in SI are more likely to feel inhibited, tense, and insecure when with others. In children, the label *behavioral inhibition* is often used to describe this tendency (Gest, 1997).

SI is more closely related to the *interpersonal dimension* of introversion/extraversion than to intrapsychic facets of extraversion such as positive affect or excitement seeking. Infants and children with an inhibited temperament tend to develop into adults who avoid people and situations that are novel or unfamiliar (Schwartz, Wright, Shin, Kagan, & Rauch, 2003). Hence, individuals who are high in SI try to avoid potential "dangers" involved in social interaction, such as disapproval or criticism by others, through the deliberate inhibition of self-expression (Asendorpf, 1993). They tend to experience discomfort in encounters with other people, may keep other people at a distance, and are less likely to actively seek social support.

Interestingly, recent imaging research demonstrates the effect of social inhibition on the *neural coding of threatening signals* in the human brain (Kret, Denollet, Grèzes, & de Gelder, 2011). Socially inhibited adults may show greater signal response in the brain to threatening stimuli than adults who are not socially inhibited (Schwartz et al., 2003). Other research also indicates that socially inhibited people tend to overactivate a broad cortical network in the brain when looking at fearful or angry facial and bodily expressions (Kret et al., 2011).

There are several reasons why it is important to account for individual differences in SI in clinical research and practice. First, SI has been related to *difficulties in coping with the challenges of everyday life*. For example, inhibited children may show a delay in establishing a first stable partnership and finding a first full-time job in early adulthood (Asendorpf, Denissen, & van Aken, 2008). Second, socially inhibited individuals may seem quiet on the surface, while they may actually *avoid interpersonal conflict* through excessive control over their emotional and behavioral responses. Third, SI has been associated with an increased long-term *risk of developing internalizing problems* (Asendorpf et al., 2008), including anxiety disorders (Rapee, 2002) and other forms of distress (Gest, 1997) in adulthood.

SI may also increase the risk of physical health problems. Individuals who are high in SI display physiologic hyperreactivity to stress

(Cole, Kemeny, Fahey, Zack, & Naliboff, 2003), and the active inhibition of emotions induces increased cardiovascular reactivity (Gross & Levenson, 1997). In clinical research, SI has been associated with the progression of HIV (Cole et al., 2003) and with an increased risk of adverse cardiac events in patients with heart disease (Denollet et al., 2006).

SI can be reliably *assessed with the 7-item SI measure of the DS14* (Denollet, 2005), a scale that was specifically designed to assess this broad and stable tendency to inhibit the expression of emotions and behaviors in social interaction.

Cross-References

▶ Type D Personality

References and Readings

Asendorpf, J. B. (1993). Social inhibition: A general-developmental perspective. In H. C. Traue & J. W. Pennebaker (Eds.), *Emotion, inhibition, and health* (pp. 80–99). Seattle, WA: Hogrefe & Huber.

Asendorpf, J. B., Denissen, J. J., & van Aken, M. A. (2008). Inhibited and aggressive preschool children at 23 years of age: Personality and social transitions into adulthood. *Developmental Psychology, 44*, 997–1011.

Cole, S. W., Kemeny, M. E., Fahey, J. L., Zack, J. A., & Naliboff, B. D. (2003). Psychological risk factors for HIV pathogenesis: Mediation by the autonomic nervous system. *Biological Psychiatry, 54*, 1444–1456.

Denollet, J. (2005). DS14: Standard assessment of negative affectivity, social inhibition, and type D personality. *Psychosomatic Medicine, 67*, 89–97.

Denollet, J., Pedersen, S. S., Ong, A. T., Erdman, R. A., Serruys, P. W., & van Domburg, R. T. (2006). Social inhibition modulates the effect of negative emotions on cardiac prognosis following percutaneous coronary intervention in the drug-eluting stent era. *European Heart Journal, 27*, 171–177.

Gest, S. D. (1997). Behavioral inhibition: Stability and associations with adaptation from childhood to early adulthood. *Journal of Personality and Social Psychology, 72*, 467–475.

Gross, J. J., & Levenson, R. W. (1997). Hiding feelings: The acute effects of inhibiting negative and positive emotion. *Journal of Abnormal Psychology, 106*, 95–103.

Kret, M. E., Denollet, J., Grèzes, J., & de Gelder, B. (2011). The role of negative affectivity and social inhibition in perceiving social threat: An fMRI study. *Neuropsychologia, 49*, 1187–1193.

Rapee, R. M. (2002). The development and modification of temperamental risk for anxiety disorders: Prevention of a lifetime of anxiety? *Biological Psychiatry, 52*, 947–957.

Schwartz, C. E., Wright, C. I., Shin, L. M., Kagan, J., & Rauch, S. L. (2003). Inhibited and uninhibited infants "grown up": Adult amygdalar response to novelty. *Science, 300*, 1952–1953.

Social Integration

▶ Social Support

Social Isolation

▶ Loneliness

Social Marketing

Sara Mijares St. George and Dawn Wilson
Department of Psychology, University of South Carolina, Columbia, SC, USA

Synonyms

Cause marketing; Noncommercial advertising; Public interest advertising; Public service advertising

Definition

Social marketing is the application of marketing principles to non-tangible "products," including ideas, attitudes, and lifestyle changes. Unlike traditional marketing, the primary goal of social marketing is to improve public health, not to increase the marketer's profitability (Lefebvre & Flora, 1988). Specifically, it is a planning and intervention model that targets audiences with marketing technologies to improve health and quality of life (Andreasen, 1995). There are five

principles addressed in the "marketing mix" of social marketing, known as the "5 Ps:" product, price, place, promotion, and positioning. "Product" is the target behavior (e.g., breastfeeding, healthy eating). "Price" refers to the social, economic, and psychological costs involved in adopting the target behavior. "Place" is the setting, community context, or distribution channels for the product. "Promotion" includes the steps taken to make the audience aware of the ideas, behaviors, and their benefits and may also involve interpersonal communication, media messages, grassroots approaches, special events, and incentives. Lastly, "positioning" describes how the product is framed, namely, to maximize perceived benefits and minimize perceived costs.

Social marketing campaigns using mass media strategies have been effective in promoting positive health behavior outcomes. For example, the Stanford Five-City Project for Heart Disease Prevention, (Farquhar et al., 1985, 1990) which targeted specific audiences, developed a product (i.e., a 6-week quitting contest), and involved local television stations, resulted in a 30% increase in smoking cessation. Similarly, the VERB Campaign (Huhman et al., 2005; Wong et al., 2004) utilized mass media techniques such as advertising on television, on billboards, through a website, and through school- and community-based promotions to increase physical activity in ethnically diverse youth and their parents. While both of the aforementioned social marketing campaigns involved mass media strategies to promote their "products," social marketing campaigns that use interpersonal channels and target the social context may maximize success in promoting health behavior change.

Indeed, effective and sustainable social marketing campaigns are driven largely by social factors (e.g., social norms, social support) and involve the target population in the social marketing processes to increase the integration of the program into established community structures (Bryant, Forthofer, Landis, & McDermott, 2000). For example, Wilson and colleagues (2010) used a collaborative community process to develop a social marketing campaign which motivated citizens in a low-income, high-crime community to use a walking path. With the assistance of a communications firm, a community steering committee guided the development of the overall social marketing objectives and approach. One of five key objectives, including increasing perceptions of safety and social connectedness, was targeted each month using corresponding print materials (e.g., a 12-month calendar, matching door hangers). Through grassroots social networking, the program engaged residents to participate in walks with peers, allowing them to feel safe and connected to their neighbors. This study is an example of how involving constituents in the process of social marketing ensures that the approach is tailored and truly fits the needs of the target audience. Furthermore, social marketing campaigns that strategically provide opportunities for interactions between neighbors, friends, and families may influence social norms and support around a particular health behavior and foster a social climate of behavior change.

Cross-References

- ▶ Health Behaviors
- ▶ Health Communication
- ▶ Social Support

References and Readings

Andreasen, A. (1995). *Marketing social change: Changing behavior to promote health, social development, and the environment*. San Francisco: Jossey-Bass.

Bryant, C., Forthofer, M., Landis, D., & McDermott, R. (2000). Community-based prevention marketing: The next steps in disseminating behavior change. *American Journal of Health Behavior, 24*, 61–68.

Farquhar, J., Fortmann, S., Flora, J., Taylor, C., Haskell, W., Williams, P., et al. (1990). Effects of communitywide education on cardiovascular disease risk factors: The Stanford five-city project. *Journal of the American Medical Association, 264*(3), 359.

Farquhar, J., Fortmann, S., MacCoby, N., Haskell, W., Williams, P., Flora, J., et al. (1985). The Stanford five-city project: Design and methods. *American Journal of Epidemiology, 122*(2), 323.

Huhman, M., Potter, L., Wong, F., Banspach, S., Duke, J., & Heitzler, C. (2005). Effects of a mass media

campaign to increase physical activity among children: Year-1 results of the VERB campaign. *Pediatrics, 116*(2), e277.
Lefebvre, C., & Flora, J. (1988). Social marketing and public health intervention. *Health Education & Behavior, 15*(3), 299.
Wilson, D. K., Trumpeter, N. N., St. George, S. M., Coulon, S. M., Griffin, S., Van Horn, M. L., et al. (2010). An overview of the "positive action for today's health" (PATH) trial for increasing walking in low income, ethnic minority communities. *Contemporary Clinical Trials, 31*, 624–633.
Wong, F., Huhman, M., Asbury, L., Bretthauer-Mueller, R., McCarthy, S., Londe, P., et al. (2004). VERB™-a social marketing campaign to increase physical activity among youth. *Preventing Chronic Disease, 1*(3), 1–7.

Social Network

▶ Family, Relationships
▶ Family Social Support
▶ Social Support

Social Networks

▶ Social Relationships

Social Norms

▶ Norms

Social Pain

▶ Loneliness

Social Problem-Solving Therapy (SPST)

▶ Problem Solving

Social Processes

▶ Interpersonal Relationships

Social Relationships

Kristin J. August[1] and Karen S. Rook[2]
[1]Department of Psychology, Rutgers University, Camden, NJ, USA
[2]Department of Psychology & Social Behavior, University of California Irvine, Irvine, CA, USA

Synonyms

Interpersonal relationships; Social networks; Social ties

Definition

Broadly defined, social relationships refer to the connections that exist between people who have recurring interactions that are perceived by the participants to have personal meaning. This definition includes relationships between family members, friends, neighbors, coworkers, and other associates but excludes social contacts and interactions that are fleeting, incidental, or perceived to have limited significance (e.g., time-limited interactions with service providers or retail employees). Scientists interested in behavioral medicine often emphasize the informal social relationships that are important in a person's life, or the person's social network, rather than formal relationships, such as those with physicians, lawyers, or clergy. Relationship phenomena of interest to scientists encompass both the specific interactions that individuals experience with members of their social networks and the global perceptions of those interactions, which are shaped by past and current interactions with important social network members. The interactions that occur with social network members are often positive, and include the provision

of emotional and material support, companionship, and encouragement of health-enhancing behaviors. Interactions with social network members also can be negative, however, and can include insensitive, unresponsive, hurtful, or intrusive actions by others.

Description

A large body of evidence suggests that social relationships are associated with health. Research has linked social relationships to mortality and morbidity (Berkman, Glass, Brissette, & Seeman, 2000; Cohen, 2004; House, Landis, & Umberson, 1988). People with fewer social network ties have been found to have an elevated risk for a number of diseases, including cardiovascular disease and stroke, some forms of cancer, infectious disease, and possibly dementia (Cohen, 2004; Uchino, 2006). Social relationships also have been linked to the onset and progression of chronic illness, as well as illness adjustment, postsurgical recovery, disability, and survival (e.g., Seeman & Crimmins, 2001). Increased confidence in the associations between social relationships and health stems from the fact that the associations emerge not only in large, well-controlled cross-sectional and longitudinal epidemiological studies, but also in experimental studies of humans and animals. Moreover, the strength of these associations is impressive, as evidenced by research suggesting that the effects of social relationships on health are comparable in size to the effects of conventional risk factors, such as smoking (House et al., 1988).

Structural Versus Functional Aspects of Social Relationships

Both structural and functional aspects of social relationships have been distinguished (Berkman et al., 2000). Structural aspects refer to the existence and objective characteristics of social relationships, whereas functional aspects refer to the functions performed by and subjective qualities of social relationships (House, Umberson, & Landis, 1988). Structural characteristics of interest to health researchers include the size of a person's social network, the frequency of contact with social network members, and the nature of the role relationships with network members (e.g., family member, friend, coworker). Research has demonstrated that some structural aspects of social relationships, such as social network size and frequency of contact, are related to health. For example, having more social ties and more frequent social interaction has been found to be associated with lower risks for mortality and poor mental and physical health outcomes. Similarly, the marital relationship has been found to be especially consequential for health, relative to other role relationships. Married individuals, compared to unmarried individuals, have a lower prevalence and incidence of both mental and physical health problems and a lower mortality risk (House et al., 1988). Furthermore, research suggests that men may derive more health benefits from marriage than do women. Poor quality marriages, however, and the disruption associated with divorce or widowhood appear to be particularly deleterious to mental and physical health (Burman & Margolin, 1992).

Although numerous robust associations between structural aspects of social relationships and health have been documented, functional aspects also need to be examined in order to understand how and why social relationships impact health. The most commonly studied social network function that contributes to health is social support. A great deal of evidence suggests that social support can help to buffer people from the adverse effects of life stress (Cohen, 2004; Uchino, Cacioppo, & Kiecolt-Glaser, 1996). Different types of social support have been distinguished (emotional, instrumental, informational), all of which are conceptualized as ways that social network members provide each other with care and aid in times of need. Different types of support are viewed as being important in the context of different stressors, although evidence suggests that emotional support is important across a very broad range of stressors (Cohen & Wills, 1985).

Beyond social support, social relationships also serve as sources of companionship, which

provides opportunities for enjoyable interaction and camaraderie. The positive affect and relief from stress afforded by companionship, in turn, help to sustain health and well-being (Rook, 1987). Social network members also monitor each other's health behavior and intervene to discourage health-compromising behavior, leading researchers to be interested in the health effects of social regulation (or social control; Umberson, 1987).

The beneficial functions of social relationships also have been posited to have negative counterparts (Rook, 1998). Specifically, social relationships not only can provide support, companionship, and social regulation, but also can fail to provide support or can provide misguided support, can reject or neglect others, and can foster bad, rather than good, health practices. Even though such negative interactions with others are relatively rare, they can take a considerable toll on health and well-being (Rook, 1998).

Pathways by Which Social Relationships Influence Health

Understanding the pathways by which social relationships influence health is a key goal for health researchers. Three main pathways have been identified (Berkman et al., 2000; Rook, August, & Sorkin, 2010): The first pathway involves psychological processes and conditions associated with social relationships, such as positive and negative emotions, feelings of self-worth and self-efficacy, coping strategies, and depression. The second pathway involves physiological processes, or activation of bodily systems (endocrine, cardiovascular, and immune) in response to various kinds of social interactions. The third pathway involves health behaviors that are fostered by interactions with social network members, including health-enhancing behaviors (e.g., exercise) as well as health-compromising behaviors (e.g., smoking). All three of these pathways have the potential to have independent and joint effects on morbidity and, ultimately, mortality.

Pathways having beneficial health effects. Social support, companionship, and social regulation are believed to affect health through unique mechanisms. Social support is thought to dampen the emotional, physiological (neuroendocrine, cardiovascular, immune), and behavioral effects of stress by improving one's perceived ability to cope with stress (Uchino, 2006; Uchino et al., 1996). Companionship, on the other hand, is thought to influence health and well-being by enhancing positive affect and providing a respite from stress (Rook, 1987). Positive affect, in turn, has been linked to lower rates of morbidity, fewer symptoms and less pain from health conditions, and greater longevity (Pressman & Cohen, 2005). Companionship also may activate physiological processes, such as the release of oxytocin, a neuropeptide that helps to counter harmful stress responses, including release of the stress hormone, cortisol. Social control is believed to affect health through two primary, but opposing, processes (Hughes & Gove, 1981). Specifically, social control may discourage health-compromising behaviors and encourage health-enhancing behaviors, thereby contributing to better health and ultimately, a lower risk of mortality. Yet, at the same time, to the extent that social control involves constraints on others' behavior, it may provoke psychological distress, erode feelings of self-efficacy, and kindle relationship tensions. The psychological and relationship costs of social control may thus reduce or cancel the health benefits of social control, although the net effects of social control on health are not yet fully understood (Rook et al., 2010).

Pathways having detrimental health effects. Persistent conflict in social relationships, as well as the absence or loss of social relationships, also impact health through a number of mechanisms. Specifically, recurring strains and conflicts in social relationships lead to repeated activation of physiological systems (e.g., hypothalamic-pituitary-adrenal axis or sympathetic nervous system activity) and impaired immune functioning. These chronically activated and dysregulated physiological systems, in turn, may accelerate disease onset and progression. Additionally, social isolation and loneliness have been linked to negative emotions, chronic stress, cardiovascular

activation, low physical activity, and impaired sleep (Cacioppo et al., 2002). Finally, it is important to recognize that social network members sometimes encourage undesirable, rather than desirable, health practices. For example, evidence suggests that adolescents sometimes recruit their peers to use illegal substances or to engage in other risky health behaviors. Thus, conflict and tensions in social relationships, social isolation and loneliness, and undesirable social influence all can increase the risk of disease onset and progression.

Conclusion

It is well established that social relationships are important for health and well-being, and research has identified key aspects of social relationships that warrant consideration in efforts to understand these links with health. The psychological, physiological, and behavioral pathways by which social relationships affect health also are beginning to be understood. As this literature evolves and expands to document patterns that exist across different sociodemographic and lifespan contexts, it may help to inform interventions designed to strengthen social relationships and, in turn, health.

Cross-References

- ▶ Family and Medical Leave Act
- ▶ Family Assistance
- ▶ Family Planning
- ▶ Family Stress
- ▶ Family Studies (Genetics)
- ▶ Family Systems Theory
- ▶ Family Violence
- ▶ Family, Caregiver
- ▶ Family, Income
- ▶ Family, Relationships
- ▶ Family, Structure
- ▶ Loneliness
- ▶ Psychosocial Characteristics
- ▶ Psychosocial Factors
- ▶ Social Capital and Health
- ▶ Social Cohesion
- ▶ Social Conflict
- ▶ Social Factors
- ▶ Social Strain
- ▶ Social Stress
- ▶ Social Support

References and Readings

Berkman, L. F., Glass, T., Brissette, I., & Seeman, T. E. (2000). From social integration to health: Durkheim in the new millennium. *Social Science and Medicine, 51*, 843–857.

Burman, B., & Margolin, G. (1992). Analysis of the association between marital relationships and health problems: An interactional perspective. *Psychological Bulletin, 112*, 39–63.

Cacioppo, J. T., Hawkley, L. C., Crawford, L. E., Ernst, J. M., Burleson, M. H., Kowalewski, R. B., et al. (2002). Loneliness and health: Potential mechanisms. *Psychosomatic Medicine, 64*, 407–417.

Cohen, S. (2004). Social relationships and health. *American Psychologist, 59*, 676–684.

Cohen, S., & Wills, T. A. (1985). Stress, social support, and the buffering hypothesis. *Psychological Bulletin, 98*, 310–357.

House, J. S., Landis, K. R., & Umberson, D. (1988). Social relationships and health. *Science, 241*, 540–545.

House, J. S., Umberson, D., & Landis, K. R. (1988). Structures and processes of social support. *Annual Review of Sociology, 14*, 293–318.

Hughes, M., & Gove, W. R. (1981). Living alone, social integration, and mental health. *The American Journal of Sociology, 87*, 48–74.

Pressman, S. D., & Cohen, S. (2005). Does positive affect influence health? *Psychological Bulletin, 131*, 925–971.

Rook, K. S. (1987). Social support versus companionship: Effects on life stress, loneliness, and evaluations by others. *Journal of Personality and Social Psychology, 52*, 1132–1147.

Rook, K. S. (1998). Investigating the positive and negative sides of personal relationships: Through a lens darkly? In B. H. Spitzberg & W. R. Cupach (Eds.), *The dark side of close relationships* (pp. 369–393). Mahwah, NJ: Lawrence Erlbaum.

Rook, K. S., August, K. J., & Sorkin, D. H. (2010). Social network functions and health. In R. J. Contrada & A. Baum (Eds.), *The handbook of stress science: Biology, psychology, and health* (pp. 123–136). New York: Springer.

Seeman, T. E., & Crimmins, E. (2001). Social environment effects on health and aging. Integrating epidemiologic and demographic approaches and perspectives. *Annals of the New York Academy of Sciences, 954*, 88–117.

Uchino, B. N. (2006). Social support and health: A review of physiological processes potentially underlying links

to disease outcomes. *Journal of Behavioral Medicine, 29*, 377–387.
Uchino, B. N., Cacioppo, J. T., & Kiecolt-Glaser, J. K. (1996). The relationship between social support and physiological processes: A review with emphasis on underlying mechanisms and implications for health. *Psychological Bulletin, 119*, 488–531.
Umberson, D. (1987). Family status and health behaviors: Social control as a dimension of social integration. *Journal of Health and Social Behavior, 28*, 306–319.

Social Resources

▶ Family Social Support
▶ Social Support

Social Strain

▶ Social Support

Social Stress

Vanessa Juth and Sally Dickerson
Department of Psychology and Social Behavior, University of California, Irvine, Irvine, CA, USA

Synonyms

Interpersonal stress or conflict; Societal stress

Definition

Social stress can be broadly defined as a situation which threatens one's relationships, esteem, or sense of belonging within a dyad, group, or larger social context. Social stress can emerge in a number of situations. Social stress can stem from difficult social interactions, for example, a conflictual or tumultuous marital or family relationship (Kiecolt-Glaser, Gouin, & Hantsoo, 2010). Social stress can also emerge in the context of evaluated performance situations, where others could be judgmental or critical, or in contexts in which one feels rejected, ostracized, or ignored (Dickerson & Kemeny, 2004). Social stress can also be more broadly construed, representing perceptions of one's lower role or standing within a group or community.

Social stress can lead to a range of observable and measurable responses related to health outcomes (Miller, Chen, & Cole, 2009). In some cases, social stress can cause increases in negative affect and distress. It can also elicit specific negative emotions. For example, rejection or social-evaluative performance stressors can elicit increases in self-conscious emotions, such as shame and embarrassment. Severe interpersonal stressors (e.g., parental neglect) may lead to pathological forms of psychological distress, such as posttraumatic stress disorder.

By nature, social stress can range from being acute and relatively benign (e.g., delivering a speech) to chronic and severe (e.g., abusive relationship). Acute social stressors can lead to physiological responses. For example, social-evaluative performance stressors can elicit strong and significant increases in cortisol, as well as increases in cardiovascular activity (e.g., heart rate, blood pressure). Acute instances of rejection or conflict can also lead to changes in a variety of physiological parameters, including how the immune system functions. Chronic social stress can have implications for health. If social stressors are experienced repeatedly or consistently, stress-responsive physiological systems could be repeatedly activated, which could have negative health consequences; for example, high cortisol levels and immune dysregulation have predicted the onset and progression of different diseases. Additionally, those with chronic diseases (e.g., HIV, rheumatoid arthritis) who experience social stressors have shown faster disease progression than those without (Lepore & Revenson, 2007). Social stressors can also have long-lasting effects, such that social stress experienced early in life (e.g., childhood) can influence stress reactivity and the regulation of stress-responsive systems in adulthood. Furthermore, sociodemographic variables related to social status within a group, community, or

society at large (e.g., socioeconomic status, ethnicity) can increase the risk for negative health outcomes and/or behaviors.

Cross-References

- ▶ Biobehavioral Mechanisms
- ▶ Psychosocial Factors
- ▶ Social Class
- ▶ Social Conflict
- ▶ Social Relationships
- ▶ Social Support
- ▶ Stress Reactivity

References and Readings

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130*, 355–391.

Kiecolt-Glaser, J. K., Gouin, J. P., & Hantsoo, L. (2010). Close relationships, inflammation, and health. *Neuroscience and Biobehavioral Reviews, 35*, 33–38.

Lepore, S., & Revenson, T. (2007). Social constraints on disclosure and adjustment to cancer. *Social and Personality Psychology Compass, 1*, 313–333.

Miller, G., Chen, E., & Cole, S. W. (2009). Health psychology: Developing biologically plausible models linking the social world and physical health. *Annual Review of Psychology, 60*, 501–524.

Social Support

John Ruiz, Courtney C. Prather and
Erin E. Kauffman
Department of Psychology,
University of North Texas, Denton, TX, USA

Synonyms

Moderators/moderating factors; Social integration; Social network; Social resources

Definition

Social support refers to the belief that one is valued, cared for, and loved by others in a social network. Social support can generally be conceptualized as reflecting two broad factors (Cohen & Syme, 1985; Cohen & Wills, 1985). Structural support reflects properties of the social network and the degree to which a person participates in that network (i.e., social integration). Functional support refers to the ways by which network members aid the individual through tangible assistance or through psychological and emotional buffering. Social support is among the most widely studied and robust psychosocial moderators of health. This entry will expand upon this description, review evidence linking it to health, examine hypothesized mechanisms, and discuss future directions.

Description

Social support is a multicomponent construct reflecting the size, quality, and availability of one's social resources to moderate stress. Social support should be distinguished from conceptually related terms. *Social capital* is a sociological term referring to stock or stored social credit. *Social network* refers to the social collectives or groups to which a person may belong. *Social integration* refers to the degree to which a person is embedded in or a part of a social network. *Relationship* is a descriptor often used to characterize a specific type of social tie.

Social support is generally conceptualized as having structural (i.e., size of the social network, degree of social integration) and functional (i.e., support processes; support received versus support perceived) characteristics. In addition, the quality of support is increasingly recognized as an important moderator of the relationship between support and health. Importantly, relationships are not uniformly positive, with some acting as sources of stress (Uchino, 2004).

Measures

Social support is generally measured by self-report. Data sources may include demographic data from hospital, census, or other records; interviews; psychometrically validated instruments; and ad hoc items and measures.

Structural Measures of Support

The simplest of the social integration measures is marital status which is consistently identified as a protective factor in large, prospective studies of mortality. Marital status is a particularly attractive measure as it is a common demographic characteristic and, thus, available for analysis with many kinds of health data. Many researchers are also interested in measuring the size of one's social networks and the degree to which a person is embedded in the networks. One well-validated multicomponent measure of social integration is the Social Network Index (SNI: Berkman & Syme, 1979) which provides an aggregate score of social integration by sampling activity in multiple relationships. Lower SNI scores are predictive of a nearly two fold increase in mortality risk. Finally, the literature is replete with numerous ad hoc structural support measures including measures of number of networks, network size, as well as the number of specific kinds of relationships such as friendships, and others.

Functional Measures of Support

Functional measures can be classified into support received and support perceived. Interestingly, measures of received and perceived support tend not to be highly correlated. Measures of received supportive behavior allow assessment of the actual social resources available to an individual. For example, the Inventory of Socially Supportive Behaviors (ISSB: Barrera, Sandler, & Ramsay, 1981) is a 40-item structural support measure assessing the frequency with which one receives a variety of supportive actions. Measures of perceived social support assess an individual's beliefs about the social resources available to them. Perceived support measures generally consist of two factors: beliefs about support available, and satisfaction with level of perceived support. A widely used example of a perceived support measure is Cohen and Wills (1985) Interpersonal Support Evaluation List (ISEL) which assesses perceptions of belonging, tangible support, support appraisals, and confidence in support perceptions.

Quality of Support

Relationship quality has generally been assessed along a single dimension ranging from positive to negative. However, emerging models hypothesize that relationships can have both positive and negative characteristics. The Quality of Relationship Inventory (QRI: Pierce, Baldwin, & Lydon, 1997) is among the most popular multidimensional measures. The 25-item, self-report measure yields discrete support and conflict scales as well as a total quality rating. Several authors have suggested that the relationship quality dimensions of positivity and negativity are orthogonal, yielding four basic relationship types: (1) high positive, low negative (i.e., supportive relationship), (2) low positive, high negative (i.e., conflictual relationship), (3) high positive, high negative (i.e., ambivalent relationship), and (4) low positive, low negative (i.e., benign/irrelevant relationship). The Social Relationships Index (SRI: Uchino, 2004) is an example of this multidimensional relationship quality assessment approach. Although health outcome data is limited, this conceptualization appears to be gaining acceptance as a more theoretically sound approach.

Social Support and Health

Social support is among the most robust predictors of disease and all-cause mortality. For example, a recent meta-analysis of 148 studies estimated the effect of social relationships to improve survival rates by 50% (Holt-Lunstad, Smith, & Layton, 2010). This effect varied by measurement, with multidimensional measures of social integration associated with more than 90% increase in survival rates. As noted by the authors, the magnitude of the effect of social support on mortality is likely diluted in these studies by unmeasured negative aspects of social relationships (e.g., a conflictual marriage). The results of this meta-analysis extend the work of previous systematic reviews and meta-analyses in identifying that social support is a substantial health and disease moderator with effects equivalent to more traditionally acknowledged risk factors such as cigarette smoking and obesity.

Cardiovascular Disease

The strongest evidence for a protective role of social support on health comes from studies of cardiovascular diseases such as coronary heart disease (CHD) morbidity and mortality (Smith & Ruiz, 2002). Multiple prospective studies of initially healthy samples have demonstrated that lower social integration and social support are associated with greater CHD incidence, faster disease progression, risk of myocardial infarction (MI), and greater risk for all-cause mortality. Among persons with diagnosed CHD, lower social support is predictive of significantly greater risk of recurrent MI and death. For example, Welin, Lappas, and Wilhelmsen (2000) found an almost three-fold increased chance of cardiac mortality in post-MI patients with low perceived emotional support at 10-year follow-up. A similar effect size was found by Berkman, Leo-Summers, and Horwitz (1992). Some studies indicate that low social integration is only associated with survival in the most severely isolated of the population. In a review of the literature, Mookadam and Arthur (2004) estimated a two to three times increased risk of 1-year mortality in the socially isolated population, with little health benefits resulting from support systems above this threshold. Moreover, there is some evidence that the beneficial effects of social support on cardiac health are stronger among women and that functional measures of support are more strongly related to cardiac disease compared to structural measures.

Effect sizes for social integration in healthy samples for future development of CHD are comparable to those of more traditional risk factors such as cigarette smoking (Orth-Gomer, Rosengren, & Wilhelmsen, 1993). Similarly, social integration is as strong a predictor of mortality among clinical populations as traditional risk factors such as cholesterol level, tobacco use, and hypertension in the patient population (Mookadam & Arthur, 2004). An important conceptual issue is whether high social support connotes a cardiovascular benefit akin to physical activity or whether it is simply the absence of support that is relevant. In addition, the relative value of structural versus functional support remains an open question. Regardless, the cumulative evidence indicates social support is an important moderator of cardiovascular risk.

Cancer

The relationship between social support and cancer is quite mixed. A recent systematic review of the prospective longitudinal literature concluded that in the context of breast cancer, greater social support was associated with slower disease progression in five of seven well-designed studies. Structural indices were the more commonly used and significant measure of social support in these studies. In contrast, there were no associations between measures of social support and other types of cancer with the paradoxical exception of more social support related to faster cancer progression in a sample of colorectal patients (Villingshoj, Ross, Thomson, & Johansen 2006). A meta-analysis of 87 studies estimated the relative risk of perceived social support and measures of social network size on mortality among cancer patients to be .82 and .80, respectively, suggesting a beneficial effect (Pinquart & Duberstein, 2010). Inconsistencies between studies may be partially explained by differences in support types measured and patient needs. The type of support associated with improved prognosis appears to vary by cancer site, with perceived social support representing a stronger predictor for leukemia and lymphoma, whereas breast cancer patients benefit more from have a large number of social ties (Pinquart & Duberstein).

In contrast to the mixed findings regarding physical outcomes, a robust literature supports the beneficial effects of social support on emotional and psychological reactions to cancer and associated treatments. Moreover, cancer is often a shared interpersonal experience – affecting supports as well as patients. A meta-analysis of these *contagion effects* estimated the correlation between patient and caregiver distress is .35 (Hodges, Humphris and Macfarlane, 2005). Importantly, partner response to illness affects patient adjustment, particularly in terms of quality of life. The type of coping employed (e.g., positive versus negative), relationship maintenance

behaviors, and the amount of communication about the relationship may also be important moderators of couples adjustment.

With respect to interventions, efforts have largely focused on increasing the patient's social network size and opportunity for emotional support. Perhaps the most well known of these interventions was conducted by Spiegel, Bloom, Kraemer, and Gottheil (1989). Women with late-stage metastatic breast cancer were randomized to either supportive group therapy or wait-list control. Findings that women in the treatment condition survived approximately 18 months longer generated interest in the possible healing role of support. Several clinical and prospective trials have failed to replicate these results, fueling doubts about the potential physical benefits of the approach. Regardless, there is substantial effort to translate socially based interventions from bench to bedside where their emotional and quality of life benefits are well recognized.

HIV/AIDS

Conclusions regarding the benefits of social support on physical outcomes in the context of HIV/AIDS are limited by the small number of published studies. A prospective study of HIV-positive men with hemophilia demonstrated that lower levels of perceived support at baseline were predictive of faster decreases in CD4 T-lymphocyte levels, a key marker of AIDS status, over a 4-year follow-up (Theorell et al. 1995). Leserman and colleagues conducted a study of 82 asymptomatic gay men, in which greater satisfaction with social support was associated with slower progression to AIDS, regardless of network size (Leserman et al. 1999). In contrast, other studies have found perceived support to be unrelated to progression to AIDS. More research is needed to adequately evaluate these relationships.

The social environment of the HIV/AIDS population may be uniquely important because of the social stigma associated with the diagnosis (Herek & Glunt, 1988). Stigma is associated with both personal distress and hesitancy to self-disclose status to sexual partners, potentiating further transmission of the infection either by the individual or the individual's un-informed sexual partners. A meta-analysis of 21 studies showed that greater perceived social support was associated with increased likelihood of self-disclosing one's HIV-positive status (Smith, Rosetto, & Peterson, 2008). Disclosure of HIV status is associated with increased social support within those relationships, indicating that disclosure may be a positive method of eliciting support from others.

Mechanisms

Social support is hypothesized to affect health and well-being through two pathways. The *main effects* hypothesis suggests that having more social resources reduces the chances of exposure to stressful circumstances or the magnitude of threat associated with certain environments. For example, one is likely to be safer walking at night with a large group of friends than when walking alone. Numerous studies have shown that having more social resources (measured as the number of social ties, the degree of social integration, etc.) is predictive of lower disease incidence, better survival following illness, lower all-cause mortality, and greater longevity irrespective of the quality of those relationships. Interestingly, these social resources need not be human to have a beneficial effect. Several studies have demonstrated that having a loving pet is associated with less stress and better survival following disease incidents such as a heart attack.

Social support may also affect health by moderating or reducing the impact of stressful circumstances (i.e., *stress buffering hypothesis*). After a stressful romantic breakup, a friend may comfort you with a hug, by providing you with an opportunity to vent your emotions, or by introducing you to someone new. Substantial laboratory data demonstrates that provision of support reduces self-reported stress and acute physiological responses to lab stressors. For example, individuals who received a note communicating emotional support from a supportive friend experience less blood pressure increase during a subsequent speech task relative to those who receive a note from a less supportive person (Uno, Uchino, & Smith, 2002). Importantly, the perception of support appears to

be more important than the actual provision of support. For example, imagining a supportive tie prior to a stressor results in less cardiovascular reactivity and less self-reported stress (i.e., buffering) compared to imagining an acquaintance (Smith, Ruiz, & Uchino, 2004). These findings support the idea that social support, received or perceived, can reduce stressful experiences.

Future Directions

Future research will continue to expand upon conceptual distinctions regarding sources of support and related actions. Longitudinal research is also needed to understand the biobehavioral mechanisms by which social support translates into disease risk. Further, more research is needed to determine whether it is better to have substantial support or simply to not be alone. Finally, emerging social phenomenon such as texting and online social networking through Facebook, Twitter, and other forums presents new challenges for researchers to conceptualize, measure, and gauge as moderators of health.

Cross-References

- ▶ Psychosocial Factors
- ▶ Psychosocial Predictors
- ▶ Psychosocial Variables
- ▶ Social Capital and Health
- ▶ Social Cohesion
- ▶ Social Conflict
- ▶ Social Factors
- ▶ Social Relationships
- ▶ Social Strain

References and Readings

Barrera, M., Sandler, I. N., & Ramsay, T. B. (1981). Preliminary development of a scale of social support: Studies on college students. *American Journal of Community Psychology, 9*(4), 435–447.

Berkman, L. F., Leo-Summers, L., & Horwitz, R. I. (1992). Emotional support and survival after myocardial infarction. A prospective, population-based study of the elderly. *Annals of Internal Medicine, 117*(12), 1003–1009.

Berkman, L. F., & Syme, S. L. (1979). Social networks, host resistance, and mortality: A nine-year follow-up study of Alameda County residents. *American Journal of Epidemiology, 109*(2), 186–204.

Cohen, S., & Syme, S. L. (1985). Issues in the study and application of social support. In S. Cohen & S. L. Syme (Eds.), *Social Support and health* (pp. 3–22). San Francisco: Academic Press Inc.

Cohen, S., & Wills, T. A. (1985). Stress, social support, and the buffering hypothesis. *Psychological Bulletin, 98*(2), 310–357.

Herek, G. M., & Glunt, E. K. (1988). An epidemic of stigma: Public reactions to AIDS. *American Psychologist, 43*(11), 886–891.

Hodges, L. J., Humphris, G. M., & Macfarlane, G. (2005). A meta-analytic investigation of the relationship between the psychological distress of cancer patients and their carers. *Social Science & Medicine, 60*(1), 1–12. 1982.

Holt-Lunstad, J., Smith, T. B., & Layton, J. B. (2010). Social relationships and mortality risk: A meta-analytic review. *PLoS Medicine, 7*(7), e1000316–e1000316.

Leserman, J., Jackson, E. D., Petitto, J. M., Golden, R. N., Silva, S. G., Perkins, D. O., et al. (1999). Progression to AIDS: The effects of stress, depressive symptoms, and social support. *Psychosomatic Medicine, 61*(3), 397–406.

Mookadam, F., & Arthur, H. M. (2004). Social support and its relationship to morbidity and mortality after acute myocardial infarction: Systematic overview. *Archives of Internal Medicine, 164*(14), 1514–1518.

Orth-Gomer, K., Rosengren, A., & Wilhelmsen, L. (1993). Lack of social support and incidence of coronary heart disease in middle-aged Swedish men. *Psychosomatic Medicine*, *55*, 37–43.

Pierce, T., Baldwin, M. W., & Lydon, J. E. (1997). A relational schema approach to social support. In G. Pierce, Lakey, Sarason, & Sarason (Eds.), *Sourcebook of social support and personality* (pp.19–47). New York: Plenum Press.

Pierce, G. R., Sarason, I. G., Sarason, B. R., & Solky-Butzel, J. A. (1997). Assessing the quality of personal relationships. *Journal of Social and Personal Relationships, 14*(3), 339–356.

Pinquart, M., & Duberstein, P. R. (2010). Associations of social networks with cancer mortality: A meta-analysis. *Critical Reviews in Oncology/Hematology, 75*(2), 122–137.

Smith, R., Rossetto, K., & Peterson, B. L. (2008). A meta-analysis of disclosure of one's HIV-positive status, stigma and social support. *AIDS Care, 20*(10), 1266–1275.

Smith, T. W., & Ruiz, J. M. (2002). Psychosocial influences on the development and course of coronary heart disease: Current status and implications for research and practice. *Journal of Consulting and Clinical Psychology, 70*(3), 548–568.

Smith, T. W., Ruiz, J. M., & Uchino, B. N. (2004). Mental activation of supportive ties, hostility, and cardiovascular reactivity to laboratory stress in young men and women. *Health Psychology, 23*(5), 476–485.

Spiegel, D., Bloom, J. R., Kraemer, H. C., & Gottheil, E. (1989). Effect of psychosocial treatment on survival of patients with metastatic breast cancer. *Lancet, 2*(8668), 888–891.

Theorell, T., Blomkvist, V., Jonsson, H., Schulman, S., Berntorp, E., & Stigendal, L. (1995). Social support and the development of immune function in human immunodeficiency virus infection. *Psychosomatic Medicine, 57*(1), 32–36.

Uchino, B. N. (2004). *Social support and physical health: Understanding the health consequences of relationships*. New Haven, CT: Yale University Press.

Uno, D., Uchino, B. N., & Smith, T. W. (2002). Relationship quality moderates the effect of social support given by close friends on cardiovascular reactivity in women. *International Journal of Behavioral Medicine, 9*, 243–262.

Villingshoj, M., Ross, L., Thomsen, B. L., & Johansen, C. (2006). Does marital status and altered contact with the social network predict colorectal cancer survival? *European Journal of Cancer (Oxford, England: 1990), 42*(17), 3022–3027.

Welin, C., Lappas, G., & Wilhelmsen, L. (2000). Independent importance of psychosocial factors for prognosis after myocardial infarction. *Journal of Internal Medicine, 247*(6), 629–639.

Social Support at Work

► Job Satisfaction/Dissatisfaction

Social Ties

► Social Relationships

Societal Stress

► Social Stress

Society of Behavioral Medicine

Stephanie Ann Hooker
Department of Psychology, University of Colorado, Denver, CO, USA

Synonyms

SBM

Definition

The Society of Behavioral Medicine (SBM) is a nonprofit organization founded in 1978. The organization strives to be multidisciplinary in nature, creating a dialogue between nursing, public health, psychological, and medical professionals to promote the study of interactions between behavior, biology, and the environment and to apply that knowledge to improve the health and well-being of individuals and communities. This is further illustrated through SBM's vision statement, which is "Better Health Through Behavior Change." In 2011, over 2,000 behavioral and biomedical researchers and clinicians were members of SBM.

SBM hosts an annual meeting for its members and other behavioral medicine researchers and clinicians to share recent research findings and clinical strategies. SBM also sponsors two journals, *Annals of Behavioral Medicine* and *Translational Behavioral Medicine: Practice, Policy, and Research*.

References and Readings

Society of Behavioral Medicine. (2011). Society of Behavioral Medicine (SBM). Retrieved July 15, 2011, from http://www.sbm.org

Sociocultural

Patricia Gonzalez[1] and Orit Birnbaum-Weitzman[2]
[1]Institute for Behavioral and Community Health (IBACH), Graduate School of Public Health, San Diego State University, San Diego, CA, USA
[2]Department of Psychology, University of Miami, Miami, FL, USA

Synonyms

Social circumstance; Sociocultural context; Sociocultural factors; Socioeconomic position; Socioeconomic status (SES)

Definition

"Sociocultural" refers to a wide array of societal and cultural influences that impact thoughts, feelings, behaviors, and ultimately health outcomes. Sociocultural determinants of health and illness encompass socioeconomic status (SES) factors (traditionally assessed by income, education, occupation) and cultural factors. There are several dimensions encompassed by the term, which can include race, ethnicity, ethnic identity, sex, acculturation, language, beliefs and value systems, attitudes, and religion.

Description

Sociocultural factors are salient determinants of health and have been found to be associated with a multitude of health outcomes including health behaviors (e.g., physical activity, diet, health screenings, and health-care utilization) and illness (e.g., cancer, diabetes, cardiovascular disease, and depression). Sociocultural factors are complex and may vary by sex, age, and racial/ethnic groups. In recent years, the term sociocultural has been extensively used in the literature in connection with physical and mental health outcomes.

Social and cultural factors play a central role in preventing illness, maintaining good health, and treating disease. Research has shown that an individual's social environment, family, neighborhood, school, and workplace have a significant impact on health. At the same time, cultural factors influence how physical and mental illness are viewed and diagnosed. A great advance in understanding the determinants of health and disease has been the identification of social and cultural factors influencing them. Social and cultural factors are pertinent not only to understanding individuals' health status but also recognizing the existing health disparities among different populations. In particular, a substantial body of research suggests that social factors stand at the root of health disparities (Marmot, 2005).

Some of the most salient sociocultural factors studied in relation to health disparities, including morbidity and mortality, are SES and race/ethnicity. Levels of health within the USA vary dramatically among different social, economic, and racial/ethnic groups. Moreover, considerable research suggests that determinants of health often reflect economic disparities (Braveman, Cubbin, Egerter, Williams, & Pamuk, 2010). Higher income levels are linked with overall better health, including self-rated health, lower cardiovascular risk factors, and lower mortality (Braveman, Egerter, & Mockenhaupt, 2011; Hajat, Kaufman, Rose, Siddiqi, & Thomas, 2011). The incidence and prevalence of many diseases (e.g., cardiovascular disease, arthritis, diabetes, and cervical cancer) increases as SES decreases. In addition, SES differences in mortality have been observed for many causes of death including some cancers, diabetes, and cardiovascular disease. Similarly, individuals with higher SES have greater life expectancy rates than individuals with lower income levels (Braveman et al., 2011). In terms of health status, adults with lower incomes are more likely to report their health status as poor or fair compared with adults with higher incomes (Braveman et al., 2011).

Several explanations have been proposed to account for the association between socioeconomic standing and health. First, economic stability enables individuals to live in safer neighborhoods, access healthier food alternatives, have more leisure time for physical activity, and endure less stress. Second, income impacts access to high-quality health care such that lower SES individuals are less likely to be covered by health insurance and to receive high-quality health care (Braveman et al., 2011). Hence, the uninsured may have less access to preventive services (e.g., health screenings) and early diagnosis. Greater education is also linked with longer life expectancy. Individuals who have completed college have a greater life expectancy (at least 5 years longer) than individuals who have not completed high school (Braveman et al., 2011). Higher education levels are associated with greater knowledge regarding health and feelings of control (Braveman et al., 2011) over different domains of one's life.

Therefore, education increases the likelihood that individuals will have the knowledge to prevent illness. As illustrated by these health patterns, SES disparities in health mirror a gradient pattern, with greater social and economic advantage being associated with better health.

Differences in health have also been observed based on race/ethnicity. For example, compared to non-Hispanic Whites, morbidity and mortality rates for cardiovascular disease (CVD) are higher among African-Americans (Payne et al., 2005). Compared to non-Hispanic Whites, African-Americans and Hispanics are more likely to have diabetes (Centers for Disease Control and Prevention, 2011). Moreover, ethnic-minority and low-income groups have a disproportionate burden of death and disability as a result of cardiovascular disease. In addition, although significant progress has been made in reducing cancer mortality rates in the USA, decreases in cancer mortality rates in ethnic minorities have been slower compared to non-Hispanic Whites (Cancer Facts & Figures, 2011).

Culture refers to the shared values, beliefs, and norms held in common by a defined group of people. Within each culture, there is a set of behaviors and values related to health and illness which may vary between different groups, causing differing viewpoints toward illness. Each culture has a set of norms for behavior with related beliefs, knowledge, and customs. Acculturation, a related cultural construct, is often used to explain ethnic disparities in health outcomes. Acculturation as a predictive variable is based on the premise that culturally based knowledge, attitudes, and beliefs influence people to behave in particular ways and to select specific health choices. For limited English proficiency (LEP) individuals, language barriers can contribute to health disparities. For example, LEP individuals may encounter difficulties communicating with medical professionals, understanding printed health information or accessing health-related services due to lack of information about available services (Racial and Ethnic Disparities in Health Care, 2010). Moreover, some individuals may fear jeopardizing their immigration status by using health services. Research also suggests that cultural norms within the USA or Western society contribute to lifestyles and behaviors associated with risk factors for diseases (e.g., cancer, diabetes, cardiovascular disease) (Thomas, Fine, & Ibrahim, 2004). Therefore, health behavior interventions must address the target group's belief systems as well as cultural values.

Although the US population is diverse, health policies and interventions are often based on Western cultural assumptions. Often, minimal attention is given to aspects of culture from the perspective of individuals from diverse ethnic or SES membership groups. It is key to acknowledge that social and cultural factors may explain related health behaviors and, in part, elucidate disparities between ethnic/racial and SES groups. More specifically, research findings examined from the perspective of sociocultural differences may provide more meaningful information and help develop innovative intervention strategies for ameliorating some of the disparities in health outcomes and access to health care.

Cross-References

- ▶ Health Disparities
- ▶ Socioeconomic Status (SES)

References and Readings

American Cancer Society. (2011). *Cancer Facts & Figures*. Atlanta: American Cancer Society.

American College of Physicians. (2010). *Racial and Ethnic Disparities in Health Care*. Philadelphia: American College of Physicians.

Braveman, P. A., Cubbin, C., Egerter, S., Williams, D. R., & Pamuk, E. (2010). Socioeconomic disparities in the United States: What the patterns tells us. *American Journal of Public Health, 100*, S186–S196.

Braveman, P. A., Egerter, S. A., & Mockenhaupt, R. E. (2011). Broadening the focus: The need to address the social determinants of health. *American Journal of Preventive Medicine, 40*, S4–S18.

Centers for Disease Control and Prevention. (2011). *National diabetes fact sheet: national estimates and general information on diabetes and prediabetes in the United States, 2011*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention.

Hajat, A., Kaufman, J. S., Rose, K. M., Siddiqi, A., & Thomas, J. C. (2011). Long-term effects of wealth on mortality and self-rated health status. *American Journal of Epidemiology, 173*, 192–200.
Marmot, M. (2005). Social determinants of health inequalities. *The Lancet, 365*, 1099–1104.
Payne, T. J., Wyatt, S. B., Mosley, T. H., Dubbert, P. M., Guiterrez-Mohammed, M. L., Calvin, R. L., et al. (2005). Sociocultural methods in the Jackson Heart Study: Conceptual and descriptive overview. *Ethnicity & Disease, 15*, S6-38–S6-48.
Thomas, S. B., Fine, M. J., & Ibrahim, S. A. (2004). Health disparities: The importance of culture and health communication. *American Journal of Public Health, 94*, 2050.

Sociocultural Context

▶ Sociocultural

Sociocultural Differences

Melissa Walls
Biobehavioral Health & Population Sciences, University of Minnesota Medical School – Duluth, Duluth, MN, USA

Definition

Sociocultural approaches to understanding differences in health call attention to the roles of and potential interdependence between social and cultural factors for health outcomes.

Cultural attitudes, beliefs, values, history, and systems of knowledge are interdependent with the social environment that includes economic status, community and family systems, and interpersonal relationships. Together, sociocultural factors may impact health in numerous ways, such as influencing access/barriers to health care and service utilization preferences/patterns as well as affecting health behaviors such as diet and exercise.

Cross-References

▶ Aerobic Exercise
▶ Sociocultural

Sociocultural Factors

▶ Sociocultural

Socioeconomic Position

▶ Social Class
▶ Sociocultural

Socioeconomic Status (SES)

▶ Education, Lack Of: As a Risk Factor
▶ Social Factors
▶ Sociocultural

Sodium

▶ Salt, Intake

Sodium Chloride

▶ Salt, Intake

Sodium, Sodium Sensitivity

Jonathan Newman
Columbia University, New York, NY, USA

Definition

Sodium chloride (NaCl), commonly known as salt, is a molecule crucial for fluid balance and free-water homeostasis. However, overconsumption of sodium/salt plays an important role in the

development of essential hypertension. Essential hypertension is seen almost exclusively in societies where average daily sodium consumption is greater than 2.3 g. In contrast, hypertension is rare in populations with low-sodium consumption (typically less than 1.2 g/day). These effects of sodium consumption appear independent of other potential causes of essential hypertension, such as obesity.

The blood pressure (BP) responsiveness to variations in sodium intake is known as salt sensitivity.

The change in BP to salt intake varies significantly between individuals and in the same individual at different times.

Salt sensitivity also increases with age and is more prominent in those with diabetes, obesity, and metabolic syndrome.

It may also be more common in African-Americans and other populations, in which excess salt intake may play an important role in the development of hypertension.

There is evidence to suggest that salt-sensitive individuals with normal blood pressure are at a greater risk of developing hypertension and at further risk of hypertension progression and poor blood pressure control. The mechanisms of salt sensitivity are incompletely understood but likely involve a combination of altered salt/water homeostasis, abnormal vascular signaling pathways, and other metabolic abnormalities such as type 2 diabetes and electrolyte abnormalities, such as hypokalemia.

References and Readings

Barba, G., Galletti, F., Cappuccio, F. P., Siani, A., Venezia, A., Versiero, M., Della Valle, E., Sorrentino, P., Tarantino, G., Farinaro, E., & Strazzullo, P. (2007). Incidence of hypertension in individuals with different blood pressure salt-sensitivity: Results of a 15-year follow-up study. *Journal of Hypertension, 25*(7), 1465–1471.

Obarzanek, E., Proschan, M. A., Vollmer, W. M., Moore, T. J., Sacks, F. M., Appel, L. J., Svetkey, L. P., Most-Windhauser, M. M., & Cutler, J. A. (2003). Individual blood pressure responses to changes in salt intake: Results from the DASH-Sodium trial. *Hypertension, 42*(4), 459–467.

Solid Fats

▶ Fat: Saturated, Unsaturated

Somatic Symptoms

Kurt Kroenke
Department of Medicine, Indiana University, Regenstrief Institute, VA HSR&D Center for Implementing Evidence-Based Practice, Indianapolis, IN, USA

Definition

Mental health professionals commonly label bodily symptoms as "somatic" to distinguish them from cognitive, emotional, or other types of non-somatic symptoms (Kroenke, 2007a). In contrast, bodily symptoms are more often referred to as "physical" symptoms by those practicing in general medical, surgical and other non-mental health care professions. Somatic symptoms are exceedingly prevalent, accounting for over half of all outpatient encounters. About half of these are pain complaints (e.g., headache, chest pain, abdominal pain, back pain, joint pains), a quarter are upper respiratory (e.g., cough, sore throat, ear or nasal symptoms), and the remainder are nonpain, non-upper-respiratory symptoms (e.g., fatigue, insomnia, dizziness, palpitations).

Description

Epidemiology

About 80% of individuals in the general population experience one or more symptoms each month, of who less than 1 in 4 seek care (Kroenke & Rosmalen, 2006). This ubiquitous nature of somatic symptoms mandates that some thresholds be set to distinguish most "persons" who experience common symptoms from the smaller subset of individuals who qualify as "patients." Some thresholds might include severity of the symptom: its duration or persistence; the degree of occupational or social

impairment; the level of patient distress, concerns or worries; the decision to seek treatment or use health care; and the direct and indirect financial costs.

An exact medical diagnosis that accounts for the symptom is often not established, with at least one-third of somatic symptoms lacking an adequate physical explanation and referred to by a variety of labels, including functional, idiopathic, atypical, somatoform, or unexplained.

About three-fourths of outpatients presenting with somatic complaints experience improvement within 2 weeks, while 20–25% of symptoms become chronic or recurrent.

Functional Somatic Syndromes

These conditions consist of a cluster of somatic symptoms for which the etiology is poorly understood and include disorders such as irritable bowel syndrome, fibromyalgia, chronic fatigue syndrome, temporomandibular disorder, interstitial cystitis, and others. Experts have questioned whether these are all separate disorders or instead part of a group of poorly explained somatic conditions sharing common features. Supporting the latter, literature syntheses have revealed that these disorders frequently overlap, both at the level of specific syndromes (half to two-thirds of patients with one syndrome also suffer from one or more additional syndromes) as well as in terms of individual symptoms (Kroenke & Rosmalen, 2006). Second, they are similar in rates of psychiatric comorbidity, particularly depression and anxiety (Henningsen, Zimmermann, & Sattel, 2003). Third, functional somatic syndromes respond similarly to certain therapies traditionally considered "psychological" treatments, such as antidepressants and cognitive-behavioral therapy.

Psychological Comorbidity

In patients presenting with poorly explained physical symptoms, a depressive disorder can be diagnosed 50–60% of the time, and an anxiety disorder 40–50% of the time, regardless of the type of symptom. While the specific type of symptom is not particularly important in terms of predicting depression or anxiety, the number of symptoms is. In two primary care studies involving 1,500 patients, those who endorsed 0–1, 2–3, 4–5, 6–8, or ≥ 9 physical symptoms on a 15-symptom scale, the proportion with a depressive or anxiety disorder was 6%, 20%, 33%, 58%, and 80% respectively, suggesting a "dose-response" effect between the number of physical symptoms and the likelihood of psychiatric comorbidity.

Two-thirds of primary care patients with major depression present exclusively with somatic complaints, and half report multiple, unexplained somatic symptoms. Also, depression is present in a quarter to a third of patients referred to medical specialty clinics and, if depressed, referred patients are only about a quarter as likely to have a physical diagnosis established as an explanation for their symptoms triggering the referral. Even disease-specific somatic symptoms (e.g., chest pain in patients with coronary artery disease, dyspnea in patients with pulmonary disease, joint pain in patients with arthritis) are at least as strongly associated with depression and anxiety as they are with objective physiologic measures of the medical disorder (Katon, Lin, & Kroenke, 2007).

Overlap among somatic, anxiety and depressive symptoms (the *SAD triad)* is more common than the "pure" form of any of the three types of symptoms (Löwe et al., 2008). For example, very high levels of depressive, anxiety and somatic symptoms are present in 7%, 8%, and 10% of primary care patients, respectively. However, only 26% of depressed patients have depression alone (i.e., without high levels of anxiety and/or somatic symptoms), 43% of anxious patients have anxiety only, and 46% of patients with high somatic symptom levels have somatization alone. Predictors of psychological comorbidity in patients with somatic symptoms are summarized in Table 1.

Somatoform Disorders

Somatoform disorders currently defined in the American Psychiatric Association's Diagnostic and Statistical Manual, 4th Edition (DSM-IV) include somatization disorder (chronic history of multiple medically unexplained symptoms), conversion disorder (unexplained neurological symptoms), hypochondriasis (preoccupation with having a serious medical illness that persists despite medical evaluations and reassurance), body dysmorphic disorder (distorted perceptions

Somatic Symptoms, Table 1 Predictors of psychological comorbidity in patients with somatic symptoms

Symptom remains medically unexplained after clinical assessment
Multiple symptoms
Three or more unexplained symptoms
Pain symptoms in two or more regions of the body
Multiple functional somatic syndromes
Chronic or recurrent symptom (s)
Excessive health care use
Medication history
Polypharmacy (especially for symptoms)
Poor response of symptoms to multiple medications
Nocebo response (nonspecific adverse effects to multiple medications)
Difficult clinician-patient relationship
Number of S4 predictors [a]
Stress recently
Symptom count is high
Self-rated health is low
Severity of symptom is high

[a]The four S4 predictors are (1) stress in past week (yes/no); (2) Patient reports being "bothered a lot" by five or more symptoms on the PHQ-15 scale of 15 somatic symptoms; (3) self-rated overall health of poor or fair on a 5-point scale (excellent, very good, good, fair, poor); (4) self-rated severity of presenting somatic symptom of $^{3}6$ on 0 (none) to 10 (unbearable) scale. The likelihood of a depressive or anxiety disorder with 0, 1, 2, 3, or 4 of these S4 predictors is 8%, 16%, 43%, 69%, and 94%, respectively

of specific bodily features), and chronic pain disorder. However, these are likely to be substantially revised in DSM-V (Kroenke, Sharpe, & Sykes, 2007). In particular, criteria for the most common type of somatoform disorder (full and abridged versions of somatization disorder) are likely to rely less on symptom counts or the degree to which symptoms are "medically explained" and more on positive psychological criteria characteristic of somatizing patients (e.g., excessive illness worry and health anxiety, inordinate health care use, catastrophizing).

Measuring Somatic Symptoms

The PHQ-15 is a brief, freely-available scale (www.phqscreeners.com) that measures 15 symptoms that account for more than 90% of non-upper-respiratory symptoms seen in primary care (Kroenke, Spitzer, Williams, & Löwe, 2010). The PHQ-15 asks patients to rate how much they have been bothered by each symptom during the past month on a 0 ("not at all") to 2 ("bothered a lot") scale. Thus, the total score ranges from 0 to 30, with cutpoints of 5, 10, and 15 representing thresholds for mild, moderate, and severe somatic symptom severity, respectively.

Increasing scores on the PHQ-15 are strongly associated with functional impairment, disability, health care use, and somatoform disorder diagnoses. Also, items on the PHQ-15 overlap better with other validated somatization screeners than any other two screeners do with one another. There is emerging evidence that the PHQ-15 is responsive to treatment.

Treatment

Treatment of somatoform disorders as well as functional somatic syndromes has been recently reviewed (Abbass, Kisely, & Kroenke, 2009; Jackson, O'Malley, & Kroenke, 2006; Kroenke, 2007b). In addition to symptom-specific treatments (e.g., analgesics for pain, medications specific to the symptoms for irritable bowel syndrome, medications recently approved for fibromyalgia), the two most evidence-based treatments for both somatoform disorders and functional somatic syndromes are cognitive behavioral therapy and antidepressants, which have a beneficial effect on the somatic symptoms in these conditions independent of their effect on psychological symptoms such as depression and anxiety. Additionally, regular visits with a primary physician, avoidance of excessive testing, and evaluation of new symptoms (but not repeated evaluation of chronic symptoms) is beneficial. A clinical approach to the patient with unexplained somatic symptoms is outlined in Table 2.

Strategies for managing chronic somatization

- Schedule regular, brief appointments that are not related to symptom exacerbations
- Limit extensive diagnostic testing and multiple subspecialty referrals, especially for symptoms previously evaluated
- When new symptoms arise, conduct focused evaluations and testing rather than exhaustive work-ups

Somatic Symptoms, Table 2 Clinical approach to the patient with unexplained somatic symptoms

Initial visit	
Symptom-specific evaluation	Focus the interview and physical examination on the relevant symptom(s) Stratify symptoms into those that are at higher risk of a serious cause (e.g., angina-like chest pain; acute abdominal pain; syncope) and those that are seldom urgent (back pain, fatigue, insomnia). Identify "red flags" of a potentially serious cause (e.g., focal neurologic findings in patient with dizziness or headache; abnormal cardiac exam in patient with syncope)
Probe for symptom-specific concerns and expectations	"Is there anything else you were worried about?" (Serious cause? How long symptom might last?) "Is there anything else you wanted or thought might be helpful?" (Test? subspecialty referral? Specific treatment?)
Consider short-term use of symptom-specific medications (often available over-the-counter)	Simple analgesics for pain Gastrointestinal medications for dyspepsia or constipation Decongestants, antihistamines, cough suppressants for upper respiratory symptoms
Watchful waiting	Have patient follow-up in 2–6 weeks if symptom is not resolved
Follow-up visits	
Psychological screening	Especially for treatable depressive and anxiety disorders (see www.phqscreeners.com) Assess for somatization (PHQ-15 or other scale)
Diagnostic evaluation	Selective testing and/or specialty referral, especially if new unexplained symptom, if findings on interview or examination are worrisome for serious cause, or if patient insists
Chronic symptom therapies	Pharmacological – analgesics; gastrointestinal medications; antidepressants, other Nonpharmacological – cognitive-behavioral therapy (CBT); other psychological or behavioral therapies; exercise; pain self-management programs; reattribution

- Empathize with the complaint. Do not dispute the reality of the symptom or associated impairment.
- Focus on symptom management/reduction rather than elimination (coping rather than cure).
- Strive for gradual rehabilitation (maximizing function despite the symptom) rather than chronic disability
- Emphasize that referral is for consultation or co-management rather than dismissal (i.e., you are not "dumping" the patient)

Cross-References

- ▶ Antidepressant Medications
- ▶ Anxiety Disorder
- ▶ Chronic Pain
- ▶ Cognitive Behavioral Therapy (CBT)
- ▶ Depression: Treatment
- ▶ Medically Unexplained Symptoms
- ▶ Pain
- ▶ Somatization
- ▶ Somatoform Disorders
- ▶ Stress
- ▶ Symptoms

References and Readings

Abbass, A., Kisely, S., & Kroenke, K. (2009). Short-term psychodynamic psychotherapy for somatic disorders: Systematic review and meta-analysis of clinical trials. *Psychotherapy and Psychosomatics, 78*, 265–274.

Dimsdale, J. E., Xin, Y., Kleinman, A., Patel, V., Narrow, W. E., Sirvatka, P. J., & Regier, D. A. (Eds.). (2009).

Somatic presentations of mental disorders: Refining the research agenda for DSM-V. Arlington, VA: American Psychiatric Association.
Henningsen, P., Zimmermann, T., & Sattel, H. (2003). Medically unexplained physical symptoms, anxiety, and depression: A meta-analytic review. *Psychosomatic Medicine, 65*, 528–533.
Jackson, J. L., O'Malley, P. G., & Kroenke, K. (2006). Antidepressants and cognitive behavioral therapy for symptom syndromes. *CNS Spectrums, 11*, 212–222.
Katon, W., Lin, E., & Kroenke, K. (2007). The association of depression and anxiety with medical symptom burden in patients with chronic medical illness. *General Hospital Psychiatry, 29*, 147–155.
Kroenke, K. (2007a). Somatoform disorders and recent diagnostic controversies. *Psychiatric Clinics of North America, 30*, 593–619.
Kroenke, K. (2007b). Efficacy of treatment for somatoform disorders: A review of randomized clinical trials. *Psychosomatic Medicine, 69*, 881–888.
Kroenke, K., & Rosmalen, J. G. M. (2006). Symptoms, syndromes and the value of psychiatric diagnostics in patients with functional somatic disorders. *Medical Clinics North America, 90*, 603–626.
Kroenke, K., Sharpe, M., & Sykes, R. (2007). Revising the classification of somatoform disorders: Key questions and preliminary recommendations. *Psychosomatics, 28*, 277–285.
Kroenke, K., Spitzer, R. L., Williams, J. B. W., & Löwe, B. (2010). The patient health questionnaire somatic, anxiety, and depressive symptom scales: A systematic review. *General Hospital Psychiatry, 32*, 345–359.
Löwe, B., Spitzer, R. L., Williams, J. B. W., Mussell, M., Schellberg, D., & Kroenke, K. (2008). Depression, anxiety, and somatization in primary care: Syndrome overlap and functional impairment. *General Hospital Psychiatry, 30*, 191–199.

Somatization

Winfried Rief
Department of Clinical Psychology and Psychotherapy, Philipps University of Marburg, Gutenbergstr, Marburg, Germany

Definition

The term "somatization" goes back to psychodynamic theory, and describes the transformation of unconscious conflicts and repressed emotions into somatic symptoms. Later on, Lipowski defined somatization as "a tendency to experience and communicate somatic distress in response to psychosocial stress and to seek medical help for it" (Lipowski, 1986). Therefore this definition postulates that psychosocial stress is the cause for somatization, and that seeking medical help is a necessary feature of this syndrome. However, current concepts of somatization use this term more descriptively. According to these modern concepts, somatization could be defined as "the tendency to experience a variety of somatic symptoms that are usually poorly described by biomedical disease processes." In this definition, the presence of multiple somatic complaints is the core feature of somatization.

Somatization itself is not a diagnosis, but is frequently related to the diagnostic group of "somatoform disorders," and especially to "somatization disorder." If multiple somatic symptoms are part of a depressive syndrome or anxiety disorder, the term could be also used. It is not recommended to use the term "somatization" for a postulated association between psychological conflicts and serious medical conditions (e.g., cancer). However, medical conditions like cancer or diabetes can also be associated with multiple medical symptoms that are not explained by the biomedical disease itself. For this subgroup of patients with serious medical conditions, the additional use of the term "somatization" could be appropriate.

Current behavioral-medical concepts prefer the process of "somatosensory amplification" to describe the development and maintenance of somatization. Somatosensory amplification summarizes the process of focusing attention to bodily perceptions; together with health worries and a catastrophizing style of interpreting bodily perceptions, selective attention leads to an amplified style of perceiving somatic symptoms. If symptoms are chronic, further sensitization processes might be involved. Further details on modern concepts of somatization can be found in Barsky (1992), Looper and Kirmayer (2002), and Rief and Broadbent (2007).

Cross-References

▶ Somatoform Disorders

References and Readings

Barsky, A. J. (1992). Amplification, somatization, and the somatoform disorders. *Psychosomatics, 33*, 28–34.
Lipowski, Z. J. (1986). Somatization: A borderland between medicine and psychiatry. *Canadian Medical Association Journal, 135*, 609–614.
Looper, K. J., & Kirmayer, L. J. (2002). Behavioral medicine approaches to somatoform disorders. *Journal of Consulting and Clinical Psychology, 70*, 810–827.
Rief, W., & Broadbent, E. (2007). Explaining medically unexplained symptoms: Models and mechanisms. *Clinical Psychology Review, 27*, 821–841.

Somatoform Disorders

Winfried Rief
Department of Clinical Psychology and Psychotherapy, Philipps University of Marburg, Gutenbergstr, Marburg, Germany

Synonyms

Functional somatic symptoms; Medically unexplained physical symptoms

Definition

The common feature of somatoform disorders is the presence of physical symptoms that could indicate a general medical condition, but the physical symptoms are not fully explained by a well-known biomedical disease, by the direct effects of a substance, or by another mental disorder. The group of somatoform disorders includes diagnoses such as somatization disorder, undifferentiated somatoform disorder, conversion disorder, pain disorder, hypochondriasis, and body dysmorphic disorder.

Description

The term "somatoform disorder" has been introduced by DSM-III (*Diagnostic and Statistical Manual of Mental Disorders*, 3rd version) as a category for a group of diagnoses. The common feature of somatoform disorders is the presence of physical symptoms that could indicate a general medical condition, but the physical symptoms are not fully explained by a well-known biomedical disease, by the direct effects of a substance, or by another mental disorder (e.g., panic disorder, depression). Symptoms must cause clinically significant distress or impairment. Somatoform disorders have to be distinguished from factitious disorders and malingering; the physical symptoms in somatoform disorders are not intentional or imagined, but patients perceive these symptoms similar to other physical symptoms caused by medical conditions.

The group of somatoform disorders includes diagnoses such as somatization disorder, undifferentiated somatoform disorder, conversion disorder, pain disorder, hypochondriasis, and body dysmorphic disorder. Somatization disorder is the prototype for patients suffering from many physical complaints including pain symptoms, gastrointestinal symptoms, sexual symptoms, and pseudoneurological symptoms. In DSM-IV, the onset of symptoms was required before age 30 years, and the diagnosis was only justified if symptoms persisted over a period of several years. The criteria for this diagnosis have been criticized because somatization disorder does not include the majority of patients with multiple somatoform symptoms, as the diagnosis is over-exclusively defined. Therefore many patients fall under the diagnosis of "undifferentiated somatoform disorder," which only requests one or more physical complaints that are medically unexplained. Both diagnoses cannot only be used in the absence of medical conditions, but these diagnoses are also justified if general medical conditions do not fully account for all somatic symptoms presented by the patient. While most people suffer from somatic symptoms from time to time, a diagnosis of somatoform disorder should be only given if the symptoms cause clinically significant distress or impairment. A diagnosis of conversion disorder is justified if people suffer from motor or sensory symptoms or deficits that are not fully accounted by a medical condition. Hypochondriasis ("fears and worries about illnesses") and body

dysmorphic disorder ("preoccupation with an imagined defect in appearance") are disorders that are considered to indicate an overlap with anxiety disorders/OCD (obssessive-compulsive disorders). Pain disorder is also classified under somatoform disorders, although many pain conditions are associated with both psychological factors and a general medical condition. In fact, in "Western" cultures pain symptoms are the most frequent somatic symptoms. If pain duration is longer than 6 months, it should be considered to be chronic. In most cases, chronic pain conditions are not sufficiently understood with pure biomedical approaches, but psychological and social factors have to be also considered.

Somatoform disorders can develop as a result of the interaction of psychological factors (selective attention to bodily processes, overinterpretation of somatic sensations, illness fears, demoralization) with biomedical factors (e.g., traumatic injuries, car accidents, biological dysregulation of stress and immune responses) and with social factors (e.g., reinforcement of symptom expression by proxies, strong biomedical orientation of health care systems, fears of doctors to overlook biomedical causes). While somatization typically refers to the experience of multiple medically unexplained symptoms, the concept of somatosensory amplification refers to the self-reinforcing circle of attention to somatic processes, overinterpretation of somatic sensations, intensified physical sensations, and behavioral consequences (Barsky, 1992). Somatoform disorders (especially somatization disorder and undifferentiated somatoform disorder) are more prevalent in women than in men. While prevalence rates for diagnoses such as somatization disorder are significantly lower than 1%, general somatoform symptoms can be found in more than 10% of the population (Wittchen & Jacobi, 2005).

The category of somatoform disorders has been criticized (e.g., Mayou, Sharpe, Kirmayer, Simon, & Kroenke, 2005). In some countries, the term "somatoform" was interpreted as indicating "not real symptoms," although the term was originally introduced as a pure descriptive term. For patients with multiple somatic symptoms, the diagnosis "somatization disorder" is overrestrictive (see above). Finally, the classification of "medically explained" versus "medically unexplained" symptoms is unreliable and medical doctors highly disagree in their ratings about causality of somatic symptoms. Some experts criticized that the concept of somatoform disorders further continues a "mind-body separation" instead of favoring a biopsychosocial model. The revision of this category in DSM-V tries to overcome these shortcomings.

To date, no well-founded pharmacological treatment is available for patients with somatoform syndromes. There is some evidence for the use of SSRIs (selective serotonin reuptake inhibitors) in patients with hypochondriasis and body dysmorphic disorders. Psychological interventions (especially behavioral-medical interventions) have been shown to be effective in all groups of somatoform disorders. However, effect sizes vary substantially, with highest effect sizes for the psychological treatment of hypochondriasis, while effect sizes for the treatment of somatization disorder or chronic pain conditions are only in the low to medium range. The rate of overlooked medical conditions that can explain the somatic symptoms does not seem to be increased compared to other mental disorders: Long-term studies reveal that less than 10% of patients with somatoform disorders must be considered to be misdiagnosed (Rief & Broadbent, 2007).

Cross-References

- ▶ Functional Somatic Syndromes
- ▶ Somatization

References and Readings

Barsky, A. J. (1992). Amplification, somatization, and the somatoform disorders. *Psychosomatics, 33*, 28–34.

Mayou, R., Sharpe, M., Kirmayer, L. J., Simon, G., & Kroenke, K. (2005). Somatoform disorders: Time for a new approach in DSM-V. *American Journal of Psychiatry, 162*, 847–855.

Rief, W., & Broadbent, E. (2007). Explaining medically unexplained symptoms: Models and mechanisms. *Clinical Psychology Review, 27*, 821–841.
Wittchen, H. U., & Jacobi, F. (2005). Size and burden of mental disorders in Europe – A critical review and appraisal of 27 studies. *European Neuropsychopharmacology, 15*, 357–376.

Spatial Analysis

▶ Geographic Information System (GIS) Technology

Spectral Analysis

▶ Quantitative EEG Including the Five Common Bandwidths (Delta, Theta, Alpha, Sigma, and Beta)

Speech and Language Pathology

▶ Speech and Language Therapy

Speech and Language Therapy

Steven Harulow
Royal College of Speech & Language Therapists, London, UK

Synonyms

Speech and language pathology; Speech therapy; Speech, language, and communication therapy

Definition

Speech and language therapy is an evidence-based discipline that anticipates and responds to the needs of individuals who experience speech, language, communication, or swallowing difficulties. Speech and language therapy works in partnership with individuals and their families and with other professions and agencies to reduce the impact of these often isolating difficulties on well-being and the ability to participate in daily life (Royal College of Speech and Language Therapists, 2005).

Speech and language therapists (SLTs) are the lead experts regarding communication and swallowing disorders. This does not mean that others do not work within these areas or that others do not have many skills that may overlap with or complement those of SLTs. Rather, SLTs, through their preregistration education, and later experience, have greater depth and breadth of knowledge and understanding of these clinical areas and associated difficulties. This enables SLTs to lead on the assessment, differential diagnosis, intervention with, and management of individuals with communication and swallowing disorders.

Speech and language therapy assistants and bilingual co-practitioners are integral members of the speech and language therapy team, employed to act in a supporting role under the direction of a professionally qualified SLT.

Description

A wide range of individuals can potentially benefit from speech and language therapy, including:

- Babies with feeding and swallowing difficulties
- Children (from neonates to school age), adolescents, and adults with special needs in communication, communication disability, and/or swallowing disorders associated with diagnosed impairments, genetic and medical conditions, trauma, developmental delays, mental health problems, and learning disability
- Children (from neonates to school age), adolescents, and adults with special needs in the following areas: speech, voice, fluency, language, psychologically based communication disorders, social skills, problem solving, literacy, swallowing functions, and alternative and augmentative communication (AAC)

- Parents and families, caregivers, communication partners, friends, and colleagues of people with communication and swallowing disorders

Speech and language therapists work in and across a variety of settings.

Within education, these settings include:

- Local education authority nurseries and schools (mainstream and special)
- Language and communication units and colleges of further education
- Independent nurseries and schools
- Play groups
- Government-funded initiatives

Within health and social care, settings include:

- Hospitals inpatient and outpatient centers and hospices
- Specialist centers: child development centers, rehabilitation centers, specialist joint consultative clinics
- Primary care: community clinics, community day centers
- Supported living homes
- Mental health services
- Initiatives in areas of social deprivation (such as Sure Start)

Speech and language therapists have an increasing role within the legal system, including within the penal system/prisons, in court tribunals, and as part of adult and child protection services.

They also work in independent practice, as part of social enterprises and for the voluntary/charitable sector.

All speech and language therapy intervention is delivered on the basis of ongoing assessment and review of progress with the individual (and/or carer as appropriate) as measured against targeted outcomes. Various approaches or models of working have been developed to meet the needs of individuals and context.

The following are key principles guiding the provision of services:

- The rights, wishes, and dignities of each individual and their carers are respected at all times.
- Effective intervention is based on a holistic understanding of the individual, including their social, cultural, economic, political, and linguistic context.
- The safety of the individual is paramount.
- Speech and language therapy intervention aims to be efficient and effective, i.e., best results against targeted outcomes within given resources.

Speech and language therapy services may operate at the level of the person (working with individuals); the level of their environment (working with people, processes, or settings); and the level of the wider community (influencing attitude, culture, or practice). The form of intervention will vary according to the changing needs of the individual and contexts.

Benefits

Speech and language therapy can contribute to the following health, educational, and psychosocial benefits:

- Improvement in general health and well-being
- Increased independence
- Improved participation in family, social, occupational, and educational activities
- Improved social and family relationships
- Reduction in the negative effects of communication disability and the harm or distress this may cause to the individual and others
- Reduced risk of surgical intervention and poor nutrition in the case of individuals with swallowing disorders
- Reduced health risks and length of hospital stay through the prevention of respiratory problems associated with swallowing difficulties
- Reduced risk of surgical intervention by maintaining healthy voice mechanisms
- Reduced risk of educational failure
- Reduced risk of social isolation
- Prevention of certain speech, language, and communication disorders

Outcomes

The outcomes of speech and language therapy include:

- Diagnosis of communication and/or swallowing disorders
- Maintenance of optimal communication and/or swallowing abilities

- Improvement in the speech, language, and communication abilities of individuals
- Improved use of existing function
- Reduction of communication anxiety and avoidance
- Provision and use of AAC where oral communication is limited or precluded by a physical condition
- Improvement in interaction and effective social communication
- Increased awareness of others about communication and/or swallowing disorders, intervention, and management
- Improved communication environment
- Greater opportunities for communication
- Improvement in the individual's understanding of the nature and implications of a communication and/or swallowing disorder

In 2010, the Royal College of Speech and Language Therapists (RCSLT) commissioned analysts Matrix Evidence to review the existing evidence and undertake an economic evaluation of the provision of speech and language therapy to four specific client groups. The aim of this was to pinpoint the benefits generated by speech and language therapy in relation to the costs of provision. The result was the UK-wide study "The economic case for speech and language therapy" (Marsh et al., 2010).

The Matrix research aimed to determine the costs and benefits for four common speech and language therapy client groups:

- Adults with dysphagia post stroke
- Adults with aphasia post stroke
- Children with speech and language impairment (SLI)
- Children with autism

Matrix Evidence undertook an evaluation of the costs and benefits of speech and language therapy intervention for each condition and compared either the effects of speech and language therapy with the effects of alternative forms of treatment, or the effects of intensive against less intensive therapy. Specifically, the analysis evaluated:

- Speech and language therapy for stroke survivors with dysphagia compared with "usual" care
- Enhanced NHS speech and language therapy for stroke survivors with aphasia compared with usual NHS therapy
- Enhanced speech and language therapy for children with SLI compared with existing therapy provision
- Enhanced speech and language therapy for children with autism compared with usual SLT treatment

The results of the Matrix report show that speech and language therapy for all four cohorts and conditions represents an efficient use of public resources. The net benefits of the interventions – including health and social care cost savings, quality of life, and productivity gains – are positive and exceed their costs. The report shows the total annual net benefit across aphasia, SLI, and autism is £765 million; it excludes dysphagia from the calculation since the two poststroke conditions are not mutually exclusive.

The RCSLT

Established as the College of Speech Therapists in 1945, the Royal College of Speech and Language Therapists (RCSLT) is the membership organization for UK SLTs, providing and promoting:

- Support and professional leadership for members, including the setting of standards
- Strategic direction for the profession
- Consistent, effective, and accurate professional representation to external bodies and the government
- Heightened public awareness of the medical, social, and emotional effects of communication, eating, drinking, and swallowing difficulties
- Heightened awareness of the contribution of speech and language therapy with the public, government, other professions, and the media

The RCSLT provides leadership so that issues concerning the profession are reflected in public policy and people with communication, eating, drinking, or swallowing difficulties receive optimum care.

Cross-References

- ▶ Alzheimer's Disease
- ▶ Brain Injury
- ▶ Brain Tumor
- ▶ Chronic Disease Management
- ▶ Cognitive Impairment
- ▶ Cost-Effectiveness
- ▶ Neurological
- ▶ Neuromuscular Diseases
- ▶ Neuromuscular Disorders
- ▶ Nutrition
- ▶ Occupational Therapy
- ▶ Rehabilitation
- ▶ Stroke Burden

References and Readings

For more information, visit the RCSLT website: www.rcslt.org

Marsh, K., Bertranou, E., Suominen, H., & Venkatachalam, M. (2010). *An economic evaluation of speech and language therapy: Final report*. December 2010. Matrix Evidence.

Royal College of Speech Therapists Language. (2005). *Communicating quality* (Vol. 3, pp. 2–28). London: RCSLT.

Speech Therapy

▶ Speech and Language Therapy

Speech, Language, and Communication Therapy

▶ Speech and Language Therapy

Sperm Donation

▶ In Vitro Fertilization, Assisted Reproductive Technology

Sperm Donor

▶ In Vitro Fertilization, Assisted Reproductive Technology

Spiritual

▶ Religion/Spirituality

Spiritual Beliefs

Afton N. Kapuscinski
Psychology Department, Syracuse University, Syracuse, NY, USA

Synonyms

Religious beliefs; Spirituality

Definition

The concept of spiritual beliefs is a critical component of the broader terms *spirituality* or *religiousness* and is to some degree inseparable from them. The meanings of these terms, however, remain elusive. In fact, the paramount importance, and difficulty, with defining spiritual concepts has proven a prominent obstacle to establishing a cohesive body of literature. The empirical research is littered with varying, and sometimes incompatible, ways of understanding spiritual constructs (Hill & Pargament, 2003; Kapuscinski & Masters, 2010). Researchers disagree, for example, regarding the relationship of religiousness and spirituality. This issue takes on special significance when *beliefs* in particular are the subject of consideration because Western notions of religion often differentiate believers from unbelievers based on their convictions (beliefs), rather than behaviors. Historically, spirituality and religiousness were considered to be,

if not the same entity, intimately tied to one other. One's personal ideas about and experiences of the sacred were both informed by and occurred in the context of institutional religious beliefs and practices. In recent years, however, a growing minority of Americans have begun to identify themselves as "spiritual but not religious." The distinction often involves the term spiritual being used to signify personal beliefs or experiences of the sacred, apart from traditional religious doctrine and organizations. Interestingly, although most people recognize religion and spirituality as somewhat different concepts, they also see the terms as sharing much in common, including traditional religious concepts of God, Christ, and the church (Zinnbauer, Pargament & Scott, 1999).

Some researchers have taken the connotation of spirituality as an individual's internal communion with the transcendent and sharply separated it from the idea of religiousness now defined narrowly as involvement with organized beliefs and practices (Hill & Pargament, 2003; Zinnbauer et al., 1999). In some cases, the supernatural or sacred is completely removed from the notion of spirituality or spiritual beliefs, leaving a search for meaning or perspective that is unrelated to the transcendent. Some researchers (Kapuscinski & Masters, 2010; Koenig, 2010) do not support this separation on several grounds, including that the removal of the sacred creates a somewhat artificial notion of spirituality, devoid of any substance that separates it from mental health variables like optimism and purpose in life. Therefore, the conceptual state of affairs on these topics does not allow spiritual beliefs to be cleanly separated from spirituality or religiousness. All of these concepts overlap significantly.

Nevertheless, despite the overlap, thematic elements in the literature suggest that spiritual beliefs per se may be defined as convictions about self, others, and the world, which emerge from a search for the transcendent or sacred, and include the values regarding lifestyle and moral conduct derived from these convictions. Spiritual beliefs may be roughly synonymous with the notion of spiritual worldview, the basis of which is a belief in the transcendent. Beliefs may or may not include doctrine associated with religious institutions (e.g., that an omnipotent God created the universe, as in the Judeo-Christian tradition).

Description

Most of the questionnaires designed to assess spirituality emphasize beliefs as a critical element of what is measured, and a few, like the Beliefs and Values Scale (King, Barnes, Low, Walker, Wilkinson, Mason, et al., 2006) and the Royal Free Interview for Spiritual and Religious Beliefs (King, Speck, & Thomas, 2001), focus on beliefs specifically. Thus, consistent with the discussion above, the spiritual beliefs component of religiousness and spirituality (R/S) is strongly embedded in research findings on R/S, even when studies do not claim to focus specifically on beliefs. A wealth of evidence demonstrates that both R/S in general, and sometimes beliefs in particular, influence physical and mental health.

Physical Health

Available research generally indicates a relationship between R/S variables and better physical health and implicates the value of incorporating this aspect of culture into health-promoting interventions. Masters and Hooker (2011) provide an overview of the R/S and health literature. Religious service attendance stands out as a variable that consistently predicts mortality. Frequent attendees are at reduced risk of mortality compared to those who never attended services, even when standard controls are included, and the relationship is especially strong for African Americans. Importantly, this relationship appears to transcend culture, with research demonstrating that R/S serves as a protective factor against mortality in a variety of countries, including non-Western societies such as China and Israel. In this light, it is very important to note that service attendance does not measure spiritual beliefs per se. Nevertheless, the literature also indicates that spiritual beliefs are related to improved outcomes for seriously ill individuals.

In cancer patients, for example, spiritual beliefs predict positive psychological adjustment and higher rates of perceived cancer-related growth. Studies also indicate spiritual beliefs and practices may be helpful for cardiac patients, putting them at reduced risk of morbidity, complications, and depression following surgery. However, the relationship between R/S and health is complex, with some studies indicating a detrimental relationship between certain R/S variables and health outcomes. For instance, cardiac patients who viewed God as responsible for their *illnesses* had more difficult recoveries, whereas individuals who attributed their recoveries to God enjoyed better outcomes. Psychologists have theorized several pathways to explain the relationship between R/S and health variables, including the idea that R/S is associated with increased social support, positive coping skills, and the adoption of a healthy lifestyle (e.g., abstinence from smoking and alcohol use, regular exercise, and utilization of preventative health care).

Mental Health

The vast majority of research also indicates a positive association between mental health and spiritual beliefs (Koenig, 2010). Individuals scoring higher on measures of spirituality are consistently less likely to have depressive symptoms or disorders, with an effect equivalent in size to that of gender and depression. Similarly, many studies indicate that anxiety is lower for individuals who are more spiritual and that spiritually based interventions result in reduced anxiety. However, the results are mixed – with some research indicating a positive correlation between certain spiritual variables (e.g., spiritual struggle) and anxiety. The relationship between spiritual beliefs and substance use is less ambiguous, with the preponderance of evidence indicating that more spiritual individuals are significantly less likely to engage in substance use, misuse, and abuse.

Despite marked disagreement regarding conceptualization of spiritual constructs, the interaction (whether beneficial or detrimental) of spiritual beliefs with both physical and mental health highlights the significance of recognizing this dimension of human experience in both the science and practice of psychology.

Cross-References

- ▶ Religion/Spirituality
- ▶ Spirituality and Health
- ▶ Spirituality, Measurement of

References and Readings

Hill, P. C., & Pargament, K. I. (2003). Advances in the conceptualization of religiousness and spirituality: Implications for physical and mental health research. *The American Psychologist, 58*, 64–74. doi:10.1037/0003-066X.58.1.64.

Kapuscinski, A. N., & Masters, K. S. (2010). The current status of measures of spirituality: A critical review of scale development. *Psychology of Religion and Spirituality, 2*, 191–205. doi:10.1037/a0020498.

King, M., Jones, L., Barnes, K., Low, J., Walker, C., Wilkinson, S., et al. (2006). Measuring spiritual belief: Development and standardization of a beliefs and values scale. *Psychological Medicine, 36*, 417–425. doi:10.1017/S003329170500629X.

King, M., Speck, P., & Thomas, A. (2001). The royal free interview for spiritual and religious beliefs: Development and validation of a self-report version. *Psychological Medicine, 31*, 1015–1023. doi:10.1017/S0033291701004160.

Koenig, H. G. (2010). Spirituality and mental health. *International Journal of Applied Psychoanalytic Studies, 7*, 116–122. doi:10.1002/aps.239.

Koenig, H. G., McCullough, M. E., & Larson, D. B. (Eds.). (2001). *Handbook of religion and health*. New York: Oxford University Press.

Masters, K. S., & Hooker, S. A. (2011). Impact of religion and spirituality on health. In J. Aten, K. O'Grady, & E. Worthington (Eds.). *The psychology of religion and spirituality for clinicians: Using research in your practice* (357–386). New York: Routledge.

Zinnbauer, B. J., Pargament, K. I., & Scott, A. B. (1999). The emerging meanings of religiousness and spirituality: Problems as prospects. *Journal of Personality, 67*(6), 889–919. doi:10.1111/1467-6494.00077.

Spiritual Coping

▶ Religious Coping

Spiritual Struggle

► Religious Coping

Spirituality

Stephen Gallagher and Warren Tierney
Department of Psychology, Faculty of Education & Health Sciences, University of Limerick, Castletroy, Limerick, Ireland

Synonyms

Religion; Religiosity; Religiousness

Definition

Spirituality is a very unclear concept that has no concrete definition. By its very nature, the concept of spirituality is deeply rooted in religion, yet in contemporary spirituality, there is an incremental divide emerging between religion and spirituality. Therefore, in present-day society, the formation of a dichotomy with spirituality representing the personal, subjective, inner-directed, unsystematic, liberating expression, and religion signifying a formal, authoritarian, institutionalized inhibiting expression is being witnessed. Spirituality has also been defined as a subjective and fluid approach to experiences which leads one to search for enlightenment whereby behaviors are practiced in accordance with these sacred beliefs. Similarly, one can also consider spirituality to be something personal, which is defined by individuals themselves and is mostly likely devoid of the rules and regulations associated with religion.

Description

Pathways Linking Spirituality to Health

Rendering a congruent spiritual outlook on life has been associated with an enhanced quality of life, better mental and physical health, and improved recovery from various illnesses. However, the precise mechanisms behind these relationships remain unclear. This can be partly attributed to several reasons: first, the lack of a clear conceptual definition of what spirituality is; second, researchers using both concepts of religion and spirituality interchangeably; third, measuring both concepts with similar assessments (e.g., denomination and frequency of religious observance) which clearly are not adequate to capture a measure of one's spiritual beliefs. Finally, there is a social acceptance that being spiritual entails performing soothing activities such as mediation, yoga or praying, etc., which may be tied to social interactions. Thus, it is necessary to distinguish social factors involved in these practices from spirituality itself. All of the above make it difficult to draw any firm conclusions; thus, caution must be warranted when interpreting the data from such studies. Nonetheless, a number of pathways linking spirituality and health have been proposed, from direct physiological mechanisms (e.g., immune functioning) to more indirect stress buffering (e.g., coping strategies) and lifestyle choices (e.g., dietary and exercise behaviors coupled to one's spiritual beliefs) (Miller & Thorensen, 2003). For example, a positive correlation between spirituality and T-cells percentages in HIV positive women has been reported, suggestive of a more direct physiological route, while other studies support a more indirect pathway; spiritual beliefs have been found to be linked to lower stress, better nutrition, and more exercise, all of which are associated with positive health indices (Koenig McCullough, & Larson, 2001). However, what is more interesting is the strong evidence coming from intervention research; a number of studies have been conducted along this line, but one study found that those taught spiritual meditation had greater decreases in anxiety, negative affect, and frequency of migraine headaches compared to those who practiced internally focused secular meditation, externally focused secular meditation, or muscle relaxation

(Wachholtz & Pargament, 2009). Taken together, these studies indicate that spirituality influences health and that interventions targeting this concept can bring health benefits.

However, despite these positive benefits, a word of caution is warranted when investigating these relationships as there is also negative health consequences associated with spirituality. For example, a spiritual struggle denotes the anxiety and pressure about spiritual concerns inside oneself, with others, and the godly. Indeed, this spiritual struggle is associated with poor mental and physical health among suffers in some traditional faith practices (Rosmarin, Pargament, Flannelly, & Koenig, 2009); hence, it is sometimes difficult to determine whether spirituality is a resource or a liability and adds to the complexity that already exists in this spirituality-health relationship, which in some instances may not be linear. Further, there have been attempts to adopt the concept of spirituality into an overall definition of health, and some now argue that spiritual health and growth are equally important for quality of life (Sawatzky, Ratner, & Chiu, 2005); thus, spirituality can now be viewed and measured as an endpoint itself.

Measuring Spirituality

Measuring spirituality is a very difficult task due to lack of agreed upon definition and its close knit ties with religion. However, there have been some admirable attempts to capture the concept using self-report methods, and some of the measures include the Daily Spiritual Experience Scale (Underwood & Teresi, 2002), the Spiritual Well-being Scale (Paloutzian & Ellison, 1991), the Theistic Spiritual Outcome Survey (Richards et al., 2005), and both the Spiritual Transcendence Scale (Piedmont, 1999), and the Beliefs and Values Scale (King et al., 2006) offers a conceptualization of spirituality which is nonreligious; these may be useful when one is dealing with individuals who are more inclined to adopt a contemporary view of spirituality.

Cross-References

- ▶ Religion
- ▶ Religiousness/Religiosity
- ▶ Religion/Spirituality
- ▶ Spiritual Beliefs

References and Readings

Hill, P. C., & Pargament, K. I. (2003). Advances in the conceptualization and measurement of religion and spirituality: Implications for physical and mental health research. *American Psychologist, 58*, 64–74.

King, M., Jones, L., Barnes, K., Low, J., Walker, C., Wilkinson, S., et al. (2006). Measuring spiritual belief: Development and standardization of a beliefs and values scale. *Psychological Medicine, 36*(3), 417–425.

Koenig, H. G. (2009). Research on religion, spirituality, and mental health: A review. *Canadian Journal of Psychiatry, 54*, 283–291.

Koenig, H. G., McCullough, M. E., & Larson, D. B. (Eds.). (2001). *Handbook of religion and health*. New York: Oxford University Press.

Miller, W. R., & Thorensen, C. E. (2003). Spirituality, religion, and health: An emerging research field. *American Psychologist, 58*, 24–35.

Paloutzian, R. E., & Ellison, C. W. (1991). *Manual for the spiritual well-being scale*. Nayack: Life Advance.

Peterman, A. H., Fitchett, G., Brady, M. J., Hernandez, L., & Cella, D. (2002). Measuring spiritual well-being in people with cancer: The functional assessment of chronic illness therapy–Spiritual Well-being Scale (FACIT-Sp). *Annals of Behavioral Medicine, 24*, 49–58.

Piedmont, R. L. (1999). Does spirituality represent the sixth factor of personality? Spiritual transcendence and the five-factor model. *Journal of Personality, 67*, 985–1013.

Richards, P. S., Smith, T., Schowalter, M., Richard, M., Berrett, M. E., & Hardman, R. K. (2005). Development and validation of the theistic spiritual outcome survey. *Psychotherapy Research, 15*, 457–469.

Rosmarin, D. H., Pargament, K. I., & Flannelly, K. J. (2009). Do spiritual struggles predict poorer physical/mental health among Jews? *The International Journal for the Psychology of Religion, 19*, 244–258.

Sawatzky, R., Ratner, P. A., & Chiu, L. (2005). A meta-analysis of the relationship between spirituality and quality of life. *Social Indicators Research, 72*, 153–188.

Underwood, L. G., & Teresi, J. A. (2002). The daily spiritual experience scale: Development, theoretical description, reliability, exploratory factor analysis and preliminary construct validity using health-related data. *Annals of Behavioral Medicine, 24*, 22–33.

Wachholtz, M. A. B., & Pargament, K. I. (2009). Migraines and meditation: Does spirituality matter? *Journal of Behavioral Medicine, 3*, 351–366.

Spirituality and Health

Kevin S. Masters
Department of Psychology, University of Colorado, Denver, CO, USA

Synonyms

Faith and health; Religiousness and health

Definition

Spirituality is an elusive term for which definitional consensus has yet to be reached. Many definitions include concepts concerning that which is beyond the material world and may include features of life that are not commonly perceptible by the physical senses. This is sometimes said to include a search for the sacred or belief in the transcendent. Common themes in definitions of spirituality include connectedness or relationship, subjectivity, personal experience, behaviors reflecting sacred or secular beliefs, and belief in something transcendent. This entry regards spirituality as related to health, an area of significantly increased research activity over the last 15–20 years.

Description

Over the last 15–20 years, scholars have become significantly more interested in determining if there is a relationship between spirituality and health, what the strength of this relationship might be, and if the relationship varies depending on the specific dimensions of both spirituality and health under consideration. Clearly both spirituality and health are multidimensional constructs, and thus, any general statement on their relationship will be an oversimplification.

The first major problem that investigators have in this area is, indeed, defining both terms. In this entry, the definitional focus is on spirituality. An important first question pertains to the similarities and differences between religion and spirituality. Most scholars currently agree that they are related, but not synonymous constructs. This understanding diverges from the historic conceptualization that viewed spirituality and religion as indivisible entities but coincides temporally with the increasing secularization of Western culture (Zinnbauer, Pargament, & Scott, 1999). Compared to spirituality, religion is often viewed as including more organized social or group practices with well-defined rituals, doctrinal creeds, and statements of faith. Spirituality, on the other hand, is thought to be more subjective and personal, lacking in the organizational elements that characterize religion. Shahabi et al. (2002) reported that 10% of individuals in a US national stratified sample classified themselves as spiritual, but not religious. Nevertheless, most highly religious individuals note that their spirituality is pursued within the context of their religion. Further, a common theme for definitions of both religion and spirituality is reference to a higher power, and 70% of spirituality definitions referred to traditional concepts of God, Christ, and the church as constituting what is sacred (Zinnbauer et al., 1997). Based on a review of the nursing literature, Emblen (1992) defined spirituality as "a personal life principle which animates a transcendent quality of relationship with God" (p. 45). More recently, Saucier and Skrzypinska (2006) demonstrated that subjective spirituality and tradition-oriented religiousness are empirically independent and correlate quite distinctly with personality dimensions. It has also been observed that neither spirituality nor religiousness can be simply reduced to a commonly measured personality variable (Piedmont, 1999; Saroglou & Muñoz-García, 2008; Saucier & Skrzypinska, 2006).

Given the definitional problems with spirituality, it is not surprising that there are measurement concerns as well. Recently, Kapuscinski and Masters (2010) reviewed work in this area. They not only observed the lack of

definitional consensus and related measurement confusion but also suggested that, given the dearth of lexical studies of spirituality, it is quite likely that the spiritual construct as defined by researchers differs from popular understanding and use of the term.

Clearly, these basic problems limit the extent that strong statements can be made regarding spirituality and health. At this point, most of the research has not focused on spirituality per se, as differentiated from religiousness, but rather has used measures such as religious service attendance as proxies for spirituality or has confounded religion and spirituality often using the R/S naming convention to represent both simultaneously and therefore demonstrate that no attempt at conceptual separation was made. There is significant research suggesting that some practices that are often associated with spirituality or considered behavioral indicators of spirituality can be effective. For example, meditation has a strong history of beneficial findings in the area of stress management and has been an important component in some major lifestyle interventions such as the Ornish Lifestyle Heart Trial (Ornish et al., 1983, 1990). Nevertheless, intervention studies that include a specifically spiritual intervention are almost nonexistent, and observational research, even if longitudinal, is severely limited in considering cause and effect relations. For example, it is clear that people tend to pray more when they are ill (negative relationship with health), but it is quite likely that it is the illness that is "causing" the increase in prayer rather than prayer having a negative impact on health. Investigations on frequency of prayer and health are, at this point, nonconclusive and largely characterized by cross-sectional research designs of self-report data. A few studies have investigated the content of prayer and found that, for example, self-esteem is higher among older adults when they pray, believing that only God knows when and how to best answer prayer (Krause, 2004). Krause (2003) also found that prayer for material things did not alleviate the burden of financial strain on physical health. But these are very preliminary findings, and much work in this area remains.

Finally, there are many behavioral and cognitive practices that could be considered either spiritual or not spiritual depending on how they are conceptualized and contextualized. These include concepts such as gratitude, forgiveness, relaxation, compassion, hope, optimism, faith, and connectedness among others. The extent that these may be considered aspects of spirituality depends on many factors notably the extent to which they are conceptualized as aspects of relating to the transcendent or sacred.

Cross-References

▶ Spiritual Beliefs
▶ Spirituality, Measurement of

References and Readings

Emblen, J. D. (1992). Religion and spirituality defined according to current use in nursing literature. *Journal of Professional Nursing, 8*, 41–47. doi:10.1016/8755-7223(92)90116-G.

Kapuscinski, A. N., & Masters, K. S. (2010). The current status of measures of spirituality: A critical review of scale development. *Psychology of Religion and Spirituality, 2*, 191–205.

Krause, N. (2003). Praying for others, financial strain, and physical health status in late life. *Journal for the Scientific Study of Religion, 42*, 377–391.

Krause, N. (2004). Assessing the relationships among prayer expectancies, race, and self-esteem in late life. *Journal for the Scientific Study of Religion, 42*, 395–408.

Ornish, D., Brown, S. E., Scherwitz, L. W., Billings, J. H., Armstrong, W. T., Ports, T. A., et al. (1990). Can lifestyle changes reverse coronary heart disease? The lifestyle heart trial. *Lancet, 336*, 129–133.

Ornish, D., Scherwitz, L. W., Doody, R. S., Kesten, D., McLanahan, S. M., Brown, S. E., et al. (1983). Effects of stress management training and dietary changes in treating ischemic heart disease. *JAMA: The Journal of the American Medical Association, 249*, 54–59.

Piedmont, R. L. (1999). Does spirituality represent the sixth factor of personality? Spiritual transcendence and the five-factor model. *Journal of Personality, 67*, 983–1013. doi:10.1111/1467-6494.0080.

Saroglou, V., & Muñoz-García, A. (2008). Individual differences in religion and spirituality: An issue of personality traits and/or values. *Journal for the Scientific Study of Religion, 47*, 83–101.

Saucier, G., & Skrzypinska, K. (2006). Spiritual but not religious? Evidence for two independent dispositions. *Journal of Personality, 74*, 1257–1292. doi:10.1111/j.1467-6494.2006.00409.x.

Shahabi, L., Powell, L. H., Musick, M. A., Pargament, K. I., Thoresen, C. E., Williams, D., et al. (2002). Correlates of self-perception of spirituality in American adults. *Annals of Behavioral Medicine, 24*, 59–68. doi:10.1207/S15324796ABM2401_07.

Zinnbauer, B. J., Pargament, K. I., Cole, B., Rye, M. S., Butter, E. M., Belavich, T. G., et al. (1997). Religion and spirituality: Unfuzzying the fuzzy. *Journal for the Scientific Study of Religion, 36*, 549–564. doi:10.2307/1387689.

Zinnbauer, B. J., Pargament, K. I., & Scott, A. B. (1999). The emerging meanings of religiousness and spirituality: Problems as prospects. *Journal of Personality, 67*, 889–919. doi:10.1111/14676494.

Spirituality, Measurement of

Afton N. Kapuscinski
Psychology Department, Syracuse University, Syracuse, NY, USA

Definition

Quantitative assessment of spirituality, typically in the form of self-report questionnaires.

Description

Measuring spirituality poses a serious challenge to research in the psychology of religion and spirituality. The principal source of difficulty is researchers' inability to reach a consensus on how to define spirituality, especially regarding its relationship to the concept of religiousness (Kapuscinski & Masters, 2010). As language in the United States has shifted over the past few decades, such that religion and spirituality are no longer considered to be synonymous, researchers have created instruments intended to measure spirituality as a concept distinct from religiousness. However, the definitions used to generate measures of spirituality are highly diverse, resulting in some instruments that appear to share little overlap in content (Kapuscinski & Masters, 2010). For instance, some measures define spirituality using language stemming from traditional religious institutions (e.g., "God"), whereas others include no reference to the transcendent or sacred in their conceptualizations. An additional concern is that some themes in the way researchers tend to understand spirituality stand in contrast to how the term is used in common language by the general public (Hill & Pargament, 2003; Zinnbauer, Pargament & Scott, 1999). Specifically, researchers tend to sharply divide spirituality from religion, such that religion is considered to be comprised of external behaviors (e.g., religious service attendance) and is associated with formal institutions, whereas spirituality is comprised of personal, inner experience that is separate from organized religion. Further, researchers are inclined to view spirituality as a health-promoting quality associated with positive psychological and societal benefits, and religiousness, in contrast, is perceived to be restrictive and unhealthy. The empirical literature, however, indicates that non-researchers regard the concepts as overlapping considerably and view both religiousness and spirituality as positive qualities. Moreover, researchers' connotations are not consistent with research linking both religion and spirituality to positive mental and physical health outcomes (Masters & Hooker, 2011).

Further, theoretical concerns arise regarding the feasibility of quantifying and operationally defining a concept that seems to be, by its nature, highly experiential and personalized. The literature indicates that like researchers, individuals differ in how they understand spirituality (Zinnbauer et al., 1999). Interestingly, the majority of researchers do not consult participants regarding conceptualization of spirituality when developing new measures (Kapuscinski & Masters, 2010). Miller and Thoresen (2003) comment that from the believer's perspective, scientists can at best explore mere reflections of

spirituality, which approximate but never fully capture its essence.

The disagreement regarding how to conceptualize spirituality is implied by the sheer number of available spirituality instruments. Reviews of measures (Hill & Hood, 1999; MacDonald, LeClair, Holland, Alter, & Friedman, 1995; MacDonald, Friedman, & Kuentzel, 1999) indicate that over 100 measures exist that are designed to measure a variety of spiritual constructs, including spirituality, intrinsic spirituality, spiritual experiences, spiritual meaning, spiritual development, spiritual transcendence, spiritual transformation, and spiritual well-being. Most measures appear to address interest in or search for the sacred and focus on assessing cognitive (e.g., meaning, beliefs, values) or emotional (e.g., peace, hope, connection) experiences associated with the sacred. Several high-quality measures should be considered as appropriate for use in health psychology research. The FACIT Spiritual Well-Being Scale (Peterman, Fitchett, Brady, Hernandez, & Cella, 2002) was designed specifically to assess aspects of spirituality relevant to quality of life for chronically ill individuals. The Daily Spiritual Experiences Scale (Underwood & Teresi, 2002) includes language that is relevant to individuals from various faith traditions and has demonstrated a relationship to important health variables such as alcohol consumption and depression. Additionally, the Spiritual Transcendence Scale (Piedmont, 1999), which does not include language associated with institutional religion, is noteworthy for its high-quality scale development and validation practices and includes an observer report form. When selecting a measure for use, researchers should carefully consider whether or not the content of scale items is consistent with the conceptualization of spirituality relevant to their study and population of interest.

Cross-References

- ▶ Spiritual Beliefs
- ▶ Spirituality
- ▶ Spirituality and Health

References and Readings

Hill, P. C., & Hood, R. W. (Eds.). (1999). *Measures of religiosity*. Birmingham, AL: Religious Education.

Hill, P. C., & Pargament, K. I. (2003). Advances in the conceptualization of religiousness and spirituality: Implications for physical and mental health research. *American Psychologist, 58*, 64–74. doi:10.1037/0003-066X.58.1.64.

Kapuscinski, A. N., & Masters, K. S. (2010). The current status of measures of spirituality: A critical review of scale development. *Psychology of Religion and Spirituality, 2*, 191–205. doi:10.1037/a0020498.

MacDonald, D. A., Friedman, H. L., & Kuentzel, J. G. (1999). A survey of measures of spiritual and transpersonal constructs: Part one – research update. *Journal of Transpersonal Psychology, 31*, 137–154.

MacDonald, D. A., LeClair, L., Holland, C. J., Alter, A., & Friedman, H. L. (1995). A survey of measures of transpersonal constructs. *Journal of Transpersonal Psychology, 27*, 171–235.

Masters, K. S., & Hooker, S. A. (2011). Impact of religion and spirituality on health. In J. Aten, K. O'Grady, & E. Worthington (Eds.). The psychology of religion and spirituality for clinicians: Using research in your practice (357–386). New York: Routledge.

Miller, W. R., & Thoresen, C. E. (2003). Spirituality, religion and health: An emerging research field. *American Psychologist, 58*, 24–35. doi:10.1037/0003-066X.58.1.24.

Peterman, A. H., Fitchett, G., Brady, M. J., Hernandez, L., & Cella, D. (2002). Measuring spiritual well-being in people with cancer: The functional assessment of chronic illness therapy – spiritual well-being scale (FACIT-Sp). *Annals of Behavioral Medicine, 24*, 49–58. doi:10.1207/S15324796ABM2401.

Piedmont, R. L. (1999). Does spirituality represent the sixth factor of personality? Spiritual transcendence and the five-factor model. *Journal of Personality, 67*, 986–1013. doi:10.1111/1467-6494.00080.

Underwood, L. G., & Teresi, J. A. (2002). The daily spiritual experience scale: Development, theoretical description, reliability, exploratory factor analysis and preliminary construct validity using health-related data. *Annals of Behavioral Medicine, 24*, 22–33. doi:10.1207/S15324796ABM2401_04.

Zinnbauer, B. J., Pargament, K. I., & Scott, A. B. (1999). The emerging meanings of religiousness and spirituality: Problems as prospects. *Journal of Personality, 67*(6), 889–919. doi:10.1111/1467-6494.00077.

Squamous Cell Carcinoma of the Cervix (SCCC)

- ▶ Cancer, Cervical

Stages-of-Change Model

Jonathan A. Shaffer
Department of Medicine/Division of General Medicine, Columbia University Medical Center, New York, NY, USA

Definition

The Stages-of-Change Model was developed by James Prochaska and Carlo DiClemente as a framework to describe the five phases through which one progresses during health-related behavior change (Prochaska & DiClemente, 1983). It is part of their broader Transtheoretical Model, which not only assesses an individual's readiness to act to eliminate a problem behavior but also includes strategies and processes of change to guide the individual through the stages. The Stages-of-Change Model originated in research related to psychotherapy and the cessation of addictive behaviors, such as smoking, alcohol and substance abuse, and issues related to weight management (Buxton, Wyse, & Mercer, 1996). Although Prochaska and DiClemente initially hypothesized that individuals progress linearly through a series of discrete stages of change, researchers now believe that a cyclical or "spiral" pattern more accurately represents how most people change unhealthy behavior over time. Since its development, the Stages-of-Change Model has been related to a variety of problem behaviors, associated with treatment outcomes, and integrated in stage-based interventions. Although most scientists and clinicians agree that the model has heuristic value, it has been criticized by some researchers.

Description

History of the Model

Stage theories have been integral to the field of psychology since its inception and include Freud's and Erikson's psychosexual stages, Kohlberg's stages of moral development, Piaget's stages of cognitive development, and Maslow's hierarchy of needs (Dolan, 2005). DiClemente and Prochaska's Stages-of-Change Model uses language similar to Horn. Specifically, Horn hypothesized four stages of change associated with health-related behavior: (1) contemplating change, (2) deciding to change, (3) short-term change, and (4) long-term change (Horn, 1976). DiClemente and Prochaska initially identified four stages of changes associated with smoking cessation and maintenance: (1) thinking about change (contemplation), (2) becoming determined to change (decision making), (3) actively modifying behavior and/or environment (action), and (4) maintaining new behaviors (maintenance). Precontemplation was later identified as a separate stage preceding contemplation.

Description of Stages

Precontemplation. The individual in the precontemplation stage has no intention to change his or her behavior in the foreseeable future (Prochaska & Norcross, 2001). Although individuals are unaware of their problems, their families, friends, neighbors, and employees are often very aware of these problems. Individuals presenting for treatment in the precontemplation stage generally do so because of pressure from others.

Contemplation. During the contemplation stage, individuals are aware that a problem exists and seriously consider overcoming it. However, they have not yet made a commitment to do so. According to Prochaska and Norcross, individuals often remain stuck in this stage for long periods.

Preparation. The preparation stage combines intention and behavioral criteria. Individuals in the preparation stage intend to enact change in the next month and have unsuccessfully attempted to do so in the past year. These individuals report small behavioral changes, but they have not yet reached a criterion for effective action, such as abstinence from smoking or sufficient weight loss. These individuals do intend to take action in the very near future.

Action. Individuals in the action stage modify their behavior, experiences, and environment in order to overcome their problems. This stage involves the most overt behavioral changes and requires considerable commitment of time and energy. Modifications of the problem behaviors made in this stage are most visible to others and tend to elicit others' recognition. Individuals in this stage must have successfully altered their problem behavior for a period of 1 day to 6 months.

Maintenance. Individuals in the maintenance stage concentrate on preventing relapse and consolidating the gains attended during the previous stage. These individuals must have remained free of their problem behavior and consistently engaged in a new incompatible behavior for more than 6 months.

Termination. Individuals who reach this stage have completed the change process and no longer have to work to prevent relapse. This stage involves total confidence or self-efficacy across all high-risk situations and no temptation to relapse.

Assessing Stages of Change

Multiple ways for measuring stage of change have been proposed and devised, and researchers/clinicians usually assign people to stages on the basis of their responses to questions concerning their prior behavior and current behavioral intentions (Weinstein, Rothman, & Sutton, 1998). A Stages-of-Change Questionnaire has been developed as a brief and reliable instrument for measuring stages of change in psychotherapy and has been adopted to evaluate stages of change for specific problem behaviors (McConnaughy, Prochaska, & Velicer, 1983). This continuous measure includes questions such as "As far as I'm concerned, I don't have problems that need changing" (precontemplation), "I have a problem and I really think I should work on it" (contemplation), "I am working really hard to change" (action), and "I may need a boost right now to help me maintain the changes I've already made" (maintenance). Given that attributes that define the stages of change are mainly internal to the individual (e.g., beliefs, plans, attributions), measurement is often imperfect (Weinstein, Rothman, & Sutton, 1998).

Stages of Change and Specific Health Behaviors

The Stages-of-Change Model has been used to understand a variety of problem behaviors including smoking cessation, cessation of cocaine use, weight control, high-fat food consumption, adolescent delinquent behaviors, risky sexual behaviors, sunscreen use, radon gas exposure, exercise acquisition, mammography screening, and physicians' preventive practices with smokers (Prochaska et al., 1994).

Stage-Based Treatments

The Stages-of-Change Model has been use to aid in treatment planning and to develop stage-based treatments. Prochaska (1991) has argued that a person's stage of change provides proscriptive and prescriptive information about appropriate treatments (Prochaska, 1991). For example, those who are in the preparation or action stages presumably benefit from action-oriented therapies, whereas those in the precontemplation or contemplation stages likely may benefit more from insight-oriented, consciousness raising interventions.

Several interventions based on stage-based models of change have been developed to modify risk behaviors. These interventions are tailored to take into account the current stage an individual has reached in the change process in contrast to "one size fits all" interventions. A systematic review of these interventions identified 37 RCTs of such interventions aimed at smoking cessation, promotion of physical activity, dietary change, multiple lifestyle changes, mammography screening, and treatment adherence (Riemsma et al., 2002). The authors of this review concluded that there is little evidence to suggest that stage-based interventions are more effective compared to non-stage-based interventions, no intervention, or usual care. Nonetheless, they recommend additional studies of tailored interventions which involve frequent reassessment of patients' readiness to change. Reviews of

stage-based lifestyle interventions in primary care (Van Sluijs, Van Poppel, & Van Mechelen, 2004) and stage-based interventions for smoking cessation (Riemsma et al., 2003) have likewise resulted in limited scientific evidence in support of these interventions.

Revised Stages-of-Change Model

Freeman and Dolan (2001) offered a revised Stages-of-Change Model with five additional changes to more precisely determine a psychotherapy patient's position on the continuum of change (Freeman & Dolan). This revised model has been recommended as a more dynamic and flexible one that provides clinicians with a more experience-centered focus from which to make treatment decisions.

Non-contemplation is the stage during which an individual is not considering or even thinking about changing. These individuals do not actively avoid, resist, or oppose change; they are rather unaware of their need to change or of the effect their behavior has on others. Anti-contemplation involves the process of becoming reactive and violently opposed to the notion of needing change, a response often seen in individuals who are legally mandated to attend treatment or who come to treatment at the urging of their family, friends, or significant others. Freeman and Dolan's precontemplation and contemplation stages are identical to those of Prochaska and DiClemente. Their action planning stage occurs when the clinician and patient have collaboratively developed a treatment focus and treatment plan. Patients in this stage are actively willing to plan change, and next progress to the action stage of Prochaska and DiClemente's model. Prelapse, lapse, and relapse stages may then occur prior to the maintenance stage. During prelapse, an individual experiences overwhelming thoughts, desires, and cravings to engage in the problem behavior. During lapse, the skills needed to maintain the action stage decrease or are ignored. During relapse, the individual returns to the problem behavior. As in Prochaska and DiClemente's model, the maintenance stage occurs when the individual actively works toward maintaining the cessation of the problem behavior.

Evaluation of the Stages-of-Change Model

The Stages-of-Change Model is not without criticism. Those who have criticized the model argue that its inherent concept of discrete stages involves arbitrary distinctions; it falsely assumes that individuals make coherent and stable plans, and it neglects the role of reward, punishment, and associative learning that contribute to the maintenance of problem behaviors (West, 2005). Others have found that minimal supportive evidence for the Stages-of-Change Model exists (Whitelaw, Baldwin, Bunton, & Flynn, 2000) and questioned the model's internal validity (Ahijevych & Wewers, 1992; Bandura, 1997; Farkas et al., 1996), external validity (Clarke & Eves, 1997), and ethical difficulties associated with interventions derived from the Stages-of-Change Model (Piper & Brown, 1998).

Notwithstanding the criticisms and absence of evidence discussed above, the Stages-of-Change Model provides a pragmatic framework for practitioners and clinical researchers, and it is intuitively appealing to many in the fields of clinical psychology, behavioral medicine, public health, and other fields. Future research promises to offer improved measurement of stages, qualitative case studies of practitioner utilization, and process-based implementation evaluation of the model in various settings (Whitelaw, Baldwin, Bunton, & Flynn, 2000).

Cross-References

- ▶ Health Beliefs/Health Belief Model
- ▶ Transtheoretical Model of Behavior Change

References and Readings

Ahijevych, K., & Wewers, M. (1992). Processes of change across five stages of smoking cessation. *Addictive Behaviors, 17*(1), 17–25.

Bandura, A. (1997). Editorial: The anatomy of stages of change. *American Journal of Health Promotion, 12*, 8–10.

Buxton, K., Wyse, J., & Mercer, T. (1996). How applicable is the stages of change model to exercise behaviour? A review. *Health Education Journal, 55*(2), 239–256.

Clarke, P., & Eves, F. (1997). Applying the transtheoretical model to the study of exercise on prescription. *Journal of Health Psychology, 2*(2), 195–207.

Dolan, M. (2005). Stages of change. In A. Freeman et al. (Eds.), *Encyclopedia of cognitive behavior therapy* (pp. 387–390). New York: Springer.

Farkas, A., Pierce, J., Zhu, S., Rosbrook, B., Gilpin, E., Berry, C., et al. (1996). Addiction versus stages of change models in predicting smoking cessation. *Addiction, 91*(9), 1271–1280.

Freeman, A., & Dolan, M. (2001). Revisiting Prochaska and DiClemente's stages of change theory: An expansion and specification to aid in treatment planning and outcome evaluation. *Cognitive and Behavioral Practice, 8*(3), 224–234.

Horn, D. (1976). A model for the study of personal choice health behavior. *International Journal of Health Education, 19*(1), 89–98.

McConnaughy, E., Prochaska, J., & Velicer, W. (1983). Stages of change in psychotherapy: Measurement and sample profiles. *Psychotherapy: Theory, Research & Practice, 20*(3), 368–375.

Piper, S., & Brown, P. (1998). Psychology as a theoretical foundation for health education in nursing: empowerment or social control? *Nurse Education Today, 18*(8), 637–641.

Prochaska, J. (1991). Prescribing to the stage and level of phobic patients. *Psychotherapy: Theory, Research, Practice, Training, 28*(3), 463.

Prochaska, J., & DiClemente, C. (1983). Stages and processes of self-change of smoking: toward an integrative model of change. *Journal of Consulting and Clinical Psychology, 51*(3), 390–395.

Prochaska, J., & Norcross, J. (2001). Stages of change. *Psychotherapy: Theory, Research, Practice, Training, 38*(4), 443.

Prochaska, J., Velicer, W., Rossi, J., Goldstein, M., Marcus, B., Rakowski, W., et al. (1994). Stages of change and decisional balance for 12 problem behaviors. *Health Psychology, 13*, 39–39.

Riemsma, R., Pattenden, J., Bridle, C., Sowden, A., Mather, L., Watt, I., et al. (2002). A systematic review of the effectiveness of interventions based on a stages-of-change approach to promote individual behaviour change. *Health Technology Assessment, 6*(24), 1–231.

Riemsma, R., Pattenden, J., Bridle, C., Sowden, A., Mather, L., Watt, I., et al. (2003). Systematic review of the effectiveness of stage based interventions to promote smoking cessation. *British Medical Journal, 326*(7400), 1175.

Van Sluijs, E., Van Poppel, M., & Van Mechelen, W. (2004). Stage-based lifestyle interventions in primary care: Are they effective? *American Journal of Preventive Medicine, 26*(4), 330–343.

Weinstein, N., Rothman, A., & Sutton, S. (1998). Stage theories of health behavior: Conceptual and methodological issues. *Health Psychology, 17*, 290–299.

West, R. (2005). Time for a change: Putting the transtheoretical (stages of change) model to rest. *Addiction, 100*(8), 1036–1039.

Whitelaw, S., Baldwin, S., Bunton, R., & Flynn, D. (2000). The status of evidence and outcomes in stages of change research. *Health Education Research, 15*(6), 707–718.

Standard Deviation

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

A simple measure of dispersion is the range, the arithmetic difference between the greatest (maximum) and the least (minimum) value in a data set. While this characteristic is easily calculated and useful in initial inspections of data sets, by definition it only uses two of the values in a data set. In a large data set most pieces of numerical information are therefore not used in the calculation of the range, and it is not known whether many data points lie close to the minimum, maximum, or mean, or in any other distribution pattern.

Two more sophisticated measures of dispersion are variance and the standard deviation. These measures are intimately related to each other and take account of all values in a data set. The calculation of variance involves calculating the deviation of each data point from the mean of the data set, squaring these values, and summing them. The process of squaring the deviation is mathematically necessary: If the raw deviations were to be summed they would always sum to zero. However, the squaring process creates the problem that the units of measurement of variance are not the same as the units of measurement of the original data. In the vast majority of cases, the data points in our studies are not simply numbers, but numerical representations of information measured in certain units. For example, a systolic blood pressure measurement of "125"

is actually a measurement of 125 millimeters of mercury (mmHg). Since the calculation of variance involves squaring certain values, the variance of a set of blood pressure data points would actually be measured in squared millimeters of mercury, a nonsensical unit.

Fortunately, this problem can be solved by simply calculating the square root of the variance. The resulting value is called the standard deviation (SD), and the unit of measurement of the SD is the same as the unit of measurement of the original data points. The SD is a very commonly presented descriptor in research studies. It is usually presented in conjunction with the mean in the form "mean ± SD."

Cross-References

► Variance

Standard Normal (Z) Distribution

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Z distribution

Definition

The Standard Normal distribution, also known as the Z distribution, is one particular form of the Normal distribution in which the mean is zero (i.e., 0) and the variance is unity (i.e., 1). This can be written as ($\mu = 0, \sigma = 1$).

Before presenting the Z distribution, it is necessary to discuss the Normal distribution in general. Imagine that the heights of a large number of adult males (or females) are measured and the results plotted as a histogram. Height is plotted in inches on the x-axis and the number of people within each height category is plotted on the y-axis. There would be many more people close to the middle of the histogram than close to either end, since more individuals are close to the mean height, and very few are very tall or very short. Given a large sample and decreasingly thin bars in the histogram (that is, the width of the measurement intervals along the *x*-axis becomes infinitely small such that the height data become continuous), a curve can be superimposed on this histogram. One particular version of a density curve is called the Normal distribution. This distribution is of considerable interest since height and many physiological variables conform very closely (but not perfectly) to this distribution. Since the word normal is used in everyday language, and since its meaning in Statistics is different and important, the word is written in this entry with an upper case N when it is used in its statistical sense.

The Normal distribution has several notable properties:

- The highest point of the Normal curve occurs for the mean of the population. The properties of the Normal distribution ensure that this point is also the median value and the mode.
- The shape of the Normal curve (relatively narrow or relatively broad) is influenced by the standard deviation (SD) of the data. The sides of the curve descend more gently as the standard deviation increases and more steeply as it decreases.
- At a distance of approximately ±2 SDs from the mean, the slopes of the downward curves change from a relatively smooth downward slope to a curve that extends out to infinity and thus never quite reaches the *x*-axis. For practical purposes, the curve is often regarded as intercepting the *x*-axis at a distance of ±3 SDs from the mean, but this is an approximation.

Area Under the Normal Curve

The area under the Normal curve is of considerable interest in the discipline of Statistics. That is, it is of considerable interest to define and quantify the area bounded by the Normal curve at the top and the *x*-axis at the bottom. This area will be defined as

1.00, or as 100%. Given this interest, the final bullet point in the previous list raised an issue that appears problematic. That is, it appears that, if the two lower slopes of the Normal curve never quite reach the *x*-axis, the area under the curve is never actually fully defined and can therefore never be calculated precisely. Fortunately, this apparent paradox can be solved mathematically.

The solution is related to the observation that the sum of an infinite series can converge to a finite solution. An example that effectively demonstrates the solution here is the geometric series "1/2 + 1/4 + 1/8 + . . . *ad infinitum*." That is, the series starts with 1/2, and every subsequent term is one half of the previous term. Given this, the terms of the series never vanish to zero. However, the sum of them is precisely 1.00. The proof of this is as follows, where the series is represented as *S*:

$$S = 1/2 + 1/4 + 1/8 + \ldots ad\ infinitum \quad (1)$$

Both sides of this equation are then multiplied by the same value, namely, 2 (multiplying both sides of an equation by a constant means that the sides are still of equal value):

$$2S = 1 + 1/2 + 1/4 + \ldots ad\ infinitum \quad (2)$$

The value *S* is then subtracted from both sides (subtracting a constant from both sides of an equation means that the sides are still of equal value). First, consider the left-hand side (LHS) of Eq. 2:

LHS of Eq. 2: $2S - S$, which equals S

Now consider the right-hand side (RHS) of Eq. 2. Subtracting *S* from this quantity can be represented as:

RHS of Eq. 2: (1 + 1/2 + 1/4 + . . . *ad infinitum*) − *S*, which equals what, exactly?

To determine the unknown value we can use Eq. 1, which shows that *S* is equal to (1/2 + 1/4 + 1/8 + . . . *ad infinitum*). Therefore, the right-hand side of Eq. 2 can be written as follows:

RHS of Eq. 2: (1 + *S*) − *S*, which equals 1

Equation 2 can therefore be rewritten as:

$$S = 1$$

Therefore, despite the initial paradoxical nature of the statement, it can indeed be shown that the sum of an infinite series can converge to a finite solution.

Returning to the topic of immediate interest, i.e., the area under the Normal curve, the statement that the terms of the geometric series never vanish to zero can be reinterpreted in this context as saying that the curves of the Normal curve never intercept the *x*-axis. Despite this statement, however, an adaptation of the proof just provided shows that the area under the Normal curve is indeed precisely equal to 1.00, or 100%. The visual equivalent of this is that there is indeed a defined area under the Normal curve, bounded by the curve and the *x*-axis, and the value of this area can be represented as 1.00, or 100%. This can be demonstrated formally using integral calculus. It can also be thought of as analogous to the statement that the probability of all mutually exclusive events must sum to 1.00.

It is of particular interest in Statistics that the means of many large samples taken from a particular population are approximately distributed in this Normal fashion, i.e., they are said to be Normally distributed. This is true even when the population data themselves are not Normally distributed. The mathematical properties of a true Normal distribution allow quantitative statements of the area under the curve between any two points on the *x*-axis. It was just demonstrated that the total area under the Normal curve is 1, or 100%. It is also of interest to know the proportion of the total area under the curve that lies between two points that are equidistant from the mean. These points are typically represented by multiples of the standard deviation (SD). From the properties of the mathematical equation that governs the shape of the Normal curve, it can be shown that:

- The central 90% of the area under the curve lies between the mean ±1.645 SDs
- The central 95% of the area under the curve lies between the mean ±1.960 SDs
- The central 99% of the area under the curve lies between the mean ±2.576 SDs

The area under the curve is representative of the number of data points falling within that

range. That is, the percentage of the area under the curve translates directly into the percentage of data points falling between the two identified points. Of particular relevance for many research studies is that 95% of the area under the curve lies between the mean ±1.960 SDs. The value of 1.960 is often rounded up to 2, leading to the statement in many practical examples in textbooks that 95% of the data points fall within the mean ±2 SDs.

The Z Distribution

The Z distribution is such that the mean and the standard deviation in the immediately preceding statements can be removed, leading to the following statements:

- The central 90% of the area under the curve lies between ±1.645.
- The central 95% of the area under the curve lies between ±1.960.
- The central 99% of the area under the curve lies between ±2.576.

This distribution is used extensively in Statistics, and underpins many more complex statistical procedures (Durham & Turner, 2008).

Cross-References

- ▶ Data
- ▶ Hypothesis Testing
- ▶ Median
- ▶ Mode
- ▶ Probability
- ▶ Standard Deviation

References and Readings

Durham, T. A., & Turner, J. R. (2008). *Introduction to statistics in pharmaceutical clinical trials*. London: Pharmaceutical Press.

State Anxiety

- ▶ Anxiety

Static Exercise

- ▶ Isometric/Isotonic Exercise

Statins

Ken Ohashi
Department of General Internal Medicine, National Cancer Center Hospital, Chuo-ku, Tokyo, Japan

Synonyms

HMG-CoA reductase inhibitors

Definition

Statins, also known as HMG-CoA reductase inhibitors, are a class of cholesterol lowering agents that are prescribed worldwide to hyperlipidemic patients who are at high risk for cardiovascular disease. Statins currently on the market include pravastatin, simvastatin, fluvastatin, atorvastatin, rosuvastatin, and pitavastatin. Statins exert their effect through inhibition of 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase, the rate-limiting enzyme of cholesterol biosynthesis in the liver. In many clinical trials, statins have been beneficial both in the primary and secondary prevention of coronary heart disease. Recent clinical and experimental data suggest that the benefit of statins may extend beyond their lipid lowering effects. Those cholesterol-independent or "pleiotropic" effects of statins involve improving endothelial dysfunction, enhancing the stability of atherosclerotic plaques, decreasing oxidative stress and inflammation, and inhibiting the thrombogenesis.

Cross-References

- ▶ Hyperlipidemia

Statistical Inference

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Inferential statistics

Definition

Statistical inference is a process of using the precise data and results from a specific group of subjects in a research study to infer the likely responses to the same treatment in the general population of patients who would receive the treatment or intervention if it entered general behavioral medicine practice.

The ultimate purpose of the results from a single behavioral medicine study, such as a randomized clinical trial, is not to tell us precisely what happened in that trial, but to gain insight into likely responses to the behavioral treatment or intervention in patients with the disease or condition of clinical concern who would receive the treatment. Inferential statistics allows us to do this.

The treatment effect calculated from the data in the single study is regarded as the treatment effect point estimate. Confidence intervals are then placed around this point estimate. The range of values between the lower limit and the upper limit of the confidence interval represents a range that covers the true but unknown population treatment effect with a specified degree of confidence.

Cross-References

► Hypothesis Testing
► Randomized Clinical Trial

Statistical Inquiry

► Surveys

Statistics

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

For present purposes, the discipline of Statistics (recognized here by the use of an upper case "S") can be usefully defined as an integrated discipline that is critically and fundamentally important in all of the following activities associated with clinical research in behavioral medicine:

- Identifying a research question that needs to be answered.
- Deciding upon the design of the study, the methodology that will be employed, and the numerical information (data) that will be collected.
- Presenting the design, methodology, and data to be collected in a study protocol. This study protocol specifies the manner of data collection and addresses all methodological considerations necessary to ensure the collection of optimum quality data for subsequent statistical analysis.
- Identifying the statistical techniques that will be used to describe and analyze the data in a section within the protocol or in an associated statistical analysis plan, which should be written in conjunction with the study protocol.
- Describing and analyzing the data. This includes analyzing the variation in the data to see if there is compelling evidence that the treatment is safe and effective. This process includes evaluation of the statistical significance of the results obtained and, very importantly, their clinical significance.

- Presenting the results of a clinical study to the research and clinical communities in conference talks and posters, and in journal publications.

Cross-References

- ▶ Clinical Decision-Making
- ▶ Hypothesis Testing
- ▶ Statistical Inference

Stem Cells

Keiki Kumano
Department of Cell Therapy and Transplantation Medicine, The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Definition

Stem cells are found in all multicellular organisms. They are defined by the ability to renew themselves through mitotic cell division (self-renewal) and to generate all the differentiated cell types of the tissue (multipotency). For this definition, one stem cell divides into one father cell that is identical to the original stem cell and another daughter cell that is differentiated.

The mammalian stem cells are divided into two broad types: embryonic stem cells and adult somatic stem cells. Embryonic stem cells are isolated from the inner cell mass of blastocysts, and adult somatic stem cells that are found in adult tissues. In a developing embryo, stem cells can differentiate into all of the specialized embryonic tissues. The stem cells can become any tissue in the body, excluding a placenta. Only the morula's cells are totipotent, able to become all tissues and a placenta.

In adult organisms, stem cells and progenitor cells act as a repair system for the body, replenishing specialized cells, but also maintain the normal turnover of regenerative organs, such as blood, skin, or intestinal tissues. In addition to the definition described above, adult stem cells are thought to be quiescent within the niche, dividing infrequently to generate one stem cell copy and a rapidly cycling cell (transient amplifying cell). Transient amplifying cells undergo a limited number of cell divisions and differentiate into the functional cells of the tissues.

Recently, the third stem cells, artificially established, induced pluripotent stem cells (iPSCs) are generated. Previously, nuclear transfer of embryo into the adult somatic cells is known to be able to reprogram the somatic cells. These are not adult stem cells but rather reprogrammed cells (e.g., epithelial cells) given pluripotent capabilities. So reprogramming factors are thought to exist in the embryo or embryonic stem cells. Using genetic reprogramming with transcription factors, pluripotent stem cells equivalent to embryonic stem cells have been derived from human adult tissue. Shinya Yamanaka and his colleagues used the transcription factors Oct3/4, Sox2, c-Myc, and Klf4 in their experiments on cells from human faces. Another groups used a different set of factors, Oct4, Sox2, Nanog, and Lin28, and more limited combination of these factors (Oct3/4, Sox2, Klf4 (OSK), OS, only Oct3/4) can reprogram the adult tissue.

Stem cell therapy has the potential to dramatically change the treatment of human disease. A number of adult stem cell therapies already exist, particularly bone marrow transplantation that is used to treat hematological disease (leukemia, lymphoma, etc.). In the future, stem cell therapy will be broadened to treat a wider variety of diseases including cancer, neurological diseases, several inherited diseases, and so on.

Cross-References

- ▶ Genetics
- ▶ Hematopoietic Stem Cell Transplantation

STEMI

- ▶ Acute Myocardial Infarction

Steptoe, Andrew (1951–)

Mika Kivimaki
Epidemiology & Public Health, University College London, London, WC1E 6BT, UK

Biographical Information

Andrew Steptoe was born in London on April 24, 1951. He is British Heart Foundation Professor of Psychology at University College London, UK, where he is also the Director of the Division of Population Health, a grouping of academic departments including Epidemiology and Public Health, Primary Care and Population Health, Infection and Population Health, and the Medical Research Council Clinical Trials Unit. Steptoe graduated in Natural Sciences from Cambridge in 1972, and completed his Doctorate at Oxford University in 1976. He was appointed lecturer in psychology at St. George's Hospital Medical School in 1977, becoming professor and chair of the Department in 1988. He moved to his present research chair at University College London in 2000, where he is the Director of the Psychobiology Group. Steptoe is also the Director of the English Longitudinal Study of Ageing, a population cohort of older men and women in England.

Major Accomplishments

Steptoe was one of the small group of behavioral medicine specialists, who developed the International Society of Behavioral Medicine (ISBM) in the 1980s. He served as the third President of the ISBM from 1994 to 1996. Additionally, he was the former President of the Society for Psychosomatic Research in the UK. He was the cofounding editor of the *British Journal of Health Psychology* along with Jane Wardle, and has served as an associate editor of *Psychophysiology*, the *Annals of Behavioral Medicine*, the *British Journal of Clinical Psychology*, and the *Journal of Psychosomatic Research*, and is on the editorial boards of six other journals.

Steptoe is the author of more than 550 journal articles and papers, and author or editor of 17 books, most recently the *Handbook of Behavioral Medicine* (Steptoe 2010) and *Stress and Cardiovascular Disease* (Hjemdahl, Rosengren, & Steptoe, 2012). His research has addressed many topics in behavioral medicine, including stress and health, socioeconomic status, the determinants of health behavior, health behavior change, cardiovascular disease, respiratory disorders, aging, and positive well-being. His collaborative research with Professor Marmot has focused on understanding the biological processes through which lower socioeconomic status and psychosocial risk factors influence cardiovascular disease risk. This work has involved laboratory studies of the influence of psychosocial factors on cardiovascular, neuroendocrine, and immune function, and naturalistic studies of blood pressure, cortisol, and other biological measures.

Steptoe has advanced our understanding of the psychobiology of health and diseases and the multiple associations between affect and biology in everyday life. He and his team found that people from lower socioeconomic groups tend to suffer the biological effects of stress for longer than more affluent people. This is a potential pathway linking low socioeconomic status with increased risk for coronary heart disease. Further studies by Steptoe's group showed that in some patients, intense episodes of anger and stress occurred in the hours immediately before the onset of chest pain. In experiment settings, episodes of mental stress, similar to those encountered in everyday life, were found to cause transient (up to 4 h) endothelial dysfunction in

healthy young individuals. These studies have been important in demonstrating the role of emotional factors in the triggering of coronary ischemia and acute coronary syndromes. Steptoe is also one of the leading scientists on the protective effects of positive affect in physical health.

Steptoe has received many honors for his work. He is a Fellow of the Society of Behavioral Medicine and the Academy of Behavioral Medicine Research in the USA, the Academy of Medical Sciences, the Academy of Learned Societies for the Social Sciences, and the British Psychological Society.

Cross-References

- ▶ Cardiovascular Disease
- ▶ Psychological Stress
- ▶ Socioeconomic Status (SES)
- ▶ Stress

References and Readings

Hjemdahl, P., Rosengren, A., & Steptoe, A. (Eds.). (2012). *Stress and cardiovascular disease*. London: Springer.

Kunz-Ebrecht, S. R., Kirschbaum, C., Marmot, M., & Steptoe, A. (2004). Differences in cortisol awakening response on work days and weekends in women and men from the Whitehall II cohort. *Psychoneuroendocrinology, 29*, 516–528.

Steptoe, A. (Ed.). (2006). *Depression and physical illness*. Cambridge: Cambridge University Press.

Steptoe, A. (Ed.). (2010). *Handbook of behavioral medicine*. New York: Springer.

Steptoe, A., & Appels, A. (Eds.). (1990). *Stress, personal control, and health*. Chichester: John Wiley.

Steptoe, A., Cropley, M., Griffith, J., & Kirschbaum, C. (2000). Job strain and anger expression predict early morning elevations in salivary cortisol. *Psychosomatic Medicine, 62*, 286–292.

Steptoe, A., Doherty, S., Rink, E., Kerry, S., Kendrick, T., & Hilton, S. (1999). Behavioural counselling in general practice for the promotion of healthy behaviour among adults at increased risk of coronary heart disease: Randomised trial. *BMJ, 319*, 943–947.

Steptoe, A., Hamer, M., & Chida, Y. (2007). The effects of acute psychological stress on circulating inflammatory factors in humans: A review and meta-analysis. *Brain, Behavior, and Immunity, 21*, 901–912.

Steptoe, A., Pollard, T. M., & Wardle, J. (1995). Development of a measure of the motives underlying the selection of food – The food choice questionnaire. *Appetite, 25*, 267–284.

Steptoe, A., & Wardle, J. (Eds.). (1994). *Psychosocial processes and health: A reader*. Cambridge: Cambridge University Press.

Steptoe, A., Wardle, J., & Marmot, M. (2005). Positive affect and health-related neuroendocrine, cardiovascular, and inflammatory processes. *PNAS, 102*, 6508–6512.

Steptoe, A., Willemsen, G., Owen, N., Flower, L., & Mohamed-Ali, V. (2001). Acute mental stress elicits delayed increases in circulating inflammatory cytokine levels. *Clinical Science, 101*, 185–192.

Stereotypes

- ▶ Stigma

Steroid Hormones

- ▶ Estrogen
- ▶ Steroids

Steroids

Sarah Aldred
School of Sport and Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

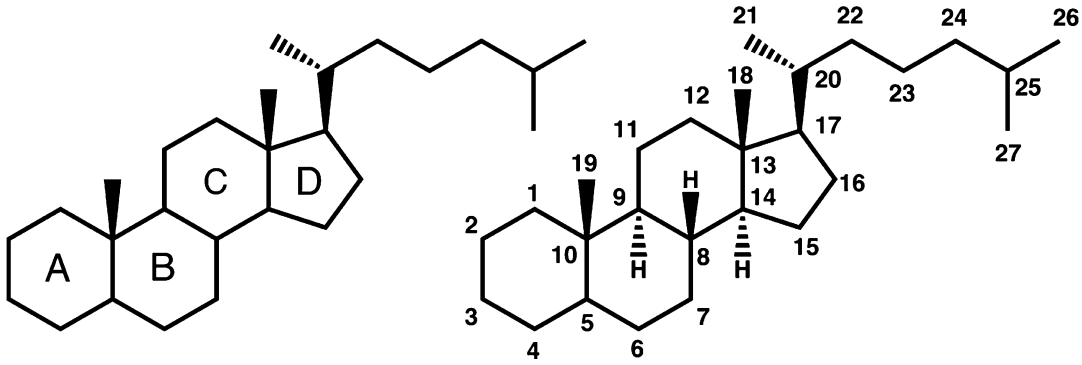
Synonyms

Steroid hormones

Definition

A steroid or steroid hormone is a biomolecule derived from cholesterol, with a characteristic structure containing four fused rings (see Fig. 1).

Cholesterol is the precursor for five major classes of steroid hormones: progestagens, glucocorticoids, mineralcorticoids, androgens, and estrogens. These hormones are powerful



Steroids, Fig. 1 Structure and carbon numbering scheme for cholesterol and other steroids

signaling molecules that are released in order to elicit a specific response.

Androgens, such as testosterone, are responsible for the development of male sex characteristics, whereas estrogens are responsible for the development of female sex characteristics. Dehydroepiandrosterone (DHEA) is the most abundant circulating steroid in humans, and is a precursor for the sex hormones, testerosterone and estradiol. In addition, DHEA is a cortisol antagonist.

Glucocorticoids, such as cortisol, promote the formation of glycogen and inhibit the inflammatory response. They enable humans (and animals) to respond to stress.

Steroids act by interaction with cellular receptors that serve as transcription factors to regulate gene expression. Steroids are incredibly potent and elicit very specific responses due to their interaction with steroid receptors.

Cross-References

- ▶ Androgen
- ▶ Cortisol
- ▶ Estrogen
- ▶ Inflammation

References and Readings

Berg, J. M., Tymoczko, J. L., & Stryer, L. (2002). *Biochemistry* (5th ed.). New York: WH Freeman.

Nussey, S., & Whitehead, S. (2001). *Endocrinology*. Oxford: BIOS Scientific Publishers.

Sterol

- ▶ Cholesterol

Stigma

Valerie Earnshaw[1] and Stephenie Chaudoir[2]
[1]Department of Public Health, Yale University, New Haven, CT, USA
[2]Department of Psychology, Bradley University, Peoria, IL, USA

Synonyms

Deviance; Discrimination; Prejudice; Stereotypes; Stigmatization

Definition

A stigma is a personal attribute, mark, or characteristic that is socially devalued and discredited (Goffman, 1963). A wide variety of attributes are stigmas, including physical illnesses (e.g., HIV/AIDS, tuberculosis, epilepsy), mental illnesses (e.g., schizophrenia, mental disability), social norm violations (e.g., homosexuality, sex work, drug use, obesity), and certain demographic characteristics (e.g., racial/ethnic background, gender, socioeconomic status).

People who possess a stigma are perceived and treated negatively by others and ultimately suffer worse physical, psychological, and behavioral outcomes than people who do not possess a stigma.

The following sections describe how certain attributes become socially devalued, how stigma impacts individuals who possess a stigma (i.e., stigmatized people) and who do not possess a stigma (i.e., nonstigmatized people) via a series of stigma mechanisms, and other considerations relevant to stigma. Because HIV/AIDS is one of the strongest stigmas throughout the world and has received a great deal of empirical attention related to stigma (e.g., Aggleton & Parker, 2002), it used as the primary example of stigma throughout these sections.

The Social Construction of Stigma

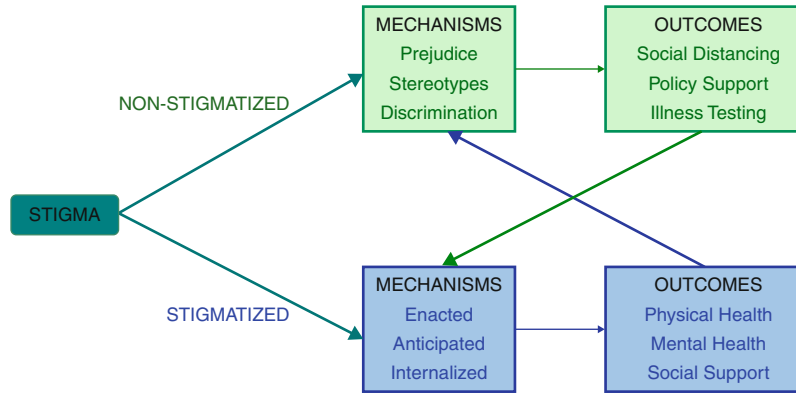
Stigmas are socially constructed (Crocker, Major, & Steele, 1998). In other words, certain attributes become devalued as the result of a social process (Link & Phelan, 2001) rather than as the result of innate differences between people who possess the attribute and people who do not possess the attribute. This social process involves stereotyping by associating the attribute with negative characteristics. For example, people living with HIV/AIDS (PLWHA) may be stereotyped to be promiscuous. The social process also involves separating the people who have the attribute into out-group categories. HIV-negative people may view other HIV-negative as part of their social group (i.e., part of "us") but PLWHA as part of a different social group (i.e., part of "them"). Finally, the social process involves experiences of status loss and discrimination by people who have the attribute. PLWHA may not be given medical care because of their HIV-status. Importantly, the social process that results in stigma relies on power. Nonstigmatized people have more power than stigmatized people and use this power to produce and reproduce social inequities and inequalities. This happens in both subtle ways (e.g., stigmatized people being paid systematically less than nonstigmatized people) and blatant ways (e.g., stigmatized people being enslaved by nonstigmatized people).

Because stigma results from a social process, the extent to which an attribute is devalued varies across different social contexts. Social contexts include both individual relationships and cultural contexts. For example, individual people (e.g., friends, family members, employers, and health-care providers) vary in the degree to which they view HIV/AIDS as a devalued attribute. Similarly, HIV/AIDS may be more devalued in some sociocultural contexts (e.g., Asian cities) than others (e.g., North American cities; Rao, Angell, Chow, & Corrigan, 2008). The extent to which an attribute is devalued also varies across time. For example, devaluation associated with HIV/AIDS decreased during the 1990s within the United States (Herek, Capitanio, & Widaman, 2002). Taken together, the degree of devaluation associated with a stigma is not universal or fixed; instead, it changes relative to specific social contexts and time periods.

Stigma Mechanisms and Outcomes

Individuals are impacted by stigma via a series of stigma mechanisms. Stigma mechanisms refer to the ways in which people react to either possessing or not possessing a particular stigma (Earnshaw & Chaudoir, 2009). Stigma mechanisms, in turn, result in physical, psychological, and behavioral outcomes for both stigmatized and nonstigmatized people. Figure 1 is adapted from the HIV Stigma Framework (Earnshaw & Chaudoir, 2009) and shows how stigma leads to stigma mechanisms, which in turn lead to outcomes. Because they represent the link between the social process of stigma and outcomes associated with stigma, stigma mechanisms are often measured by researchers.

Individuals who do not possess the stigma experience the stigma mechanisms of prejudice, stereotyping, and discrimination. Prejudice refers to negative emotions and feelings toward people who possess the stigma. For example, HIV-negative people may feel disgust toward PLWHA. Stereotypes refer to group-based beliefs about people who possess the stigma. HIV-negative people may believe that PLWHA are mostly gay men. Finally, discrimination refers to behavioral expressions of prejudice



Stigma, Fig. 1 Stigma framework

directed toward people who possess the stigma. HIV-negative people might refuse to hire, medically treat, or give housing to PLWHA. Although people who do not possess the stigma have more power than people who possess the stigma, people who do not possess the stigma may still suffer negative consequences due to stigma mechanisms. For example, an HIV-negative person who endorses the stereotype that PLWHA are mostly gay men may be less likely to engage in safe sex, be tested for HIV, or seek health care for HIV-related symptoms if they do not identify as a gay man. Consequently, belief in stereotypes can put HIV-negative people at risk for contracting HIV and not receiving treatment.

Individuals who possess the stigma experience the stigma mechanisms of anticipated stigma, enacted stigma, and internalized stigma. Enacted stigma, also called experienced stigma, refers to experiences of prejudice, stereotyping, and/or discrimination. For example, PLWHA may experience social rejection from friends and family members. Anticipated stigma refers to expectations of prejudice, stereotyping, and/or discrimination. PLWHA may expect that they will not be hired by a potential employer. Finally, internalized stigma refers to the endorsement of prejudice and stereotypes associated with one's stigma and applying them to the self. PLWHA may feel that they are dirty due to their HIV status. Further, stigma mechanisms experienced by individuals who do not possess the stigma may impact the outcomes of individuals who do possess the stigma. Discrimination perpetuated by HIV-negative people may be experienced as enacted stigma by PLWHA.

Stigma mechanisms profoundly impact the physical, psychological, and behavioral outcomes of stigmatized individuals. Meta-analytic evidence has shown that stigma mechanisms are associated with decreased physical health (e.g., increased physical illnesses and illness symptoms; Pascoe & Smart Richman, 2009) and mental health (e.g., increased depression and decreased self-esteem; Mak, Poon, Pun, & Cheung, 2007). Additionally, stigma mechanisms are associated with maladaptive behaviors which may further undermine the health of stigmatized individuals. For example, stigma mechanisms are related to behaviors such as delayed HIV treatment initiation and nonadherence to medication regimens (Chesney & Smith, 1999). Stigma mechanisms are further related to decreased likelihood of disclosure among individuals living with concealable stigmatized identities including HIV/AIDS (Smith, Rossetto, & Peterson, 2008).

Other Considerations

Intersectional stigma and layered stigma refer to the possession of multiple stigmas. For example, a gay man living with HIV possesses two stigmas: homosexuality and HIV. Possessing multiple stigmas may exacerbate the impact of stigma on individuals.

Associative stigma, courtesy stigma, and affiliate stigma refer to being connected to someone who possesses a stigma. For example, an HIV-negative man who has a daughter living

with HIV/AIDS may experience associative stigma due to his connection to his daughter. People who possess an associative stigma may experience negative outcomes via stigma mechanisms typically reserved for people who possess a stigma.

Concealable stigmas refer to devalued attributes that cannot be seen by others. Examples of concealable stigmas include many physical and mental illnesses and some social norm violations such as homosexuality and drug use. In contrast, visible stigmas refer to devalued attributes that can be seen by others. Examples of visible stigmas include many demographic characteristics such as racial/ethnic background and gender. Whereas people with concealable stigmas can hide their stigma from others in some social interactions, people with visible stigmas cannot.

Cross-References

- ▶ Discrimination and Health
- ▶ Health Disparities

References and Readings

Aggleton, P., & Parker, R. (2002). *A conceptual framework and basis for action: HIV/AIDS stigma and discrimination*. Geneva, Switzerland: Joint United Nations Programme on HIV/AIDS.

Allport, G. W. (1954). *The nature of prejudice*. Oxford, UK: Addison-Wesley.

Brewer, M. B., & Brown, R. J. (1998). Intergroup relations. In D. T. Gilbert, S. T. Fiske, & G. Lindzey (Eds.), *The handbook of social psychology* (4th ed., pp. 554–593). Boston: McGraw-Hill.

Chesney, M. A., & Smith, A. W. (1999). Critical delays in HIV testing and care: The potential role of stigma. *The American Behavioral Scientist, 47*, 1162–1174. doi:10.1177/00027649921954822.

Crocker, J., Major, B., & Steele, C. (1998). Social stigma. In D. T. Gilbert, S. T. Fiske, & G. Lindzey (Eds.), *The handbook of social psychology* (4th ed., Vol. 2, pp. 504–553). Boston: McGraw-Hill/Distributed exclusively by Oxford University Press.

Earnshaw, V. A., & Chaudoir, S. R. (2009). From conceptualizing to measuring HIV stigma: A review of HIV stigma mechanism measures. *AIDS and Behavior, 13*, 1160–1177. doi:10.1007/s10461-009-9593-3.

Goffman, E. (1963). *Stigma: Notes on the management of spoiled identity*. New York: Simon & Schuster.

Herek, G. M., Capitanio, J. P., & Widaman, K. F. (2002). HIV-related stigma and knowledge in the United States: Prevalence and trends, 1991–1999. *American Journal of Public Health, 92*, 371–377. doi:10.2105/ajph.92.3.371.

Jones, E. E., Farina, A., Hastorf, A. H., Markus, H., Miller, D. T., & Scott, R. A. (1984). *Social stigma: The psychology of marked relationships*. New York: W. H. Freeman and Company.

Link, B. G., & Phelan, J. C. (2001). Conceptualizing stigma. *Annual Review of Sociology, 27*, 363–385. doi:10.1146/annurev.soc.27.1.363.

Major, B., & O'Brien, L. T. (2005). The social psychology of stigma. *Annual Review of Psychology, 56*, 393–421. doi:10.1146/annurev.psych.56.091103.070137.

Mak, W. W. S., Poon, C. Y. M., Pun, L. Y. K., & Cheung, S. F. (2007). Meta-analysis of stigma and mental health. *Social Science & Medicine, 65*(2), 245–261. doi:10.1016/j.socscimed.2007.03.015.

Pascoe, E. A., & Smart Richman, L. (2009). Perceived discrimination and health: A meta-analytic review. *Psychological Bulletin, 135*(4), 531–554. doi:10.1037/a0016059. supp (Supplemental).

Rao, D., Angell, B., Chow, L., & Corrigan, P. (2008). Stigma in the workplace: Employer attitudes about people with HIV in Beijing, Hong Kong, and Chicago. *Social Science & Medicine, 67*, 1541–1549. doi:10.1016/j.socscimed.2008.07.024.

Smith, R., Rossetto, K., & Peterson, B. L. (2008). A meta-analysis of disclosure of one's HIV-positive status, stigma and social support. *AIDS Care, 20*(10), 1266–1275. doi:10.1080/09540120801926977.

Stigmatization

▶ Stigma

Stop Smoking

▶ Smoking Cessation

Strain

▶ Psychological Stress

Stranger Anxiety

▶ Anxiety

Strength Model of Self-Control

▶ Self-Regulatory Capacity

Stress

Kristen Salomon
Department of Psychology, University of South Florida College of Arts & Sciences, Tampa, FL, USA

Synonyms

Anxiety; Distress; Mental stress; Pressure; Psychological stress

Definition

Stress is a transactional process occurring when an event is perceived as relevant to an individual's well-being, has the potential for harm or loss, and requires psychological, physiological, and/or behavioral efforts to manage the event and its outcomes (Lazarus & Folkman, 1984). The stimuli or events that cause stress are referred to as stressors (Mason, 1975). Stress often results in psychological distress and efforts to cope with the event. Physiological stress responses are often in support of efforts to manage the stressful event and protect the organism from harm (McEwen & Seeman, 1999). Stress may not be a uniformly negative experience, as stressful events may also include the potential for benefit and growth (Lazarus & Folkman). Early views of stress focused on physical stress – events that perturb the resting homeostasis of the body, such as changes in temperature or physical injury (McEwen & Seeman, 1999). Current views of stress focus heavily on social and psychological sources, with appraisals of the event and the perceived coping resources as key features. Stress may be categorized by its severity, its time course (acute, repeated, or chronic), and degree of control over the stressor. Distinctions also have been made between active coping stressors and passive coping stressors (Obrist, 1981). Active coping stressors require overt behavioral action, such as giving a speech or performing a reaction time task, whereas passive coping stressors require that the individual endure without behavioral action, such as watching gruesome photos. Psychological and physiological stress responses have been shown to differ based upon these dimensions (Tomaka, Blascovich, Kelsey, & Leitten, 1993).

Cross-References

▶ Coping
▶ Family Stress
▶ Perceived Stress
▶ Perceptions of Stress
▶ Physiological Reactivity
▶ Stress, Emotional
▶ Stress Responses

References and Readings

Cohen, S., Kessler, R. C., & Gordon, L. U. (1995). *Measuring stress: A guide for health and social scientists*. New York: Oxford University Press.

Hobfoll, S. E. (1989). Conservation of resources: A new attempt at conceptualizing stress. *American Psychologist, 44*, 513–524.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal and coping*. New York: Springer.

Mason, J. W. (1975). A historical view of the stress field (Parts I and II). *Journal of Human Stress, 1*, 6–12 & 22–36.

McEwen, B. S., & Seeman, T. (1999). Protective and damaging effects of mediators of stress. Elaborating and testing the concepts of allostasis and allostatic load. *Annals of the NY Academy of Science, 896*, 30–47.

Obrist, P. A. (1981). *Cardiovascular psychophysiology: A perspective*. New York: Plenum Press.

Selye, H. (1956). *The stress of life*. New York: McGraw-Hill.

Tomaka, J., Blascovich, J., Kelsey, R. M., & Leitten, C. L. (1993). Subjective, physiological, and behavioral effects of threat and challenge appraisals. *Journal of Personality and Social Psychology, 65*, 248–260.

Stress and Occupational Health

▶ Psychosocial Work Environment

Stress Appraisals

▶ Perceptions of Stress

Stress Cascade

▶ Neuroendocrine Activation

Stress Diathesis Models

▶ Stress Vulnerability Models

Stress Disorder

▶ Anxiety Disorder

Stress Management

Catherine Benedict
Department of Psychology, University of Miami, Coral Gables, FL, USA

Synonyms

Coping with stress; Relaxation techniques; Stress reduction

Definition

Stress management techniques and interventions can be broadly categorized into skills that are provided via education, relaxation training, psychosocial interventions, and group formats, alone or in combination, that aim to reduce the stress response by targeting coping strategies and relaxation skills.

Description

The deleterious effects of stress on health and well-being are well documented and have led to the incorporation of stress management techniques into many psychosocial treatment protocols, alone or in combination with medical treatments. Chronic stress has been linked to physiologic changes such as neuroendocrine and immune dysregulation, and worsened disease profiles. One of the most prominent associations between stress and poor health has been in the area of cardiovascular risk. For example, evidence suggests that chronic stress is associated with increased sympathetic cardiovascular activity and damage to endothelial functioning, which increases the risk of several cardiovascular conditions, such as arterial hypertension, coronary artery disease, and arrhythmias. Therefore, incorporation of stress management into clinical and research protocols designed to improve health and well-being has become increasingly popular.

The field of stress management is comprised of a variety of methods and techniques. Relaxation skills training methods with the most empirical support include progressive relaxation or progressive muscle relaxation, autogenic training, biofeedback, mental imagery, and other Eastern or Westernized meditation methods. Various cognitive techniques and in some cases, pharmacotherapy, are also part of stress management programs. Other methods that have been incorporated into stress management include listening to relaxing music, massage, aerobic exercise, diaphragmatic breathing, and postural relaxation methods. Some evidence suggests that "active" stress management techniques (e.g., breathing-guided relaxation training) may be more effective in reducing stress symptoms and inducing improved autonomic cardiovascular regulation than "passive" techniques (e.g., massage). Several

standardized stress management interventions have been developed and shown to be efficacious in reducing symptoms of stress and improving various indicators of physical and mental health and quality of life. Empirical evidence supports the use of stress management techniques in the treatment of migraines, pain, and other somatic complaints, and have also been used to reduce stress symptoms, improve adjustment, well-being, and quality of life in a range of patient populations, including those diagnosed with type 2 diabetes, coronary heart disease, fibromyalgia, chronic fatigue syndrome (CFS), human immunodeficiency virus (HIV), and cancer.

Stress management intervention strategies range from single session psychosocial treatments to multifaceted treatments involving psychosocial and behavioral modification components. Likewise, intervention aims and target outcomes also range from behavior and lifestyle changes (e.g., diet, exercise, and utilization of stress management techniques) to changes in psychological and physical well-being (e.g., relief of depressive or anxious symptoms, cortisol regulation). It is hypothesized that if one can manage stress effectively, an individual's ability to adopt lifestyle changes that positively impact health outcomes will be maximized. Conversely, chronic stress has been associated with negative lifestyle factors including poor diet, sedentary lifestyle, alcohol and substance use, and poor adherence to medical regimens. Therefore, many stress management programs include medical endpoints such as disease risk factors, disease morbidity, and mortality. For example, stress management interventions in coronary heart disease may include known risk factors as relevant endpoints, such as being overweight or obese, smoking status, blood pressure, cholesterol, lipids, cardiac ischemia (e.g., angina), and number of cardiac events and/or procedures (e.g., myocardial infarction, angioplasty). Endpoints may be measured at proximal, intermediate, or distance time points, depending on the outcome of interest.

Cultural factors have also been shown to be associated with stress and stress management. Although all cultures experience stress and its sequelae, psychosocial sources of stress, the expression of stress symptoms, and the use and acceptability of stress management techniques varies across cultures. For example, evidence suggests that Hispanics are more likely to experience somatic symptoms in response to stress, compared to non-Hispanic Whites. Cultural differences in stress symptoms will likely lead to varying intervention and treatment approaches. Individuals who present with emotional symptoms of stress without a somatic component, for example, receive more treatment in the United States compared to South Korea, a collectivist culture in which individuals are less likely to express signs of emotional distress directly. However, there is no cross-cultural difference between the United States and South Korea in treatment of somatic symptoms of stress, suggesting that treatment methods may be culturally biased.

Stress Management Techniques and Interventions

Many different methods of stress management have been employed with empirical support across a range of populations. Progressive relaxation or progressive muscle relaxation consists of consecutively tensing and relaxing different sets of muscle groups throughout the body, generally starting with the feet and systematically progressing up to the head. Diaphragmatic breathing consists of taking deep breaths in which the diaphragm contracts and the abdomen, rather than the chest, is extended. This type of breathing involves a slow and deep inhalation through the nose, usually to a count of 10, followed by slow and complete exhalation for a similar count; the process is repeated for a preferred number of times to facilitate relaxation. Using mental imagery as a stress management tool involves imagining a scene, place, or event that is considered safe, peaceful, and restful; one that is associated with affective feelings of happiness, job, and contentment. Alternatively, images may involve mental pictures of stress flowing out of the body or being locked away in a padlocked chest. All of the senses are incorporated into the mental imagery exercise

and it is encouraged to develop details and complex images that invoke sensual perceptions. The imagined place is used as a retreat from environmental stressors, with the goal of having the body react to imagined scenes of peace and tranquility as if they were real, counteracting the adrenergic effects of stress. These methods have been shown to be beneficial through self-report measures of stress and well-being and physical measures of the body's stress response through biofeedback methods.

Biofeedback is a method that keeps track of the body's physiological responses in real time, generally through machines that measure heart rate, muscle tension, or brain waves. Most often, biofeedback is used as a tool to facilitate control over the stress response sequelae. Individuals are taught to recognize the stress response when it is underway and employ relaxation techniques (e.g., deep breathing, mental imagery, or adaptive cognitive replacement) to calm physiological arousal. Theory and empirical evidence suggest that the real-time feedback of physiological changes in response to stress and the utilization of stress management techniques facilitate learning and adoption of effective relaxation methods into daily life.

Stress Management Programs

Many stress management interventions for clinical populations consist of a combination of methods that frequently incorporate a variety of cognitive and behavioral techniques. For example, cognitive-behavioral stress management (CBSM) interventions have been employed and been shown to have beneficial effects in stressed nonclinical subjects and a range of patient populations. These interventions typically aim to improve adaptive coping and reduce psychological distress through the use of emotion regulation strategies and relaxation training. Didactic training typically addresses stress appraisal and cognitive coping strategies to improve stress management of general- and disease-specific stressors. Often, a psychoeducational component about the nature and consequences of stress and disease processes is also incorporated into intervention protocols. The CBSM protocol consists of a 10-week manualized group intervention in which groups meet for 2 h per week; sessions consist of 90 min of didactic discussion and exercises and 30 min of relaxation training. During the didactic portion of each session, participants are provided information regarding stress awareness, physical responses to stress, and the appraisal process and are taught a variety of cognitive-behavioral techniques designed to manage general- and disease-specific stress. Cognitive techniques include learning to identify cognitive distortions and cognitive restructuring processes (e.g., rational thought replacement), effective coping strategies (e.g., emotional-focused vs. problem-focused coping), and anger management and assertiveness training (e.g., effective communication). Information related to disease physiology, diagnosis, and treatment are also provided and health maintenance strategies are reviewed. During the relaxation portion, participants are taught a variety of techniques through group relaxation exercises, including progressive muscle relaxation, guided imagery, meditation, and diaphragmatic breathing. Participants are encouraged to practice the techniques at home on a daily basis. The CBSM intervention has been shown to be effective in increasing stress management skills, which has been related to improvements in a number of quality-of-life domains, in HIV, cancer, and CFS populations.

Similarly, some have demonstrated the efficacy of a group-based stress management program that included progressive muscle relaxation training, didactic training in the use of cognitive and behavioral skills to bring awareness to and reduce physiological symptoms of stress (e.g., guided imagery, deep breathing techniques, thought stopping, and recognition of life stressors), and education on the negative effects of stress on health in improving glycemic control in patients with type 2 diabetes. Participants learned stress management techniques in a group-based intervention format and were instructed to practice muscle relaxation at home twice daily with the aid of an audiotape. Specific instructions were given to encourage "mini-practices"

(i.e., brief, 30-s versions of a progressive relaxation session) to facilitate the use of stress management and relaxation techniques into daily life. Although similar interventions have been conducted and shown positive effects on blood glucose, findings have been mixed as several others have failed to show a therapeutic effect of stress management on diabetes control.

Mindfulness-based stress reduction (MBSR) is a clinically standardized meditation program, originally developed as a group-based intervention for chronic pain but has since demonstrated efficacy for patients with a range of mental and physical disorders, as well as healthy subjects. The MBSR protocol consists of three different stress management techniques: body scan, which involves focusing on different parts of the body, bringing attention and awareness to sensations and feelings, breath awareness, and relaxation; sitting meditation, which involves focusing on body sensations and breathe, as well as nonjudgmental awareness of the cognitions and the stream of thoughts and distractions that pass through the mind; and Hatha yoga practice, which consists of breathing exercises, stretching, and postural exercises designed to strengthen and relax the musculoskeletal system. The MBSR program is an 8- to 10-week group intervention in which sessions typically last 2.5 h, with an additional single all-day session per course. Homework of at least 45 min a day, 6 days a week, is also encouraged, which may consist of meditation practice, mindful yoga, and/or incorporating mindfulness into daily life. Groups may be either homogenous or heterogeneous with regard to illnesses or presenting problems of participants. Empirical evidence provides consistent and relatively strong effect sizes regarding the efficacy of MBSR on a number of psychological (e.g., depressive and anxiety symptoms, coping style) and physical (e.g., medical symptoms, sensory pain, physical impairment, and functional quality of life) well-being in clinical and nonclinical populations. Clinical populations have included patients diagnosed with anxiety disorders, depression, chronic pain, fibromyalgia, cancer, and stress related to environmental contexts (e.g., medical school, prison life), as well as relatively healthy individuals interested in improving their ability to cope with normal stressors of daily living.

Stress management interventions for healthy and patient populations are promising. However, findings have been mixed and the literature is limited by measurement and research design problems, insufficient information regarding intervention components and protocol fidelity, clinical relevance of statistically significant effects, and feasibility and dissemination concerns regarding the translation of research protocols into clinical practice. Nevertheless, given the detrimental effects of stress on psychological and physical well-being, further research is needed using large-scale, randomized clinical trials, with sound methodological procedures that include objective markers of health and disease status, in addition to self-report measures of psychosocial well-being and functional indicators of distress.

Cross-References

▶ Anger Management

References and Readings

Antoni, M. H., Lechner, S. C., Kazi, A., Wimberly, S. R., Sifre, T., Urcuyo, K. R., Phillips, K., Glück, S., & Carver, C. S. (2006). How stress management improves quality of life after treatment for breast cancer. *Journal of Consulting and Clinical Psychology, 74*(6), 1143–1152.

Blumenthal, J. A., Sherwood, A., Babyak, M. A., Watkins, L. L., Waugh, R., Georgiades, A., Bacon, S. L., Hayano, J., Coleman, R. E., & Hinderliter, A. (2005). Effects of exercise and stress management training on markers of cardiovascular risk in patients with ischemic heart disease. *Journal of the American Medical Association, 293*(13), 1626–1634.

Brown, J. L., & Vanable, P. A. (2008). Cognitive-behavioral stress management interventions for personal living with HIV: A review and critique of the literature. *Annals of Behavioral Medicine, 35*, 26–40.

Chiesa, A., & Serretti, A. (2009). Mindfulness-based stress reduction for stress management in healthy people: A review and meta-analysis. *The Journal*

of Alternative and Complementary Medicine, 15(5), 593–600.
Gaab, J., Blattler, N., Menzi, T., Pabst, B., Stoyer, S., & Ehlert, U. (2003). Randomized controlled evaluation of the effects of cognitive-behavioral stress management on cortisol responses to acute stress in healthy subjects. *Psychoneuroendocrinology, 28*, 767–779.
Lehrer, P. M., Carr, R., Sargunaraj, D., & Woolfolk, R. L. (1994). Stress management techniques: Are they all equivalent, or do they have specific effects? *Biofeedback and Self-Regulation, 19*(4), 353–401.
Lehrer, P. M., Woolfolk, R. L., & Sime, W. E. (Eds.). (2007). *Principles and practice of stress management*. New York: Guildford.
Lucini, D., Malacarne, M., Solaro, N., Busin, S., & Pagani, M. (2009). Complementary medicine for the management of chronic stress: Superiority of active versus passive techniques. *Journal of Hypertension, 27*, 2421–2428.
McEwen, B. S. (2007). Physiology and neurobiology of stress and adaptation: Central role of the brain. *Physiological Review, 87*, 873–904.
Smith, J. E., Richardson, J., Hoffman, C., & Pilkington, K. (2005). Mindfulness-based stress reduction as supportive therapy in cancer care: Systematic review. *Journal of Advanced Nursing, 52*(3), 315–327.
Surwit, R. S., van Tilburg, M. A., Zucker, N., McCaskill, C. C., Parekh, P., Feinglos, M. N., Edwards, C. L., Williams, P., & Lane, J. D. (2002). Stress management improves long-term glycemic control in type 2 diabetes. *Diabetes Care, 25*(1), 30–34.

Stress Reactivity

Wolff Schlotz
Institute of Experimental Psychology, University of Regensburg, Regensburg, Germany

Synonyms

Stress responsivity

Definition

Stress reactivity is the capacity or tendency to respond to a stressor. It is a disposition that underlies individual differences in responses to stressors and is assumed to be a vulnerability factor for the development of diseases.

Description

People respond differently when exposed to the same stressor. Such differences can be observed in all four major stress response domains, namely, physiology, behavior, subjective experience, and cognitive function. Within the physiological domain, two response systems are of particular importance: cardiovascular responses (indicated by blood pressure and heart rate), driven by sympathetic nervous system (SNS) activity, and output of the glucocorticoid hormone cortisol from the adrenal cortex, driven by hypothalamic-pituitary-adrenal (HPA) axis activity. Stress reactivity is assumed to be stable over time, i.e., persons showing high responses at an initial assessment also show high responses when the assessment is repeated at a later time. Stress reactivity can be conceptualized as specific or general. Whereas specific stress reactivity reflects reactivity of a particular response system (e.g., cardiovascular stress reactivity; endocrine stress reactivity; affective stress reactivity), general stress reactivity is indicated by aggregation of responses across domains and/or stressors.

The relevance of stress reactivity for behavioral medicine rests primarily on the assumption that high stress reactivity is assumed to be a vulnerability factor for disease that predicts disease outcome variance independently of well-established risk factors. As early as in the first half of the twentieth century it was proposed that the size of blood pressure responses to placing the hand in cold water (cold pressor test) would indicate the risk of later development of hypertension. More recently, a growing body of evidence from longitudinal studies that used laboratory stress tests support the assumption that cardiovascular stress reactivity is indeed a risk factor for subclinical and clinical cardiovascular disease. This has been shown for both physiological stressors such as the cold pressor test and psychological stressors such as pressure to perform in a social situation (e.g., public speaking). On the basis of this evidence it has been concluded that cardiovascular stress reactivity is a risk factor for cardiovascular disease in addition

to the classic risk factors of family history, obesity, smoking, diabetes mellitus, and hypercholesterolemia. As for endocrine stress reactivity, a few longitudinal studies found evidence for associations between endocrine stress reactivity and increased risk for disease. However, this research area is much less developed than that for cardiovascular stress reactivity, and the evidence is less robust. Finally, it has been suggested that endocrine and affective stress reactivity might be a risk factor for the development of mental disorders such as psychosis, depression, and anxiety disorders. Again, research in this area is still relatively scarce and the evidence mostly relies on cross-sectional studies.

In contrast to specific stress reactivity, the concept of general stress reactivity emphasizes generalizability of responses across response systems and stressors. It is based on the notion that stress responses have a common origin in brain areas that mediate activation of the HPA axis and the SNS, as well as behavioral and subjective-emotional responses. In particular, hippocampus, amygdala, and prefrontal cortex are higher central mediators of subjective-emotional responses. These areas are functionally connected to the hypothalamic and brainstem nuclei which are critical in activating the HPA axis and the SNS. If a stressor is processed in higher brain areas, stress responses are expected to show relatively high covariance. This is to be expected for psychological stressors, but less so for physiological stressors. Thus, dissociations between responses systems might reflect individual differences at both levels. In addition, observed physiological stress responses are influenced by peripheral factors such as receptor sensitivity in the periphery or vascular resistance. Although high covariance between response systems would be expected, a number of factors such as different dynamics of the response systems, habituation effects, measurement error, and limited variance due to limited stressor intensity act at attenuating associations between responses.

Complementary to the notion of general stress reactivity, the concepts of individual response specificity (IRS) and stimulus response specificity (SRS) reflect observations of dissociation between response systems or stressors. The concept of IRS describes individual differences in patterns of responses, for example, one person might respond to stressors with a high blood pressure but low cortisol increase, whereas another person might also show a high cortisol increase. SRS describes such response patterns as related to stressors, for example, it has been proposed that the HPA axis in humans is activated by stressors that include social evaluative threat, but not by cognitive effort without that component. In research, it is often difficult to reliably detect general stress reactivity, IRS or SRS response patterns. Although there is now increasing evidence for the concept of general stress reactivity, associations between responses are usually moderate. Therefore, it is important to note that it is not possible to use the stress response in one domain or system as a general indicator of responses in other domains. Although it has been suggested that such dissociations might present useful information about psychobiological responses systems of an individual that could be valuable for behavioral medicine, to date little is known about the stability and implications of response dissociations.

Stress reactivity can be assessed both in the laboratory and in daily life. The major advantage of laboratory stress tests is the high degree of standardization over the conditions implemented. However, stress responses in the laboratory have limited ecological validity, i.e., do not necessarily reflect stress reactivity in daily life. For that reason, ambulatory assessment methods are increasingly used and present the opportunity to assess real-time stress reactivity in daily life for both research and clinical practice. A number of factors influence stress reactivity, with implications for clinical decisions. For example, it is known that stress reactivity is associated with sex, age, ethnicity, personality factors, preexisting disease, and the presence or absence of chronic stress. Due to such moderating factors there is a wide range of associations between stress reactivity and disease outcome. Therefore, the predictive value varies across individuals, making it difficult to use stress reactivity scores in clinical practice. Although the reliability of

stress reactivity assessments could be increased by assessing aggregated stress responses in a repeated measure/multiple stressors design, few studies have implemented this design due to the high demands on resources. It would be expected that stronger and more consistent association with disease could be observed with such a design. Finally, an important conclusion from the variety of findings on associations between stress response systems is that, as mentioned above, a single assessment of one response system cannot be used as an indicator for stress responses in other systems. The notion of a consistent "gold-standard" indicator of stress reactivity is not supported by the research literature.

As the assessment of physiological stress responses is relatively expensive and fraught with practical problems, clinicians often rely on retrospective self-reports of individual stress experience to assess stress reactivity. However, such subjective measures of stress experience confound individual stress reactivity with frequency of exposure to daily life stress. Recently, a self-report instrument for the assessment of perceived stress reactivity has been developed and evaluated. Although retrospective self-report methods cannot replace real-time measures, they present an opportunity to assess patients' perceptions of their own stress reactivity in daily life when resources to assess real-time responses are lacking or when such self-observations are of primary interest.

Stress reactivity is assumed to be a consequence of the individual genetic makeup and early environmental factors. Quantitative genetic studies using mainly twin designs have concluded that approximately 50% of the variance in stress responses is due to genetic factors. However, there is some variability of estimates between studies, and the amount of heritability seems to change with repeated exposure to the same stressor. Molecular genetic studies have revealed a number of single gene variants that might influence stress responses in different systems, although many of these effects of candidate genes need replication before valid conclusions can be drawn. In addition to genetic makeup, factors of the environment early in life have been shown to influence stress reactivity. A number of studies suggest that an adverse prenatal environment might exert long-term effects on stress reactivity in the offspring. Similarly, adverse early postnatal environmental factors such as maternal care or abuse have been shown to affect stress reactivity, with consequences for the risk of mental disorder later in life. Animal studies have suggested that such early environmental effects might be mediated by epigenetic changes in specific brain areas.

Despite good evidence for the prediction of cardiovascular disease by cardiovascular stress reactivity, potential pathways and causal chains of effects are unclear. In addition, not all individuals with high cardiovascular stress reactivity later develop cardiovascular disease. This points at another factor implicated in the pathway. It is likely that high stress reactivity leads to disease particularly if a highly stress reactive individual is exposed to chronic stress in daily life (diathesis-stress model). Other areas of discussion are the potential adaptive function of high stress reactivity in an evolutionary context, and the significance of low levels of stress reactivity for the development of diseases and disorders.

Cross-References

- ▶ General Adaptation Syndrome
- ▶ Hypothalamic-Pituitary-Adrenal Axis
- ▶ Individual Differences
- ▶ Physiological Reactivity
- ▶ Stress
- ▶ Stress Responses
- ▶ Stress Test
- ▶ Stress Vulnerability Models
- ▶ Stressor
- ▶ Sympathetic Nervous System (SNS)

References and Readings

Chida, Y., & Steptoe, A. (2010). Greater cardiovascular responses to laboratory mental stress are associated

with poor subsequent cardiovascular risk status: A meta-analysis of prospective evidence. *Hypertension, 55*(4), 1026–1032.
Contrada, R. J., & Baum, A. (Eds.). (2010). *The handbook of stress science: Biology, psychology, and health*. New York: Springer.
Heim, C., & Nemeroff, C. B. (2001). The role of childhood trauma in the neurobiology of mood and anxiety disorders: preclinical and clinical studies. *Biological Psychiatry, 49*(12), 1023–1039.
Kudielka, B. M., Hellhammer, D. H., & Wust, S. (2009). Why do we respond so differently? Reviewing determinants of human salivary cortisol responses to challenge. *Psychoneuroendocrinology, 34*(1), 2–18.
Lovallo, W. R. (2005). *Stress and health: Biological and psychological interactions* (2nd ed.). Thousand Oaks, CA: Sage.
Manuck, S. B., & McCaffery, J. M. (2010). Genetics of stress: Gene-stress correlation and interaction. In A. Steptoe (Ed.), *Handbook of behavioral medicine: Methods and applications* (pp. 455–478). New York: Springer.
Myin-Germeys, I., & van Os, J. (2007). Stress-reactivity in psychosis: Evidence for an affective pathway to psychosis. *Clinical Psychology Review, 27*(4), 409–424.
Phillips, D. I. (2007). Programming of the stress response: A fundamental mechanism underlying the long-term effects of the fetal environment? *Journal of Internal Medicine, 261*(5), 453–460.
Schlotz, W., Yim, I.S., Zoccola, P.M., Jansen, L., & Schulz, P. (2011). The perceived stress reactivity scale: Measurement invariance, stability and validity in three countries. *Psychological Assessment*, *23*(1), 80–94.
Treiber, F. A., Kamarck, T., Schneiderman, N., Sheffield, D., Kapuku, G., & Taylor, T. (2003). Cardiovascular reactivity and development of preclinical and clinical disease states. *Psychosomatic Medicine, 65*(1), 46–62.
Ulrich-Lai, Y. M., & Herman, J. P. (2009). Neural regulation of endocrine and autonomic stress responses. *Nature Reviews Neuroscience, 10*(6), 397–409.

Stress Reduction

▶ Stress Management

Stress Response

▶ Immune Responses to Stress
▶ Neuroendocrine Activation
▶ Psychophysiologic Reactivity

Stress Responses

Kristen Salomon
Department of Psychology, University of South Florida College of Arts & Sciences, Tampa, FL, USA

Synonyms

Stress; Stress reactivity

Definition

Stress responses are psychological, physiological, and behavioral responses to an event perceived as relevant to one's well being with some potential for harm or loss and requiring adaptation. Psychological stress responses often include negative emotions, such as anxiety, distress, or anger, although positive emotional states related to feeling challenged and driven may also occur. Cognitive efforts aimed at coping with the stressor, such as planning, distancing, and/or reinterpreting, also occur (Lazarus & Folkman, 1984). Physiological stress responses are often those that are in support of coping with or fleeing from the stressor, and protecting the organism from potential harm. These responses include, but are not limited to, changes in heart rate, blood pressure, cortisol, and immune function (Sapolsky, 1994). Behavioral stress responses involve actions also aimed at coping with or fleeing from the stressful event, such as actively performing a task or withdrawing effort from a situation perceived as impossible (Lazarus & Folkman, 1984).

Cross-References

▶ Coping
▶ Physiological Reactivity

References and Readings

Cacioppo, J. T. (1994). Social neuroscience: Autonomic, neuroendocrine, and immune responses to stress. *Psychophysiology, 31*, 113–128.
Cohen, S., Kessler, R. C., & Gordon, L. U. (1995). *Measuring stress: A guide for health and social scientists*. New York: Oxford University Press.
Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal and coping*. New York: Springer.
Sapolsky, R. M. (1994). *Why zebras don't get ulcers*. New York: Holt.

Stress Responsivity

► Stress Reactivity

Stress Test

Jet Veldhuijzen van Zanten
School of Sport and Exercise Sciences,
The University of Birmingham, Edgbaston,
Birmingham, UK

Synonyms

Mental stress task; Psychological stress task; Psychological stressor

Definition

Laboratory mental stress tasks are commonly used in behavioral medicine to assess the physiological responses to a standardized stressor in a controlled setting (Turner, 1994).

Description

Even though originally it was thought that particularly exaggerated physiological responses to mental stress can be predictive of cardiovascular disease (Obrist, 1981), there is now growing evidence that blunted physiological responses can also be associated with poor health (Carroll, Lovallo, & Phillips, 2009). Other evidence is available that not the responses to mental stress itself, but the physiological recovery upon completion of the stress task can be related with the poor health outcomes (Larsen & Cristenfeld, 2011). There is a large body of research that explores the associations between psychological traits (e.g., competitiveness and hostility) as well as mental disorders (e.g., depression and anxiety) with the individual differences in physiological responses to mental stress (Lovallo, 1997). Participants that have been included in these stress studies comprise of young healthy participants, elderly people, and a wide variety of clinical populations. This section will describe the general setup of a laboratory stress task and the most commonly used mental stress tasks in behavioral medicine. Even though the focus will be on mental stress tasks, it is worth noting that physical tests, such as exercise, tilt test, and cold pressor, are also readily used in laboratory settings.

In order to assess the physiological responses to a mental stress task, it is important to assess the resting physiological state of the participant. To quantify the stress response, a reactivity score is calculated, which is the difference between the physiological activity during the stress task and the activity during the rest period (Turner, 1994). A typical stress session starts with a resting period of 15–30 min, during which the participant is relaxed. Relaxation can be facilitated by listening to music, reading magazines, or watching a low-stimulating video. This is followed by an explanation of the stress task with, where appropriate, a brief practice session and the actual stress task. Upon completion of the stress task, a recovery period is started, during which the participant is asked again to relax, similar to the baseline rest period. The duration of the recovery period is depending on the variables that are under investigation. Whereas heart rate is known to return to baseline relatively soon after the end of the stress task, changes in other variables, in particular blood based measures such as cytokines, will not be seen until 30 min or longer

(Steptoe, Hamer, & Chida, 2007). In general, physiological data collection is conducted throughout each of these periods.

When exploring the effects of individual differences in physiological responses to mental stress, it is crucial that the testing procedures are identical between participants (Turner, 1994). The conditions in the laboratory, such as temperature and number of experimenters present, but also time of day, should be kept consistent. Care should also be taken to standardize the instructions of the task, which can be done by having the instructions prerecorded. Finally, adherence of the participants to the presession instructions is important. These involve most commonly avoiding strenuous exercise, food, caffeine, and smoking, as well as instructions about the medication which could influence the physiological measurements. The presession instructions are dependent on the physiological measures that are under investigation.

Mental Stress Tasks

Public Speaking – The participant is asked to give a speech in front of an audience and/or a video camera, following a brief preparation period. The topic of speech is psychologically stressful such as "pretend that you are falsely accused of shoplifting and that you have to defend yourself to the shop owner" or "describe your personal strengths and weaknesses" or "describe a recent event that caused anger." The participant will be told that the audience will be critically evaluating the content and delivery of the speech (Van Eck, Nicolson, Berkhof, & Sulon, 1996).

Mental Arithmetic – Different varieties are available for mental arithmetic tasks, which include serial subtraction or addition of double digit numbers or serial addition of single digit numbers with an element of retention. These tasks, even though not complicated in nature, have been developed to be provocative by adding components of increased time pressure, competition, harassment when a wrong answer is given, and social evaluation (Veldhuijzen van Zanten et al., 2004).

Trier Social Stress Test – This is a combination of mental arithmetic task followed by a public speech, all under conditions of social evaluation. In addition to the two varieties of mental stress, this task also has a postural component as the speech is conducted while upright (Kirschbaum & Hellhammer, 1993).

Computer Games – A variety of computer games have been used to induce stress in participants, which has been mainly conducted in younger participants. These tasks often have a strong component of competition; participants are either directly competing against the experimenter (often in a modified situation to standardize the success rate between participants) or competing against the other participants in the study.

Stroop Color Word Task – The participants are presented with words which describe colors, but the color of the letters is incongruent with the color that the word is written in. For example, the word red is written with yellow ink, and the word yellow is written with red ink. The participant is asked to call out the color of the ink (Stroop, 1935).

Mental stress tasks are subject to the effects task novelty and habituation (Turner, 1994). For example, even though all of these tasks provoke an increase of heart rate throughout the task, typically, the peak heart rate response is seen at the start of the test. Particularly when a participant is asked to complete the task on different occasions, it is important to maintain the engagement of the participant in each session. It has been shown that the addition of stressful elements such as social evaluation and competition will help to facilitate this. To ensure that the desired levels of stress are obtained, it is common practice to add a measure of self-reported perceptions of the task to each session. These can vary from a simple Likert scale related to perceived stressfulness and difficulty or measures of state stress and anxiety levels both before and after the stress task.

The stress tasks vary in terms of generalizability to real-life settings. Interestingly, an overview of various stress task revealed that public

speaking tasks were most consistently effective in inducing myocardial ischemia in patients with coronary heart disease (Strike & Steptoe, 2003). It is possible that this is due to the more naturalistic nature of the task than, for example, mental arithmetic or Stroop task. However, care should be taken when interpreting the effectiveness of a certain task to induce physiological changes between studies, as it is hard to compare the stressfulness of tasks between studies. Ambulatory recording techniques are available for the assessment of physiological measurements in real-life setting. Even though these field studies cannot be standardized between participants, it is worth noting that there is evidence that the laboratory cardiovascular responses to mental stress were predictive of ambulatory physiological assessments (Strike & Steptoe, 2003).

Cross-References

- ▶ Cardiovascular Recovery
- ▶ Immune Responses to Stress
- ▶ Mental Stress
- ▶ Psychological Stress
- ▶ Psychophysiologic Reactivity
- ▶ Stressor
- ▶ Trier Social Stress Test

References and Readings

Carroll, D., Lovallo, W. R., & Phillips, A. C. (2009). Are large physiological reactions to acute psychological stress always bad for health? *Social and Personality Psychology Compass, 3*, 725–743.

Kirschbaum, C., & Hellhammer, D. H. (1993). The 'Trier Social Stress Test' – A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology, 28*, 76–81.

Larsen, B. A., & Cristenfeld, N. J. (2011). Cognitive distancing, cognitive restructuring, and cardiovascular recovery from stress. *Biological Psychology, 86*, 143–148.

Lovallo, W. R. (1997). *Stress & health, biological and psychological interactions*. Thousand Oaks: Sage.

Obrist, P. A. (1981). *Cardiovascular psychophysiology: A perspective*. New York: Plenum Press.

Steptoe, A., Hamer, M., & Chida, Y. (2007). The effects of acute psychological stress on circulating inflammatory factors in humans: A review and meta-analysis. *Brain, Behavior, and Immunity, 21*, 901–912.

Strike, P. C., & Steptoe, A. (2003). Systematic review of mental stress-induced myocardial ischaemia. *European Heart Journal, 24*, 690–703.

Stroop, J. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology, 18*, 643–662.

Turner, J. R. (1994). *Cardiovascular reactivity and stress*. New York: Plenum Press.

Van Eck, M. M., Nicolson, N. A., Berkhof, H., & Sulon, J. (1996). Individual differences in cortisol responses to a laboratory speech task and their relationship to responses to stressful daily events. *Biological Psychology, 43*, 69–84.

Veldhuijzen van Zanten, J. J. C. S., Ring, C., Burns, V. E., Edwards, K. M., Drayson, M., & Carroll, D. (2004). Mental stress-induced hemoconcentration: Sex differences and mechanisms. *Psychophysiology, 41*, 541–551.

Stress Testing

- ▶ Exercise Testing

Stress Vulnerability Models

Conny W. E. M. Quaedflieg and Tom Smeets
Faculty of Psychology and Neuroscience, Maastricht University, Maastricht, MD, The Netherlands

Synonyms

Stress diathesis models

Definition

Vulnerability models are used to identify factors that are causally related to symptom development. Stress vulnerability models describe the relation between stress and the development of (psycho-)pathology. They propose an association

between (1) latent endogenous *vulnerability factors* that interact with stress to increase the adverse impact of stressful conditions, (2) *environmental factors* that influence the onset and course of (psycho-)pathology, and (3) *protective factors* that buffer against or mitigate the effects of stress on pathological responses.

Description

The prevalence of stress-related mental disorders encompassing mood and anxiety disorders in Europe is above 20%. This morbidity is associated with high health care costs, disability, and potential mortality. It is widely acknowledged that there are individual differences in how stressful people judge a particular event to be as well as in their ability to cope with adverse stressful life events. While historically stress was said to play an initiating role in the development of pathology, only a minority of people who experience adverse stressful life events go on to develop pathology. To distinguish people who develop pathology from people who do not (i.e., are resilient), vulnerability processes are suggested that predispose individuals to psychopathology when confronted with severe stressors. In the late 1970s, Zubin and Spring were the first to introduce this idea in the field of behavioral medicine by postulating a vulnerability model for schizophrenia. Specifically, they suggested that humans inherit a genetic predisposition to mental illness. However, an interaction between the genetic vulnerability and biological or psychosocial stressors is necessary to develop the disorder. The relationship between predispositional factors (or diathesis) and development of pathology has been described in four basic stress vulnerability models.

Stress Vulnerability Models

The first and most simple stress vulnerability model, the *dichotomous interactive* model, suggests that when predispositional factors are absent, even severe stress will not result in pathology. Instead, it is only when predispositional factors are present that stress may, depending on the severity of the stress, lead to the expression of pathology. Alternatively, the *quasi-continuous* model suggests varying degrees of predisposition with a continuous effect of predispositional factors on pathology once a threshold has been exceeded. The third, more extensive *threshold* model incorporates an individually specific threshold that is determined by the degree of vulnerability and the level of experienced stress. Finally, perhaps the most comprehensive model is the *risk-resilience continuum* model in which vulnerability is viewed as a continuum ranging from vulnerability to resilience, integrating different levels of severity of pathology into the model. Here, resilient characteristics that can make people more resistant to the impact of stress are also emphasized. Note that according to this latter model, even highly resilient individuals might still be at risk for developing pathology when experiencing extreme stress, but their individual threshold will be higher and the symptomatology likely less severe. Collectively, these four models are used to describe the relation between predispositional factors and the development of various pathologies.

Vulnerability Factors

In general, stress vulnerability models postulate that a genetic vulnerability interacts with adverse life events or stressors to produce pathology. This gene-environment interaction with regard to stress and the development of pathology has been most extensively investigated in mood disorders such as depression. Gene-environment interaction studies use monozygotic twin, adoption, and family studies as tools to identify predispositional factors in shared and non-shared environments in order to differentiate genetic from environmental influences. In twin studies, a higher prevalence of pathology in monozygotic twins reared in different environments is used to confirm a genetic predisposition, whereas in adoption studies the effect of the environment

(adoptive parents) can be offset against the effect of genes (biological parents). Using these methods the heritability of major depression, has been estimated at around 40%.

At the neurochemical level, the serotonin (5-HT) system has been implicated in depression. 5-HT regulates among others mood, activity, sleep, and appetite. Accumulating evidence indicates that individuals with a serotonergic vulnerability, manifested in a more sensitive brain serotonergic system, have an increased likelihood of developing mood-related disorders. Specifically, polymorphisms in the 5HT transporter system (5-HTT) have been associated with stressful life events, a heightened risk for depression, and reactivity to negative emotional stimuli. Individuals carrying two copies of the short variant of the 5-HTT allele (i.e., 5-HTTLPR), a less active gene resulting in fewer 5-HTT transporters, display an increased sensitivity to the impact of mild stressful life events, an excessive amygdala activity to fearful faces and produce elevated and prolonged levels of cortisol in response to a laboratory stressor compared to individuals with the long variant of the 5-HTT allele. The heritability of the stress hormone response has also been investigated with family studies in relatives of patients with depression using neuroendocrine functioning tests. For example, studies with the dexamethasone suppression test, a drug test used to measure the effectiveness of the negative feedback mechanism of the hypothalamic-pituitary-adrenal (HPA) axis at the level of the pituitary, have found an amplified set point of the HPA axis in relatives of depressed patients compared to healthy controls.

Moreover, 5-HT is also involved in the modulation of the HPA axis and its associated regulatory actions in the secretion of cortisol, the major human glucocorticoid stress hormone. Cortisol binds to two corticosteroid receptors in the brain, namely, the mineralocorticoid receptor (MR) and the glucocorticoid receptor (GR). Two mechanisms of cortisol binding are known. First, cortisol can bind to the hormone response element on DNA to influence gene expression (intracellular MR and GR binding properties). Secondly, cortisol can bind to membrane versions of the corticosteroid receptors to influence glutamate transmission and gene expression in the brain. The MR controls the basal HPA activity through inhibition of the HPA axis, facilitating the selection of adaptive behavioral responses and preventing minor adverse stressful life events to disturb homeostasis. In contrast, the GR promotes recovery after stress as well as the storage of information for future events. The balance between the MR and GR receptors determines the threshold and termination of the HPA axis response to stress. Studies have demonstrated that individuals with polymorphisms in the GR gene display higher cortisol responses and inefficient recovery of the HPA axis following standardized laboratory stress tests, thus revealing predisposition factors for stress-related pathology.

Genes can have a direct effect on the development of various brain systems. To illustrate this point, altered gene expression can reduce plasticity in brain circuits regulating mood, anxiety, and aggression and thereby decrease one's ability to cope with stressful life events. Moreover, genes can bias brain circuits to inefficient information processing which can result in the expression of pathology (e.g., intrusive memories in patients suffering from posttraumatic stress disorder). Genetic polymorphisms are then viewed as vulnerability factors given that they produce an increased sensitivity to the impact of stressful life events. However, it should be kept in mind that replication studies of candidate gene associations in pathology are relatively sparse and that most disorders are polygenetic. Additionally, the net outcome of a stressor is at least in part determined by the individual's personality traits that may be formed by genes, potentially indirectly influencing the selection of environments and thus the risk of exposure to adverse effects.

Lifespan models have examined the relation between early life stressful events, later stressful life events and pathology development. Undifferentiated neuronal systems are dependent on early experience during development. It is suggested that early life stress results in inefficient information processing and sensitization of brain circuits involved in regulating stress reactivity, which

may ultimately render people more vulnerable. Different brain structures have specific developmental trajectories resulting in a variety of pathological response after stress across the lifespan. For example, prenatal stress originating from maternal stress or postnatal environmental stress such as the quality of parental care influences the regulation of the HPA axis. However, exposure to a manageable stressor during childhood can also desensitize the stress circuits, producing experience-based resilience in which brain systems tend to become less reactive to future stress. Early life stress can hence be protective in that it can negate or diminish the negative outcomes or alternatively promote adaptive functioning in the context of adverse stressful life events. Additionally, other psychosocial factors during development like social support, parental care, and affective style have been identified as potentially protective factors that can enhance adaptive coping during or after stress. In a similar vein, brain frontal alpha asymmetry has been suggested to bias individuals' affective style and emotion regulation capacities. Specifically, left frontal activation has been linked to approach behavior and suggested to be an indicator of decreased vulnerability to depression whereas right frontal activation is viewed as a predispositional factor, lowering the threshold for adverse impact of stressful conditions.

In sum, stress vulnerability models underscore that the nature and intensity of the stressor in combination with genetic vulnerability factors, phenotypic vulnerability factors (personality, neuroendocrine reactivity), and both genetic and phenotypic protective (resilience) factors determine the impact and sequela of adverse stressful life events.

Cross-References

- ▶ Corticosteroids
- ▶ Cortisol
- ▶ Family Studies (genetics)
- ▶ Family Stress
- ▶ Gene-Environment Interaction
- ▶ Glucocorticoids
- ▶ Hypothalamic-Pituitary-Adrenal Axis
- ▶ Individual Differences
- ▶ Resilience
- ▶ Stress
- ▶ Stress Reactivity
- ▶ Stress Responses
- ▶ Stress Test
- ▶ Stress: Appraisal and Coping
- ▶ Stress, Caregiver
- ▶ Stressor
- ▶ Twin Studies

References and Readings

Coan, J. A., & Allen, J. J. B. (2003). The state and trait nature of frontal EEG asymmetry in emotion. In K. Hugdahl & R. J. Davidson (Eds.), *The asymmetrical brain* (pp. 565–616). Cambridge, MA\London: MIT Press.

Curtis, W. J., & Cicchetti, D. (2003). Moving research on resilience into the 21st century: Theoretical and methodological considerations in examining the biological contributors to resilience. *Development and Psychopathology, 15*, 773–810.

DeRijk, R. H., & de Kloet, E. R. (2008). Corticosteroid receptor polymorphisms: Determinants of vulnerability and resilience. *European Journal of Pharmacology, 583*, 303–311.

Gotlib, I. H., Joormann, J., Minor, K. L., & Hallmayer, J. (2008). HPA axis reactivity: A mechanism underlying the associations among 5-HTTLPR, stress, and depression. *Biological Psychiatry, 63*, 847–851.

Ingram, R. E., & Luxton, D. D. (2005). Vulnerability-stress models. In B. L. Hankin & J. R. Z. Abela (Eds.), *Development of psychopathology: A vulnerability-stress perspective* (pp. 32–46). Thousand Oaks, CA: Sage.

Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience, 10*(6), 434–445.

Oitzl, M. S., Champagne, D. L., van der Veen, R., & de Kloet, E. R. (2010). Brain development under stress: Hypotheses of glucocorticoid actions revisited. *Neuroscience and Biobehavioral Reviews, 34*, 853–866.

Stahl, S. M. (2008). *Stahl's essential psychopharmacology: Neuroscientific basis and practical applications* (3rd ed.). New York: Cambridge University Press.

Van Praag, H. M., de Kloet, E. R., & van Os, J. (2004). *Stress, the brain and depression*. New York: Cambridge University Press.

Zubin, J., & Spring, B. (1977). Vulnerability-a new view of schizophrenia. *Journal of Abnormal Psychology, 86*, 103–126.

Stress, Caregiver

Youngmee Kim
Department of Psychology, University of Miami, Coral Gables, Miami, FL, USA

Synonyms

Caregiver burden; Caregiver hassle; Caregiver strain

Definition

The stress of caregivers is defined as a feeling experienced when a person thinks that the demands of caregiving exceed the personal and social resources the individual is able to mobilize (Lazarus & Folkman, 1984).

Description

An illness affects not only the quality of life of individuals with the disease but also that of their family members and close friends who care for the patients. The stress of caregivers is defined as a feeling experienced when a person thinks that the demands of caregiving exceed the personal and social resources the individual is able to mobilize (Lazarus & Folkman, 1984). The caregiver role of family members incorporates diverse aspects involved in dealing with an illness of the relative. This role includes providing the patient with cognitive/informational, emotional, financial/legal, daily activity, medical, and spiritual support, as well as facilitating communication with medical professionals and other family members and assisting in the maintenance of social relationships (Kim & Given, 2008). All of these aspects can contribute to caregivers' stress when they perceive it difficult to mobilize their personal and social resources to carry out each of the caregiving-related tasks. Therefore, identifying the gaps between resources available for caregiving and the caregiving demands, unmet needs in caregiving, should be the initial step in the development of programs designed to reduce caregivers' stress and enhance their quality of life.

In addition to assessing the diverse aspects of caregivers' stress and unmet needs, understanding how caregivers' stress varies across the illness trajectory is an importance concern (Kim, Kashy, Spillers, & Evans, 2010). For example, in the early phase of caregivership, caregivers' stress is often associated with providing informational and medical support to the patients. During the remission phase, dealing with uncertainty about the future, fear that the disease may come back, the financial burden of extended treatment needs of the patients, and changes in social relationships are major sources of caregivers' stress. After the death of the patients, spiritual concerns and psychological and physical recovery efforts from caregiving strain are the challenges caregivers face.

Another important aspect of caregivers' stress is their own unmet needs—things that are not directly related to caring for the patient but represent important personal needs to the caregivers. That is, in addition to caring for the individual with an illness, family caregivers likely have responsibilities for self-care and care for other family members that may have to be set aside or ignored in order to carry out the caregiver role.

This complex construct of caregiver stress has been associated with caregivers' demographic characteristics (Kim et al., 2010; Pinquart & Sörensen, 2003, 2005). For example, younger caregivers have reported greater stress in providing psychosocial, medical, financial, and daily activity support during the early phase of the illness trajectory. During the remission years after the illness onset, however, younger caregivers have reported greater stress only in daily activity. Gender has been also an important factor. Female caregivers have reported greater stress from dealing with psychosocial concerns of the patients, other family members, and themselves. Ethnic minorities tend to report lower levels of psychological stress but greater levels

of physical stress from caregiving. Studies have found mixed associations of caregiver stress to other demographic characteristics, such as education, income, employment status.

Perceived level of stress from providing care has been significantly related to the caregivers' quality of life, after taking into consideration the variations in caregiving stress related to the demographic characteristics mentioned above (Kim et al., 2010; Pinquart & Sörensen, 2003). Caregivers who reported higher levels of psychosocial stress from caregiving have shown poorer mental health consistently and strongly across different phases of the illness trajectory. Caregivers' poorer mental health has also been related to higher levels of stress from meeting the medical needs of the patients during the early phase of illness, whereas during remission, poorer mental health has been related to financial stress from caregiving.

With regard to the self-reported physical health of the caregivers, caregivers' perceived stress has been a fairly weak contributor beyond contributions of demographic factors (Kim et al., 2010; Pinquart & Sörensen, 2003). However, the physical burden of caregiving, documented in objective measures, is considerable. For example, compared with matched non-caregivers, caregivers for a spouse with dementia report more infectious illness episodes, have poorer immune responses to influenza virus and pneumococcal pneumonia vaccines (Glaser, Sheridan, Malarkey, MacCaullum, & Kiecolt-Glaser, 2000), show slower healing for small standardized wounds, have greater depressive symptoms, and are at greater risk for coronary heart disease (2000; Vitaliano, Zhang, & Scanlan, 2003). A recent meta-analysis (2003) concluded that compared with demographically similar non-caregivers, caregivers of dementia patients had a 9% greater risk of health problems, a 23% higher level of stress hormones, and a 15% poorer antibody production. Moreover, caregivers' relative risk for all-cause mortality was 63% higher than non-caregiver controls.

Immune dysregulation has been identified as a key mechanism linking caregiving stress to physical health. Chronically stressed dementia caregivers have numerous immune deficits compared to demographically matched non-caregivers, including lower T-cell proliferation, higher production of immune regulatory cytokines (interleukin-2 [IL-2], C-reactive protein [CRP], tumor necrosis factor-alpha [TNF- α], IL-10, IL-6, D-dimer), decreased antibody and virus-specific T-cell responses to influenza virus vaccination, and a shift from a Th1 to Th2 cytokine response (i.e., an increase in the percentage and total number of IL10+/CD4+ and IL10+/CD8+ cells) (Segerstrom & Miller, 2004; Vitaliano et al., 2003). A 6-year longitudinal community study (Kiecolt-Glaser et al., 2003) documented that caregivers' average rate of increase in IL-6 was about four times as large as that of non-caregivers. The mean annual change in IL-6 among former caregivers did not differ from that of current caregivers, even several years after the death of the spouse. There were no systematic group differences in chronic health problems, medications, or health-relevant behaviors that might otherwise account for changes in caregivers' IL-6 levels during the 6 years of the study period (2003).

Another mechanism linking caregiving stress to poor physical health is lifestyle behaviors. Family members with chronic strain from caring for dementia patients increase health-risk behaviors, such as smoking and alcohol consumption (Carter, 2002). They also get inadequate rest, inadequate exercise, and forget to take prescription drugs to manage their own health conditions, resulting in poorer physical health (Beach, Schulz, Yee, & Jackson, 2000; Burton, Newsom, Schulz, Hirsch, & German, 1997).

In summary, caregiver stress is a multidimensional construct that varies in nature across the illness trajectory. Certain caregivers by their demographic characteristics can be identified as a vulnerable sub-group to greater caregiving stress. Overall, however, caregiving stress takes a considerable toll on the caregivers' mental and physical health. Such effects deserve further systematic study to understand

their psychological, biological, and behavioral pathways.

Cross-References

- Alzheimer's Disease
- Cancer Survivorship
- Caregiver/Caregiving and Stress
- Daily Stress
- Dementia
- Family Stress
- Family, Caregiver
- Family, Relationships
- Quality of Life

References and Readings

Beach, S. R., Schulz, R., Yee, J. L., & Jackson, S. (2000). Negative and positive health effects of caring for a disabled spouse: Longitudinal findings from the caregiver health effects study. *Psychology and Aging, 15*(2), 259–271.

Burton, L. C., Newsom, J. T., Schulz, R., Hirsch, C. H., & German, P. S. (1997). Preventive health behaviors among spousal caregivers. *Preventive Medicine, 26*(2), 162–169.

Carter, P. A. (2002). Caregivers' descriptions of sleep changes and depressive symptoms. *Oncology Nursing Forum, 29*(9), 1277–1283.

Glaser, R., Sheridan, J., Malarkey, W. B., MacCallum, R. C., & Kiecolt-Glaser, J. K. (2000). Chronic stress modulates the immune response to a pneumococcal pneumonia vaccine. *Psychosomatic Medicine, 62*, 804–807.

Kiecolt-Glaser, J. K., Preacher, K. J., MacCallum, R. C., Atkinson, C., Malarkey, W. B., & Glaser, R. (2003). Chronic stress and age-related increases in the proinflammatory cytokine IL-6. *Proceedings of the National Academy of Sciences of the United States of America, 100*, 9090–9095.

Kim, Y., & Given, B. A. (2008). Quality of life of family caregivers of cancer survivors across the trajectory of the illness. *Cancer, 112*(11 suppl), 2556–2568.

Kim, Y., Kashy, D. A., Spillers, R. L., & Evans, T. V. (2010). Needs assessment of family caregivers of cancer survivors: Three cohorts comparison. *Psycho-Oncology, 19*, 573–582.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal and coping*. New York: Springer.

Pinquart, M., & Sörensen, S. (2003). Differences between caregivers and noncaregivers in psychological health and physical health: A meta-analysis. *Psychology and Aging, 18*(2), 250–267.

Pinquart, M., & Sörensen, S. (2005). Ethnic differences in stressors, resources, and psychological outcomes of family caregiving: A meta-analysis. *The Gerontologist, 45*(1), 90–106.

Segerstrom, S. C., & Miller, G. E. (2004). Psychological stress and the human immune system: A meta-analytic study of 30 years of inquiry. *Psychological Bulletin, 130*, 601–630.

Vitaliano, P. P., Zhang, J., & Scanlan, J. M. (2003). Is caregiving hazardous to one's physical health? A meta-analysis. *Psychological Bulletin, 129*, 946–972.

Stress, Early Life

Christine Heim
Institute of Medical Psychology, Charité University Medicine Berlin, Berlin, Germany

Synonyms

Adversity, early life; Trauma, early life

Definition

In order to define early life stress in humans, two main criteria must be considered: (a) the developmental age range that is subsumed under "early life" and (b) the characteristics of the events that would be considered as "stressful" during early life. There is no such generally agreed upon definition (Heim, Meinlschmidt, & Nemeroff 2003).

Many investigators use an upper age limit to define the early life criterion, usually between 12 and 18 years. An alternative approach is to define the early life period by developmental stage, using, for example, sexual maturation (such as menarche in girls) as a cutoff criterion.

As for the stress criterion, prevailing models suggest that stress is generally experienced when an individual is confronted with a situation, which is appraised as personally threatening and for which adequate coping resources are

unavailable. In addition, threats to physiological homeostasis, such as injury or illness, elicit stress responses. Any such situation occurring within the defined developmental period may be classified as early life stress. The most salient forms of early life stress in humans are abuse (sexual, physical, emotional), neglect (emotional, physical), and parental loss (death, separation). Other forms of early life stress include accidents, physical illness, surgeries, natural disasters, and war or terrorism-related events. Less obvious experiences, which pose significant distress on a child, include unstable families, inadequate parental care, dysfunctional relationships between parent and child, and poverty.

Early life stress is often complex, inasmuch as various forms coexist or are associated among each other. While early life stress may be a single event, it more typically occurs as chronic or ongoing adversity in most cases. Taken together, there remains substantial ambiguity in the definition of early life stress in humans (Heim, Meinlschmidt, & Nemeroff 2003).

Description

It is well established that early life stress, such as childhood abuse, neglect, or loss, dramatically increases the risk for developing a wide range of psychiatric disorders as well as certain medical diseases later in life. Among the major psychiatric disorders, depression and anxiety disorders have been most prominently linked to early life stress. Medical disorders, for which early-life stress induces risk, include ischemic heart disease, lung disease, cancer, gastrointestinal disorders, and chronic fatigue and pain syndromes among others. Early life stress has further been linked to a variety of risk behaviors, including smoking, alcohol, or drug abuse, impulsive behavior, promiscuity, teen pregnancy, and suicide (for further reading, see Anda et al., 2006). Many of the above disorders and risk behaviors are elicited or aggravated by acute stress, and individuals with early life stress experiences have decreased thresholds to exhibit symptoms and risk behaviors even upon mild challenge (Hammen, Henry, & Daley, 2000).

The precise mechanisms that mediate the detrimental and persistent impact of early adversity on long-term adaptation and health have been the subject of intense inquiry over decades. Advances from neuroscience research have provided compelling insights into the enormous plasticity of the developing brain as a function of experience. For example, visual sensory input early in life is required for normal development of the visual cortex and perception, and disruptive experiences during such critical periods of plasticity can lead to lifelong and sometimes irreversible damage. The same principle may be applied to stress experiences during critical periods early in life that may permanently impact on the development of brain regions implicated in the regulation of emotion and stress responses (for further reading, see Weiss & Wagner, 1998). Enduring effects of early life stress on the brain and its regulatory outflow systems, including the autonomic, endocrine, and immune systems, may then lead to the development of a vulnerable phenotype with increased sensitivity to stress and risk for a range of behavioral and somatic disorders (for further reading, see Heim, Plotsky, & Nemeroff, 2004).

Compelling support for this hypothesis comes from a burgeoning literature of studies in animal models that provide the direct and causal evidence that early adverse experience, such as prolonged maternal separation or naturally occurring low maternal care, leads to structural, functional, and epigenetic changes in a connected network of brain regions that is implicated in neuroendocrine control, autonomic regulation and vigilance, and emotional regulation or fear conditioning. These neural changes converge into lifelong increased physiological and behavioral responses to subsequent stress in animal models (see Heim et al., 2004; Lupien, McEwen, Gunnar, & Heim, 2009; Meaney, 2001). These effects appear to be present across species and in different models of adversity, while the unifying element across studies is timing of the stressor in early life. Particularly intriguing are results

from animal studies suggesting that epigenetic changes, stress sensitization, and maternal care behavior are transmitted into the next generation (Francis, Diorio, Liu, & Meaney, 1999; Franklin et al., 2010).

Accumulating evidence suggests that these preclinical findings can be translated to humans. For example, adult women with histories of childhood sexual or physical abuse exhibit markedly increased neuroendocrine and autonomic responses to psychosocial laboratory stress, particularly those with depression (Heim et al., 2000). Other alterations in humans with early life stress experiences include glucocorticoid resistance, increased levels of inflammation, increased central corticotropin-releasing hormone activity and decreased activity of the prosocial neuropeptide, oxytocin (Carpenter et al. 2004; Danese, Pariante, Caspi, Taylor, & Poulton, 2007; Heim, Newport, Mletzko, Miller, & Nemeroff, 2008; Heim et al., 2009). A small hippocampus has also been linked to early life stress in humans (Vythilingam et al., 2002). Early adversity has also been found to be associated with epigenetic changes of the glucocorticoid receptor gene in hippocampal tissue obtained by postmortem from suicide victims, leading to reduced glucocorticoid receptor expression and enhanced stress responses (McGowan et al., 2008).

Taken together, these neurobiological and epigenetic changes secondary to early life stress likely reflect risk to develop depression and a host of other disorders in response to additional challenge. In several studies, these changes were not present in depressed persons without early life stress, suggesting the existence of biologically distinguishable subtypes of depression as a function of early life stress (Heim et al., 2008, 2004). These subtypes of depression were also found to be responsive to differential treatments (Nemeroff et al., 2003). Therefore, consideration of early life stress might be critical to guide treatment decisions.

Several genes moderate the link between childhood trauma and adult risk for depression and other disorders, including the serotonin transporter, corticotropin-releasing hormone receptor 1, FK506 binding protein 5, and oxytocin receptor genes. A more recent idea is that such genetic factors might reflect general sensitivity to the environment, inasmuch as persons who are susceptible to the detrimental effects of trauma might also be particularly amenable to the beneficial effects of a positive social environment or early psychological intervention (Binder et al., 2008; Bradley et al., 2008; Bradley, Westen, Binder, Jovanovic, & Heim, 2011; for further reading, see Caspi, Hariri, Holmes, Uher, & Moffitt, 2010).

References and Readings

Anda, R. F., Felitti, V. J., Bremner, J. D., Walker, J. D., Whitfield, C., Perry, B. D., et al. (2006). The enduring effects of abuse and related adverse experiences in childhood. *European Archives of Psychiatry and Clinical Neuroscience, 256*, 174–186.

Binder, E. B., Bradley, R. G., Liu, W., Epstein, M. P., Deveau, T. C., Mercer, K. B., et al. (2008). Association of FKBP5 polymorphisms and childhood abuse with risk of posttraumatic stress disorder symptoms in adults. *Journal of the American Medical Association, 299*, 1291–1305.

Bradley, R. G., Binder, E. B., Epstein, M. P., Tang, Y., Nair, H. P., Liu, W., et al. (2008). Influence of child abuse on adult depression: Moderation by the corticotropin-releasing hormone receptor gene. *Archives of General Psychiatry, 65*, 190–200.

Bradley, B., Westen, D., Binder, E. B., Jovanovic, T., & Heim, C. (2011). Association between childhood maltreatment and adult emotional dysregulation: Moderation by oxytocin receptor gene. *Developmental Psychopathology, 23*(2), 439–452.

Carpenter, L., Tyrka, A., McDougle, C. J., Malison, R. T., Owens, M. J., Nemeroff, C. B., et al. (2004). CSF corticotropin-releasing factor and perceived early-life stress in depressed patients and healthy control subjects. *Neuropsychopharmacology, 29*, 777–784.

Caspi, A., Hariri, A. R., Holmes, A., Uher, R., & Moffitt, T. E. (2010). Genetic sensitivity to the environment: The case of the serotonin transporter gene and its implications for studying complex diseases and traits. *American Journal of Psychiatry, 167*, 509–527.

Danese, A., Pariante, C. M., Caspi, A., Taylor, A., & Poulton, R. (2007). Childhood maltreatment predicts adult inflammation in a life-course study. *Proceedings of the National Academy of Sciences United States of America, 104*, 1319–1324.

Francis, D., Diorio, J., Liu, D., & Meaney, M. J. (1999). Nongenomic transmission across generations of maternal behavior and stress responses in the rat. *Science, 286*, 1155–1158.
Franklin, T. B., Russig, H., Weiss, I. C., Gräff, J., Linder, N., Michalon, A., et al. (2010). Epigenetic transmission of the impact of early stress across generations. *Biological Psychiatry, 68*, 408–415.
Hammen, C., Henry, R., & Daley, S. E. (2000). Depression and sensitization to stressors among young women as a function of childhood adversity. *Journal of Consulting and Clinical Psychology, 68*, 782–787.
Heim, C., Meinlschmidt, G. & Nemeroff, C. B. (2003). Neurobiology of early-life stress and its relationship to PTSD. *Psychiatric Annals, 33*, 1–10.
Heim, C., Newport, D. J., Heit, S., Graham, Y. P., Wilcox, M., Bonsall, R., et al. (2000). Pituitary-adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. *Journal of the American Medical Association, 284*, 592–597.
Heim, C., Newport, D. J., Mletzko, T., Miller, A. H., & Nemeroff, C. B. (2008). The link between childhood trauma and depression: Insights from HPA axis studies in humans. *Psychoneuroendocrinology, 33*, 693–710.
Heim, C., Plotsky, P. M., & Nemeroff, C. B. (2004). Importance of studying the contributions of early adverse experience to neurobiological findings in depression. *Neuropsychopharmacology, 29*, 641–648.
Heim, C., Young, L. J., Newport, D. J., Mletzko, T., Miller, A. H., & Nemeroff, C. B. (2009). Lower cerebrospinal fluid oxytocin concentrations in women with a history of childhood abuse. *Molecular Psychiatry, 14*, 954–958.
Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience, 10*, 434–445.
McGowan, P. O., Sasaki, A., D'Alessio, A. C., Dymov, S., Labonté, B., Szyf, M., et al. (2008). Epigenetic regulation of the glucocorticoid receptor in human brain associates with childhood abuse. *Nature Neuroscience, 12*, 342–348.
Meaney, M. J. (2001). Maternal care, gene expression, and the transmission of individual differences in stress reactivity across generations. *Annual Reviews in Neuroscience, 24*, 1161–1192.
Nemeroff, C. B., Heim, C., Thase, M. E., Klein, D. N., Rush, A. J., Schatzberg, A. F., et al. (2003). Differential responses to psychotherapy versus pharmacotherapy in patients with chronic forms of major depression and childhood trauma. *Proceedings of the National Academy of Sciences United States of America, 100*, 14293–14396.
Vythilingam, M., Heim, C., Newport, J., Miller, A. H., Anderson, E., Bronen, R., et al. (2002). Childhood trauma associated with smaller hippocampal volume in women with major depression. *American Journal of Psychiatry, 159*, 2072–2080.
Weiss, M. J., & Wagner, S. H. (1998). What explains the negative consequences of adverse childhood experiences on adult health? Insights from cognitive and neuroscience research. *American Journal of Preventive Medicine, 14*, 356–360.

Stress, Emotional

Tamar Mendelson
Mental Health, Johns Hopkins Bloomberg School of Public Health Johns Hopkins University, Baltimore, MD, USA

Synonyms

Emotional distress; Mental stress; Psychological stress; Stress

Definition

Emotional stress involves the experience of negative affect, such as anxiety, in the context of a physiological stress response that includes cardiovascular and hormonal changes. Emotional stress commonly occurs when an individual perceives that he or she does not have adequate personal resources to meet situational demands effectively (Lazarus, 1966).

Description

Early conceptions of stress characterized its physical properties, with a focus on the disruption of homeostasis in an organism (Selye, 1956). The stress concept subsequently evolved to include a greater emphasis on the influence of psychological factors on the stress process. The term "emotional stress" reflects the fact that the stress process in humans involves a substantial affective component.

Emotional stress includes both negative affect, such as anxiety and distress, as well as a cascade of physiological responses associated with the

stress-response system. Physiological responses promote "fight or flight" and include activation of the hypothalamic pituitary adrenal (HPA) axis, which stimulates secretion of cortisol, and activation of the sympathetic nervous system, which increases heart rate (Sapolsky, 1994). Behavioral responses may include attempts to flee or avoid the stressor or to actively address it.

Emotional stress can be triggered by various stress exposures, including major life events, chronic stressful situations, and daily hassles. Certain objective features of a stressor influence the likelihood that it will produce emotional stress. For instance, emotional stress is more likely to result from stressors that are not within an individual's control (e.g., a death) and affect central aspects of an individual's life (Dohrenwend, 2000).

Individual differences are also critical components in predicting levels of emotional stress, particularly when the stressor is not extremely traumatic. Thus, the same stressor may produce emotional stress in one individual but not in another. Richard Lazarus and Susan Folkman's work has established the importance of appraisal processes in generating or buffering against stress (Lazarus & Folkman, 1984). Emotional stress results from an appraisal that the situation is threatening and that efforts to address it effectively are not likely to be successful. In contrast, a sense of positive challenge may arise if the situation is not perceived as overly threatening or if the perceiver feels capable of an effective response. Similarly, a number of other factors can increase risk for, or protect against, emotional stress. These factors include the ability to employ effective coping strategies and the presence of positive social supports (Kessler, Price, & Wortman, 1985).

A life course perspective is important for understanding the etiology of vulnerability to emotional stress. Both vulnerability to stress and resilience are likely shaped over the life course by complex interactions of genetic factors, biological mechanisms, and environmental exposures. Emerging research suggests that exposure to stress and adversity during sensitive periods early in the life course (prenatal, early postnatal, childhood) may be especially critical in influencing genetic expression and impacting the developing stress-response system, with long-term effects on vulnerability to emotional stress (Anderson & Teicher, 2009; Dudley, Li, Kobor, Kippin, & Bredy, 2011; Shonkoff, Boyce, & McEwen, 2009).

A key reason for continued interest in the study of emotional stress is its well-documented link with development of both mental and physical disorders. Major depressive disorder and posttraumatic stress disorder are two commonly studied psychiatric sequelae of emotional stress. Emotional stress has also been found to predict cardiovascular disease and other physical health problems (Brotman, Golden, & Wittstein, 2007; Rozanski, Blumenthal, & Kaplan, 1999). Putative mechanisms linking emotional stress with psychiatric and physical disorders include stress-related neurobiological changes (e.g., dysregulation of the HPA axis) and increased cardiovascular reactivity to stress with slow recovery (Chida & Steptoe, 2010; Hammen, 2005); detailed understanding of these pathways requires more study.

A variety of psychosocial stress management interventions have been developed to reduce emotional stress and prevent its negative effects on health. Such interventions generally aim to enhance positive coping methods, including the use of relaxation techniques, exercise, and cognitive strategies for managing stress. Some stress management interventions have been shown to have positive effects on emotional and physical outcomes (e.g., Blumenthal et al., 2005).

Cross-References

- ▶ Cardiovascular Disease
- ▶ Coping
- ▶ Depression: Symptoms
- ▶ Relaxation
- ▶ Stress Responses
- ▶ Stress Vulnerability Models
- ▶ Stress, Early Life

- ▶ Stress Reactivity
- ▶ Stress: Appraisal and Coping
- ▶ Stressor
- ▶ Sympathetic Nervous System (SNS)

References and Readings

Andersen, S. L., & Teicher, M. H. (2009). Desperately driven and no brakes: Developmental stress exposure and subsequent risk for substance use. *Neuroscience and Behavioral Reviews, 33*, 516–524.

Blumenthal, J. A., Sherwood, A., Babyak, M. A., Watkins, L. L., Waugh, R., Georgiades, A., et al. (2005). Effects of exercise and stress management training on markers of cardiovascular risk in patients with ischemic heart disease: A randomized controlled trial. *Journal of the American Medical Association, 293*, 1626–1634.

Brotman, D. J., Golden, S. H., & Wittstein, I. S. (2007). The cardiovascular toll of stress. *Lancet, 370*, 1089–1100.

Chida, Y., & Steptoe, A. (2010). Greater cardiovascular responses to laboratory mental stress are associated with poor subsequent cardiovascular risk status: A meta-analysis of prospective evidence. *Hypertension, 55*, 1026–1032.

Dohrenwend, B. P. (2000). The role of adversity and stress in psychopathology: Some evidence and its implications for theory ad research. *Journal of Health and Social Behavior, 41*, 1–19.

Dudley, K. J., Li, X., Kobor, M. S., Kippin, T. E., & Bredy, T. W. (2011). Epigenetic mechanisms mediating vulnerability and resilience to psychiatric disorders. *Neuroscience and Biobehavioral Reviews, 35*, 1544–1551.

Hammen, C. (2005). Stress and depression. *Annual Review of Clinical Psychology, 1*, 293–319.

Kessler, R. C., Price, R. H., & Wortman, C. B. (1985). Social factors in psychopathology: Stress, coping, and coping processes. *Annual Review of Psychology, 36*, 531–572.

Lazarus, R. S. (1966). *Psychological stress and the coping process*. New York: McGraw Hill.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.

Rozanski, A., Blumenthal, J. A., & Kaplan, J. (1999). Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation, 99*, 2192–2217.

Sapolsky, R. M. (1994). *Why zebras don't get ulcers*. New York: Holt.

Selye, H. (1956). *The stress of life*. New York: Mc-Graw-Hill.

Shonkoff, J. P., Boyce, W. T., & McEwen, B. S. (2009). Neuroscience, molecular biology, and the childhood roots of health disparities. Building a new framework for health promotion and disease prevention. *Journal of the American Medical Association, 301*, 2252–2259.

Stress, Exercise

Rick LaCaille[1] and Marc Taylor[2]
[1]Psychology Department, University of Minnesota Duluth, Duluth, MN, USA
[2]Department 163, Behavioral Sciences & Epidemiology, Naval Health Research Center, San Diego, CA, USA

Definition

Exercise is a form of physical activity that involves repeated body movements that are both structured and planned with the intention of maintaining or enhancing one's health or physical fitness. Typically, exercise is characterized as either aerobic or anaerobic with the former emphasizing the use of oxygen for sustained movements such as jogging or swimming, whereas the latter emphasizes the use of muscle glycogen supply and metabolism as sources of energy for higher-intensity activities such as strength and resistance training. Moreover, exercise has been defined in terms of being chronic/regular/habitual or as acute/single bout.

Stress represents a response among biopsychosocial systems in an effort to adapt to a challenge. The nature of the challenge has been delineated along several dimensions, including but not limited to its intensity, duration, frequency, quality, and familiarity. The stress response has been measured in naturally occurring situations as well as in laboratory settings and ranged from self-reported questionnaires to physiological indices of neuroendocrine, heart rate, and blood pressure reactivity.

Description

The health benefits of exercise and physical fitness have been well documented in the literature. In particular, the effects of exercise on reduced morbidity and mortality from cardiovascular disease have received a great deal of attention, in part, due to the association between stress and

cardiovascular disease and the potential attenuation of physiological reactivity to stressors. Notably, an acute bout of exercise may also act as a stressor and elicit the same cardiovascular and neuroendocrine responses as psychosocial stressors without the detrimental effects health. As exercise becomes chronic and habitual, the physiological adaptations (e.g., reduced heart rate and blood pressure and increased parasympathetic activity) that occur are thought to yield similar responses and adaptations in the presence of psychosocial stressors. This beneficial adaptation and reduced sensitivity to stress is characterized in the literature as the cross-stressor-adaptation hypothesis. Thus, the effect of exercise on stress response is somewhat paradoxical as it is considered to be both a stressor and a potential modifier of stress. Most investigations of exercise-stress adaptations have relied upon laboratory stressors (e.g., mental arithmetic, public speaking and evaluative scenarios, cold pressor tests, reaction time, Stroop color-word test), cardiorespiratory responses, and cross-sectional designs. Although a number of studies have examined the association between exercise/fitness and psychophysiological stress responses, the findings have been rather mixed even among the reviews and quantitative analyses of the literature.

In an initial meta-analysis of the relationship between aerobic fitness and resistance to stress reactivity by Crews and Landers (1987), the overall effect size across multiple indices was d = 0.48 with effects ranging from 0.15 to 0.87. The findings from this review suggested that fitness/exercise training was beneficial in reducing reactivity to stressors with regards to heart rate d = (0.39), diastolic blood pressure d = (0.40), systolic blood pressure d = (0.42), self-reported stress d = (0.57), skin response d = (0.67), and muscle tension d = (0.87). The review has since been criticized for a number of methodological limitations including confounding reactivity with recovery. Later qualitative reviews reported no beneficial effect for fitness on stress reactivity in terms of heart rate, blood pressure, or catecholamine responses, with the effect on stress recovery determined to be inconclusive (Claytor, 1991; De Geus & Van Doornen, 1993). More recently, Jackson and Dishman (2006) revealed in a meta-analytic review that fitness was associated with a slight increase in stress reactivity to laboratory psychosocial stressors. Notably, a small effect size was present between cardiorespiratory fitness and stress response recovery, indicating that physically fitter individuals appeared to have an enhanced and quicker recovery following their peak stress response. In contrast to these findings, Forcier and colleague's (2006) meta-analytic review examining the effects of aerobic fitness on stress reactivity and recovery revealed that, despite considerable heterogeneity present in the analyses and no significant moderation effects (e.g., gender, stressor intensity), significant effects for exercise/fitness were found for decreased heart rate and systolic blood pressure reactivity. Additionally, a significant effect was found for fitness and heart rate recovery, though no such effect was present for systolic blood pressure. Thus, the findings from this meta-analysis suggest a beneficial attenuated physiological reactivity and improved recovery from psychosocial stressors as a consequence of fitness/chronic exercise. Although the effects between fitness and reactivity/recovery appeared small in magnitude (e.g., 1.8 bpm heart rate reactivity, 3.7 mmHg systolic blood pressure reactivity), the differences are equivalent to 15–25% reductions in reactivity which may still be of clinical importance.

Evidence from a recent meta-analysis (Hamer, Taylor, & Steptoe, 2006) examining blood pressure response to a psychosocial stressor suggested that an acute bout of aerobic exercise may result in a significant reduction in reactivity. That is, medium effect sizes revealed that exercise resulted in beneficial attenuated stress reactivity for both diastolic and systolic blood pressure with reductions of a 3.0 mmHg and 3.7 mmHg in diastolic and systolic responses, respectively. The observed effects appeared most robust with psychosocial stressors administered up to 30 min postexercise with moderate to vigorous exercise intensity and durations lasting from 20 min to 2 h. The findings from this review offer the possibility that the effects of acute exercise may account for some

of the mixed results from studies examining chronic exercise and blood pressure reactivity because habitually exercising may place an individual in the postexercise "window" more frequently when encountering daily stressors and thereby result in attenuated stress responses.

Although evidence from meta-analytic reviews is lacking, some studies have reported relationships between physical fitness and reduced hypothalamic-pituitary-adrenal (HPA) axis psychosocial stress reactivity, such as cortisol and inflammatory cytokine production. However, the ability to draw solid conclusions in this area is currently limited. With regard to sympathetic-adrenal-medullary responses, the findings appear conflicting as a result of sampling methodology and exercise intensity and durations employed. Some studies have found no effect of fitness on norepinephrine and epinephrine levels, whereas other studies have reported higher norepinephrine levels in the early phases of a stress response or, conversely, an association between lower levels of fitness and an increased norepinephrine response. In animal analogue studies, levels of norepinephrine have been shown to increase in the frontal cortex following an acute bout of exercise, suggesting increased vigilance to threat and reactivity; however, reduced levels have been found in the hypothalamus and hippocampus which suggests diminished stress reactivity.

In summary, although it appears that acute and chronic exercise and fitness may favorably influence an individual's response to psychosocial stress, further research is needed to clarify this relationship. Reviews of the literature suggest that there is a need for greater methodological rigor and reporting as well as examination of potential moderators of exercise-reactivity and exercise-recovery associations.

Cross-References

- ▶ Benefits of Exercise
- ▶ Physical Activity and Health
- ▶ Stress
- ▶ Stress Reactivity
- ▶ Stressor

References and Readings

Buckworth, J., & Dishman, R. K. (2002). *Exercise psychology*. Champaign, IL: Human Kinetics.

Claytor, R. P. (1991). Stress reactivity: Hemodynamic adjustments in trained and untrained humans. *Medicine and Science in Sports and Exercise, 23*, 873–881.

Crews, D. J., & Landers, D. M. (1987). A meta-analytic review of aerobic fitness and reactivity to psychosocial stressors. *Medicine and Science in Sports and Exercise, 19*(Suppl), S114–S120.

De Geus, E. J. C., & Van Doornen, L. J. P. (1993). The effects of fitness training on the physiological stress response. *Work and Stress, 7*, 141–159.

Edenfield, T. M., & Blumenthal, J. A. (2011). Exercise and stress reduction. In A. Baum & R. Contrada (Eds.), *The handbook of stress science: Biology, psychology, and health* (pp. 301–319). New York: Springer.

Forcier, K., Stroud, L. R., Papandonatos, G. D., Hitsman, B., Reiches, M., Krishnamoorthy, J., et al. (2006). Links between physical fitness and cardiovascular reactivity and recovery to psychological stressors: A meta-analysis. *Health Psychology, 25*, 723–739.

Hamer, M., Taylor, A., & Steptoe, A. (2006). The effect of acute aerobic exercise on stress related blood pressure responses: A systematic review and meta-analysis. *Biological Psychology, 71*, 183–190.

Hand, G. A., Phillips, K. D., & Wilson, M. A. (2006). Central regulation of stress reactivity and physical activity. In E. Acevado & P. Ekkekakis (Eds.), *Psychobiology of physical activity* (pp. 189–201). Champaign, IL: Human Kinetics.

Jackson, E. M., & Dishman, R. K. (2006). Cardiorespiratory fitness and laboratory stress: A meta-regression analysis. *Psychophysiology, 43*, 57–72.

Sothmann, M. S., Buckworth, J., Claytor, R. P., Cox, R. H., White-Welkley, J. E., & Dishman, R. K. (1996). Exercise training and the cross-stressor adaptation hypothesis. *Exercise and Sport Sciences Reviews, 24*, 267–287.

Stress, Posttraumatic

Viana Turcios-Cotto
Department of Psychology, University of Connecticut, Storrs, CT, USA

Synonyms

PTS

Definition

Posttraumatic stress is a stress reaction characterized by a multitude of symptoms following a traumatic event. Symptoms can be affective, behavioral, cognitive, and/or physiological in nature. These symptoms appear only after exposure to an event that involved a threat to one's physical integrity such as actual or threatened death or serious injury to oneself, witnessing such a threat on another person, or learning of such a threat experienced by a family member or other close person. A traumatic event can come in many forms including but not limited to a natural disaster (e.g., earthquake, tornado), violent personal attack, (e.g., rape, robbery), military combat, terrorist attack, severe automobile accident, or diagnosis of life-threatening illness (American Psychiatric Association [APA], 1994). Although posttraumatic stress is similar to posttraumatic stress disorder (PTSD), a diagnosable psychological condition, it is distinctly different in that the symptoms of posttraumatic stress may not be as severe or as numerous as those of PTSD. Thus, not all of the criteria necessary for diagnosis of PTSD are met, and the symptoms typically do not cause clinically significant distress or impairment in important areas of functioning.

Description

Rates of trauma exposure are unclear, but it is estimated that a majority of people, over 60%, living within the United States have experienced at least one traumatic event (Resick, Monson, & Rizvi, 2008). Some of those affected will develop no signs of distress, whereas some will develop clinically significant, diagnosable signs of distress, and yet others, people experiencing posttraumatic stress, can be categorized as developing subclinical levels of distress. Like many other psychological difficulties, whether or not someone develops significant signs of distress after a traumatic event depends on many variables such as the individual's coping abilities, trauma history, support system, type of traumatic event, intensity of the event, proximity to the event, and so on. Individuals with diminished coping abilities, extensive trauma histories, weak support systems, and who experience more intense and physical traumas closer to their bodies will be at greater risk for suffering from more severe posttraumatic distress (APA, 1994; Resick et al., 2008). Since the subclinical population does not warrant a diagnosis and most likely does not seek treatment due to the lack of or low level of impairment, it is difficult to estimate how many are afflicted by posttraumatic stress. However, individuals living with posttraumatic stress experience various difficulties, which can be categorized into groups of affective, behavioral, cognitive, and physiological symptoms.

Affective Symptoms

After a traumatic event, one can feel a variety of stressful emotions as a consequence for days, weeks, or even months after the experience. Intense fear and helplessness are characteristic markers of posttraumatic stress (APA, 1994; Beidel & Stipelman, 2007; Resick et al., 2008). A person can feel scared that the event might reoccur at any moment and horror that he or she will be unable to stop it from happening again. One might fear for their life and worry that death or severe injury is imminent, creating constant anxiety, and perhaps a sense of a foreshortened future (APA, 1994; Beidel & Stipelman, 2007). Some people may react to a traumatic incident with guilt and/or shame, unwilling to talk to others about the incident believing that they are at fault for the event. Sadness and even depression can also arise as a result from experiencing a trauma. This can lead to no longer finding pleasure in activities that the person once enjoyed or in diminished social involvement (Beidel & Stipelman, 2007). Lastly, an individual may experience a feeling of anger, manifested through irritability or outbursts (APA, 1994). This anger may be focused on the event itself; it may also be geared toward others, perhaps blaming others for the occurrence of the event or fueled by mistrust of others. Overall, the affective symptoms that result from posttraumatic stress are typically negative and can become harmful if they linger for too long.

Behavioral Symptoms

Certain behaviors that were not present before a traumatic event can also surface as a result. Hypervigilance, a constant watchful eye on one's surroundings, is a hallmark behavior used to protect oneself from unexpected threats. This hypervigilance includes heightened sensory sensitivity with an intense, somewhat irrational reaction or an exaggerated startle response (APA, 1994; Beidel & Stipelman, 2007). For example, an individual who has personally experienced an earthquake may become more aware of slight tremors, flickering lights, rattling windows, or rumbling sounds that others do not notice. These stimuli might arouse fear or anxiety in the individual who might feel as if they are reexperiencing the traumatic event. A person may also purposely work toward not reexperiencing or remembering the event. He or she might avoid certain places, people, smells, sounds, topics of conversation, or anything else that might trigger memories of the incident and may even be unable to recall certain aspects of the event altogether (Beidel & Stipelman, 2007; Resick et al., 2008). One might also learn to numb their feelings so that they no longer experience fear, anxiety, or anger as a protective measure from the various affective symptoms that may have developed after the trauma. However, this typically also leads to numbing positive affect such as joy and excitement as well, restricting one's range of affect (APA, 1994; Beidel & Stipelman, 2007). Avoidance and numbing can further cause a person to become somewhat reclusive and decrease involvement in social situations, resulting in a feeling of detachment or estrangement from friends and family. In general, the behavioral symptoms that appear after a traumatic event are used as coping mechanisms to either protect oneself to keep such an event from reoccurring or to distance oneself from the event that has occurred.

Cognitive Symptoms

There are key cognitions that are representative of those individuals who have experienced a trauma. Most often, there is an overgeneralization of the event and the harm that it caused (Resick et al., 2008). In other words, an individual might believe that harmful or deadly events happen more often and in more places than previously thought. He or she might feel that he or she is always at risk and lacks safety, misappraising minor events or stimuli as much more dangerous than they really are. A victim might lose trust in others and think that others are out to harm him or her, particularly if another human being caused the trauma. Other distorted cognitions about power and control may surface as well (Resick et al.). Such beliefs might be that one has no control or power over events in their daily life, perhaps lowering self-esteem. Another distortion might be self-blame where the victim believes he or she had complete control and power in the situation, bringing the traumatic event upon him or herself. This can result in a feeling of shame or guilt.

Although cognitive symptoms usually only include cognitions or beliefs that a person holds, in the case of posttraumatic stress, they also include ideas, images, and impulses that might materialize in one's mind after a traumatic event. Individuals might have sudden, intrusive memories of images, smells, sounds, etc., of the event flash in his or her mind (APA, 1994). The victim might also have distressing dreams about the incident or feel and act as if he or she is reliving the terrible moment (Resick et al., 2008). Due to the many stressors experienced after a traumatic event, concentration may be difficult for individuals with posttraumatic stress.

Physiological Symptoms

Posttraumatic stress causes physiological changes as well. Most notably, there is an increase in heart rate and sweat gland activity. Levels of cortisol may also rise. These symptoms may lead to exhaustion or various serious illnesses such as heart disease (Resick et al., 2008; Sarafino, 2008).

Summary

Posttraumatic stress is a psychological stress reaction following a traumatic event. It is characterized by a multitude of affective, behavioral, cognitive, and physiological symptoms that

negatively impact an individual. However, the symptoms are not severe enough to cause impairment in social, occupational, or other areas of functioning and therefore do not warrant a diagnosis. Nevertheless, if symptoms become more severe and do not diminish within a month's time, they may be a sign of a diagnosable psychological condition, posttraumatic stress disorder. In such a case, the individual should seek treatment from a professional psychologist or counselor.

Cross-References

- ▶ Posttraumatic Growth
- ▶ Posttraumatic Stress Disorder
- ▶ Stress

References and Readings

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Beidel, D. C., & Stipelman, B. (2007). Anxiety disorders, Chapter 11. In M. Hersen, S. M. Turner, & D. C. Beidel (Eds.), *Adult psychopathology and diagnosis* (5th ed., pp. 349–409). Hoboken, NJ: John Wiley & Sons.

Resick, P. A., Monson, C. M., & Rizvi, S. L. (2008). Posstraumatic stress disorder, Chapter 2. In D. H. Barlow (Ed.), *Clinical handbook of psychological disorders: A step-by-step treatment manual* (4th ed., pp. 65–122). New York: Guilford Press.

Sarafino, E. P. (2008). Stress – Its meaning, impact, and sources, Chapter 3. In E. P. Sarafino (Ed.), *Healh psychology: Biopsychosocial interactions* (6th ed., pp. 61–86). Hoboken, NJ: John Wiley & Sons.

Stress: Appraisal and Coping

Susan Folkman
Department of Medicine, School of Medicine, University of California San Francisco, San Mateo, CA, USA

Definition

Stress has been defined traditionally either as a *stimulus*, often referred to as a *stressor*, that happens to the person such as a laboratory shock or loss of a job, or as a *response* characterized by physiological arousal and negative affect, especially anxiety. In his 1966 book, *Psychological Stress and the Coping Process* (Lazarus, 1966), Richard Lazarus defined stress as a relationship between the person and the environment that is appraised as personally significant and as taxing or exceeding resources for coping. This definition is the foundation of stress and coping theory (Lazarus & Folkman, 1984).

Description

Stress and coping theory provides a framework that is useful for formulating and testing hypotheses about the stress process and its relation to physical and mental health. The framework emphasizes the importance of two processes, appraisal and coping, as mediators of the ongoing relationship between the person and the environment. Stress and coping theory is relevant to the stress process as it is experienced in the ordinary events of daily life, major life events, and chronic stressful conditions that stretch out over years.

Appraisal refers to the individual's continuous evaluation of how things are going in relation to his or her personal goals, values, and beliefs. Primary appraisal asks "Am I okay?" Secondary appraisal asks "What can I do?" Situations that signal harm or potential harm that is personally significant and in which there are few options for controlling what happens are appraised as stressful. Stress appraisals include harm or loss, which refer to damage already done; appraisals of threat, which refer to the judgment that something bad might happen; and appraisals of challenge, which refer to something that may happen that offers the opportunity for mastery or gain as well as some risk of an unwelcome outcome. Situations that are appraised as high in personal significance and low in controllability, for example, are usually appraised as threats, and situations that are high in personal significance and high in controllability are more likely to be appraised as challenges.

The concept of appraisal addresses the issue of variability of responses among people experiencing a similar stressor and why a given situation may be more stressful for one person than another. The situation may involve goals, values, or beliefs that are more personally significant for one person than for another, or one person may be better equipped than another to control the situation's outcome. Appraisal-based approaches now dominate the field (Pearlin, Lieberman, Menaghan, & Mullan, 1981).

Appraisals generate emotions that vary in quality and intensity according to the person's evaluation of personal significance (primary appraisal) and options for coping (secondary appraisal). Threat appraisals, for example, are often accompanied by fear, anxiety, and worry; harm/loss appraisals are often accompanied by anger, sadness, or guilt; and challenge appraisals are often accompanied by eagerness and excitement as well as a touch of threat.

People experience a complex array of emotions during real-life stressful events, including positive as well as negative emotions (Folkman, 1997, 2008). Emotions indicate that something is happening that matters to the individual. Emotions also often signal what the person intends to do. Negative emotions have long been associated with the individual's preparation to approach or avoid, fight or flee (Lazarus, 1991). Positive emotions have more recently been examined for their roles in the stress process. Positive emotions, for example, are associated with widened focus of attention, motivating meaning-focused coping, and eliciting social support (Folkman, 2008; Fredrickson, 1998).

Coping refers to the thoughts and actions people use to manage distress (emotion-focused coping), manage the problem causing the distress (problem-focused coping), and sustain positive well-being (meaning-focused coping). Emotion-focused coping includes strategies such as distancing, humor, and seeking social support that are generally considered adaptive, and strategies such as escape-avoidance, day dreaming, and blaming others that are generally considered maladaptive. Problem-focused coping includes strategies such as information gathering, seeking advice, drawing on previous experience, negotiating, and problem solving. Meaning-focused coping includes strategies such as focusing on deeply held values, beliefs, and goals; reframing or reappraising situations in positive ways; and amplifying positive moments over the course of a day (Folkman & Moskowitz, 2000).

Coping is influenced by the person's coping resources including psychological, spiritual, social, environmental, and material resources, and by the nature of the situation, especially whether its outcome is controllable or has to be accepted. Problem-focused coping is used more in situations that are controllable, and emotion-focused coping is used more in situations that have to be accepted. Meaning-focused coping appears to be used more in situations that are chronic and not resolvable, such as in caregiving or serious illness. It is hypothesized that meaning-focused coping becomes more active when initial coping efforts fail to make the situation better (Folkman, 2011). Meaning-focused coping sustains other coping efforts and restores coping resources.

People use an array of coping strategies in real-life situations. Most situations involve more than one coping task or goal, each of which requires a coping strategy tailored to that task or goal. And people switch coping strategies when the ones they are using do not have the desired effect. Coping also changes as an encounter unfolds in response to changes in the environment, the situation, or to changes within the person.

Coping effectiveness is determined contextually because effective coping in one situation may be ineffective in another. For instance, distancing may be ineffective when a person should be problem solving or preparing for an upcoming challenge, whereas it may be effective when there is nothing to be done, as when waiting for a test result. Researchers often identify on an a priori basis the outcome that is desired, such as improved mood. In such cases, effective coping is the coping that is associated with the desired outcome.

Another approach to evaluating coping is to examine the goodness of the fit between the

appraised options for coping and the choice of coping strategy. Problem-focused coping that is used when the situation is appraised as controllable, for example, would be a good fit, whereas the same form of coping in situation where nothing can be done would be a poor fit. Conversely, distancing that is used when there is nothing that can be done would be a good fit, whereas the same form of coping in a controllable situation that called for attention would be a poor fit.

Like appraisal, coping is key to understanding why the outcomes of given stressful situations can vary from person to person. Two people may cope quite differently with the same stressful situation because of differences in their resources, experiences, motivation, preferences, and skills for coping.

The dynamic quality of the stress process is evident in changes in the appraisal and reappraisal process, the fluidity of emotions, and changes in coping thoughts and actions as an encounter unfolds. The processes are also in reciprocal relationships. An outcome of appraisal and coping at Time 1, such as mood, for example, can become a predictor of appraisal and coping at Time 2.

References and Readings

Folkman, S. (1997). Positive psychological states and coping with severe stress. *Social Science and Medicine, 45*, 1207–1221.

Folkman, S. (2008). The case for positive emotions in the stress process. *Anxiety Stress Coping, 21*, 3–14.

Folkman, S. (Ed.). (2011). *The Oxford handbook of stress, health, and coping*. New York: Oxford University Press.

Folkman, S., & Moskowitz, J. T. (2000). Positive affect and the other side of coping. *American Psychologist, 55*, 647–654.

Fredrickson, B. L. (1998). What good are positive emotions? *Review of General Psychology Special Issue: New Directions in Research on Emotion, 2*, 300–319.

Lazarus, R. S. (1966). *Psychological stress and the coping process*. New York: McGraw Hill.

Lazarus, R. S. (1991). *Emotion and adaptation*. New York: Oxford University Press.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.

Pearlin, L. I., Lieberman, M. A., Menaghan, E. G., & Mullan, J. T. (1981). The stress process. *Journal of Health and Social Behavior, 22*, 337–356.

Stressful Events

▶ Life Events

Stressful Life Event

▶ Psychosocial Factors and Traumatic Events

Stressor

▶ Psychological Stress

Stress-Related Growth

▶ Benefit Finding
▶ Perceived Benefits
▶ Posttraumatic Growth

Stroke Burden

Jonathan Newman
Columbia University, New York, NY, USA

Definition

Description

Strokes are one of the leading causes of death and disability: each year, nearly 800,000 Americans experience a new or recurrent stroke. Approximately 600,000 of these are first events and roughly 180,000 are recurrent attacks. In the United States, strokes accounted for 1 out of every 16 deaths in 2004, and more than 50% of the deaths attributable to strokes occurred outside of the hospital. When separated from other cardiovascular diseases, stroke is the third leading cause of death, behind heart disease and cancer. Importantly, strides have been made in reduction

of stroke mortality: the stroke death rate has fallen more than 20% from 1994 to 2004. However, important disparities remain. While Hispanic, Latino, American Indian, and Pacific Islander populations have somewhat lower stroke death rates than whites, black men and women continue to have significantly higher stroke death rates than all other populations, and the prevalence of stroke remains higher in minority populations than among whites. Lastly, because women live longer than men, in 2004, women accounted for greater than 60% of stroke deaths in the United States.

In addition to the significant mortality, the morbidity associated with stroke is considerable: more than 25% of stroke survivors older than age 65 are disabled 6 months later. The length of time to recover from a stroke depends on its initial severity. From 50% to 70% of stroke survivors regain functional independence, but 15–30% are permanently disabled, and 20% require institutional care at 3 months after onset. Although 70% of strokes are a first cardiovascular event, 15% of survivors will have a recurrent event within 1 year, and 30% will have a recurrent event within 5 years. The period soon after an acute stroke is associated with the highest rate of stroke recurrence, and the risk of stroke following a transient ischemic attack (TIA) is over 10% for the following 90 days.

The medical costs of stroke are high: the estimated direct and indirect costs of stroke for 2008 are $65.5 billion USD. In comparison, the 2008 costs for coronary heart disease were estimated at $156.4 billion USD, making stroke one of the leading US health-care expenditures. The mean lifetime cost of ischemic stroke is over $140,000 USD, and 70% of the first-year costs following an acute stroke are due to inpatient hospital costs.

Finally, the burden associated with stroke contains an important association with other cardiovascular disease processes and risk. Many risk factors for stroke overlap with those of cardiac and peripheral vascular disease. This overlap has led to the concept of "global risk" for cardiovascular disease in general, of which stroke is one component. High blood pressure is the greatest risk factor for stroke, but factors like smoking, diabetes, and low high-density lipoprotein levels are important risk factors. Depression has been suggested to be an independent risk factor for stroke. The occurrence of a stroke is therefore likely to be the initial manifestation of a global cardiovascular disease process, with high morbidity, mortality, and cost, and risk factors that overlap with traditional cardiovascular risk factor categories.

References and Readings

Asplund, K., Stegmayr, B., & Peltonen, M. (1998). From the twentieth to the twenty-first century: A public health perspective on stroke. In M. D. Ginsberg & J. Bogousslavsky (Eds.), *Cerebrovascular disease pathophysiology, diagnosis, and management* (Vol. 2). Malden, MA: Blackwell. Chap 64.

Rosamond, W., Flegal, K., Furie, K., et al. (2008). Heart disease and stroke statistics-2008 update: A report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation, 117*, e25–e146.

Rosamond, W. D., Folsom, A. R., Chambless, L. E., Wang, C. H., McGovern, P. G., Howard, G., Copper, L. S., & Shahar, E. (1999). Stroke incidence and survival among middle-aged adults: 9-year follow-up of the Atherosclerotic Risk in Communities (ARIC) cohort. *Stroke, 30*, 736–743.

Stroop Color-Word Test

Mark Hamer
Epidemiology and Public Health, Division of Population Health, University College London, London, UK

Synonyms

Mental stressor; Problem solving

Definition

The Stroop test is commonly used in psychophysiological studies as a problem-solving task to elicit mental stress. The test is an incongruent task that requires participants to identify the

name of a color (e.g., "blue," "green," or "red") that is printed in a conflicting color not denoted by the name (e.g., the word "red" printed in blue ink instead of red ink). The task primarily evokes beta-adrenergically driven responses, resulting in increased heart rate and cardiac output. Functional neuroimaging studies of the Stroop effect have consistently revealed activation in the frontal lobe and more specifically in the anterior cingulate cortex and dorsolateral prefrontal cortex, two structures hypothesized to be responsible for conflict monitoring and resolution.

Cross-References

▶ Blood Pressure Reactivity or Responses
▶ Heart Rate

Structural Equation Modeling (SEM)

Maria Magdalena Llabre and William Arguelles
Department of Psychology, University of Miami, Coral Gables, FL, USA

Synonyms

SEM

Definition

Structural equation modeling (SEM) is a multivariate statistical methodology for the estimation of a system of simultaneous linear equations that may include both observed and latent variables. With origins in the path analysis work of the biometrician Sewell Wright and the factor analysis tradition of Charles Spearman, over the past 40 years, SEM has transitioned from a novel methodology for linear models to a mainstream statistical framework for the analysis of latent variable models. SEM-related techniques may be used to examine a wide variety of structures, including causal models, measurement models, growth models, latent classes or mixtures, and combinations of these. The generality and flexibility of SEM, the development of efficient estimation methods, and the availability of computer programs contribute to the utility of this methodology for addressing important research questions in behavioral medicine.

Description

The first step in an SEM analysis is the specification of the model. SEM models are specified via either a system of structural equations or, more commonly, a path diagram. Included in the path diagram are the observed variables to be analyzed, the constructs or latent variables to be inferred, the unobserved but ever-present errors or disturbances, and the ways in which these observed and unobserved variables are related to one another. The path diagram is a valuable tool in itself, helping investigators better understand their research questions and their data. A path diagram forces the investigator to think critically about every variable relevant to the phenomenon under study.

Observed variables (also called indicators) are the variables we measure directly. These could be exogenous (variables whose causes are not included in the model) or endogenous (variables whose causes are posited in the model). *Path analysis* is a special case of SEM in which all variables specified in a model are observed (except for the errors) and assumed to be perfectly reliable. However, when reliability is not perfect and indicators contain measurement error, as is often the case, parameter estimates may be biased. One of the key features of SEM is the possibility of combining multiple observed variables into *latent variables* (the constructs of interest). A latent variable, like a construct, is not observed directly, but is rather inferred from the covariances shared among its corresponding indicators. For example, we could combine multiple measures of depression into a latent variable to improve reliability. Thus, a general SEM model may be viewed as having two components: a structural model (which allows for the specification and testing of relationships among

variables) and a measurement model (which offers the advantage of using latent variables to represent constructs of interest, modeling sources of measurement error and bias associated with directly observed variables).

Generally speaking, the purpose of analyzing SEM models is twofold. First, we wish to test whether the specified model fits the data. Second and simultaneously, we want to estimate the parameters of interest and test them for significance. The most common *method of estimation* used by available computer programs is maximum likelihood (ML), performed iteratively to arrive at an admissible solution. ML parameter estimates are unbiased, consistent, efficient, and normally distributed in large samples. Given a particular model specification, this method is used to generate and compare a model-implied variance-covariance matrix to the data-based variance-covariance matrix. ML estimates are those that minimize the discrepancy between those two matrices, and as such, yield parameter values that have the greatest likelihood of having given rise to the sample values obtained, assuming a multivariate normal distribution. The typical output from a computer analysis will have indices of model fit, as well as the parameter estimates, their standard errors, and *z*-values used to test them for significance.

The primary index used to test model fit is a χ^2 statistic. However, given this statistic's direct dependence on sample size, with large sample sizes, even small differences between the two matrices may yield a significant χ^2 indicative of poor model fit. Several other indices have been developed and proposed as either alternatives or companions to the χ^2 in such cases, including the comparative fit index (CFI), the root mean squared error of approximation (RMSEA), and the standardized root mean residual (SRMR). Beyond overall measures of fit, it is important to make sure that parameter estimates make sense in relation to the problem being investigated.

In terms of the structural aspect of SEM, the *parameters of primary interest* are the path coefficients (or the direct effects among the variables). In SEM, the variances and covariances among the exogenous variables are also estimated, as well as the variances and covariances of the disturbances. In terms of the measurement aspect of SEM, the parameters of primary interest are the factor (or latent variable) loadings, the measurement error variances, and the variances and covariances of the latent variables.

When working within the SEM framework using ML estimation, it is possible to take advantage of its full information capabilities to include all of the available data. Often referred to as full information maximum likelihood (FIML), this approach to *missing data* has been shown to yield unbiased parameter estimates when missingness is related to variables that are accessible for analysis (Little & Rubin, 2002; Schafer & Graham, 2002). Comparable to multiple imputation, this method is superior to other missing data techniques such as listwise or pairwise deletion, or mean, regression, or hotdeck imputation, particularly when data are not missing completely at random (Enders, 2006).

SEM is a large sample methodology and the appropriate sample size must be considered keeping in mind several issues including model complexity, estimation method, and statistical power. With samples less than 100 participants, models must be simple and the variables normally distributed, otherwise problems with model convergence are likely to arise. As a general rule, more complex models or non-normal data will require more participants.

Of note, testing of *alternative models* is necessary to strengthen the causal inferences often associated with SEM. Sometimes researchers improperly assume that models that fit the data represent reality, without recognizing there are always multiple alternative models that fit just as well. Models can be rejected but not proven. It is important to consider design features such as randomization, experimentation, longitudinal designs, instrumental variables, or inclusion of other variables to strengthen model interpretation.

In addition to testing relationships among variables, means and mean structures may be analyzed. For example, multiple groups may be compared with respect to means of latent

variables, or mean changes over time may be modeled using *latent growth modeling*. Many other extensions are possible but their description is beyond the scope of this entry. However, this is an active area of research and readers are encouraged to learn more by consulting available textbooks, Web sites, and papers. A very readable conceptual introduction to SEM is provided by Kline (2010). A more detailed presentation of its use in behavioral medicine may be found in Llabre (2010). The Web site for Mplus (Muthen & Muthen, 2011) – one of the more popular computer programs – contains a lot of useful information on the basics of SEM, as well as extensions to more complex models.

Cross-References

- ▶ Hierarchical Linear Modeling (HLM)
- ▶ Latent Variable
- ▶ Missing Data
- ▶ Randomization

References and Readings

Enders, C. K. (2006). Analyzing structural equation models with missing data. In G. R. Hancock & R. O. Mueller (Eds.), *Structural equation modeling: A second course* (pp. 313–344). Greenwich, CT: Information Age Publishing.

Kline, R. (2010). *Principles and practice of structural equation modeling* (2nd ed.). New York: Guilford Press.

Little, R. J., & Rubin, D. B. (2002). *Statistical analysis with missing data* (2nd ed.). Hoboken, NJ: Wiley.

Llabre, M. M. (2010). Structural equation modeling in behavioral medicine research. In A. Steptoe (Ed.), *Handbook of behavioral medicine: Methods and applications* (pp. 895–908). New York: Springer.

Muthen, L., & Muthen, B. (1998–2011). *Mplus User's Guide*. Los Angeles: Muthen & Muthen.

Schafer, J. L., & Graham, J. W. (2002). Missing data: Our view of the state of art. *Psychological Methods, 7*, 147–177.

Structural Variant

- ▶ Copy Number Variant (CNV)

Structured Clinical Interview for DSM-IV (SCID)

Ulrike Kübler
Department of Psychology, University of Zurich, Binzmuehlestrasse, Zurich, Switzerland

Definition

The Structured Clinical Interview for DSM-IV (SCID) is a semistructured interview created to make reliable psychiatric diagnoses in adults according to the Diagnostic and Statistical Manual, fourth edition (DSM-IV). The SCID has two parts: one for DSM-IV Axis I Disorders (SCID-I) and another for DSM-IV Axis II Personality Disorders (SCID-II).

In order to meet different needs, the SCID-I is available in two versions: the Research Version (SCID-I-RV; First, Spitzer, Gibbon, & Williams, 2002) and the Clinician Version (SCID-CV; First, Spitzer, Gibbon, & Williams, 1996). In contrast to the SCID-CV, the SCID-I-RV comprises more disorders, subtypes, severity, and course specifiers, and is easier to modify. The SCID-I-RV itself is also available in different versions. The broadest SCID-I-RV version comprises 10 self-contained diagnostic modules: Mood Episodes, Psychotic and Associated Symptoms, Psychotic Disorders, Mood Disorders, Substance Use Disorders, Anxiety Disorders, Somatoform Disorders, Eating Disorders, Adjustment Disorders, and an optional module which allows psychiatric diagnoses that may be of the interviewer's interest, such as the module on Acute Stress Disorder and on Minor Depressive Disorder.

The SCID-I starts with an open-ended overview that includes questions about demographic information, work history, chief complaint, past and present periods of psychopathology, treatment history, and current functioning. This is followed by the diagnostic modules, which are presented in a three-column format: the left-hand column contains the interview questions, the

middle column contains the corresponding DSM-IV criteria and in the right-hand column ratings for the criteria are indicated. Besides rating the presence of the DSM-IV criteria for Axis I Disorders, the SCID-I also enables rating of Axis III, IV, and IV of the DSM (see DSM-IV for more details).

The SCID-II (First, Gibbon, Spitzer, Williams, & Benjamin, 1997) is only offered in a single version. It covers the ten standard DSM-IV Axis II Personality Disorders (Avoidant, Dependent, Obsessive-Compulsive, Paranoid, Schizotypal, Schizoid, Histrionic, Narcissistic, Borderline, Antisocial Personality Disorder), as well as Personality Disorder Not Otherwise Specified, and the appendix categories Depressive Personality Disorder and Passive-Aggressive Personality Disorder. The item format and the conventions of the SCID-II are very similar to those of the SCID-I. The SCID-II consists of several questions organized in sections in accordance with the DSM-IV diagnoses for personality disorders. In most cases, the questions correspond accurately with the criteria. To shorten overall administration time, the SCID-II is also provided with a self-report screening questionnaire that is intended to be administered at first. After this questionnaire has been filled out, only those items indicating personality abnormalities need to be inquired in more detail during the interview.

The SCID-II is often used in conjunction with the SCID-I. While administration of SCID-I typically takes between 45 and 90 min, the complete administration time of the SCID-II usually lasts about 1 h. Ideally, the SCID is administered by a trained interviewer familiar with the diagnostic criteria used in the DSM-IV. The SCID can be used in both healthy individuals and psychiatric patients. In individuals with either severe psychotic symptoms or severe cognitive impairments, the administration of the SCID is not recommended.

Overall, the SCID is a widely used assessment tool in both research and clinical settings in many countries. Various versions of the SCID have been translated into multiple languages, including Mandarin, Spanish, French, and German. The psychometric properties of the SCID-I and the SCID-II have been evaluated in several adult populations in numerous investigations, with encouraging results for most Axis I and Axis II disorders (e.g., Lobbestael, Leurgans, & Arntz, 2010). Computer-assisted versions of the SCID are also available. For more information on the SCID, the reader is referred to the SCID website.

References and Readings

American Psychiatric Association. (2000). *Diagnostic and statisticial manual of mental disorders* (4th ed.). Washington, DC: Author. Text revision.

First, M. B., Gibbon, M., Spitzer, R. L., Williams, J. B. W., & Benjamin, L. S. (1997). *Structured clinical interview for DSM-IV axis II personality disorders, (SCID-II)*. Washington, DC: American Psychiatric Press.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1996). *Structured clinical interview for DSM-IV axis I disorders, clinician version (SCID-CV)*. Washington, DC: American Psychiatric Press.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (2002). *Structured clinical interview for DSM-IV-TR axis I disorders, research version, patient edition. (SCID-I/P)*. New York: Biometrics Research, New York State Psychiatric Institute.

Lobbestael, J., Leurgans, M., & Arntz, A. (2010). Inter-rater reliability of the structured clinical interview for DSM-IV axis I disorders (SCID I) and axis II disorders (SCID II). *Clinical Psychology & Psychotherapy, 18*(1), 75–79. doi: 10.1002/cpp.693.

www.scid4.org

Study

▶ Job Diagnostic Survey

Study Methodology

▶ Research Methodology

Study Protocol

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

The study protocol is "the most important document in clinical trials, since it ensures the quality and integrity of the clinical investigation in terms of its planning, execution, conduct, and the analysis of the data" (Chow & Chang, 2007). The study protocol is a comprehensive plan of action that contains information concerning the goals of the study, details of subject recruitment, details of safety monitoring, and all aspects of design, methodology, and analysis. (In some cases, a Statistical Analysis Plan, associated with and written at the same time as the study protocol, will contain the detailed description of the analyses to be conducted.)

Description

The creation of study protocol requires input from many individuals. Consider a study protocol for a pharmaceutical clinical trial, since this is likely to be more extensive (and complex) than some smaller trials for behavioral medicine interventions and treatments. By considering this more extensive version, one will be able to judge which parts are and are not needed on a case by case basis.

In the case of a pharmaceutical trial, input will be needed from clinical scientists, medical safety officers, study managers, data managers, and statisticians. Consequently, while one clinical scientist or medical writer may take primary responsibility for the protocol's preparation, many members of the study team make important contributions.

The requirements of a study protocol include:

- Objectives (usually primary and secondary objectives). These goals of the study are stated as precisely as possible.
- Measurements related to the drug's safety, and procedures to ensure the safety of all subjects while participating in the trial.
- Inclusion and exclusion criteria. These provide detailed criteria for subject eligibility for participation in the trial.
- Details of the procedures for physical examinations.
- Laboratory procedures. Full details of the nature and timing of all procedures and tests are provided.
- Electrocardiogram (ECG) measurement and any other measurements such as imaging.
- Drug treatment schedule. Route of administration, dosage, and dosing regimen are detailed. This information is also provided for the control treatment.
- In the case of later-phase trials, measurements of efficacy. The criteria to be used to determine efficacy are provided.
- In the case of later-phase trials, details of the method of diagnosis of the disease or condition of clinical concern for which the drug is intended.
- Statistical analysis. The precise analytical strategy needs to be detailed, here and/or in an associated statistical analysis plan.

Inclusion and exclusion criteria are central components of clinical trials. A study's inclusion and exclusion criteria govern which individuals interested in participating in the trial are admitted to the study as subjects. Criteria for inclusion in the study include items such as the following:

- Reliable evidence of a diagnosis of the disease or condition of clinical concern.
- Being within a specified age range.
- Willingness to take measures to prevent pregnancy during the course of treatment. This includes a female in the trial not becoming pregnant (she may be receiving the drug being tested), and a male participating in the

trial not causing a female to become pregnant (he may be receiving the drug being tested).

Criteria for exclusion from the study may include:

- Taking certain medications for other medical conditions and which therefore cannot safely be stopped during the trial.
- Participation in another clinical trial within so many months prior to the commencement of this study.
- Liver or kidney disease.

While inclusion and exclusion criteria are typically provided in two separate lists in regulatory documentation, exclusion criteria can be regarded as further refinements of the inclusion criteria. Meeting all the inclusion criteria allows a person to be considered as a study participant, while not meeting any exclusion criteria is also necessary to allow the person to become a participant.

Cross-References

▶ Informed Consent

References and Readings

Chow, S.-C., & Chang, M. (2007). *Adaptive design methods in clinical trials: Concepts and methodologies*. Boca Raton, FL: CRC/Taylor Francis.

Study Size

▶ Sample Size Estimation

Subethnic Groups

▶ Ethnicity
▶ Minority Subgroups

Subgroup Heterogeneity

▶ Minority Subgroups

Subject Characteristics

▶ Demographics

Subjective Well-Being

▶ Happiness and Health

Submissiveness

▶ Interpersonal Circumplex

Substance Abuse

▶ Alcohol Abuse and Dependence
▶ Dependence, Drug
▶ Lifestyle Changes

Substance Abuse: Treatment

John Grabowski
Department of Psychiatry, Medical School, University of Minnesota, Minneapolis, MN, USA

Synonyms

Addiction rehabilitation; Chemical dependency treatment; Drug abuse: treatment; Drug and alcohol treatment; Drug dependence treatment; Drug rehabilitation; Inpatient treatment; Outpatient treatment; Residential treatment

Definition

Treatment refers to a defined, empirically evaluated, data-based intervention intended to manage, remediate, or cure a diagnosed condition, here,

impairing or problematic drug use. Historically, there have been two frameworks underpinning substance use treatment, the "disease" and "learning/conditioning" models (Higgins, 1997). While some assumptions are divergent and perhaps irreconcilable, a broad integrative view dictates that genetic and other biological factors interact with behavior and environmental factors as composite determinants of substance use disorders. The diagnostic criteria applied to determining need for treatment are found in the International Statistical Classification of Diseases and Related Health Problems (ICD) 10 (*"Mental and behavioral disorders due to psychoactive substance use"–"a wide variety of disorders that differ in severity and clinical form but that are all attributable to the use of one or more psychoactive substances, which may or may not have been medically prescribed"*) and The American Psychiatric Association Diagnostic and Statistical Manual (DSM) IV, (*"Substance Related Disorders"–"The Substance-Related Disorders include disorders related to the taking of a drug of abuse (including alcohol), to the side effects of a medication, and to toxin exposure"*). The intervention may include behavioral, psychological, social, and pharmacological components delivered by skilled practitioners. There is a wide range of self-help and other efforts with little or no documented efficacy that are beyond the purview of this entry as are a number of criminal justice–based interventions.

Description

Underlying Disciplines, Principles, Goals, and Focus

In the public and lay domain, putative treatments for substance use disorders (i.e., "drug abuse" or "addiction") are legend and varied in their underpinnings. However, the core principles for systematic treatment of substance use disorders reside in empirically based interventions of psychology, psychiatry, pharmacology, behavioral science, and neuroscience. Despite diverse theoretical, conceptual, and terminological differences, there are many commonalities in both practice and behaviors and thoughts that are the focus of treatment. Emphasis is typically placed on avoiding drug use–related circumstances (physical or social) and replacing drug-seeking, drug use, and drug-related thoughts with other behaviors (e.g., coping skills, problem-solving skills). The interventions focus on altering biology, behavior, and social interaction through behavioral/psychological and pharmacological techniques. Longer term pharmacotherapy focuses on substituting a therapeutic medication (e.g., methadone) at controlled doses for the drug used (opiate) or a medication blocking (e.g., naltrexone) the effects of the drug used (opiate) with untoward consequences. Each strategy produces blunting or elimination subjective drug effects. Short-term alleviation of withdrawal symptoms or disturbed behavior is achieved with specific symptomatic treatment (e.g., anxiolytics, sedatives, antidepressants, antipsychotics).

Two important considerations in discussion of treatment are the determinants and correlates of the disorders and the not uncommon existence of co-occurring psychiatric/behavioral or medical problems. These may predate or be a consequence of the substance use disorder(s). The most common comorbid condition for treatment is multiple drug use (e.g., cocaine, heroin, alcohol). Next, the psychological/psychiatric diagnoses of problematic drug use or dependence are not uncommon among individuals also diagnosed for schizophrenia, depression, posttraumatic stress disorder, etc. Indeed, observation of common symptoms associated with extreme drug use, for example, disorganization, paranoid ideation, hallucinations, may reveal existence of the other psychiatric condition. Increasing awareness of co-occurrence of other psychiatric conditions with substance use disorders has resulted in extensive research, discussion, and review of conceptualization of treatment. For example, should specialized single interventions be applied to each presenting condition or should integrated treatments be devised? Questions arise as to correlation, association, and causation. Did the substance use cause psychiatric illness or precipitate its onset and reveal its existence or a predisposition? Did the psychiatric illness predispose to drug use, perhaps

as a means to self-medicate the underlying condition? Finally, in instances of severe drug use, medical consequences (e.g., respiratory depression, cardiovascular event, accident-induced trauma) rather than psychiatric symptoms may lead to identification of problematic drug use and need for treatment.

Critical in establishing treatment strategies and regimens is understanding that the substance use disorder is the consequence of multiple determinants, some amenable to manipulation or intervention, others not. As with forms of some other common conditions, for example, hypertension, diabetes, obesity, a complex interplay of genetic, biological, behavioral, social, and other environmental factors contributes to the observed disease. However, limits must exist on diagnosis and treatment. Thus, while some patients present with reasonably stable life styles, educational backgrounds, and work histories, others using the same drugs may have limited education and skills. In both cases, systematic focus on diagnosed conditions is essential. However, the myriad collateral circumstances are the purview of other domains (e.g., social service networks, job counselors, educational systems), which can be addressed more effectively, for example, by adept case managers and other experts, rather than health care providers. The two historical models continue to influence the perspective and goals of treatment. Underpinning an integrative behavioral learning perspective is the concept that just as one learns other behaviors (reading, using the internet, sports), one learns to use drugs. The behavior is maintained by biological, behavioral, and social rewards or reinforcers. The treatment strategy is establishment or learning of other behaviors sustained by non-drug reinforcers, while drug seeking and taking are diminished or eliminated. The disease model assumes a "chronic relapsing disease" evidenced when a predisposed individual is exposed to and begins using psychoactive drugs; complete and perpetual abstinence is the goal and relapse is always imminent (Higgins, 1997). To the extent that biology underlies behavior, the integrated learning model accounts for genetic and biological differences but focuses on new behaviors and reinforcers rather than the chronicity of disease.

Treatment Settings, Dose, and Costs

Distinct from the intervention is the setting or environment in which treatment is provided. Settings include outpatient (e.g., office, clinics, emergency rooms), inpatient (e.g., hospital facilities), and residential (e.g., community-like living arrangements). The opportunity for access to patients is increased in controlled residential settings and some supplement the professionally driven therapy with a variety of "self-help" activities such as group discussions. Still others may have options for attending work or school. However, many of the same fundamental interventions can be applied in any of these settings. For example, a course of cognitive behavior therapy could be applied weekly in a mental health care practitioner's office, an inpatient facility, a residential care setting, or for that matter a prison. Another dimension of "setting" is whether care is provided to a single patient by a therapist, or with a group of patients, having the therapist interacting with individual members and also facilitating discussion among members. Attempts to match individuals to particular settings for treatment have been flawed and generally ineffective. For example, no data point to advantages of inpatient compared to outpatient care, regardless of severity for treatment of the behavior of substance use itself.

Similarly distinct is the actual amount or "dose" of intervention. Inpatient settings in which the patient resides continuously for days or weeks do not necessarily deliver more, or more efficacious therapeutic care than treatment delivered to a patient residing at home and functioning in her or his natural environment while receiving office-based care a few hours a week. For example, while inpatient or residential care may shield a patient from access to drugs, including alcohol and nicotine, it also prevents them from having exposure to the very environments in which it will be necessary to engage in life without problematic drug use; this exposure is both essential and therapeutic. As an aside, inpatient and residential care are extremely costly strategies that

are well beyond the resources of most individuals and, with increasing health cost burdens for society, will be of diminishing importance except in unusual circumstances.

Evaluation and Monitoring

Drug use severity, as determined by substance used, frequency and amount of use, may be critical to determining treatment required. Thorough diagnostics and history determination permit tailoring treatment. An interview method termed Timeline Follow Back (Sobel, Sobel, Klajner, Pavan, & Basian, 1986) permits careful structuring of a history of use. The accepted diagnostic interviews conforming to elements of the DSM or ICD criteria are necessary to determine severity, existence of comorbid conditions, and plausible treatment plans. A collateral finding in many studies of therapeutic interventions for substance use disorders is that individuals presenting with less severe conditions are more successful in treatment. For example, individuals who on entry for evaluation have negative biological screening tests for the drug used (see below) are more likely to continue successfully for a full course of treatment. Comorbid conditions and individual differences in a range of social and environmental circumstances must always be considered.

Both at intake and during treatment, objective measures of determining current use are essential. For alcohol use, this may be a breath test or urine screen. Tobacco smoking can be readily determined using CO monitoring while other tobacco use can be monitored with saliva or urine screens for cotinine, primary metabolite of nicotine. Virtually all other drug use can be monitored with simple and widely available urine screen procedures and this is essential, much as regular blood pressure evaluation is critical in treatment of hypertension or glucose level monitoring is critical to diabetes treatment.

Interventions: Behavioral, Pharmacological, and Combined

Behavioral therapy/psychotherapy and pharmacotherapy are the two broad classes of intervention applied to psychiatric disorders including substance use disorders. Current science points to remarkably effective interventions for specific types of substance use disorders, limited efficacy for some forms of substance use disorders, and for others, little efficacy or even exacerbation of drug use (e.g., olanzapine and cocaine use).

Some therapies are designed primarily to promote understanding of an existing problem or motivate entry into more extended or intensive treatment (e.g., motivational enhancement therapy). Other treatments are intended to provide a systematic course of intervention addressing the gamut of problems linked to substance use disorder and cessation of use (e.g., cognitive behavior therapy, contingency management). Still other interventions are composites with elements incorporated from a number of conceptual and pragmatic frameworks (e.g., community reinforcement approach, matrix model).

There is a clear need to distinguish between treatment of acute drug-related symptoms, notably withdrawal syndromes, and the complex behaviors of drug seeking and drug taking. A patient can achieve a "drug free" state within days by enforced abstinence. Sleep patterns and other biological functions and a variety of disrupted behaviors may stabilize within days or weeks. This normalization does not directly address the problems of drug seeking and drug use; however, for a small minority of individuals, a period of abstinence/cessation, however achieved, may be sufficient. Even this observation may be confounded since these individuals often undergo repeated cessation attempts before achieving behavioral change. For most, some supplemental intervention, ranging from brief, structured therapies to multiple structured sessions, will be required at some point in the drug use career to attain meaningful beneficial outcomes. Indeed, some scientists conceptualize substance use disorders as chronic for many individuals, requiring ongoing treatment and support, similar to obesity (McLellan, 2002).

There are several behavioral, or psychotherapy, interventions for which strong supporting data of efficacy exist. One is cognitive behavior therapy; another is contingency management sometimes applied within the broader

community-based reinforcement approach; and a third encompassing a range of theoretical frameworks entails variants of "talk therapy." Components or variants exist with labels such as "the matrix model" (Shoptaw, Rawson, McCann, & Obert, 1994), "relapse prevention," and "dialectical behavior therapy." Diverse techniques (e.g., hypnotism, acupuncture) have been incorporated into treatment models with little evidence of added efficacy compared to well-constructed behavioral therapies. All effective nonpharmacological therapies ultimately focus on behavior and thought directed at minimizing or eliminating drug use while increasing the frequency of a range of socially appropriate constructive behaviors. Among adults, these goals are more readily achieved when the individual arrives at therapy with an age and ability appropriate set of social skills, training, job history, and experience. Still, there are times when additional focused therapeutic elements may be included, for example, parenting or marital counseling.

In the realm of pharmacotherapy, opiate replacement therapy (methadone, buprenorphine) provides a clear example of robust and effective treatment applicable to use and dependence ranging from iv heroin use to oral oxycodone use. Some promising data suggest potential efficacy of a similar replacement, or agonist-like strategy for stimulant dependence though there are currently no FDA-approved medications. Possible substitute medications for stimulant dependence include amphetamine analogs. Similarly, while there are currently no approved medications for treatment of marijuana use and dependence, a promising agent that represents substitution or replacement, tetrahydrocannabinol/cannabidiol, is currently being evaluated. A variety of strategies addressing different behaviors and pharmacological mechanisms have been applied to alcohol use (disulfiram, benzodiazepines, naltrexone, acamprosate) with greater or lesser efficacy. Similarly, several medications (nicotine preparations, bupropion, varenicline) have been shown to have varying degrees, but nevertheless modest effectiveness in treatment of tobacco use and nicotine dependence. There are several nicotine preparations (gum or polacrilex, lozenges, nasal spray, patches, inhalers) that alone produce relatively modest rates of cessation across broad populations but have meaningful public health consequences due to the prevalence of tobacco use and the costs of associated diseases. Disulfiram for alcohol dependence has a unique profile: through metabolic interference, it results in high levels of acetaldehyde and induced severe discomfort; integrated into a well-controlled regimen, it can be very effective but is not widely used. Naltrexone and acamprosate appear to have modest effects in reducing alcohol intake or sustaining abstinence. For individuals whose alcohol use is found to be highly correlated with diagnoses of comorbid depression or anxiety, effective treatment may stem from administration of anxiolytics or antidepressants. In some instances, use of two or more medications may be a useful therapeutic approach. A dilemma across most medical and psychiatric is nonadherence or noncompliance with medication regimens. This is particularly true for antagonist medications such as disulfiram and naltrexone.

Ultimately, for many instances of substance use disorders, benefit of joint action of concurrently applied behavioral and pharmacological interventions may be important in all but the least severe cases. This is analogous to treatment of other disorders with clear biological and behavioral determinants. For example, hypertension may be treated with combined behavioral (e.g., exercise, diet) and pharmacological (e.g., ACE inhibitors) interventions. Similarly, depression, while amenable to both behavioral and pharmacological treatments, may be most effectively treated with a combination (e.g., CBT and SSRIs). The need for continuing intervention in treatment of substance use disorders may vary. For example, pharmacotherapy with an effective course of behavior therapy establishing (or reestablishing) alternative behaviors and eliminating drug use may still entail long-term maintenance pharmacotherapy. The latter may be necessitated by biological perturbations that predated or were a consequence of drug use. Here continued medication sustains biological and behavioral stability that would otherwise not be achieved or would only continue with

unnecessary behavioral, emotional, or biological burden on the patient. Exemplifying this is treatment of heroin dependence. On completing initial behavioral treatment combined with methadone or buprenorphine maintenance, some patients may successfully undergo gradual reduction in medication dose. In other instances, perhaps more severe and involving many years of heroin use, maintenance pharmacotherapy for years or decades is desirable and warranted. The need for maintenance may reside in engrained perturbations in biology and behavior, resulting from prolonged heroin use. Generally, the costs and inconvenience of maintenance replacement are outweighed by nearly inevitable return to drug dependence and associated dire psychosocial and medical consequences when abstinence without further treatment is undertaken.

Often discussed is the concept of "relapse," "return to drug use," or in some instances, "development of new drug use." Early in treatment (often within days but up to 3–6 months), there may be graded but rapid return to previous baseline levels of use. Some individuals may use a drug a few times, for example, smoke a cigarette, or consume alcohol. They may even engage in periodic use, heavy use, or a binge, but through sustained therapy eventually refrain from further use. The likelihood of return to use greatly decreases after a year. Confounding these general observations are individuals who enter treatment and never use the drug again as well as those who are abstinent for years and then resume use. In some cases, individuals treated for heavy use, for example, heavy alcohol drinking, may resume nominal social use without return to heavy drinking. Here the legal status of a drug is germane. Treatment with a goal of abstinence from cocaine or heroin use has positive implications beyond those related to reducing impairment from drug effects, for example, no risk of incarceration. For those engaged in heavy alcohol use, reduced use may be a realistic, achievable, beneficial goal. However, as with cigarette smoking, the entrenched habitual behavior combined with reinforcing effects of the drug may preclude a goal of moderation. Finally, a small subset of individuals who have well-established behaviors of problematic use may be successfully treated for use of one drug, but at some later date develop similarly problematic use of a different drug. This would reflect reestablishment of drug seeking and taking, but not "relapse" as the word is commonly used. As with any complex disorder with multiple determinants, the core constellation of symptoms, here drug seeking and drug taking, can be addressed directly with supplemental specialty intervention when appropriate.

References and Readings

Higgins, S. T. (1997). Applying learning and conditioning theory to the treatment of alcohol and cocaine abuse. In B. Johnson & J. Roache (Eds.), *Drug addiction and its treatment* (pp. 367–386). Philadelphia: Lippencott-Raven.

McLellan, A. T. (2002). Have we evaluated addiction treatment correctly? Implications from a chronic care perspective. *Addiction, 97*(3), 249–252.

Shoptaw, S., Rawson, R. A., McCann, M. J., & Obert, J. L. (1994). The matrix model of outpatient stimulant abuse treatment: Evidence of efficacy. *Journal of Addictive Diseases, 13*(4), 129–141.

Sobell, M. B., Sobell, L. C., Klajner, F., Pavan, D., & Basian, E. (1986). The reliability of atimeline method for assessing normal drinker college students' recent drinkinghistory: Utility for alcohol research. *Addictive Behaviors, 11*(2), 149–161.

Substance Dependence

▶ Alcohol Abuse and Dependence

Substance H

▶ Histamine

Substance Use Disorders

▶ Dependence, Drug

Success

▶ Attribution Theory

Successful Aging

Barbara Resnick
School of Nursing, University of Maryland, Baltimore, MD, USA

Synonyms

Optimal aging

Definition

Successful aging has been addressed and discussed repeatedly with at least 29 different definitions articulated from both cross-sectional and longitudinal research studies. Common themes across all of this work describe successful aging as the absence of physical and mental disability. Generally, successful aging is noted to occur when the individual has perceived "good health."

Description

Successful Aging

The concept of successful aging emerged in the late 1980s and early 1990s as a departure from the loss-focused geriatric and gerontological research that preceded the concept. In their groundbreaking 1987 article, Human Aging: Usual and Successful, Rowe and Kahn argued that the cognitive and physiological losses documented in the literature as age-related changes were mischaracterizations of the natural aging process. "We believe that the role of aging per se in these losses has often been overstated and that a major component of many age-associated declines can be explained in terms of lifestyle, habits, diet, and an array of psychosocial factors extrinsic to the aging process."

Definition

Successful aging has been addressed and there are at least 29 different definitions articulated from both cross-sectional and longitudinal research studies. Common themes across all of this work consider successful aging as the absence of physical and mental disability and perceived "good health." In addition, successful aging has increasingly been associated with resilience and maintenance of an active lifestyle and having good supportive relationships. Everyone has the opportunity to age successfully. From the outside, objectively, this may look very different across people. For some, aging successfully is still working at age 98. For others, it is sitting quietly in a room in assisted living and reliving past memories reviewing very rich, fulfilling lives.

More important than the absolute state of health of the individual is the notion of one's conceptualization and acceptance of his or her health status. Acceptance of changes and optimization of physical and mental health are the most critical aspects of aging successfully. Changes that occur as part of normal aging must be accepted, addressed, and adjusted to. Vision changes that occur starting around age 40 provide the first experience adults have to cope with and age successfully. Successful adaptation includes buying reading glasses over the counter as needed or getting eye examinations and vision testing done and getting new glasses! Increasingly baby boomers carry small lights with magnification to read restaurant menus, telephone books, and other pertinent information. Other changes, such as painful degenerative joint disease, are not so easily or commonly recognized, accepted, and adjusted to. It is being able to accept and adjust to changes that are at the core of successful aging.

As noted, resilience is central to successful aging. The word "resilience" comes from the Latin world "salire," which means to spring up and the word "resilire" means to spring back. Resilience, therefore, refers to the capacity to spring back from a physical, emotional, financial, or social challenge. Being resilient indicates that

the individual has the human ability to adapt in the face of tragedy, trauma, adversity, hardship, and ongoing significant life stressors. Resilient individuals are able to adapt to all types of situations, especially with regard to social functioning, morale, and somatic health, and are less likely to succumb to illness. That is, a resilient individual may still get ill but will respond in a way in which he or she optimizes recovery, maintenance, or even death in a way that defines resilience. Resilience, as a component of the individual's personality, develops and changes over time through ongoing experiences with the physical and social environment. Resilience can, therefore, be perceived as a dynamic process that is influenced by life events and challenges. Increasingly, there is evidence that resilience is related to motivation, specifically the motivation to age successfully and to recover from physical or psychological traumatic events.

Resilience research has helped to uncover the many factors or qualities within individuals that are associated with resilience. These include such things as positive interpersonal relationships, incorporating social connectedness with a willingness to extend oneself to others, strong internal resources, having an optimistic or positive affect, keeping things in perspective, setting goals and taking steps to achieve those goals, high self-esteem, high self-efficacy, determination, and spirituality which includes purpose of life, religiousness or a belief in a higher power, creativity, humor, and a sense of curiosity. Strengthening any of these factors will help individuals to become more resilient when faced with a challenge.

Older women who have successfully recovered from orthopedic or other stressful events describe themselves as resilient and determined and tend to have better function, mood, and quality of life than those who are less resilient. These are the women who at 97 engage in rehabilitation services post–hip fracture to their fullest ability and then following discharge home are insistent on getting back on their exercise equipment at the gym. Resilience has also been associated with adjustments following the diagnosis of dementia, widowhood, management of chronic pain, and overall adjustment to the stressors associated with aging. Thus, it is through resilience he or she adjusts, adapts, and addresses the physical, emotional, and mental challenges that are to be anticipated as one ages.

Older adults accrue a lifetime of experiences through which resilience develops. Resilient individuals generally age successfully. A resilient older adult is exemplified by such things as accepting the loss of a spouse; symptoms associated with an acute medical event; or a fracture and responding with determination to recover and grow through the experience. He or she does not waste energy on complaining about what has happened but rather on pooling the resources needed to overcome the challenging event.

Resilience alone is not sufficient to assure successful aging. It is believed that all individuals have the innate ability to be resilient and return to homeostasis successfully and to transform, change, and grow, regardless of age. He or she must, however, summon motivation in the face of adversity to be resilient. Thus, motivation may be present independent of resilience, but resilience depends on being motivated to successfully reintegrate. Resilient reintegration requires increased energy, or motivation, for resilience to successfully occur.

Motivation comes from within and is based on an inner urge that moves or prompts a person to action. This is in contrast to resilience which is stimulated in response to adversity or challenge. Motivation refers to the need, drive, or desire to act in a certain way to achieve a certain end. We do not need to be challenged to be motivated. We do have to be motivated to respond, with resilience, to a challenging event. There are some factors that are associated with both resilience and motivation such as determination, self-efficacy, being open and willing to experience new things, and social supports. The capacity to be resilient and/or motivated is present in everyone and choices are made in the face of routine and challenging situations to be motivated and/or resilient. Motivation related to engaging in physical activities may be high for some individuals while others are motivated to sit

in a chair or lie in a bed. Conversely, some older adults are motivated to take classes in a senior center while others refuse to even consider this and are motivated to sit daily and watch television alone. Some individuals are resilient with regard to physical challenges but cannot cope with challenges associated with finances or cognitive changes. Thus, there are traits and characteristics of individuals associated with resilience and motivation as well as external factors that can impact resilience and motivation as individuals respond to challenges or activities within their lives.

Resilience, unlike motivation, relies on the individual experiencing a life challenge or some type of adversity. These challenges may be developmental challenges such as those associated with normal aging (e.g., vision changes), or they maybe social and/or economic challenges such as those experienced by the loss of employment, the loss of a spouse, or a move into an assisted living facility. Conversely, motivation is not dependent on an adverse event or challenge; rather motivation is a necessary component for all activity. Routine personal care activities such as bathing and dressing require motivation, as do making plans to have dinner with a friend or play cards. Resilience would be required, however, when he or she is faced with bathing and dressing challenges following a wrist fracture.

Keys to Aging Well

An individual has the ability to be resilient as long as he or she is motivated to do so. The first step in the process is to engage in appropriate health promotion activities, particularly exercise. Due to the changes that can occur with aging, underlying physical capability will vary among older individuals. For some individuals, maintaining a regular exercise program will involve running for 40 min on the treadmill. For others, it may be walking within their apartment building or long-term care facility, doing a sitting exercise program, or swimming or walking in the water. There is, however, an exercise program that matches each individual's needs. This is critically important to appreciate and recognize. The benefit of physical activity for older adults should not focus on preventing cardiovascular disease and managing blood pressure to prevent a stroke. Rather, it should be geared toward the mental health benefits associated with physical activity as well as the sense of achievement and physical benefit of maintaining function and improving balance and strength.

Successful Aging Guidelines

Meta-analytic reviews provide strong evidence to support the many benefits of exercise including decreased risk of coronary heart disease and stroke; decreased progression of degenerative joint disease; prevention of osteoporosis of the lumbar spine; decreased incidences of falls; increased gait speed if the activity is of sufficient intensity and dosage; improved cognitive function in sedentary older adults and in those with dementia; a modest benefit in quality of life for frail older adults; and a positive association with successful aging. Current guidelines from the American College of Sports Medicine and the American Heart Association recommend that all older adults engage in moderately intense aerobic exercise for 30 min daily at least 5 days a week or vigorous exercise 20 min a day, 3 days a week. In addition, each should do eight to ten strength training exercises, 10–15 repetitions of each exercise two to three times per week. These exercises are well described in a book published by the National Institute of Aging, *Exercise: A Guide from the National Institute of Aging*. For older adults at risk for falling, balance exercise is also recommended. It is not known, however, what dose of exercise is needed for each individual to age successfully. For some, a walk to get the mail is sufficient psychologically to constitute an exercise program. For others, one activity a day (e.g., playing bridge, going to dinner) makes them feel successful and engaged. The focus should be on helping the individual understand what successful aging means to them and helping them develop a plan to achieve that.

Unfortunately, all too many older adults assume that they cannot exercise or engage actively in routine life activities and social activities because of underlying disease, pain, shortness of breath, or other limiting symptoms.

Health outcomes can be achieved at even relatively low levels of exercise intensity, particularly for those who have previously been sedentary. Thus, initiation of physical activity even at 1-min intervals is an important step to successful aging. Adjustments can be made to engage them in social activities via chair positioning that will help them maintain independence in public or remain comfortable to sit for a game of bridge or an evening eating with friends. Solving these challenges is part of the necessary resilience needed to age successfully. Health-care providers can serve as sources to help with overcoming such challenges and barriers.

In addition to physical activity, mental stimulation may also be important for successful aging. In a recent meta-analysis, there was not statistical support to indicate that cognitive stimulation through structured programs prevents or slows the progression of Alzheimer's disease. The current research, however, is limited by a lack of consensus on what constitutes the most effective type of cognitive training, insufficient follow-up times, a lack of matched active controls, and few outcome measures showing changes in daily functioning, global cognitive skills, or a decrease in disease progression. Keeping actively engaged mentally through volunteer work or activities within one's own living space certainly will build resilience and likewise assure successful aging. Opportunities abound for such activities, however, as with physical activity the older individual must be motivated to initiate and engage in these activities.

Older adults should be encouraged to move beyond their level of comfort and engage in new and different activities to stimulate their minds and their bodies. If playing bridge or doing a crossword puzzle is lifelong acquired skill, newer activities such as learning a new language or playing an instrument will provide important mind stimulation and encourage plasticity and growth.

The Health-Care Provider's Role in Successful Aging

Informing young and older adults about the ways in which to age successfully is the first step toward facilitating successful aging for any individual. There are ways in which to measure resilience and motivation, although at a clinical level they will not necessarily direct interventions. Thus, the best approach is to motivate individuals toward resilient behaviors.

Cross-References

- ▶ Aging
- ▶ Elderly
- ▶ Exercise
- ▶ Geriatric Medicine
- ▶ Gerontology
- ▶ Physical Activity

References and Readings

Boardman, J., Blalock, C., & Button, T. (2008). Sex differences in the heritability of resilience. *Twin Research and Human Genetics, 11*(1), 12–27.

Bonanno, G., Galea, S., Bucciarelli, A., & Vlahov, D. (2007). What predicts psychological resilience after disaster? The role of demographics, resources, and life stress. *Death Studies, 31*(10), 863–883.

Chow, S., Hamagani, F., & Nesselroade, J. (2007). Age differences in dynamical emotion-cognition linkages. *Psychology and Aging, 22*(4), 765–780.

Hardy, S., Concato, J., & Gill, T. (2002). Stressful life events among community-living older persons. *The Journal of General Internal Medicine, 17*(11), 832–838.

Hardy, S., Concato, J., & Gill, T. (2004). Resilience of community-dwelling older persons. *Journal of the American Geriatrics Society, 52*(2), 257–262.

Harris, P. (2008). Another wrinkle in the debate about successful aging: The undervalued concept of resilience and the lived experience of dementia. *International Journal of Aging and Human Development, 67*(1), 43–61.

Hegney, D., Buikstra, E., Baker, P., Rogers-Clark, C., Pearce, S., Ross, H., et al. (2007). Individual resilience in rural people: A Queensland study, Australia. *Rural and Remote Health, 7*(4), 620–625.

Hicks, G., Simonsick, E. M., Harris, T. B., Newman, A. B., Weiner, D. K., Nevitt, M. A., & Tylavsky, F. A. (2005). Trunk muscle composition as a predictor of reduced functional capacity in the health, aging and body composition study: The moderating role of back pain. *The Journal of Gerontology. Series A, Biological Sciences and Medical Sciences, 60*(11), 1420–1424.

Karoly, P., & Ruehlman, L. (2006). Psychological "resilience" and its correlates in chronic pain: Findings from a national community sample. *Pain, 123*(1–2), 90–97.
Lee, H., Brown, S., Mitchell, M., & Schiraldi, G. (2008). Correlates of resilience in the face of adversity for Korean women immigrating to the US. *Journal of Immigrant and Minority Health, 10*(5), 415–422.
O'Connell, R., & Mayo, J. (1998). The role of social factors in affective disorders: A review. *Hospital & Community Psychiatry, 39*, 842–851.
Ong, A., Bergeman, C., Bisconti, T., & Wallace, K. (2006). Psychological resilience, positive emotions, and successful adaptation to stress in later life. *Journal of Personality and Social Psychology, 91*(4), 730–749.
Resnick, B., Orwig, D., Zimmerman, S., Simpson, M., & Magaziner, J. (2005). The exercise plus program for older women post hip fracture: Participant perspectives. *The Gerontologist, 45*(4), 539–544.
Rossi, N., Bisconti, T., & Bergeman, C. (2007). The role of dispositional resilience in regaining life satisfaction after the loss of a spouse. *Death Studies, 31*(10), 863–883.
Sanders, A., Lim, S., & Sohn, W. (2008). Resilience to urban poverty: Theoretical and empirical considerations for population health. *American Journal of Public Health, 98*(6), 1101–1106.
Wagnild, G., & Young, H. (1993). Development and psychometric evaluation of the resilience scale. *Journal of Nursing Measurement, 1*(2), 165–177.
Werner, E., & Smith, R. (1992). *Overcoming the odds: High risk children from birth to adulthood*. Ithaca, NY: Cornell University Press.

Sudden Cardiac Death

▶ Cardiac Death
▶ Death, Sudden

Suicidal Ideation, Thoughts

Orit Birnbaum-Weitzman[1] and Mariam Dum[2]
[1]Department of Psychology, University of Miami, Miami, FL, USA
[2]Jackson Memorial Hospital, Miami, FL, USA

Synonyms

Suicidal impulses; Suicidal thoughts

Definition

Suicidal ideation refers to thoughts or impulses of engaging in behavior intended to end one's life (Nock, Borges, Bromet, Cha, Kessler, and Lee 2008). Suicidal ideation should be distinguished from suicidal plan, which refers to the formulation of a specific method through which one intends to die, and from suicide attempt, which refers to an actual engagement in potentially self-injurious behavior in which there is at least some intent to die (Nock et al., 2008).

Description

Ongoing suicidal ideation is a chronic risk factor and has been considered predictive of suicidal behavior especially if accompanied with severe hopelessness, prior suicide attempts, not having a child under 18 years old living at home, and a history of alcohol and drug abuse (Tishler and Reiss, 2009). Suicidal ideation represents an important phase in the suicidal process and often precedes suicide attempts or completed suicide. However, not all patients experiencing suicidal ideation attempt suicide (Weissman et al., 1999). While approximately 10–20% of the population across diverse countries report suicidal ideation and 3–5% have made a suicide attempt at some time in their life, only 0.01% will complete suicide (Kessler, Berglund, Borger, Nock, and Wang, 2005). Acute risk factors including severe anxiety, agitation, and severe anhedonia are most predictive of whether someone will commit suicide in inpatients (Tishler and Reiss, 2009, Bostwick and Rackley, 2007).

Physical as well as mental health problems have been associated with suicidal ideation. Patients with chronic medical illnesses, such as HIV and cancer, and especially those experiencing physical pain are more likely to report suicidal thoughts (Tang and Crane, 2006). Certain medical illnesses, such as neurological disorders and some cancers, appear to have higher rates of suicidal ideation and suicide compared to other medical disorders (Hugues and Kleespies, 2001). A higher prevalence of suicidal ideation has been

observed in patients experiencing pain associated with a variety of medical conditions including migraines, musculoskeletal pain, fibromyalgia, and arthritis (Tang and Crane, 2006). Research suggests that the location and type of pain as well as the intensity and duration of the pain may have implications for the risk of suicidal ideation (Tang and Crane, 2006). In addition, sleep-onset insomnia associated with pain is also a significant discriminator of the presence or absence of suicidal ideation (Tang and Crane, 2006).

Research in the elderly suggests that physical illness plays an important role in suicidal ideation and behavior (Szanto, Gildengers, Mulsant, Brown, Alexopoulos, and Reynolds, 2002). In the elderly with physical illness, untreated or undertreated physical pain, anticipatory anxiety regarding the progression of the physical illness, fear of dependence, and fear of burdening the family have been reported as the major contributing factors for suicidal ideation (Szanto et al., 2002). For all age groups, social isolation is considered another important risk factor that has been shown to be associated with suicidal ideation.

Sociodemographic factors including age and income level have also been associated with suicidal ideation (Kessler et al., 2005). In general, younger age, lack of education, and unemployment have been associated with higher rates of suicidal ideation and may represent an increased risk associated with social disadvantage (Nock et al., 2008). For all age groups, social isolation is considered another important risk factor that has been shown to be associated with suicidal ideation (Van Orden, Witte, Cukrowicz, Braithwaite, Selby, and Joiner, 2010).

Prior suicide attempts and the presence of a psychiatric disorder are the most consistently reported risk factors associated with suicidal ideation and behavior (Nock et al., 2008). Mood disorders such as anxiety and particularly depression significantly increase the risk of suicidal ideation in the general population and in medical patients in particular. Suicidal ideation has also been associated with other mental illnesses including severe anxiety, psychotic and personality disorders, and alcohol and substance abuse (Van Orden et al., 2010). A number of psychological processes have been found to exacerbate suicidal ideation. Specifically, findings from cross-sectional as well as longitudinal studies have attested to the role of hopelessness and helplessness, feelings of defeat and entrapment, deficits in problem solving abilities, and avoidant coping in the development of suicidal ideation (Van Orden et al., 2010, Nock et al., 2008, Szanto et al., 2002).

The presence of suicidal ideation in a clinical or medical setting typically requires a thorough risk assessment including chronic and acute risk factors as well as the frequency, intensity, and duration of suicidal thoughts (see Tishler and Reiss, 2009 for prevention in medical settings).

References and Readings

Kessler, R. C., Berglund, P., Borges, G., Nocke, M., & Wang, P. S. (2005). Trends in suicide ideation, plans, gestures, and attempts in the United States, 1990–1992 to 2001–2003. *JAMA: The Journal of the American Medical Association, 293*, 2487–2495.

Nock, M. K., Borges, G., Bromet, E. J., Cha, C. B., Kessler, R. C., & Lee, S. (2008). Suicide and suicidal behavior. *Epidemiologic Reviews, 30*, 133–154.

Szanto, K., Gildengers, A., Mulsant, B. H., Brown, G., Alexopoulus, G. S., & Reynoldsm, C. F. (2002). Identification of suicidal Ideation and prevention of suicidal behavior in the elderly. *Drugs & Aging, 19*, 11–24.

Tang, N. K., & Crane, C. (2006). Suicidality in chronic pain: A review of the prevalence, risk factors, and psychological links. *Psychological Medicine, 36*, 575–586.

Tishler, C.L. and Reiss, N.S. (2009). Inpatient suicide: preventing a common sentinel event. *General Hospital Psychiatry*, *31*, 103–109.

Van Orden, K.A., Witte, T.K., Cukrowicz, K.C., Braithwaite, S.R., Selby, E.A., and Joiner, T.E. (2010). The interpersonal theory of suicide. *Psychological Review*, *117*, 575–600.

Weissman, M. M., Bland, R. C., Canino, G. J., Greenwald, S., Hwu, H. G., Joyce, P. R., et al. (1999). Prevalence of suicide ideation and suicide attempts in nine countries. *Psychological Medicine, 29*, 9–17.

Suicidal Impulses

▶ Suicidal Ideation, Thoughts

Suicidal Thoughts

▶ Suicidal Ideation, Thoughts

Suicide

Mariam Dum[1] and Orit Birnbaum-Weitzman[2]
[1]Jackson Memorial Hospital, Miami, FL, USA
[2]Department of Psychology, University of Miami, Miami, FL, USA

Synonyms

Deliberate self-harm; Self-directed violence; Self-Inflicted injurious behavior; Self-murder

Definition

Suicide is the act of intentionally ending one's own life. The definition of suicide reflects three important components (Rudd, Joiner & Rejab 2001): (a) that the person died, (b) that the person's behavior caused death, and (c) that the person intended to cause his or her own death. While intentionality is the most precise characteristic that distinguishes those who have died by suicide and those who had died by other causes, its assessment remains controversial.

Description

Suicide is a major public health problem, and reports from the World Health Organization (WHO) indicate that suicide is projected to become an increasingly important contributor to the global burden of disease over the coming decades. Suicide is the 11th leading cause of death among all ages in the United States and the 13th leading cause of death worldwide. Suicide rates vary significantly cross-nationally (Center for Disease Control and Prevention, 2007). In general, rates are highest in Eastern Europe and lowest in Central and South America, with the United States, Western Europe, and Asia falling in the middle. Epidemiological surveys showed that 2.7% of the US population has made a suicide attempt (Nock et al., 2008). Most individuals that attempt suicide die during their first suicide attempt (Nock et al., 2008). Within medical settings, according to Joint Commission on Accreditation of Healthcare Organizations (JCAHO 2010), suicide ranks among the five top most frequent sentinel events in hospitals. Seeking help from mental health professionals can assist in the reduction of distressing psychological symptoms. However, according to a recent review, while 45% of people who successfully committed suicide contacted a primary care provider within 1 month of their death, only 20% contacted mental health services within the same time period (Lauma, Martin & Pearson 2002).

Rudd, Joiner, and Rejab (2001) provide in their book a good description on a number of psychiatric, psychological, demographic, and biological variables have been recognized as suicide risk factors. The presence of one or more psychiatric problem is a central variable explaining suicide acts. At least 90% of individuals who have died from suicide have had at least one psychiatric disturbance. In addition, past suicide attempts, history of childhood abuse, and family history of suicide are associated with increased risk of suicide. Other variables that contribute to suicide risk are psychological variables, such as hopelessness, impulsivity, problem-solving deficits, and perfectionism. In terms of demographic variables, men are more likely to die by suicide than are women. Death by suicide is more common in older, lower socioeconomic status, and veterans. Similarly, non-Hispanic White individuals have a higher rate of suicide than individuals from other ethnical background. In addition, individuals who are unemployed, single, divorced, or widowed are also at a higher risk. Biological variables have also been found to be associated with suicide behaviors. Family, twin, and adoption studies have found evidence

for heritable risk of suicidality. Biological factors related to lower levels of serotonin metabolites in the cerebrospinal fluid, higher serotonin receptor binding in platelets, and fewer presynaptic serotonin transporter sites were found in individuals who died by suicide. In addition, biological factors that inhibit impulsive behaviors, such as greater postsynaptic serotonin receptors in specific brain areas, such as the prefrontal cortex, have been associated with suicide behaviors.

The high rate of medical illness among individuals who committed suicide shows that both mental disorders and physical illness are important risk factors. Research shows variability in the risk of suicide according to the type of medical diagnosis. According to Hughes and Kleespies (2001), medical illnesses such as AIDS, cancer, chronic pain, end-stage renal diseases, severe neurological disorders, and chronic obstructive pulmonary disease have been correlated with increased risk of suicide. In contrast, they reported that other medical conditions including sclerosis, heart transplant, hypertension, rheumatoid arthritis, neoplasms, and cervix and prostate cancer have not been associated with increased risk. Studies have shown that at least one quarter of inpatients that have committed suicide in medical/surgical units did not report any previous psychiatric history (JCAHO 2010). Furthermore, suicide among these patients tends to happen within the first 2 weeks of their initial diagnosis (JCAHO 2010). This highlights the need of assessing suicidality in individuals who have chronic medical problems or who have been recently diagnosed with a life-threatening illness. Additionally, significant differences have been found between individuals who have completed suicide in inpatient psychiatric facilities and inpatient medical units. According to these studies, individuals who committed suicide in medical units tend to be older, their death did not include careful planning, and their means of committing suicide were significantly more violent (Nock et al., 2008).

In contrast to the vast literature on variables associated with suicide, there are some studies that have identified protective factors. Protective factors are those that decrease the probability of suicide in the presence of elevated risk. Rudd and colleagues (2001) summarized some of the most consistent findings in the literature are supportive social network or family, reasons for living, and religious beliefs. Being pregnant and having young children in the home are also protective against suicide.

Clinical providers face the difficulty of recognizing individuals at risk of committing suicide as no single factor is sufficient to trigger or protect an individual from a suicidal act. Suicide warning signs are the earliest detectable signs or symptoms that indicate high risk for suicide in the near term (i.e., within minutes, hours, or days before the suicidal act; Rudd et al., 2001). They provide immediate cues to loved ones or clinical providers of the imminent risk of attempting to end one's life. Some suicide warning signs include hopelessness, anger, dramatic changes in mood, acting recklessly or engaging in risky activities, reports of feeling trapped, increased alcohol or drug use, withdrawal from loved ones, agitation or anxiety, and drastic sleep changes (Rudd et al., 2001).

Due to the difficulty of preventing and recognizing suicide, the assessment of suicide risk should be completed in a standardized and systematic way. In addition to assessing risk and protective factors, as well as warning signs to understand the risk level of an individual, suicide ideation, suicide plan, intent to act, previous suicide attempts, and the medical lethality of means need to be considered. When assessing suicide risk, it is important to differentiate between chronic risk and acute risk (Rudd et al., 2001). Chronic risk involves the presence of risk factors. Acute risk is determined by suicide ideation, intention, and a suicide plan in combination with warning signs. Acute risk can be further classified into low, moderate, and high. Low acute risk involves the presence of suicide thoughts with no specific plan, intent, or behavior. Moderate acute risk involves the presence of suicide ideation and a plan without intent or behavior. High acute risk refers to the presence of persistent suicide ideation and a specific plan with the intent to die.

References and Readings

Centers for Disease Control and Prevention. (2007). *Web-based injury statistics query and reporting system (WISQARS)*. Retrieved June 20, 2011, from Centers for Disease Control and Prevention, National Center for Injury and Prevention Control Website: http://www.cdc.gov/ncipc/WISQARS

Hughes, D., & Kleespies, P. (2001). Suicide in the medically ill. *Suicide and Life Threatening Behaviors, 31*, 48–59.

Joint Commission on Accreditation of Healthcare Organizations. (2010). *Sentinel event statistics data*. Retrieved June 20, 2011, from the Joint Commission Website: http://www.jointcommission.org/sentinel_event_statistics_quarterly/

Lauma, J. B., Martin, C. E., & Pearson, J. L. (2002). Contact with mental health and primary care providers before suicide: A review of the evidence. *The American Journal of Psychiatry, 159*(6), 909–916.

Nock, M. K., Borges, G., Bromet, E. J., Cha, C. B., Kessler, R. C., & Lee, S. (2008). Suicide and Suicidal Behavior. *Epidemiologic Reviews, 30*, 133–154.

Rudd, M. D., Joiner, T., & Rajab, M. H. (2001). *Treating suicide behavior: An affective, time-limited approach*. New York: Guilford Press.

Wenzel, A., Brown, G. K., & Beck, A. T. (2009). *Cognitive therapy for suicide patients: Scientific and clinical applications*. Washington, DC: American Psychological Association.

Suicide Risk, Suicide Risk Factors

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

Death from suicide in 1998 was ranked by the World Health Organization (WHO) as the 12th leading cause of mortality worldwide. Suicide is the cause of death of one million people a year worldwide (Lineberry, 2009). Suicide has been committed via several methods, which vary across geographic regions (Ajdacic-Gross et al., 2008). It is a main cause of death in later adolescence (ages 15–24 years; Shields, Hunsaker & Hunsaker, 2006).

The main difficulty in suicide prevention is the prediction of rare events and the multiple risk factors of suicide. These include situational factors such as social stressors and life events (e.g., unemployment, poverty), psychological factors such as hopelessness and hostility, biological factors (e.g., reduced brain-derived neurotrophic factor, protein-kinase A; Dwivedi & Pandey, 2011), and mere access to means of suicide (e.g., arms). One main difficulty in predicting suicidal behavior is the reliance on self-reported instruments, where people either conceal their real replies or are unaware of them.

Conversely, some recent studies have shown that assessing implicit associations between the "self" and "life" versus "death," with the Implicit Association Test, predicted suicidal behavior beyond what was detected by traditional risk factors (e.g., depression, past attempts, clinicians' ratings; Nock et al., 2010). Similarly, attention biases to suicide-related words relative to neutral words, using the "emotional stroop," predicted suicidal behavior better than traditional risk factors (Cha, Najmi, Park, Finn, & Nock, 2010). More research needs to utilize such instruments to identify additional suicide risk factors assessed in such indirect manners. Often, an accumulation of risk factors occurs, culminating in the tragic event. To conclude, explicit and implicit psychosocial factors and biological and situational factors serve as risk factors for suicide and need to be considered in the important attempt to prevent this severe health outcome.

Cross-References

▶ Hopelessness
▶ Suicide

References and Readings

Ajdacic-Gross, V., Weiss, M. G., Ring, M., Hepp, U., Bopp, M., Gutzwiller, F., et al. (2008). Methods of suicide: International suicide patterns derived from

the WHO mortality database. *Bulletin of the World Health Organization, 86*, 726–732.
Cha, C. B., Najmi, S., Park, J. M., Finn, C. T., & Nock, M. K. (2010). Attentional bias toward suicide-related stimuli predicts suicidal behavior. *Journal of Abnormal Psychology, 119*, 616–622.
Dwivedi Y., & Pandey G. N. (2011). Elucidating biological risk factors in suicide: role of protein kinase A. *Prog Neuropsychopharmacol Biol Psychiatry, 35*, 831–841.
Lineberry, T. W. (2009). Suicide rates in 2009. Do the economy and wars have an effect? *Minnesota Medicine, 92*, 49–52.
Nock, M. K., Park, J. M., Finn, C. T., Deliberto, T. L., Dour, H. J., & Banaji, M. R. (2010). Measuring the suicidal mind: Implicit cognition predicts suicidal behavior. *Psychological Science, 21*, 511–517.
Shields, L. B., Hunsaker, D. M., & Hunsaker, J. C., 3rd. (2006). Adolescent and young adult suicide: A 10-year retrospective review of Kentucky medical examiner cases. *Journal of Forensic Sciences, 51*, 874–879.

Sulcus

▶ Brain, Cortex

Summary Data

▶ Aggregate Data

Sun Exposure

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

Sun exposure refers to the amount and manner in which people expose themselves to sunlight. This is a highly complex parameter for quantification, as it relies on self-report and its effects depend on multiple environmental and personal factors, as well as frequency and location of exposure on the body. Most skin cancers are related to sun exposure, and a great majority of exposure to sun takes place before adulthood, making children and adolescents central target groups for assessment and prevention of sun exposure. Multiple studies have linked sun exposure to various cancers, particularly to skin cancer. In a review of 57 studies, intermittent sun exposure and history of burns were related to risk of melanoma. In contrast, high occupational sun exposure was inversely related to melanoma. Furthermore, factors such as country of study seem to moderate effects of sun exposure on melanoma, suggesting that effects of sun exposure depend on geographical factors as well. Indeed, latitude synergistically interacts with history of sunburn in relation to melanoma (Gandini et al., 2005). A major cause of melanoma due to sun exposure is ultraviolet light (Armstrong & Kricker, 1993). Various scales exist for assessing sun exposure, which consider the manner and duration of sun exposure, context (working vs nonworking days), and cumulating measures in relation to various time frames (years or one's life time; Kricker, Vajdic, & Armstrong, 2005). Importantly, to achieve greater test-retest reliability, it may be beneficial to assess sun exposure in relation to activities (e.g., with family, at work) rather than in relation to specific time periods (Yu et al., 2009). In children, important social factors related to usage of sun protective agents include parental reminders (Donavan & Singh, 1999). In children, sun protective behavior decreases with age though sun exposure increases with age, possibly reflecting greater peer pressure and reduced parental control in older children (Pichora & Marrett, 2010). Thus, sun exposure reflects a major and complex cause of various skin cancers and must be properly assessed and better controlled in attempt to prevent skin cancers.

Cross-References

▶ Cancer Screening/Detection/surveillance

References and Readings

Armstrong, B. K., & Kricker, A. (1993). How much melanoma is caused by sun exposure? *Melanoma Research, 3*, 395–401.

Donavan, D. T., & Singh, S. N. (1999). Sun-safety behavior among elementary school children: the role of knowledge, social norms, and parental involvement. *Psychological Reports, 84*, 831–836.

Gandini, S., Sera, F., Cattaruzza, M. S., Pasquini, P., Picconi, O., Boyle, P., et al. (2005). Meta-analysis of risk factors for cutaneous melanoma: II. Sun exposure. *European Journal of Cancer, 41*, 45–60.

Kricker, A., Vajdic, C. M., & Armstrong, B. K. (2005). Reliability and validity of a telephone questionnaire for estimating lifetime personal sun exposure in epidemiologic studies. *Cancer Epidemiology, Biomarkers & Prevention, 14*, 2427–2432.

Pichora, E. C., & Marrett, L. D. (2010). Sun behaviour in Canadian children: Results of the 2006 national sun survey. *Canadian Journal of Public Health, 101*, 14–18.

Yu, C. L., Li, Y., Freedman, D. M., Fears, T. R., Kwok, R., Chodick, G., et al. (2009). Assessment of lifetime cumulative sun exposure using a self-administered questionnaire: Reliability of two approaches. *Cancer Epidemiology, Biomarkers & Prevention, 18*, 464–471.

Supervisory Attentional System

► Executive Function

Supplication

► Prayer

Supportive Care

► Palliative Care

Suprachiasmatic Nucleus

► Hypothalamus

Supraoptic Nucleus

► Hypothalamus

Surgery

► Cancer Treatment and Management

Surgical Resection

► Cancer Treatment and Management

Surrogacy

► In Vitro Fertilization, Assisted Reproductive Technology

Surrogate

► In Vitro Fertilization, Assisted Reproductive Technology

Surrogate Decision Making

Howard Sollins
Attorneys at Law, Ober, Kaler, Grimes & Shriver, Baltimore, MD, USA

Synonyms

Proxy

Definition

Surrogate Decision Making

If an individual is unable to make decisions about personal health care, some other individual can be authorized to provide direction. Such a person is called the surrogate decision maker, a proxy, or some other term specific to the type of authorization. For example, under an advance directive, the decision maker is typically an "agent"; under a durable power of attorney for health care, the decision maker is the "attorney in fact" (although in slang, some health-care providers refers to such persons as "the POA"); an individual may be judicially appointed as a "guardian," or state law may identify certain next of kin or other close persons as eligible to serve as a surrogate. State law will determine the role of domestic partners in the absence of an advance directive appointing someone as an agent.

Each type of surrogate or proxy has a scope of authority to make health-care decisions for an individual depending on the source of the surrogate or proxy's authority and state law. For example, the surrogate may have broad authority to consent to health care and to refuse or direct the withdrawal of health care but with limitations if the care is life-sustaining. An agent under an advance directive from an individual may have immediate, broader authority to direct the withholding or withdrawal of life-sustaining treatment than a surrogate acting only under state law. Guardians may nor may not need court approval for certain actions involving life-sustaining treatment.

Surrogates or proxies appointed by the individual patient have decision making priority. Where there is no such person, state law determines the process for consulting with next of kin or others, which can include close friends, potentially in an order of hierarchy as to who priority, and how to resolve disagreements among those with the same level of relationship. Where there are no such persons or there is an irreconcilable disagreement, a judicial guardianship may be needed. Ethics committees or Patient Care Advisory Committees are examples of bodies within a health-care facility that can be very helpful in gathering an evaluating information about an individual's clinical condition, treatment options and prognosis, previously expressed wishes, interpretations of available documents, varying points of view, and related considerations.

In a recent study, it was noted that surrogate decision making is often required for older Americans at the end of life. Among a sample of 4,246 deaths of respondents in the Health and Retirement Study, proxies reported that 42.5% of these individuals needed decision making about medical treatment before death; 70.3% of subjects lacked the capacity to make those decisions themselves and, overall, 29.8% required decision making at the end of life but lacked decision-making capacity. These findings suggest that more than a quarter of elderly adults may need surrogate decision making before death.

Cross-References

▶ End-of-Life Care

References and Readings

Silveira, M. J., Kim, S. Y. H., & Langa, K. M. (2010). Advance directives and outcomes of surrogate decision making before death. *The New England Journal of Medicine, 362*, 1211–1218.

Surveys

Seppo Laaksonen
University of Helsinki, Helsinki, Finland

Synonyms

Micro data collection and analysis system; Opinion poll; Statistical inquiry

Definition

Survey is a methodology and a practical tool used to collect, handle, and analyze in a systematic way information from individuals. These individuals or micro units can be of various types, such as people, households, hospitals, schools, businesses, or other corporations. The units can be simultaneously available from two or more levels such from households and their members. Information in surveys may be concerned various topics such as people's personal characteristics, their behavior, health, salary, attitudes and opinions, incomes, poverty and housing environments, or characteristics and performance of businesses. Survey research is unavoidably interdisciplinary, although the role of statistics is most influential since the data for surveys is constructed in a quantitative form. Correspondingly, many survey methods are special statistical applications. However, surveys exploit substantially many other sciences such as informatics, mathematics, cognitive psychology, and theory of submatter sciences of each survey topic.

Basic Survey Concepts

A key concept in surveys is *target population* the universe of which should be exactly determined and realistic. It is possible that there are more than one target population (e.g., hospitals of certain types and their clients during a specific period). Before determining a strict target population, a researcher can have in mind *population of interest*, but this is often too difficult to reach, and hence, a realistic population is chosen. For example, more or less heavy alcohol drinkers may be interest for a researcher, but such people cannot be found from any data source. Respectively, such a target population is not realistic, but fortunately, one can try with a larger target population where there are also nondrinkers or light drinkers. The study itself can, among others, concentrate on those heavy drinkers, and results are correct if there are in data enough such people in order to get appropriate results. This requires also that we have a good frame and *frame population* from which reasonable data are downloaded. A drawback is that although the frame seems to be ideal, it includes such people who do not belong to our realistic target population, nevertheless, and secondly, we cannot get responses from all selected people due to *nonresponse*. Thus, when designing data collection, it is necessary to predict nonresponse and other gaps as well as possible in order to get enough respondents for the study.

Sampling

Survey data can cover the whole target population, but if it is large, it is rational to use sampling. This leads to plan an optimal sampling design. The design may be more or less complex. The simplest one is to use completely random selection in which case every frame unit has an equal *inclusion probability* to be selected in the sample. Such a design is rarely rational since the data collection may be too expensive or such a frame is not available. For these reasons, the most common strategy in people surveys is in the first stage, to select so-called *clusters* (small areas, service houses, households), and in the second stage, the desired sample units (target people, clients, household members) are to be interviewed and studied. This strategy is called *two-stage sampling*, but *three-stage sampling* is also applied. These both strategies lead to *probability sampling* that is definitely a valid method. Naturally, next steps must have been done successfully too.

Two- and three-stage samplings are generally called multistage sampling, but if the study units are selected directly from the frame, it is one-stage sampling, but this term is not much used. In contrast, the term *element sampling* is common. Multistage sampling is *hierarchical* in its nature, that is, we approach to study units by stage by stage. This strategy is different from *multiphase sampling* in which case after one sample selection, a new sample from the first sample has been selected. This is much used in *panels* in which case the second or the

consecutive samples have been selected even with 100% from those who have responded in the first phase. This panel approach gives opportunity to follow respondents over time. It is common in health, poverty, and living condition research, for instance. Panel designs can be very complex too. *Rotating design* is much used when needed to analyze data both *cross-sectionally* (for a specific time point or time period) and *longitudinally* (for following individuals over time). Long panels might be hard to do well, and there can be met too much nonresponse that leads a worsening data quality.

Two-phase sampling is also useful when analyzing how respondents differ from nonrespondents. This information is necessary for survey quality documentation, but it can be used also for improving the quality. The second phase could in this case lead to draw a subsample of the nonrespondents, and then attempts to get some information from them. Naturally, this is not easy since they are reluctant, but most nonrespondents still are willing to answer to some simple but key questions of the survey.

Moreover, it is often advantageous to exploit *stratification* in surveys. This leads to create a number of strata that are like subpopulations. Sampling designs for each stratum may be similar or different. The main varying point is maybe that the sample size has been allocated for each stratum so that the research targets are satisfied ideally. Typically, the relative sample size is larger for a smaller stratum population and smaller for a larger population, respectively. This is due to an ordinary target to get about as accurate estimates (results) for each stratum.

Data Collection Tools and Methods

Data collection is not ready after selection a sample (or the full data). Three other major tasks are needed: (1) design the questionnaire, (2) decide the data collection mode (mail or phone or face-to-face interviewing or web or mixed mode such as web plus phone), (3) collect supported data (auxiliary) both for sampling and further data handling. Such data are required both from respondents and nonrespondents. *Auxiliary data* are very advantageous for adjusting *sampling weights* from the initial ones. Consequently, the bias in estimates will be reduced.

Data Cleaning and Analysis

Survey data should be cleaned before starting the analysis. In addition to computing the sampling weights, the following tasks are needed: data editing, imputation of missing data, data documentation (called metadata), data collection process documentation (called paradata), and selection of a good IT format (e.g., SPSS or SAS). The sampling design strategy is necessary to correctly take into account in the analysis.

Cross-References

- ▶ Clusters
- ▶ Cohort Study
- ▶ Data
- ▶ Internet-Based Studies
- ▶ Interview
- ▶ Methodology
- ▶ Multivariate Analysis
- ▶ Odds Ratio
- ▶ Participation Bias
- ▶ Population Stratification
- ▶ Probability
- ▶ Randomization
- ▶ Regression Analysis
- ▶ Response Bias
- ▶ Retrospective Study
- ▶ Sample Size Estimation
- ▶ Selection Bias
- ▶ Standard Deviation
- ▶ Statistics

References and Readings

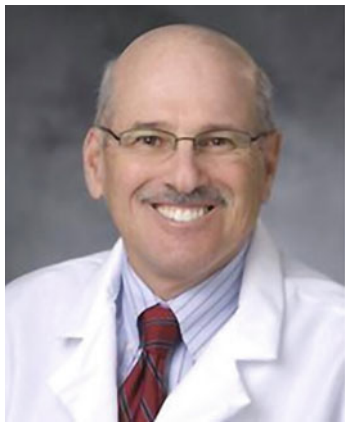
Bethlehem, J. G. (2009). *Applied survey methods: a statistical perspective*. Hoboken, NJ: Wiley. 375 pp.

de Leeuw, E. D., Hox, J. J., & Dillman, D. A. (Eds.). (2009). *International handbook of survey methodology*. New York\London: Lawrence Erlbaum Associates\Taylor and Francis Group. 549 pp.

Surwit, Richard S.

James A. Blumenthal
Department of Psychiatry & Behavioral Sciences, Duke University Medical Center, Durham, NC, USA

Biographical Information



Richard S. Surwit received his B.A. from Earlham College and Ph.D. in clinical psychology from McGill University. He completed a postdoctoral fellowship in psychophysiology at Harvard University and joined the Duke faculty in 1977. He is currently professor of medical psychology and vice chair for research in the Department of Psychiatry and Behavioral Science at Duke University Medical Center, Durham, NC.

Major Accomplishments

Surwit's early work focused on the utility of biofeedback in the treatment of medical disorders, including Raynaud's disease and hypertension, and he was instrumental in establishing one of the first clinical biofeedback facilities in the country. Over the course of a long and productive career, his scientific contributions range from basic research on the use of mouse models to examine genetic and behavioral interactions in the development of obesity to clinical investigations of the effects of stress and hostility on glucose metabolism and diabetes. He also pioneered the use of computers in the medical management of such chronic diseases as diabetes and congestive heart failure. He has received multiple grants from the National Institutes of Health, private foundations, and industry to support his research program. He has played a key administrative role in the Department of Psychiatry and Behavioral Sciences at Duke University as vice chair for research and is a former president of the Society of Behavioral Medicine. He also has served on the editorial boards of such journals as *Health Psychology*, the *Journal of Consulting and Clinical Psychology*, *Obesity*, and *Metabolism*. He is the recipient of numerous awards and honors including the Research Career Award in Health Psychology from the American Psychological Association.

Cross-References

► Diabetes

Sustainability

► Ecosystems, Stable and Sustainable

Sympathetic

► Autonomic Balance
► Heart Rate Variability

Dr. Surwit recently received the Distinguished Scientist Award from the Society of Behavioral Medicine.

Sympathetic Nervous System (SNS)

Michael Richter[1] and Rex A. Wright[2]
[1]Department of Psychology, University of Geneva, Geneva, Switzerland
[2]College of Arts and Sciences, Department of Psychology, University of North Texas, Denton, TX, USA

Definition

The sympathetic nervous system (SNS) is one of two main branches or subsystems of the autonomic nervous system (ANS). It originates in the thoracic and upper lumbar segments of the spinal cord and commonly – but not always – yields peripheral adjustments that are complementary to those produced by its counterpart, the parasympathetic nervous system (PNS).

Description

The sympathetic nervous system is one of two main branches or subsystems of the autonomic nervous system, the physical system responsible for unconsciously maintaining bodily homeostasis and coordinating bodily responses. Working with the second main branch, the parasympathetic nervous system, the sympathetic nervous system regulates a wide range of functions such as blood circulation, body temperature, respiration, and digestion. Sympathetic activation commonly leads to adjustments on organs and glands that are complementary to those produced by parasympathetic activation and suitable for high activity ("fight and flight" as opposed to "rest and digest"). Examples of high-activity adjustments are constriction of blood vessels in the skin, dilation of blood vessels in the skeletal muscles and lungs, and increased heart rate and contraction force. Although sympathetic adjustments tend to complement parasympathetic adjustments, they do not always. For example, both sympathetic nervous system arousal and parasympathetic nervous system arousal increase salivary flow, although to different degrees and yielding different compositions of saliva.

Basic functional units of the sympathetic nervous system are preganglionic and postganglionic neurons. Preganglionic neurons have cell bodies in the thoracic and upper lumbar segments of the spinal cord and axons that extend to cell bodies of postganglionic neurons. Postganglionic neurons have cell bodies that are clustered in the so-called ganglia and relatively long axons that innervate target organs and glands. The major neurotransmitters of the sympathetic nervous system are acetylcholine and norepinephrine. Acetylcholine is the neurotransmitter of all preganglionic neurons. Stimulation of cholinergic receptors of the nicotinergic subtype located on the cell bodies of the postganglionic neurons by acetylcholine leads to an opening of nonspecific ion channels. This opening permits transfer of potassium and sodium ions, which depolarizes the postganglionic cell and initiates an action potential. Norepinephrine is the neurotransmitter of most sympathetic postganglionic neurons and stimulates adrenergic receptors lying on targeted visceral structures. All adrenergic receptors are coupled with G-proteins, but transmission pathways depend on the receptor subtype. Activation of alpha-1 receptors changes the calcium concentration in the cell, which in turn triggers the specific effect on the targeted visceral structure. Alpha-2 and beta receptors trigger visceral responses by affecting cAMP production in the cell. Specific effects depend on the receptor subtype and on the innervated visceral structure. For instance, stimulation of alpha-1 receptors on blood vessels of skeletal muscles leads to vasoconstriction and reduced blood flow, whereas stimulation of alpha-1 receptors on the radial pupil muscle leads to muscle contraction and increased pupil size. Stimulation of beta-2 receptors on the heart leads to increased heart rate and contraction force, whereas stimulation of beta-2 receptors on skeletal muscle blood vessels leads to vasodilation and increased blood flow.

In working jointly with the parasympathetic nervous system, the sympathetic nervous system

does not function in an all-or-none fashion, but rather activates to different degrees. Depending on the affected visceral structure and situation, it may be more or less active than the parasympathetic nervous system. Shifts in the magnitude of sympathetic and parasympathetic influence can occur locally within a single visceral structure (e.g., the eye) or across visceral structures, with local shifts occurring to meet highly specialized demands (e.g., a change in ambient light) and global shifts adapting the body to large-scale environmental changes (e.g., the appearance of a substantial physical threat). Autonomic control is maintained by structures in the central nervous system that receives visceral information from an afferent (incoming) nervous system. A key central nervous system structure is the hypothalamus, which integrates autonomic, somatic, and endocrine responses that accompany different organism states.

Cross-References

- ▶ Acetylcholine
- ▶ Adrenaline
- ▶ Autonomic Activation
- ▶ Autonomic Balance
- ▶ Autonomic Nervous System (ANS)
- ▶ Epinephrine
- ▶ Parasympathetic Nervous System (PNS)

References and Readings

Berne, R. M., Levy, M. N., Koeppen, B. M., & Stanton, B. A. (2004). *Physiology* (5th ed.). St. Louis, MO: Mosby.

Cacioppo, J. T., & Tassinary, L. G. (1990). *Principles of psychophysiology: Physical, social, and inferential elements*. New York: Cambridge University Press.

Cacioppo, J. T., Tassinary, L. G., & Berntson, G. G. (2000). *Handbook of psychophysiology* (2nd ed.). New York: Cambridge University Press.

Ganong, W. F. (2005). *Review of medical physiology* (22nd ed.). New York: McGraw-Hill.

Levick, J. R. (2009). *An introduction to cardiovascular physiology* (5th ed.). London: Hodder.

Sympathetic Nervous System (SNS) Activation

▶ Sympatho-Adrenergic Stimulation

Sympatho-Adrenergic Stimulation

Sabrina Segal
Department of Neurobiology and Behavior, University of California, Irvine, CA, USA

Synonyms

Adrenergic activation; Sympathetic nervous system (SNS) activation

Definition

Activation of one of the three branches (the sympathetic nervous system) of the autonomic nervous system via disruption of physiological homeostasis which results in the release of epinephrine/adrenaline and norepinephrine/noradrenaline from the adrenal medulla.

Description

The sympatho-adrenomedullary (SAM) system is one of two major components of the stress system. Stress activates the sympathetic nervous system and the goal of this response is to return the individual to physiological homeostasis. Epinephrine release from the adrenal medulla causes physiological alterations in cardiovascular tone, respiration rate, and blood flow to the muscles. Epinephrine does not cross the blood–brain barrier, but acts indirectly on the brain via the vagus nerve, which projects to the nucleus of the solitary tract (NTS), resulting in noradrenergic projections to the amygdala, as well as other brain regions. The release of epinephrine and norepinephrine from the adrenal medulla increases

blood glucose levels and enhances alertness, learning, and memory.

Cross-References

► Norepinephrine/Noradrenaline

References and Readings

Cannon, W. B. (1914). The interrelations of emotions as suggested by recent physiological research. *The American Journal of Psychology, 25*(2), 256–282.
Chrousos, G. P., & Gold, P. W. (1992). The concepts of stress and stress system disorders. Overview of physical and behavioral homeostasis. *Journal of the American Medical Association, 267*(9), 1244–1252.
Hollenstein, T., McNeely, A., Eastabrook, J., Mackey, A., & Flynn, J. (2011). Sympathetic and parasympathetic responses to social stress across adolescence. *Developmental Psychobiology*. doi:10.1002/dev.20582.
Sherwood, L. (2008). *Human physiology*: *From cells to systems* (7th ed., p. 240). Stamford: Cengage Learning.
Tilders, F. J. H., & Berkenbosch, F. (1986). CRF and catecholamines; their place in the central and peripheral regulation of the stress response. *Acta Endocrinology, 113*, S63–S75.

Symptom Magnification Syndrome

Karen Jacobs
Occupational Therapy, College of Health and Rehabilitation Science, Sargent College, Boston University, Boston, MA, USA

Synonyms

Learned symptom behavior; Maladaptation of symptom behaviors to chronic illness; Malingering; Martyr behavior; Secondary gain

Definition

Symptom magnification is a self-destructive, socially reinforced behavioral response pattern consisting of reports or displays of symptoms which function to control the life of circumstances of the sufferer.

Description

Symptom magnification syndrome (SMS) may be described as a conscious or unconscious self-destructive learned pattern of behavior which is maintained through social reinforcement and typically controls the individual's life activities. SMS may be labeled "malingering" or exaggerated psychological complaints which can be associated with individuals who are seeking financial compensation or a secondary gain, e.g., increased attention from a family member, from symptom reporting. The validity scales of the Minnesota Multiphasic Personality Inventory-2 (MMPI-2) are widely used for the detection of exaggerated psychological complaints.

Cross-References

► Psychosocial Adjustment
► Psychosocial Predictors
► Psychosocial Variables
► Psychosocial Work Environment
► Psychosomatic
► Psychosomatic Disorder
► Somatic Symptoms
► Somatization
► Somatoform Disorders

References and Readings

Kopel, S., Walders-Abramson, N., MsQuaid, E., Seifer, R., Koinis-Mitchell, D., Klein, R., et al. (2010). Asthma symptom perception and obseity in children. *Biological Psychology, 84*, 135–141.
Matheson, L. N. (1986). *Work capacity evaluation: Systematic approach to industrial rehabilitation*. Anaheim, California: Employment and Rehabilitation Institute of California.
Matheson, L. N. (1987). *Symptom magnification casebook*. Matheson, LN: Employment and Rehabilitation Institute of California.
Theodore, B., Kishino, N., & Gatchel, R. (2008). Biopsychosocial factors that perpetuate chronic pain,

impairment, and disability. *Psychological Injury and Law, 1*, 182–190.
Tsushima, W., & Tsushima, V. (2009). Comparison of MMPI-2 validity scales among compensation-seekin Caucasian and Asian American medical patients. *Assessment, 16*, 159–164.

Symptom-Limited Exercise Test

▶ Maximal Exercise Stress Test

Symptoms

Tana M. Luger
Department of Psychology, University of Iowa, Iowa City, IA, USA

Synonyms

Indicators

Definition

Symptoms are physical sensations or changes in internal state that a person recognizes, interprets, and reports (Pennebaker, 1982). Although symptoms are a key indicator of disease, it has been shown that perceived symptoms do not always correspond with objective, physiological pathology. Thus, researchers have sought to examine the various factors that can influence the perceptual processing and attributing of physical symptoms.

Because perception involves attention to certain cues while ignoring others, a person is more likely to recognize symptoms if their external world is not supplying them with information or distractions (Pennebaker, 1982). For example, a person with a boring job is more likely to report symptoms than one with a fast-paced job. The assumption is that the person with the boring job can focus more attention on his internal, physical state rather than managing the external.

There is much evidence that people selectively search for information when interpreting their symptoms. People tend to focus on information which either confirms their expectations or shows the potential symptoms to be benign (Leventhal, Leventhal, & Contrada, 1998; Pennebaker, 1982). Situational cues may also influence a person's search for information. For example, a recent outbreak of influenza in one's town may make a person more sensitive to his physiological changes and more likely to interpret his symptoms as signs of the flu. Previous experience with similar symptoms may also prime a person to attribute symptoms as being indicators of a particular disease (Jemmott, Croyle, & Ditto, 1988).

Finally, researchers have found that individual differences like gender, age, and personality can affect the amount that people report symptoms (Pennebaker, 1982). For example, women tend to report more physical symptoms than men, older adults report more than young adults, and those high in the personality trait of negative affectivity report more than those high in positive affectivity. One reason suggested for these differences are differing tendencies to focus on one's internal state, resulting in more attention and recognition of symptoms.

Cross-References

▶ Cognitive Appraisal
▶ Common-Sense Model of Self-regulation
▶ Illness Cognitions and Perceptions

References and Readings

Jemmott, J. B., III, Croyle, R. T., & Ditto, P. H. (1988). Commonsense epidemiology: Self-based judgments from laypersons and physicians. *Health Psychology, 7*, 55–73.
Leventhal, H., Leventhal, E. A., & Contrada, R. J. (1998). Self-regulation, health, and behavior: A perceptual-cognitive approach. *Psychology & Health, 13*, 717–733.
Pennebaker, J. W. (1982). *The psychology of physical symptoms*. New York: Springer.

Symptoms Scale

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

Symptoms scales are psychometric instruments aimed at assessing the frequency or severity of any type of symptom associated with a mental or physical health condition. Development of symptom scales requires the same type of rigor as any other self-report instrument requires, including tests of internal reliability and test-retest reliability and face, concurrent, construct and predictive validity.Questions on such scales can be asked in relation to a specific time frame (e.g., the present moment, the past week) and in relation to certain severity levels. For example, in the assessment of pain symptoms, patients may be asked to rate their level of average and worse pain in a given time frame. Finally, some scales also ask the extent to which certain symptoms interfere with one's daily functioning, as is often done in the domain of quality of life or pain. One of the earliest developed psychological symptoms scale is the symptom check list 90 (SCL-90) which was designed to assess psychological symptoms for evaluating the outcomes of mental health interventions and for research purposes. The symptoms are assessed in relation to the past 7 days and are categorized into nine dimensions (e.g., psychoticism, depression). Its internal reliability is adequate (e.g., Cronbach's alpha of .77 to .90 on its dimensions). Its concurrent, construct, and predictive validities have been shown as well. A physical symptoms scale is the Patient Health Questionnaire 15 (PHQ-15; Kroenke, Spitzer, & Williams, 2002). This scale assesses 15 common physical symptoms including stomach, back, head and chest pains, dizziness, shortness of breath, etc. Scores on the PHQ-15 correlate with functional status and with healthcare utilization. Numerous other instruments exist for assessment of various psychiatric symptoms including depression, anxiety, post-traumatic stress disorder, and for disease-specific symptoms. The latter include symptoms scales of upper respiratory infections (Orts et al., 1995), the Rose chest pain questionnaire (Rose, McCartney, & Reid, 1977), and others. Symptom scales are a basic element in diagnosis and monitoring of treatment effects and in research in many health disciplines including medicine and behavior medicine. However, it is noteworthy to consider the limitations of symptom scales, as in most self-report scales. These include reporting biases, memory, and lack of self-awareness. One important factor known to underlie reporting biases includes neuroticism or negative affectivity, which, when elevated, often leads to inflated symptom reporting and needs to be considered when patients complete symptoms scales (Watson & Pennebaker, 1989).

Cross-References

▶ Somatic Symptoms

References and Readings

Kroenke, K., Spitzer, R. L., & Williams, J. B. (2002). The PHQ-15: Validity of a new measure for evaluating the severity of somatic symptoms. *Psychosomatic Medicine, 64*, 258–266.

Orts, K., Sheridan, J. F., Robinson-Whelen, S., Glaser, R., Malarkey, W. B., & Kiecolt-Glaser, J. K. (1995). The reliability and validity of a structured interview for the assessment of infectious illness symptoms. *Journal of Behavioral Medicine, 18*, 517–529.

Rose, G., McCartney, P., & Reid, D. D. (1977). Self-administration of a questionnaire on chest pain and intermittent claudication. *British Journal of Preventive & Social Medicine, 31*, 42–48.

Watson, D., & Pennebaker, J. W. (1989). Health complaints, stress, and distress: Exploring the central role of negative affectivity. *Psychological Review, 96*, 234–254.

Syndrome X

▶ Metabolic Syndrome

Syntocinon (Synthetic Forms)

► Oxytocin

Syringe Exchange Programs

► Needle Exchange Programs

Systematic Bias

► Bias

Systematic Desensitization

Faisal Mir
School of Sport & Exercise Sciences, University of Birmingham, Edgbaston, BHAM, UK

Synonyms

Graded exposure counterconditioning

Definition

Systematic desensitization or graded exposure is a behavioral intervention commonly used in the treatment of phobias and other anxiety-related disorders. Individuals with phobias tend to possess irrational fears of stimuli such as heights, close spaces, dogs, and snakes. In order to cope, the individual avoids such stimuli. Since escaping from the phobic object reduces anxiety temporarily, the individual's behavior to reduce the perceived fear is negatively reinforced. The aim of systematic desensitization is to overcome this avoidance by gradually exposing individuals to the phobic stimulus until their anxiety to the fear is extinguished (Sturmey, 2008).

Description

Joseph Wolpe (1915–1997)

Joseph Wolpe was a South African–born American doctor. During his work as a medical officer, Wolpe's task was to treat soldiers who were diagnosed with "war neurosis" which is now referred to as post-traumatic stress disorder. It was argued at the time that by talking about their war experiences would lead to a resolution of their symptoms. However, this was not found to be the case, and Wolpe became increasingly disillusioned by Freud's psychoanalytic therapy. It was this which served as a catalyst for Wolpe to discover other more effective treatment strategies (Wolpe, 1973).

Wolpe began to investigate behavioral strategies through laboratory experiments. One of his concepts was reciprocal inhibition by which anxiety is inhibited by a feeling which is incompatible such as relaxation (Wolpe, 1961). He pioneered the intervention assertiveness training and deciphered that this approach was useful for people who were anxious about social situations. As they learned assertiveness skills, this assisted to minimize the anxiety associated with such situations and in turn relaxed. As this proved to be highly fruitful, it further led to the development of systematic desensitization (Wolpe, 1973).

Systematic Desensitization

It was discovered that fears could be learned through the behavioral model of classical conditioning (see ► Classical Conditioning). Therefore, Wolpe (1973) sought to eliminate the fear response generated by the stimulus and replace it with a competing response of relaxation. The notion of systematic desensitization was based upon two principles of conditioning. The first was that an individual could not produce two different responses to stimuli such as fear and relaxation. Secondly, classical conditioning often involves stimulus generalization which refers to stimuli which are similar and lead to the learned response of fear (Sturmey, 2008).

During the process of systematic desensitization, the therapist works in conjunction with the

person who has a phobia to ascertain the exact stimulus which triggers the phobia. Next, the individual is taught relaxation-inducing techniques often related to a cue for relaxing. After this stage, the therapist and individual develop a list of fear-evoking stimuli, ranging from very mild to very intense anxiety. This list is referred to as a hierarchy of fears as the stimulus items are listed in order of the intensity of fear they evoke. Then, working gradually the therapist attempts to recondition the person so that the stimuli in the hierarchy become associated with a relaxed response rather than fear. Once the individual can eventually confront the stimulus which originally evoked the greatest anxiety and remains relaxed, then the phobia has been effectively extinguished (Sturmey, 2008).

Systematic desensitization has been found to be highly effective in the treatment of phobias (Clark, 1963), sexual disorders (Obler, 1973), and traumatic nightmares (Schindler, 1980).

Cross-References

- ▶ Anxiety Disorder
- ▶ Behavior Change
- ▶ Behavior Modification
- ▶ Behavioral Intervention
- ▶ Behavioral Therapy
- ▶ Classical Conditioning
- ▶ Cognitive Behavioral Therapy (CBT)

References and Readings

Clark, D. F. (1963). The treatment of monosymptomatic phobia by systematic desensitization. *Behaviour Research and Therapy, 1*(1), 63–68.

Obler, M. (1973). Systematic desensitization in sexual disorders. *Journal of Behavior Therapy and Experimental Psychiatry, 4*(2), 93–101.

Schindler, F. E. (1980). Treatment by systematic desensitization of a recurring nightmare of a real life trauma. *Journal of Behavior Therapy and Experimental Psychiatry, 11*(1), 53–54.

Sturmey, P. (2008). *Behavioral case formulation and intervention: A functional analytic approach*. Chichester: John Wiley.

Wolpe, J. (1961). The systematic desensitization treatment of neuroses. *The Journal of Nervous and Mental Disease, 132*(3), 189–203.

Wolpe, J. (1973). *The practice of behavior therapy* (2nd ed.). New York: Pergamon Press.

Systematic Review

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

Systematic reviews present a descriptive assessment of a collection of original research articles related to a specific research question. These reviews "collate, compare, discuss, and summarize the current results in that field" (Matthews, 2006). Campbell, Machin, and Walters (2007) noted that "It has now been recognised that to obtain the best current evidence with respect to a particular therapy all pertinent clinical trial information needs to be obtained." This "overview process" (Campbell et al., 2007) has led to many changes in the way clinical trial programs are developed. They have become an integral part of evidence-based medicine, impacting decisions that affect patient care.

A considerable problem in writing such reviews is the retrieval of all relevant publications in the behavioral medicine literature, although the advent of computerized searchable databases has made this task much less arduous.

While such a narrative review can be very useful in its own right, it can also be the first step in a two-step process that also includes conducting a meta-analysis. This provides a statistical (quantitative) answer, whereas the authors' conclusions in a systematic review will largely be qualitative.

Cross-References

- ▶ Meta-analysis

References and Readings

Campbell, M. J., Machin, D., & Walters, S. J. (2007). *Medical statistics: A textbook for the health sciences* (4th ed.). Chichester, UK: Wiley.

Matthews, J. N. S. (2006). *Introduction to randomized controlled clinical trials* (2nd ed.). Boca Raton: Chapman & Hall/CRC Press.

Systems Theory

Afton N. Kapuscinski
Psychology Department, Syracuse University, Syracuse, NY, USA

Definition

An approach to science that emphasizes unity and wholeness (Bertalanffy, 1968) and views factors that influence phenomena as mutually affecting each other at various levels of complexity.

Description

Systems theory, which became popular in the mid-twentieth century, developed in opposition to some of the philosophical assumptions that permeated the sciences across a variety of disciplines (Bertalanffy, 1968). Specifically, systems theory opposed the idea that knowledge about the universe is best obtained through a perspective rooted in notions of reductionism, mechanism, and objectivism (Midgley, 2000). Mechanism assumes that the universe and all phenomena contained within it can be likened to machines, composed of parts that operate in predictable, logical ways. Therefore, traditional scientific theory and methodology seeks to reduce complex phenomena to the functions of the smallest possible parts (reductionism), assuming that such analysis will yield complete understanding and ultimately control, in the area under study (Midgley, 2000). For example, a reductionistic approach to psychology may pose that all behavior can ultimately be traced to genetic endowment or chemical reactions occurring at the level of individual brain cells.

Systems theorists, however, argue that the search for simple, linear, cause and effect relationships between variables may lead to the neglect of developing more holistic, comprehensive conceptual models (Midgley, 2000), leaving the field of psychology impoverished in a couple of ways. First, mechanistic and reductionist approaches may struggle to explain emergent properties of systems, that is, those qualities of a system that cannot be explained merely as the sum of its individual parts (Bertalanffy, 1968), such as the human capacity for agency, creativity, or love. Even the concept of life itself cannot be explained by the activity of individual cells (Bertalanffy), but emerges from a complex system of interacting cells. Second, reductionistic scientific disciplines tend to exist in relative isolation from each other, without attempts at integration that may benefit the individual field, as well as facilitate broader societal improvement. Since system thinking views the boundaries between disciplines as somewhat unnecessary and artificial, a behavioral medicine investigator working from a systems perspective may draw from many sciences to obtain a more holistic understanding of a given topic. Therefore, in contrast to the "zooming in" approach of reductionism, systems thinking values panning outward to examine different levels of contextual factors that may be influencing and be influenced by a particular aspect of a system – including concepts across disciplines.

Ecological models of health behavior (see Sallis, Owen, & Fisher, 2008) are a good example of research grounded in systems thinking. These models assume multiple influences on health behaviors at various levels, with influences interacting across the levels (Sallis et al., 2008). For example, consider the problem of cardiovascular disease. A systemic approach to reducing the incidence of cardiovascular disease would aim to target a variety of factors that are interacting to determine one's degree of risk for developing the condition, including biological (e.g., hypertension, high cholesterol), psychological (e.g., hostility), social (interpersonal

relationships and support), and behavioral (e.g., diet, exercise, medication compliance) contributors. Such conceptualization in behavioral medicine is often referred to as taking a biopsychosocial approach, a form of systems thinking. A systemic model would also examine contextual factors that affect the individual, such as health care utilization or level of education, which may influence the individual's exposure to preventative medicine and information about healthy eating habits. Further, the approach would likely be mindful of broader social and economic concerns (e.g., unemployment rate, racial inequality) that may, in turn, influence one's access to these resources and thus would also be considered relevant to intervention efforts. These types of ecological models have been developed to further understanding of various health behaviors such as physical activity, tobacco control, and diabetes management (Sallis et al.).

Just as systems theory questions the validity of boundaries between disciplines, it also disputes the boundary between scientists and their objects of study (Midgley, 2000). Systems theorists reject the position that one can passively observe reality, standing separate from the object of study, but holds that scientists interact with these objects to actively create reality and interpret observations based on the lens through which they view them (Bertalanffy, 1968). If objectivism is not possible and scientists can never capture "reality," then theories and methodologies become "ways of seeing" that are full of value (Midgley, 2000). A critical implication of this shift in thinking is the possibility for theoretical pluralism, wherein investigators value contributions from various perspectives and types of research.

Influence on Psychotherapy

The principles inherent in the systems approach have influenced important shifts in the way psychotherapists understand the process of facilitating client change. First, there has been a shift away from the traditional psychoanalytic approach, which viewed the therapist as a relatively objective figure who could successfully remove his or her self from the process, providing the patient with a "blank screen" on which to project unconscious material for interpretation by the therapist. Modern psychoanalytic approaches, such as the object relations or two-person paradigms, assert that the therapist cannot avoid revealing the self and influencing the therapeutic encounter so as to create it along with the patient (Levenson, 1995). When viewing the therapeutic relationship as a system, the interaction between the therapist and patient, including the therapists' behaviors and feelings, becomes critically important and should be considered to aid case formulation and planning interventions. Since a change in any part of a system affects the whole of the system (Bertalanffy, 1968), the therapist's attempts to foster a healthy relationship can encourage improved interpersonal functioning in the patient (Levenson, 1995). Second, systems theory gave rise to another popular paradigm for understanding individual and family dysfunction, known as family systems theory. Family systems theory holds that an individual's or family's distress cannot be understood fully by looking at any one person in isolation, but must be viewed as the symptom of problematic patterns of interactions within the larger family structure (Smith-Acuña, 2010). Systems therapists therefore seek to improve the functioning of the family as a unit.

Cross-References

- ▶ Ecological Models: Application to Physical Activity
- ▶ Family Systems Theory

References and Readings

Breunlin, D. C., Schwartz, R. C., & Mac Kune-Karrer, B. (2001). *Metaframeworks: Transcending the models of family therapy*. San Francisco: John Wiley & Sons.

Levenson, H. (1995). *Time-limited dynamic psychotherapy: A guide to clinical practice*. New York: Basic Books.

Midgley, G. (2000). *Systemic intervention: Philosophy, methodology and practice*. New York: Plenum.

Sallis, J. F., Owen, N., & Fisher, E. B. (2008). Ecological models of health behavior. In K. Glanz,

B. K. Rimer, & K. Viswanath (Eds.), *Health behavior and health education: Theory, research and practice* (pp. 465–485). San Francisco: John Wiley & Sons.
Smith-Acuña, S. (2010). *Systems theory in action: Applications to individual, couple, and family therapy*. Hoboken, NJ: John Wiley & Sons.
von Bertalanffy, L. (1968). *General systems theory*. London: Penguin.

Systolic Blood Pressure (SBP)

Annie T. Ginty
School of Sport and Exercise Sciences, The University of Birmingham, Edgbaston, Birmingham, UK

Synonyms

Blood pressure

Definition

Systolic blood pressure is the force exerted by blood on arterial walls during ventricular contraction measured in millimeters of mercury (see Tortora & Grabowski, 1996). It is the highest pressure measured; normal range for systolic blood pressure is <120 mmHg.

Cross-References

▶ Blood Pressure, Measurement of
▶ Diastolic Blood Pressure (DBP)

References and Readings

Tortora, G. J., & Grabowski, S. R. (1996). *Principles of anatomy and physiology* (8th ed.). New York: Harper Collins College.

T

Tachycardia

Lois Jane Heller
Department of Biomedical Sciences, University of Minnesota Medical School – Duluth, Duluth, MN, USA

Definition

The word "tachycardia" means rapid heart rate. This condition is more precisely defined as a heart rate that is above the age-adjusted range of normal heart rates (see Table 1). Children normally have higher heart rates than adults and can tolerate rapid heart rates more easily.

Description

Normal Determinants of Heart Rate

Heart rate is normally established by the rate of spontaneous generation of an electrical signal (action potential) by "pacemaker" cells located in the sinoatrial (SA) node of the heart (in the wall of the right atrium near the entry of the superior vena cava) (Mohrman & Heller, 2011). These electrical signals are normally generated at a rate of 60–100 beats per min in a human adult. They are propagated from the SA node through the atrial and ventricular muscle cells in a set pathway that stimulates contraction in a pattern that optimizes pumping of blood from the heart. Most other cardiac muscle cells have the potential to act as pacemakers, but the SA nodal cells drive the heart at a slightly faster rate than any of these other "latent" pacemakers.

The normal fluctuations in heart rate that occur in response to normal changes in the body's metabolic demands are accomplished by altering autonomic neural influences on the SA nodal pacemaker cells from the parasympathetic and sympathetic nervous system. Increased sympathetic activity causes an increase in heart rate whereas increase in parasympathetic activity causes a decrease in heart rate.

Tachycardia, Table 1 Age-associated upper limit to normal human heart rates (Adapted from Greene, 1991)

Less than 1 year	~170 bpm
1–2 years	~150 bpm
3–5 years	~135 bpm
6–12 years	~130 bpm
12–15 years	~120 bpm
15 years through adulthood	~100 bpm

Symptoms of Tachycardia

A person with significant tachycardia may or may not be aware that their heart rate is fast without actually measuring their pulse rate (Valentin, O'Rourke, Walsh, & Poole-Wilson, 2008). However, tachycardia often results in a feeling of light-headedness, dizziness, tunnel vision, or fainting. Other symptoms may include muscle weakness, nervousness, sweating, pallor, or a feeling of fullness in the chest. The cause of

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

many of these symptoms is often related to a decrease in arterial blood pressure and therefore blood flow to the brain and other tissues.

Physiological Cause of the Symptoms

The amount of blood that the heart pumps in a minute is called the cardiac output (Mohrman & Heller, 2011). This is determined by the volume of blood ejected in each beat *(stroke volume)* and the number of beats per minute *(heart rate)*:

$$\text{Cardiac output} = \text{stroke volume} \times \text{heart rate}$$

One might predict from this equation that the cardiac output would increase whenever heart rate increased. This is true over the normal range of heart rates. However, when the heart rate exceeds this normal range, the cardiac output may actually fall. There are three primary reasons for this: (1) There is insufficient time between beats to allow the heart to fill adequately. (2) The coronary circulation to the cardiac muscle may be compromised by the compressive forces in the ventricular wall associated with each individual beat. (3) The energy requirements for the heart increase enormously and may not be met by the compromised coronary blood flow.

Physiological Causes of Tachycardia

1. *Atrial Tachycardia or Supraventricular Tachycardia* (i.e., associated with a narrow QRS complex on an ECG) (Valentin, O'Rourke, Walsh, & Poole-Wilson, 2008). This condition is more common than ventricular tachycardia (see below) and can often be successfully treated. It is often uncomfortable and alarming to the individual, but does not usually portend immediate, possibly fatal consequences.
   (a) *Sinus Tachycardia* – Elevated sympathetic neural activity or an increase in circulating catecholamines stimulates the normal pacemaker cells in the SA node to fire at a very rapid rate and this electrical signal is then carried via normal pathways through the entire heart.
   (b) *Ectopic Atrial Pacemakers* – Atrial cells that are not part of the SA node can sometimes become irritable and generate electrical signals that spread throughout the cardiac tissue.
   (c) *AV Nodal Reentrant Tachycardia* – This is a conduction defect rather than a pacemaker defect. In this case, a rapid heart rate may result from an abnormal portion of conduction pathway usually found in the AV node in which the electrical signal circles back on itself to rapidly re-excite the downstream tissue.
   (d) *Atrial Flutter* – An ectopic pacemaker or reentrant pathway in the atria evokes in a very rapid atrial rate such that AV node fails to conduct every signal. The atria may beat three or more times for each ventricular beat. The ventricular rate may be faster than normal or within the normal range but the atrial rate is faster.
   (e) *Atrial Fibrillation* – The atrial conduction pathways become disorganized and the normal synchronized excitation of the atrial tissue is disrupted. Electrical signals are initiated and conducted in bizarre patterns, resulting in unpredictable intermittent conduction through the AV node. This results in an irregular ventricular rhythm that may, on the average, be faster than normal (tachycardia), normal, or slower than normal (bradycardia).
2. *Ventricular Tachycardias* (i.e., associated with wide QRS complex on an ECG). This is a more serious condition than atrial tachycardia and needs immediate attention. The heart is still operating as a pump but the possibility of a sudden deterioration to ventricular fibrillation is high.
   (a) *Ectopic Ventricular Pacemakers* – Ventricular cells can sometimes become irritable and generate electrical signals that spread throughout the cardiac tissue. This may occur if blood flow to a portion of the ventricular wall is inadequate and the tissue becomes ischemic.
   (b) Ventricular Reentrant Pathways – In this case, a rapid heart rate may result from

abnormal conduction of the electrical signal through a small portion of ventricular muscle such that the electrical signal circles back on itself to rapidly re-excite the cardiac tissue. Because all cardiac muscle cells are electrically connected, this rapidly firing small group of cells can drive the ventricles at a fast rate.

Cross-References

- ▶ Arrhythmia
- ▶ Maximal Exercise Heart Rate

References and Readings

Greene, M. G. (Ed.). (1991). *The Harriet Lane handbook* (12th ed.). St Louis, MO: Mosby Yearbook.
Mohrman, D. E., & Heller, L. J. (2011). *Cardiovascular physiology* (Lange series 7th ed.). New York: McGraw-Hill.
Valentin, F., O'Rourke, R. A., Walsh, R. A., & Poole-Wilson, P. (2008). *Hurst's the heart* (12th ed.). New York: McGraw-Hill.

Tailored Communications

Celette Sugg Skinner
Clinical Sciences, The University of Texas Southwestern Medical Center at Dallas Harold C. Simmons Cancer Center, Dallas, TX, USA

Synonyms

Tailored health behavior change interventions

Definition

"Tailored communications are any combination of information intended to reach one specific person, based on characteristics unique to that person, related to the outcome of interests, and derived from an individual assessment" (Kreuter & Farrell, 2000).

Description

Background

The field of behavioral medicine studies how behaviors affect health and medical conditions, as well as how behaviors can be changed. Good behavior-change interventions are, of course, guided by strong health behavior theories that elucidate factors (variables) affecting individuals' behaviors. One of most basic of these, the Health Belief Model (HBM), was developed in the 1950s when the US public health service consulted with psychologists to understand why people were not availing themselves of free tuberculosis screenings. This simple model delineates three major factors (or theoretical constructs) affecting whether people take a health action. They must feel susceptible to a health threat serious enough to warrant attempts to reduce the risk, believe that changing their behavior would reduce their risk, and overcome perceived barriers to behavior change (Champion & Skinner, 2008). Individuals can vary widely on these beliefs. One's threat perception may be low whereas another may perceive the threat but not believe the behavior change would have benefit for lowering it. Among those who perceive barriers to behavior change, specific types of barriers may vary widely. For example, not having a ride to a screening site is very different from not being able to afford screening or of being afraid the screen would find a problem.

Message Customization

Face-to-face communications are usually customized for different people. For example, nurses seeking to facilitate medication adherence communicate with different patients differently, depending on factors influencing *that persons'* behavior. The message differs when talking with someone who cannot remember to take the pills versus someone avoiding the medication due to side effects. But only recently did this kind of message customization become possible in mass-produced media such as print and video. From the 1950s through the 1980s, mass-media communications such as brochures and videos were developed to address an array of variables,

with the hope that at least some of the message components would be relevant to most audience members.

Mass-Produced Tailored Communications

In the 1980s, the rise of micro-computing capabilities opened possibilities for mass-producing communications that retained advantages of customized face-to-face interactions. These "tailored communications" are reminiscent of tailor-made clothing that is based on numerous measurements taken by the tailor before beginning his work. Tailored communications begin with measures of people's behavior-influencing factors such as the HBM variables of perceived risks and beliefs about the behavior's benefits and barriers. The "fabric" of tailored communications is distinct text, audio, or graphic components that are "sewn" together to fit the measurements of a particular message recipient.

In their 2000 book, *Tailored Health Messages; Customizing Communication with Computer Technology* Kreuter, Farrell et al. provide this elegant definition of tailored communications: "any combination of information intended to reach one specific person, based on characteristics unique to that person, related to the outcome of interests, and derived from an individual assessment." Continuing the clothing analogy, this definition distinguishes between tailored and targeted communications, as follows. *Tailored communications* – and tailored clothing – are intended to fit one individual. *Targeted communications* – and off-the-rack clothing – are intended to fit any one of a group sharing some common characteristics (e.g., men who wear a size 40 long, like pinstripes, and shop in a certain price range). Targeted messages are usually directed to a particular demographic group (i.e., African American church members) and address factors known to be important for *many* members of that group. In contrast, tailored communications are based on individual-level assessment of theory-based behavior-influencing variables, with a unique combination of messages assembled for each individual within the group (Kreuter & Skinner, 2000).

The original rational for tailoring was that tailored communications would be more noticeable and compelling and less burdensome because they are streamlined to only include content relevant to the recipient. According to Petty and Cacioppo (Petty, Cacioppo, & Goldman, 1981), the more personal involvement with the message, the more careful consideration (i.e., "central processing") and elaboration on the message which, in turn, increases likelihood of attitude and behavior change.

Early Trials of Tailored Print Communications

Several initial randomized trials compared printed communications that were v. were not tailored on theory-derived behavior-influencing variables (Skinner, Campbell, Rimer, Curry, & Prochaska, 1999). Three of these, targeting mammography screening (Skinner, Strecher, & Hospers, 1994), smoking cessation (Strecher et al., 1994), and dietary change (Campbell et al., 1994), were conducted among primary-care patients who completed telephone surveys and were randomly assigned to receive tailored or non-tailored printed newsletters. In addition to age, race, and risk factors, messages in the tailored letters directly addressed variables such as the recipients' perceived risk, benefits and barriers, their stage of behavior adoption, characteristics such as age, race, and risk factors and, for smokers, causal attributions for past failed quite attempts. In each of these three studies, the non-tailored letter version addressed a number of factors that had been shown to influence people's behaviors, in general. For example, the non-tailored mammography letter was adapted from a letter from US Surgeon General's office mailed to women who requested information about breast cancer screening. Because these three trials were designed to compare tailored versus non-tailored *content*, the tailored letters did not include statements such as "the information was prepared especially for you," as have later studies. Tailored and non-tailored letters looked very much alike. They were printed in black and white on two-column 8½ × 11 paper and included a head-and-shoulders line drawing. Therefore, it is remarkable that recipients of the tailored letters

were more likely to report remembering and reading their letters. Receipt of a tailored letter was also associated with behavior change (Campbell et al., 1994), at least among important subgroups (Skinner et al., 1994; Strecher et al., 1994). Indeed, of the seven initial comparisons of tailored versus. non-tailored print communications, six (Skinner et al., 1999) found more behavior change among tailored recipients.

Movement Toward Different Comparisons

Given these impressive results from studies comparing tailored versus similar non-tailored print, and with tailoring technology expanding rapidly, tailoring researchers moved on to different comparisons. Studies tested tailored communications as an adjunct to other intervention components such as self-help smoking manuals, and compared tailored messages delivered through different media, such as tailored print versus tailored telephone counseling (Champion et al., 2007; Rimer et al., 2002), tailored on different types of variables (Kreuter et al., 2005), and with and without booster doses (Skinner, 2006). Even within the tailored print medium, researchers moved beyond the newsletter format to tailored booklets and magazines with tailored features such as cartoons and advice columns (Rimer et al., 2002; Kreuter et al., 2005). Because there were so many kinds of comparisons of different tailoring approaches and for different target behaviors, data do not exist from head-to-head comparisons of every feature (e.g., more vs. less content, fewer vs. more graphics). As reported in a 1998 review of the "first generation" of tailored communications, these various comparisons showed mixed results, and "when (tailored print communications) are only one component of a complex intervention strategy without a factorial design, it is more difficult to isolate their relative contribution to the overall intervention effect" (Skinner et al., 1999, p 296). Nonetheless, this initial review and several others published subsequently (Kreuter & Farrell, 2000; Kroeze, Werkman, & Brug, 2006; Revere & Dunbar, 2001; Rimer & Glassman, 1999) reported that tailoring seems to "work" both for attracting and retaining attention and for facilitating behavior change. Further, delivering tailored messages via print plus another medium such as telephone generally is more effective than tailored print alone.

Interactive "Real-Time" Tailoring

Original print-tailored interventions collected questionnaire data and then used that entire "batch" of data to put together specific messages that, depending on algorithms, were selected from a library of potential messages. More recently, CD-ROMs, DVDs, and the internet have allowed for interactive tailoring based on data collected during program use rather than in questionnaire form before the intervention is delivered. For example, a DVD or CD-ROM can ask a question about barriers to behavior change, then immediately show videos, selected from a tailored video library, that address the specific barriers named by the user (Skinner et al., 2011). Some web-based programs provide tailored content based on predetermined algorithms, others allow users to "self-tailor" by selecting any content of interest to them. As described in Lustria, Cortese, Noar, & Glueckauf, 2009 review of computer-tailored health interventions delivered over the web, these interventions "have involved a great diversity of features and formats," and "further outcome research is needed to enhance our understanding of how and under what conditions computer-tailoring leads to positive health outcomes in online behavioral interventions" (Lustria et al., 2009, p. 156). Therefore, as with the print-tailored communications, it is difficult to draw conclusions across studies.

Conclusions and Challenges

One problem in understanding findings and implications of tailored communication intervention studies is that journals often severely limit the amount of space for intervention descriptions. As a result, we have many statistics associated with the intervention outcomes, but we know little about the interventions themselves. In other words, we may learn that "it worked" without knowing exactly what "it" was or with what "it" was compared. Tailoring researchers have,

therefore, recently called for new reporting standards through which intervention studies will report similar descriptions and metrics, which should help in evaluation and dissemination of best practices in tailored interventions (Harrington & Noar, 2011).

In summary, communication interventions that are tailored to include theory-based messages relevant to individual recipients, based on data collected from them, are generally better than non-tailored communications in drawing the attention of message recipients and facilitating their health behavior change. However, questions still remain about optimal amounts and types of tailoring for different behavioral targets and through different media.

Cross-References

- ▶ Health Behavior Change
- ▶ Health Behaviors
- ▶ Health Beliefs/Health Belief Model
- ▶ Health Communication
- ▶ Health Risk (Behavior)
- ▶ Stages-of-Change Model

References and Readings

Campbell, M. K., DeVellis, B. M., Strecher, V. J., Ammerman, A. S., DeVellis, R. F., & Sandler, R. S. (1994). Improving dietary behavior: The effectiveness of tailored messages in primary care settings. *American Journal of Public Health, 84*, 783–787.

Champion, V. L., & Skinner, C. S. (2008). The health belief model. In K. Glanz, B. K. Rimer, & K. Viswanath (Eds.), *Health behavior and health education: Theory, research and practice* (4th ed., pp. 45–65). San Francisco: Jossey-Bass.

Champion, V., Skinner, C. S., Hui, S., Monahan, P., Juliar, B., Daggy, J., et al. (2007). The effect of telephone versus print tailoring for mammography adherence. *Patient Education and Counseling, 65*, 416–423.

Harrington, N. G., & Noar, S. M. (2011). Reporting standards for studies of tailored interventions. *Health Education Research*, *27*(2), 331–342.

Kreuter, M. W., & Farrell, D. (2000). *Tailoring health messages: Customizing communication with computer technology*. Mahwah, NJ: Lawrence Erlbaum.

Kreuter, M. W., & Skinner, C. S. (2000). Tailoring: What's in a name? *Health Education Research, 15*, 1–4.

Kreuter, M. W., Skinner, C. S., Holt, C. L., Clark, E. M., Haire-Joshu, D., Fu, Q., et al. (2005). Cultural tailoring for mammography and fruit and vegetable intake among low-income African-American women in urban public health centers. *Preventive Medicine, 41*, 53–62.

Kroeze, W., Werkman, A., & Brug, J. (2006). A systematic review of randomized trials on the effectiveness of computer-tailored education on physical activity and dietary behaviors. *Annals of Behavioral Medicine, 31*, 205–223.

Lustria, M. L., Cortese, J., Noar, S. M., & Glueckauf, R. L. (2009). Computer-tailored health interventions delivered over the Web: Review and analysis of key components. *Patient Education and Counseling, 74*, 156–173.

Petty, R. E., Cacioppo, J. R., & Goldman, R. (1981). Personal involvement as a determinant of argument-based persuasion. *Journal of Personality and Social Psychology, 41*, 847–855.

Revere, D., & Dunbar, P. J. (2001). Review of computer-generated outpatient health behavior interventions: Clinical encounters "in absentia". *Journal of the American Medical Informatics Association, 8*, 62–79.

Rimer, B. K., & Glassman, B. (1999). Is there a use for tailored print communications in cancer risk communication? *Journal of the National Cancer Institute Monographs, 25*, 140–148.

Rimer, B. K., Halabi, S., Skinner, C. S., Lipkus, I. M., Strigo, T. S., Kaplan, E. B., et al. (2002). Effects of a mammography decision-making intervention at 12 and 24 months. *American Journal of Preventive Medicine, 22*, 247–257.

Skinner, C. S. (2006). Tailored interventions for screening mammography: When is a booster dose important? *Patient Education and Counseling, 65*, 87–94.

Skinner, C. S., Buchanan, A., Champion, V., Monahan, P., Rawl, S., Springston, J., et al. (2011). Process outcomes from a randomized controlled trial comparing tailored mammography interventions delivered via telephone vs. DVD. *Patient Education & Counseling, 85*, 308–312.

Skinner, C. S., Campbell, M. K., Rimer, B. K., Curry, S., & Prochaska, J. O. (1999). How effective is tailored print communication? *Annals of Behavioral Medicine, 21*, 290–298.

Skinner, C. S., Strecher, V. J., & Hospers, H. (1994). Physicians' recommendations for mammography: Do tailored messages make a difference? *American Journal of Public Health, 84*, 43–49.

Strecher, V. J., Kreuter, M., Den Boer, D. J., Kobrin, S., Hospers, H. J., & Skinner, C. S. (1994). The effects of computer-tailored smoking cessation messages in family practice settings. *Journal of Family Practice, 39*, 262–270.

Tailored Health Behavior Change Interventions

▶ Tailored Communications

Teens

▶ Williams LifeSkills Program

Telehealth

▶ eHealth and Behavioral Intervention Technologies

Telemedicine

▶ eHealth and Behavioral Intervention Technologies

Telencephalon

▶ Brain, Cortex

Telephone Coaching

▶ Williams LifeSkills Program

Telomere and Telomerase

A. Janet Tomiyama[1] and Elissa S. Epel[2]
[1]Rutgers University, NJ, USA
[2]University of California, San Francisco, CA, USA

Definition

Telomeres are noncoding repeat DNA sequences (consisting of TTAGGG) that cap the ends of eukaryotic chromosomes. Telomerase is an enzyme that adds basepairs to telomeres.

Description

Like the plastic tips that protect shoelaces from unravelling, telomeres protect DNA material. When cells divide, the enzymes that replicate the chromosomes are unable to do so fully. The main purpose of telomeres is to form a buffer so that genetic material is not lost in this process. With each cell division, telomeres can shorten, and when telomeres become critically short, a cell undergoes senescence (cell arrest). Telomeres also function to protect chromosomes from genomic instability, end-to-end chromosome fusion, less efficient cell division, loss of ability for cell replenishment, and apoptosis or cell death (Blackburn, 2000).

Because of these important functions, telomeres are related to a number of health outcomes and disease states. Shorter telomere length is associated with chronic diseases of aging such as cardiovascular disease, cancer development, and Alzheimer's disease, and is related to earlier mortality. Telomere length is also thought to be an indicator of biological, rather than chronological, age (Epel, 2009).

Telomere length is associated with a number of psychosocial factors. For example, depression, higher perceived stress, longer stress duration, caregiving, lower socioeconomic status, pessimism, childhood adversity, and lower subjective well-being are correlated with shorter telomere length. Health behaviors such as smoking, lack of physical activity, and repeated dieting as well as altered metabolic states such as obesity and insulin resistance are also linked with shorter telomere length (Lin, Epel, & Blackburn, 2009).

Telomerase is the enzyme that adds base pairs back onto telomeres. This serves to protect telomeres from shortening. Recent work suggests telomerase may have future applications for antiaging therapies as evidenced by telomerase knockout mice displaying reversed aging with telomerase treatment (Jaskelioff et al., 2010).

Telomerase, like telomeres, appears to be responsive to psychological states, and may

serve to protect cells under stress. Exposure to acute stress (Epel et al., 2010) as well as states of chronic psychosocial adversity are associated with high telomerase. Because telomerase levels change more dynamically than telomere length, psychosocial interventions such as meditation (Jacobs et al., 2010) and comprehensive lifestyle changes (Ornish et al., 2008) have been tested in preliminary studies as a potential treatment to increase telomerase, with promising results.

Cross-References

- ▶ Aging
- ▶ Alzheimer's Disease
- ▶ Cardiovascular Disease
- ▶ Chromosomes
- ▶ Depression
- ▶ Insulin Resistance
- ▶ Lifestyle changes
- ▶ Meditation
- ▶ Obesity
- ▶ Pessimism
- ▶ Physical Activity
- ▶ Smoking and Health
- ▶ Socioeconomic Status (SES)
- ▶ Subjective Well-Being

References and Readings

Blackburn, E. H. (2000). Telomere states and cell fates. *Nature, 408*, 53–56.

Epel, E. (2009). Telomeres in a life-span perspective: A new "psychobiomarker?". *Current Directions in Psychological Science, 18*, 6–10.

Epel, E. S., Lin, J., Dhabhar, F. S., Wolkowitz, O. M., Puterman, E., Karan, L., et al. (2010). Dynamics of telomerase activity in response to acute psychological stress. *Brain, Behavior, and Immunity, 24*, 531–539.

Jacobs, T. L., Epel, E. S., Lin, J., Blackburn, E. H., Wolkowitz, O. M., Bridwell, D. A., et al. (2010). Intensive meditation training, immune cell telomerase activity, and psychological mediators. *Psychoneuroendocrinology, 36*(5), 664–681.

Jaskelioff, M., Muller, F. L., Paik, J., Thomas, E., Jian, S., Adams, A. C., et al. (2010). Telomerase reactivation reverses tissue degeneration in aged telomerase-deficient mice. *Nature, 469*, 102–106.

Lin, J., Epel, E., & Blackburn, E. (2009). Telomeres, telomerase, stress, and aging. In G. G. Benton & J. T. Cacioppo (Eds.), *Handbook of neuroscience for the behavioural sciences*. New York: Wiley.

Ornish, D., Lin, J., Daubenmier, J., Weidner, G., Epel, E., Kemp, C., et al. (2008). Increased telomerase activity and comprehensive lifestyle changes: A pilot study. *The Lancet Oncology, 9*, 1048–1057.

Temporal

- ▶ Brain, Cortex

Temporal Self-Regulation Theory

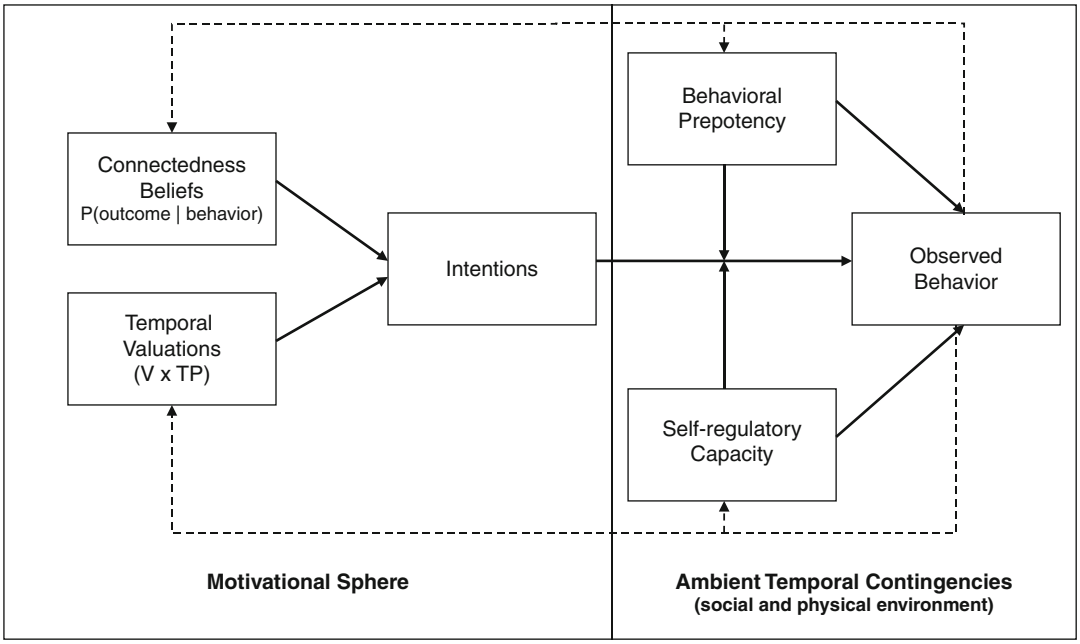
Peter A. Hall
Faculty of Applied Health Sciences, University of Waterloo, Waterloo, ON, Canada

Synonyms

Dual process models of health behavior; Dual systems models

Definition

Temporal self-regulation theory (TST; Hall & Fong, 2007; Fig. 1) is a theoretical framework for explaining individual health behavior. TST posits that health behavior is proximally determined by three factors: *intention strength*, *behavioral prepotency*, and *self-regulatory capacity*. The latter two constructs are theorized to have direct influences on behavior and also to moderate the intention-behavior link. Specifically, intentions are proposed to have a stronger influence on behavioral performance in the presence of stronger self-regulatory capacity and/or when the behavioral prepotency is weak. Also included in the model is consideration of ecological context in the form of contingencies supplied to the behavior by the social and physical environment



Temporal Self-Regulation Theory, Fig. 1 Arrows between Behavioral Prepotency and Self-regulatory Capacity to the Intentions-Behavior arrow implies moderation; V = value; TP = perceived temporal proximity. Broken arrows denote weaker (i.e., secondary) hypothesized effects. Adapted from (Hall & Fong 2007).

(i.e., *ambient temporal contingencies*). In the TST model, *intention strength* is a function of anticipated connections between one's behavior and salient outcomes (i.e., *connectedness beliefs*); the valence of the latter can range from negative (i.e., costs) to positive (i.e., benefits). These beliefs are weighted by temporal proximity (i.e., *temporal valuations*). For example, the perceived self-relevant contingencies for making a healthy dietary choice might include eventual benefits (e.g., improved appearance, better health status), but more temporally proximal – therefore more heavily influential-immediate costs (e.g., inconvenience, monetary costs, time costs). The sum of the perceived contingencies weighted by their respective temporal proximities should determine intention strength to make a healthy dietary choice, according to the TST model.

Description

The aim of the TST is to explain variability in health behavior in a manner that is sensitive to biological capacities for self-control, motivation level, and the ecological context in which the behavior takes place. Given the complexities of the model and the fact that it crosses many levels of analysis (from biological to social to ecological), it is expected that the model is not testable in its entirety. Rather, individual components of the model may be tested individually or in relation to each other (e.g., hypothesized moderating effects).

The TST model was initially offered as an improvement over traditional models of individual health behavior which posited that behavior was most proximally determined by social cognitive variables, without direct or indirect links to neurobiological resources. While TST preserves the central role of intention strength, it adds two important moderating and direct effects on health behavior performance: (1) *self-regulatory capacity* (SRC) and (2) *behavioral prepotency* (BPP).

SRC is composed primarily of executive control resources and therefore ascribed to operation of the prefrontal cortex and associated neural

systems implicated in the neurobiology of self-control (Miller & Cohen, 2001). BPP is the psychological inertia of a given behavior, by virtue of frequent past performance in similar contexts, or via the presence of strong cues (which may be social or visceral in nature) to perform the behavior at a given time. The combination of SRC and BPP determines the likelihood that intentions will be translated into behavior, and each also has direct influences on behavior itself regardless of intention.

Additional components of TST that differentiate it from its predecessors are (1) an explicit focus on temporal proximity of behavioral contingencies as determinants of their relative potency and (2) a consideration of ecological factors as causal agents in health behavior performance. These two components are conceptually linked, as ecological contexts often determine what kinds of consequences (positive, neutral, or negative) are experienced following performance of a behavior, as well as the relative proximity of those consequences (immediate vs. long-term).

The primary contribution of the TST model has been to provide some basis for understanding the possibility of brain-behavior relationships as being partial determinants of health behavior trajectories, and to provide an interface for individual models of health behavior with ecological and social-level determinants of behavior. Given that intention strength (Armitage & Connor 2001) and behavioral prepotency (Ouellette & Wood, 1998) are among the most well-established determinants of behavior in the extant research literature, the construct within TST that has required the most empirical justification is the inclusion of biologically based SRC.

Though few studies of SRC as a determinant of health behavior existed prior to the introduction of the TST model, the early findings have been promising. In recent studies, it has been found that SRC predicts health behavior patterns directly and exerts an additional moderating influence on health behavior performance, both of which are hypothesized by TST (Hall, 2012). In addition, evidence has emerged to indicate that individual differences in SRC may be selectively responsible for the previously demonstrated association between IQ and longevity, and also predicts survival time among those living with chronic illnesses that carry significant self-care demands. Finally, recent research shows that SRC interacts with intentions such that intentions predict various health behaviors, only when SRC is high, not low.

Together these findings provide support for the inclusion of biologically based SRC in contemporary models of health behavior. It is expected that the next generation of research involving TST will include experimental designs that can isolate causal relations between SRC and health behavior performance (both directly and via its intention-moderating effect) in the laboratory setting, and test the efficacy of SRC-augmenting activities in the interventional context. Among the most promising interventions for augmenting SRC is the use of aerobic training, which has been shown to enhance structural and functional components of the brain regions that are known to support self-regulatory processes and self-control (McAuley & Hillman, 2012). One final novelty of this model is its use of various types of assessment methods (self-report and neuropsychological or reaction-time tests), which may overcome problems of shared-method variance and enhance its predictive validity.

Cross-References

- ▶ Executive Function
- ▶ Self-Regulatory Capacity
- ▶ Theory

References and Readings

Armitage, C. J., & Connor, M. (2001). Efficacy of the theory of planned behaviour: A meta-analytic review. *British Journal of Social Psychology, 40*, 471–499.

Hall, P. A. (2012). Temporal self-regulation theory: Integrating biological, psychological, and ecological determinants of health behavior performance.

In P. Hall (Ed.), *Social neuroscience and public health*. New York: Springer.
Hall, P. A., & Fong, G. T. (2007). Temporal self-regulation theory: A model for individual health behavior. *Health Psychology Review, 1*, 6–52.
McAuley, E., & Hillman, C. K. (2012). Exercise and enhancement of cognitive function. In P. Hall (Ed.), *Social neuroscience and public health*. New York: Springer.
Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience, 24*, 167–202.
Ouellette, J. A., & Wood, W. (1998). Habit and intention in everyday life: The multiple processes by which past behavior predicts future behavior. *Psychological Bulletin, 24*, 54–74.

Tension

▶ Affect Arousal
▶ Asthma and Stress

Terminal Care

▶ End-of-Life Care
▶ Palliative Care

Tertiary Care

▶ Clinical Settings

Testicular Cancer

▶ Cancer, Testicular

Testicular Neoplasms

▶ Cancer, Testicular

Testoid

▶ Androgen

Thanatophobia

▶ Death Anxiety

Thanksgiving

▶ Prayer

Theories of Behavior Change

▶ Intervention Theories

Theory

Julia Allan
School of Medicine and Dentistry, University of Aberdeen, Foresterhill, Aberdeen, Scotland, UK

Synonyms

Conjecture; Model

Definition

A theory is a coherent set of statements or ideas used to organize, generalize, explain, and predict phenomena. Theories are based on observations, experimentation, and abstract reasoning, and play a fundamental role in scientific research.

Theories must (a) successfully describe and explain existing observations, (b) make predictions about future observations, and (c) be falsifiable, that is, they must be refutable by some conceivable event or observation.

While not directly verifiable, theories gain support as empirical evidence accumulates in their favor, particularly if such evidence results from "risky" predictions where the outcome could conceivably have been different (Popper, 1963/2004). An accumulation of contradictory empirical evidence results in the theory being abandoned, modified, or superseded by a new theory. This dynamic process of testing, evaluation, and change allows research to move forwards toward the "truth." Theories provide a shared language for researchers to use, enabling the development of a cumulative science. The most useful theories are those that make specific and relevant predictions that can be tested in a straightforward manner.

Prominent theories in behavioral medicine cover a range of domains and include the Transactional Theory of Stress and Coping (Lazarus & Folkman, 1984), Type D Personality (Denollet et al., 1996), the Theory of Planned Behavior (Ajzen, 1991), and the Common Sense Model of Self-Regulation of Health and Illness (Leventhal, Diefenbach, & Leventhal, 1992).

Cross-References

- ▶ Causal Diagrams
- ▶ Hypothesis Testing

References and Readings

Ajzen, I. (1991). The theory of planned behaviour. *Organizational Behavior and Human Decision Processes, 50*, 179–211.

Denollet, J., Sys, S. U., Stroobant, N., Rombouts, H., Gillebert, T. C., & Brutsaert, D. L. (1996). Personality as independent predictor of long term mortality in patients with coronary heart disease. *Lancet, 347*, 417–421.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal and coping*. New York: Springer.

Leventhal, H., Diefenbach, M., & Leventhal, E. A. (1992). Illness cognition: Using common sense to understand treatment adherence and affect cognition interactions. *Cognitive Therapy and Research, 16*, 143–163.

Popper, K. R. (1963/2004). *Conjectures and refutations: The growth of scientific knowledge*. London: Routledge.

Theory of Planned Behavior

- ▶ Theory of Reasoned Action

Theory of Reasoned Action

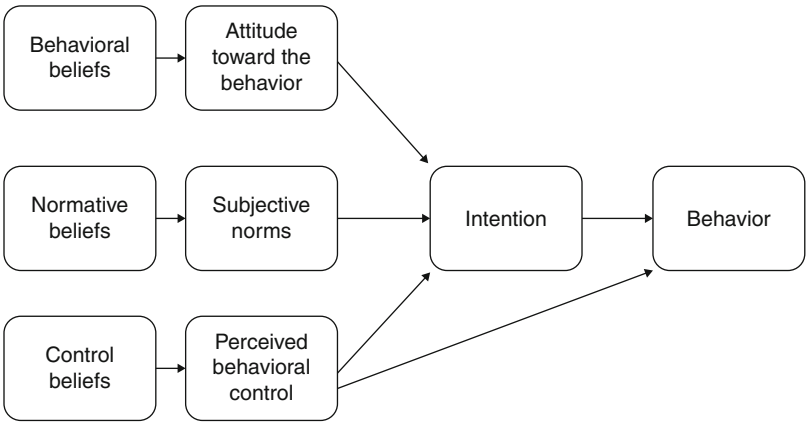
Lara LaCaille
Department of Psychology, University of Minnesota Duluth, Duluth, MN, USA

Definition

The Theory of Reasoned Action (TRA; Ajzen & Fishbein, 1980; Fishbein & Ajzen, 1975) and its extension, the Theory of Planned Behavior (TPB; Ajzen, 1985, 1991), are cognitive theories that offer a conceptual framework for understanding human behavior in specific contexts. In particular, the theory of planned behavior has been widely used to assist in the prediction and explanation of several health behaviors.

Description

According to the initial Theory of Reasoned Action, an *intention* to engage in a certain behavior is considered the best predictor of whether or not a person actually engages in that behavior. Intentions, in turn, are predicted by *attitudes* and *subjective norms*. That is, the more positively a person regards a certain behavior or action and the more they perceive the behavior as being important to their friends, family, or society, the more likely they are to form intentions to engage in the behavior. Azjen, however, noted the importance of a behavior being under volitional control in both forming intentions and engaging in the actual behavior. Therefore, he added *perceived behavioral control* to the model, which is now known as the Theory of Planned Behavior. See Fig. 1.



Theory of Reasoned Action, Fig. 1 Theory of planned behavior

Description of the Theoretical Factors

Behavioral beliefs and attitudes: A person first forms beliefs about the outcomes of a given behavior (e.g., "If I exercise, I will improve my health, lose weight, and be more attractive"). These beliefs contribute to his or her attitude or evaluation of the outcome of the behavior (e.g., "Being healthy and attractive is good/valuable"). The more favorable the attitude, the stronger the intention.

Normative beliefs and subjective norms: Normative beliefs refer to a person's perception about the expectations of important others (e.g., "My friends think I should exercise"). These beliefs contribute to the perception of social pressure and contribute to motivation to comply (e.g., "I feel pressured to exercise and I want to fit in with my friends"). The more powerful the perceived norm/pressure, the stronger the intention.

Control beliefs and perceived behavioral control: A person forms beliefs about the factors that may facilitate or be barriers to engaging in the specific behavior (e.g., "I have time before work, I have access to a gym, and I am physically able to exercise"). These beliefs lead to a perception of behavioral control or sense of ease/difficulty in engaging in the behavior (e.g., "I will be able to exercise"). Although many researchers have used the terms self-efficacy and perceived behavioral control interchangeably, including Ajzen (1991), these concepts are not quite synonymous. Whereas self-efficacy reflects individuals' beliefs about their competence or internal control, perceived behavioral control also incorporates other external/environmental factors (e.g., time, resources, social support). The greater the perceived behavioral control, the stronger the intention and the greater the likelihood of engaging in the behavior.

Intention: Intentions refer to peoples' plan of action and represent their expressed motivation to perform the behavior.

Evaluation of the Model

Meta-analytic reviews have supported the predictive efficacy of the TPB model for both behavioral intentions and behaviors (Armitage & Conner, 2001; Godin & Kok, 1996; Sheeran, 2002). The theory typically accounts for about 40–50% of the variance in intentions and 20–40% of the variance in behavior. The relative importance of each of the three factors (attitudes, norms, perceived behavioral control) varies across behaviors and situations. Subjective norms are usually the weakest predictor, though this may reflect measurement issues or people's denial of the effects of social pressures. With regard to health behaviors, the model is better at predicting some behaviors (exercise, condom use, drug use, and cigarette smoking) than others

(weight loss and dietary behavior, clinical and screening behavior, oral hygiene; see Godin & Kok). Intentions are usually more potent than perceived behavioral control in predicting health behaviors, suggesting that such behaviors are largely driven by personal motivation.

Although TPB is widely used and offers one of the most robust set of predicators of human behavior, it has been criticized for failing to include emotional variables, such as perceptions of threat, mood, and affect, which may limit is predictive power, particularly with certain health behaviors. It has been argued that many behaviors are not rational and that one's affect may be counter to one's cognitions about engaging in a particular behavior. Thus, attitude may be shaped by affect in addition to beliefs. Another criticism of the TRA/TPB model is that the majority of research testing the theory has been correlational (cross-sectional or longitudinal, typically with brief follow-up periods). The experimental studies testing the theory have provided less support for the model (Webb & Sheeran, 2006).

Measurement of the Theory of Planned Behavior Constructs

It is common practice to assess each of the constructs with only one or two items, though multi-item measures are often recommended. Differences in the way the constructs are assessed have led to confusion and may account for some of the variation between studies (see Armitage & Conner (2001) for a meta-analytic review of such differences).

Attitude: Attitude is typically assessed via the use of semantic differential scales that tap into both affective and cognitive attitude. Pairs of adjectives (reflecting a positive and negative component of an attitude) that have relevance to the health behavior being studied are provided as anchors. For example, "To me, engaging in regular exercise is…" [*unpleasant-pleasant; unsatisfying-satisfying*(affective); *harmful-beneficial; useless-useful* (cognitive)].

Subjective norms: When assessing subjective norms, it is recommended to measure both injunctive norms (what others think) and descriptive norms (what others actually do). Social groups are often identified as peers, family, friends, or important people. Examples of items include the following: "People who are important to me think I should exercise regularly" (injunctive); "My friends exercise regularly" (descriptive). Items are usually measured using a 7-point Likert scale.

Perceived behavior control: Perceived behavioral control is often measured with Likert-scale items assessing both internal control (i.e., self-efficacy), such as "I am confident that I can exercise regularly," a perception of ease/difficulty of engaging in the behavior, such as "It is easy for me to exercise," and perception of control, such as "How much personal control do you feel you have over engaging in regular exercise?" In general, measures of self-efficacy account for the most added variance to intentions.

Intentions: Many researchers assess intentions with one Likert-scale item, such as "I intend to engage in regular exercise for the next 3 months." Although intentions tend to be highly reliable, additional items may strengthen the measurement of this construct. Items are sometimes phrased as, "I will try to…," "I plan to…," and "I will make an effort to…." This construct has also been assessed with questions related to self-prediction (e.g., "It is likely that I will engage in regular exercise over the next 3 months"), though such a conceptualization probably incorporates perceived behavioral control.

Behavior: Behavior is often assessed via self-report, but observed behavior is clearly preferred. The TPB model typically predicts more of the variance in self-reported behavior than observed behavior.

Specific recommendations and guidelines for developing questionnaires for use in a particular study are offered by Ajzen (see http://people.umass.edu/aizen/tpb.html), Fishbein and Ajzen (2010), Godin and Kok (1996), and the National Cancer Institute (see http://cancercontrol.cancer.gov/brp/constructs/index.html).

Cross-References

- ▶ Attitudes
- ▶ Behavior
- ▶ Self-efficacy

References and Readings

Ajzen, I. (1985). From intentions to action: A theory of planned behavior. In J. Kuhl & J. Beckman (Eds.), *Action control: From cognitions to behaviors* (pp. 11–39). New York: Springer.

Ajzen, I. (1991). The theory of planned behavior. *Organizational Behavior and Human Decision Processes, 50*, 179–211.

Ajzen, I. (n.d.) *Theory of planned behavior*. Retrieved 6 July, 2011 from http://people.umass.edu/aizen/tpb.html

Ajzen, I., & Fishbein, M. (1980). *Understanding attitudes and predicting social behavior*. Englewood Cliffs, NJ: Prentice-Hall.

Armitage, C. J., & Conner, M. (2001). Efficacy of the theory of planned behavior: A meta-analytic review. *British Journal of Social Psychology, 40*, 471–499.

Fishbein, M., & Ajzen, I. (1975). *Belief, attitude, intention and behavior: An introduction to theory and research*. Reading, MA: Addison-Wesley.

Fishbein, M., & Ajzen, I. (2010). *Predicting and changing behavior: The reasoned action approach*. New York: Psychology Press.

Godin, G., & Kok, G. (1996). The theory of planned behavior: A review of its applications to health-related behaviors. *American Journal of Health Promotion, 11*(2), 87–98.

National Cancer Institute, US National Institutes of Health. (n.d.) *Health behavior constructs: Theory, measurement, and research*. Retrieved 6 July, 2011 from http://cancercontrol.cancer.gov/brp/constructs/index.html

Sheeran, P. (2002). Intention-behaviour relations: A conceptual and empirical review. In W. Stroebe & M. Hewstone (Eds.), *European review of social psychology* (Vol. 12, pp. 1–36). London: Wiley.

Webb, T. L., & Sheeran, P. (2006). Does changing behavioral intentions engender behavior change? A meta-analysis of the experimental evidence. *Psychological Bulletin, 132*, 249–268.

Therapy

- ▶ Occupational Therapy

Therapy, Family and Marital

Ashley K. Randall[1] and Guy Bodenmann[2]
[1]Family Studies & Human Development, University of Arizona, Tucson, AZ, USA
[2]Department of Psychology, University of Zurich, Binzmuehlestrasse, Zurich, Switzerland

Synonyms

Couple therapy; Family therapy; Interventions therapy

Definition

Both family and marital therapy are branches of psychotherapy that aim to facilitate positive change, by addressing the dyad and family as a whole. Broadly, these branches of psychotherapy help teaching communication and problem-solving techniques in an effort to ameliorate distress.

Description

There are a wide range of therapeutic interventions that work with families and couples during times of distress. The goal of each therapeutic approach is to alleviate the distress, to strengthen dyadic and family resources, and to improve well-being of the couple or family members. Although family and couple therapies can be applied with various techniques or theoretical approaches, there are common approaches that have been shown effective in alleviating family distress, such as behavioral family and couple therapy.

Family Therapy Techniques

Therapeutic techniques for families are grounded in family systems theory, which states that each member of the family is interconnected and one cannot just treat one family member independently (Segrin & Flora, 2005). The most common family therapy techniques address adolescent concerns in respect to the family dynamic.

Below, we review some of the commonly used therapies.

Bowen's family approach. Bowen's family systems theory views behavior within the family as an emotional unit. This theory operates under the balance of two forces: togetherness and individuality, whereby too much of any one force can create an imbalance in the system. Bowen's approach also uses eight concepts that aim to explain family development and functioning: (1) differentiation of self, (2) triangles, (3) nuclear emotional process, (4) family projection process, (5) multigenerational transmission process, (6) sibling position, (7) emotional cutoff, and (8) societal emotion process. Some of the main goals of therapy include reframing the family problem as a multigenerational problem that is caused by factors outside the individual and to lower the family "emotional turmoil" (Bowen, 2004; Bowen Center for the Study of the Family, 2011).

Structural family therapy (*SFT*). SFT is a way to address problems within the family by looking at the invisible rules or boundaries which help its functioning (family rules). The role of the therapist is to manipulate the therapy session in a way to accelerate change in the family, for example, by changing the seating of each family member. This helps the family members see the unbalance of the family system and the dysfunctional patterns that have developed (Minuchin, 1974).

Brief strategic family therapy (*BSFT*). BSFT aims to improve and change the family interaction patterns that have led to the disruptive behaviors in adolescents. Specifically, BSFT targets children between the ages of 6 and 17 that display or are at risk for developing behavior, conduct, and substance abuse problems. Specifically, BSFT focuses on inappropriate family alliances, poor boundaries (open vs. closed), and allows the parents to recognize the adolescent is not necessarily the cause for familial problems but the expression of dysfunctional family patterns (Szapocznik & Williams, 2000).

Functional family therapy (*FFT*). FFT is an intervention program that focuses on adolescents with disruptive behavior problems (conduct, alcohol, and/or substance abuse). The goal is to reduce the problem behavior while using an individualized nonjudgmental attitude, focusing on strengths and protective factors specific to each client. This intervention can be implemented in a variety of settings such as schools, probation and aftercare systems, and well as in mental health facilities (Alexander & Parsons, 1982).

Multisystemic therapy (*MST*). MST is an intensive family- and community-based treatment program that focuses on the entire "world" of chronic and violent juvenile offenders – their homes and families, schools and teachers, neighborhoods and friends (Heneggler, Schoenwald, Borduin, Rowland & Cunningham, 1998).

Other techniques, such as contextual therapy, focal family therapy, systemic therapy, and symbolic-experimental family therapy have also been used in family therapy (see Gurman & Kniskern, 1991, for an overview).

Marital Therapy

Traditional behavioral couple therapy (*TBCT*). TBCT attempts to increase the level of reinforcing exchange between the two partners. This approach classically aims at teaching more effective communication and problem-solving skills that will enhance the ability of the couple to effectively communicate as well as minimize punishment and maximize reward (Jacobson & Christensen, 1994; Jacobson & Margolin, 1979).

Cognitive behavioral couple therapy (*CBCT*). CBCT takes the basic principles of TCBT and incorporates partner's relationship assumptions, standards, expectancies, and attributions that contribute to the relationship distress (Baucom, Shoham, Mueser, Daiuto & Stickle, 1998; Epstein & Baucom, 2002).

Integrated behavioral couple therapy (*IBCT*). ICBT also takes the basic principles of CBCT and expands them to emphasize interventions aimed at increasing acceptance. The three components, empathetic joining, tolerance building, and detachment from the problem, are focused at enhancing the couple's ability to appreciate the differences in their marriage

(Christensen, Jacobson & Babcock, 1995; Jacobson & Christensen, 1998).

Emotion-focused couple therapy (EFCT). EFCT focuses on inner emotional experiences in combination with self-reinforcing interactions, based on adult attachments and attachment bonds. Therapists in this approach try to (1) access and reprocess the emotional experience of the partners' and (2) restructure the partner's interaction patterns. Partners are thought to learn new aspects about themselves and develop a more functional pattern of interaction with their partner that is cohesive with their specific attachment needs (Greenberg & Johnson, 1988; Johnson & Greenberg, 1985).

Integrated systematic couple therapy (*ISCT*). The specific goal of ISCT is not to resolve all of the issues causing distress but rather instigated a reversal of the negative interaction. ISCT is based on procedures from family and marital systems therapy and primarily targets the problem at the interaction level. Specifically, ISCT tries to initiate a reversal in the "fight cycle" by changing the meaning attributed to the situation. Empirical evidence exists for the efficacy of ISCT showing greater maintenance of marital satisfaction and goal attainment after the intervention (Greenberg & Goldman, 1985).

Insight-oriented therapy (*IOMT*). IOMT focuses on the interpretation of underlying intrapersonal and interpersonal dynamics between the couple partners contributing to the marital distress. IOMT also examines developmental issues, interactions, expectations, and maladaptive relationship patterns that may exist in the relationship. The role of the therapist is to guide the couple to gain a better understanding and clarification of each partner's unconscious feelings and beliefs that may be affecting the relationship (Snyder & Wills, 1989; Wills, 1982).

Coping-oriented couple therapy (*COCT*). COCT is based on stress and coping research in couples and cognitive behavioral couple therapy. It aims to enhance communication, problem-solving, and dyadic coping in both partners (Bodenmann, 2007, 2010). A main focus lies on dyadic coping and building mutual intimacy and understanding. A key element of this approach is the three-phase method. By means of this method the therapist aims to enhance mutual understanding for each partner's personal functioning (that becomes most evident in stressful situations) and its impact on the close relationship as well as the enhancement of mutual support that matches personal needs of the partner. Overall, this approach fosters (a) understanding for each partner's personality, (b) mutual dyadic coping, and (c) mutual intimacy, trust in the partner, cohesion, and emotional security (Table 1).

Prevention Programs

Several evidence-based programs aim to prevent marital distress (e.g., Couples Communication Program (CCP); Miller, Wackman & Nunnally, 1983; Premarital-Relationship Enhancement Program (PREP): Markman, Stanley & Blumberg, 1994, Couples Coping Enhancement Training (CCET); Bodenmann & Shantinath, 2004).

Efficacy of Marital and Family Therapy

The efficacy of family and couple therapies are well documented (Dunn & Schwebel, 1995; Shadish & Baldwin, 2005) including substantial mean effect sizes which demonstrate the effectiveness in relieving distress ($d = .74–.95$; Shadish, Montgomery, Wilson, Wilson, Bright & Okwumabua, 1993; $d = .50–1.30$; Shadish & Baldwin, 2003). Prior research has shown that approximately 70% of couples seeking evidence-based couple therapy report an improvement after therapy (Baucom et al., 1998; Christensen & Heavey, 1999), and more recent numbers suggest 46–56% of couples show significant clinical improvement (Christensen, Atkins, Baucom & Yi, 2010).

Application to Behavioral Medicine

Family and marital therapy techniques have been used to alleviate distress within the family, stemming from things such as communication issues or family dynamics. Nevertheless, some of these therapeutic approaches have been used for a range of behavioral and physical health problems (see Snyder, Castellani & Whisman, 2006 for

Therapy, Family and Marital, Table 1 Marital and family therapies

Marital and family therapies	Goal	Content
Family therapies		
Bowenian Family Therapy	Individuals are encouraged to look at the view they play in the family system, patterns of emotional reactivity, and interlocking triangles. In addition, the goal is to decrease anxiety	Examines how the family may operate as an "emotional system" using key concepts such as : triangles, differentiation of the self, emotional cutoff, and sibling position
Structural Family Therapy (SFT)	Address the problem in functioning within a family, in an attempt to restructure the family system's rules to become more flexible (Minuchin, 1974)	Therapist helps to show family how their family system may be unbalanced and to help the family see the dysfunctional patterns they have created. SFT also helps families move toward an understanding of how the behavior has developed into a positive feedback loop
Brief Strategic Family Therapy (BSFT)	Aims to improve family interaction and change family interaction patterns that have led to disruptive behaviors in adolescents (e.g., conduct problems, delinquency, and drug abuse) (Szapocznik & Williams, 2000)	BSFT operates on five basic concepts: (1) *context* – the behavior cannot be understood outside the context to which it occurs; (2) *systems* – the family is an interconnected entity that cannot be understood by just examining one member; (3) *structure* – provides the habitual and repetitive patterns of family interaction; (4) *strategy* – interventions tend to be practical, problem-focused, and planned; and (5) *content versus process* – the therapist's focus is on **how** the family members' interaction, not **what** the family members discuss, is the problem
Functional Family Therapy (FFT)	Family intervention program for adolescents with disrupting behavior problems (conduct, alcohol, and/or substance abuse). The goal is to reduce the problem behavior while using an individualized nonjudgmental attitude, focusing on strength/protective factors that are linked to each client (Alexander & Parsons, 1982; Alexander et al., 2000)	FFT operates on five major components: (1) *engagement phase* – the goal of this phase is to demonstrate a desire to listen and help. The therapist focuses on immediate responsiveness and maintains a strength-based focus; (2) *motivational phase* – the goals of this phase include creating a positive motivational context that will facilitate change, minimizing hopelessness, and changing the meaning of the family context to promote change; (3) *relational assessment* – the focus of this phase is on intra- and extra-familial context (e.g., values, interaction patterns, and sources of resistance, resources, and limitation); (4) *behavior change phase* – this phase helps build coping patterns, such as teaching communication as well as training conflict resolution; and (5) *generalization phase* – this phase focuses on extending positive family functions and helps the family plan for relapse prevention (e.g., using other family and community members for support)
Multisystemic Therapy (MST)	Intensive family and community-based treatment program that focuses on violent and criminal youth behavior (Heneggler et al., 1998)	MST views the child/adolescent embedded within interconnected systems: family, peers, school, neighborhood, and community/culture. Focus is on increasing parenting skills: spending time with children, teaching communication techniques and how to develop boundaries/ discipline, and teaching skills on how to deal with conflict. Help adolescents participate in positive activities (sports or extracurricular clubs) and create a supportive social network among family, peers, and community to maintain change

(*continued*)

Therapy, Family and Marital, Table 1 (continued)

Marital and family therapies	Goal	Content
Couple therapies		
Traditional Behavioral Couple Therapy	Attempts to increase the level of reinforcing exchange between partners Minimize punishment and maximize reinforcement (Jacobson & Margolin, 1979; Jacobson & Christensen, 1994)	Help couples identify positive behaviors that they can do for one another (e.g., show more affection). Help guide couples to engage in these behaviors and acknowledge when they occur (e.g., to give praise)
Cognitive Behavioral Couple Therapy (CBCT)	Examined the evidence about their thoughts about their partner Alter assumptions and standards, couple evaluates consequences of living according to their standards and assumptions about their partner (Baucom et al., 1998; Epstein & Baucom, 2002)	Use of cognitive restructuring strategies to modify different types of dysfunctional cognitions. Examine the interplay between thoughts, emotions, and behavior
Integrated Behavioral Couple Therapy (IBCT)	To help the couple think about the problem and identify feelings associated with that issue before one can accept them (Christensen et al., 1995; Jacobson & Christensen, 1998)	Educate couples that partners need to learn a way to alter negative emotional responses that make them and their partners unhappy Teaches couple new ways to resolve problems and emotions through three steps: (1) Empathetic joining, (2) tolerance building, and (3) detachment from the problem
Emotion-Focused Couple Therapy (EFCT)	Conceptualizes distress in adult romantic relationships in terms of attachment theory. Focus on re-establishing attachment bonds (Johnson & Greenberg, 1985; Greenberg & Johnson, 1988)	(1) Identify the negative interaction cycle of the conflict, (2) access unacknowledged feelings, (3) reframe the problem(s) in terms of underlying feelings, (4) promote identification with disowned needs and aspects of self, (5) promote acceptance by each partner; (6) facilitate the expression of needs and wants to restructure the interaction based on the new understandings, (7) establish the emergence of new solutions (cycles), and (8) consolidate new positions
Integrated Systematic Couple Therapy (ISCT)	Try to change meaning attributed to situation that caused distress. Aims to initiate a reversal in the "fight cycle" (self-perpetuating negative cycles that lead to changes in behavior) (Greenberg & Goldman, 1985)	(1) Define the issue presented, (2) identify the negative interactional cycle, (3) attempt restructuring, (4) reframe the problem using positive connotation followed by prescribing the symptom, (5) restrain using "go slow" and dangers of improvement, (6) consolidate the frame, and (7) prescribe a relapse
Insight-Oriented Marital Therapy (IOMT)	Emphasis is on the interpretation of underlying intra- and interpersonal dynamics that influence relationship distress (Wills, 1982; Snyder et al., 1989)	Focus on expectations, interactions, and maladaptive relationship rules using clarification and interpretation, to highlight unconscious feelings, beliefs, or thoughts causing the marital discord
Copying-Oriented Couple Techniques (COCT)	Teach couples the notion the better the partners *together* can cope with stress, the higher their chance for optimal marital satisfaction and stability. Bodenmann, Plancherel, Beach, Widmer, Gabriel & Meuwly, 2008)	Focus on educating the couple about the effects stress has on the relationship. Couples become aware of the influence of environmental factors on their close relationship. Focus on teaching dyadic copying that goes beyond the traditional models of interpersonal communication and social support in close relationships

a review). Additionally, CanCOPE is an education program developed for people facing cancer (personally or within the family). The focus of this program is to strengthen the resources within the family so that they can more effectively deal with the realities of cancer management (Scott, Halford & Ward, 2004). Family-based obesity treatment programs have also been

developed, and show that child–parent interactions can influence the outcome of obesity treatment (Temple, Wrotniak, Paluch, Roemmich & Epstein, 2006). Osterman, Sher, Hales, Canar, Singla and Tilton (2003) encourage health psychologists to incorporate more of a couple's perspective into illness, so that the partner can facilitate health-promoting attitudes and behaviors.

Cross-References

► Family Stress

References and Readings

Alexander, J. F., & Parsons, B. V. (1982). *Functional family therapy*. Monterey, CA: Brooks/Cole.

Alexander, J. F., Pugh, C., Parsons, B. V., & Sexton, T. L. (2000). *Functional family therapy* (2nd ed.). Boulder, CO: Center for the Study and Prevention of Violence, University of Colorado.

Baucom, D. H., Shoham, V., Mueser, K. T., Daiuto, A. D., & Stickle, T. R. (1998). Empirically supported couple and family interventions for marital distress and adult mental health problems. *Journal of Consulting and Clinical Psychology, 66*, 53–88.

Bodenmann, G. (2007). Dyadic coping and the 3-phase-method in working with couples. In L. VandeCreek (Ed.), *Innovations in clinical practice: Focus on group and family therapy* (pp. 235–252). Sarasota: Professional Resources Press.

Bodenmann, G. (2010). New themes in couple therapy: The role of stress, coping and social support. In K. Hahlweg, M. Grawe, & D. H. Baucom (Eds.), *Enhancing couples. The shape of couple therapy to come* (pp. 142–156). Cambridge, MA: Hogrefe Publishing.

Bodenmann, G., Plancherel, B., Beach, S. R. H., Widmer, K., Gabriel, B., & Meuwly, N. (2008). Effects of coping-oriented couples therapy on depression. A randomized clinical trial. *Journal of Consulting and Clinical Psychology, 76*, 944–954.

Bodenmann, G. & Shantinath, S. D. (2004). The Couples Coping Enhancement Training (CCET): A new approach to prevention of marital distress based upon stress and coping. *Family Relations*, 53(5), 477–484.

Bowen, M. (2004). Family psychotherapy in office practice. *Family Systems, 7*(1), 29–44.

Bowen Center for the Study of the Family. (2011). *Bowen family therapy*. Retrieved October 2011 from: www.thebowencenter.org

Christensen, A., Atkins, D. C., Baucom, B., & Yi, J. (2010). Marital status and satisfaction five years following a randomized clinical trial comparing traditional versus integrative behavioral couple therapy. *Journal of Consulting and Clinical Psychology, 78*, 225–235.

Christensen, A., & Heavey, C. L. (1999). Interventions for couples. *Annual Review Psychology, 50*, 165–190.

Christensen, A., Jacobson, N. S., & Babcock, J. C. (1995). Integrative behavioral couple therapy. In N. S. Jacobson & A. S. Gurman (Eds.), *Clinical handbook of marital therapy* (2nd ed., pp. 31–64). New York: Guilford Press.

Dunn, R. L., & Schwebel, A. I. (1995). Meta-analytic review of marital therapy outcome research. *Journal of Family Psychology, 9*, 58–68.

Epstein, N. B., & Baucom, D. H. (2002). *Enhanced cognitive-behavioral therapy for couples: A contextual approach*. Washington, DC: American Psychological Association.

Greenberg, L. S., & Goldman, A. (1985). *Integrated systemic couples' therapy: A treatment manual*. Unpublished manuscript, University of British Columbia, Vancouver, Canada.

Greenberg, L. S., & Johnson, S. M. (1988). *Emotionally focused therapy for couples*. New York: Guilford Press.

Gurman, A. S., & Kniskern, D. P. (1991). *Handbook of family therapy (Volume I and II)* (Vol. I and II). New York: Brunner & Mazel.

Heneggler, S. W., Schoenwald, S. K., Borduin, C. M., Rowland, M. D., & Cunningham, P. B. (1998). *Multisystemic treatment of antisocial behavior in children and adolescents*. New York: Guilford Press.

Jacobson, N. S., & Christensen, A. (1994). *Traditional behavioral couple therapy manual*. Unpublished manuscript, University of Washington.

Jacobson, N. S., & Christensen, A. (1998). *Acceptance and change in couple therapy: A therapist's guide to transforming relationships*. New York: Norton.

Jacobson, N. S., & Margolin, G. (1979). *Marital therapy: Strategies based on social learning and behavior exchange principles*. New York: Brunner/Mazel.

Johnson, S. M., & Greenberg, L. S. (1985). Emotionally focused couples therapy: An outcome study. *Journal of Marital and Family Therapy, 11*, 313–317.

Markman, H. J., Stanley, S., & Blumberg, S. L. (1994). *Fighting for your marriage: Positive steps for preventing divorce and preserving a lasting love*. San Francisco: Jossey Bass.

Miller, S., Wackman, D. B., & Nunnally, E. W. (1983). Couple communication: Equipping couples to be their own best problem solvers. *The Counseling Psychologist, 11*, 73–77.

Minuchin, S. (1974). *Families and family therapy*. Cambridge, MA: Harvard University Press.

Osterman, G. P., Sher, T. G., Hales, G., Canar, W. J., Singla, R., & Tilton, T. (2003). *Physical illness*. New York: Guilford Press.

Scott, J. L., Halford, W. K., & Ward, B. (2004). United we stand? The effects of a couple-coping intervention on

adjustment to breast or gynecological cancer. *Journal of Consulting and Clinical Psychology, 76*, 1122–1135.
Segrin, C., & Flora, J. (2005). Theoretical perspectives on family communication: Family systems theory. *Family communication* (pp. 28–33). Mahwah, NJ: Erlbaum.
Shadish, W. R., & Baldwin, S. A. (2003). Meta-analysis of MFT interventions. *Journal of Marital and Family Therapy, 29*, 547–570.
Shadish, W. R., & Baldwin, S. A. (2005). Effects of behavioral marital therapy: A meta-analysis of randomized controlled trials. *Journal of Consulting and Clinical Psychology, 73*, 6–14.
Shadish, W. R., Montgomery, L. M., Wilson, P., Wilson, M. R., Bright, I., & Okwumabua, T. (1993). Effects of family and marital psychotherapies: A meta-analysis. *Journal of Consulting and Clinical Psychology, 61*, 992–1002.
Snyder, D. K., Castellani, A. M., & Whisman, M. A. (2006). Current status and future directions in couple therapy. *Annual Review of Psychology, 57*, 317–344.
Snyder, D. K., & Wills, R. M. (1989). Behavioral vs. insight oriented marital therapy: A controlled comparative outcome study. *Journal of Consulting and Clinical Psychology, 57*, 39–46.
Szapocznik, J., & Williams, R. A. (2000). Brief strategic family therapy: Twenty-five years of interplay among theory, research and practice in adolescent behavior problems and drug abuse. *Clinical Child and Family Psychology Review, 3*(2), 117–134.
Temple, J. L., Wrotniak, B. H., Paluch, R. A., Roemmich, J. N., & Epstein, L. H. (2006). Relationship between sex of parent and child on weight loss and maintenance in a family-based obesity treatment program. *International Journal of Obesity, 30*, 1260–1264.
Wills, R. M. (1982). *Insight oriented marital therapy* [Treatment manual]. Unpublished manuscript, Wayne State University, Detroit.

Therapy, Occupational

Cecilia W. P. Li-Tsang[1], Kit Sinclair[1] and Jennifer Creek[2]
[1]Department of Rehabilitation Sciences, The Hong Kong Polytechnic University, Kowloon, Hong Kong, China
[2]Occupational Therapist, Guisborough, North Yorkshire, UK

Synonyms

Ergotherapist; Occupational therapist

Definition

Therapy with focus on people's occupational performances and behavior that is using occupation as the therapeutic medium is carried out by registered or legitimated occupational therapists (ergotherapists). Major goals of occupational therapy and major roles of occupational therapists are to (a) manage environmental, temporal, and occupational adaptations that facilitate clients' way of life and well-being, (b) teach clients to learn or relearn performance of activities in daily life and work, (c) enable clients to be meaningfully occupied, and (d) promote clients' health and wellness through preventive measures (Söderback, 2009).

Description

Occupational therapy is concerned with the activities and occupations that make up the pattern of people's lives, and with people's capacity to carry out those activities in ways that support their health and well-being. For occupational therapists, the concept of *occupation* encompasses everything that people do in their daily lives, including self-care, domestic activities, interactions with others, work and leisure activities (Fidler & Fidler, 1978; WFOT, 2010).

Occupational therapists understand that occupation is essential to human development and learning, to social relations, to individual health and well-being and to the well-being of communities. Occupation enables the development and integration of bodily systems, promotes socialization, and verifies the individual's identity as a contributing member of society. Further, purposeful activity can mitigate the effects of disease or injury and prevent secondary disability (Creek, 2003). Occupational therapists are concerned with the impact of illness, injury, or disability on people's ability to carry out the activities and occupations that they want to do, need to do, or expect to do. The focus of intervention is not on the client's impairment or diagnosis but on the impact that illness or

disability can have on the individual's ability to carry out his or her expected daily activities. This means that the occupational therapist works not only with the client's functional problems but also with the *meaning* of illness or injury to his or her life. This has been called a two-body practice, because occupational therapy is concerned both with the physical body and with the whole person, including social, cultural, and psychological issues. The knowledge base that supports a two-body practice is necessarily broad and varied (Mattingly, 1994). Occupational therapists have a deep understanding of the importance of activity and occupation in people's lives and they also make use of relevant knowledge from the biological, medical, psychological, social, and technological sciences. Areas of knowledge include theory, research, policy, and legislation. In most countries, occupational therapy education is at bachelors or master's degree level, although the qualification is a diploma in some countries (College of Occupational Therapists, 2009). Occupational therapists are experts in assessing function in the activities of daily life, including personal care, mobility, domestic activities, social interactions, education, leisure, and work. When a person is unable to perform the activities of daily life to an acceptable standard, the occupational therapist can assist him or her to relearn the necessary skills, develop new skills, or adapt activities so that they are within the individual's capabilities. The occupational therapist can also recommend aids, equipment, and environmental modifications to support function or compensate for loss of function.

Occupational therapists practice a set of principles that influence both what is done and how the therapeutic relationship works. These principles include respect for diversity, customs and preferences; recognition of the client's rights; incorporation of the client's perspective at all stages of intervention; promotion of client autonomy and choice, and sharing power with the client and/or carer. Intervention does not follow a standard process but is highly individualized and specific to the client, the context, and the environment. This means that the way in which occupational therapy is practiced is strongly influenced by the social context in which the therapist is working, the work setting, government policies and standards, local norms and procedures, and the available research evidence (White, 2007). Person-centered intervention takes account of the individual's current circumstances, cultural background, social context, educational experiences, employment status, personal beliefs and values, skills, interests, needs, and aspirations. Interventions are designed to suit the person's living environment, family, neighborhood, workplace, financial situation, social networks, and support systems. An expert occupational therapist is able to engage with each service user within his or her own environments, to identify the activities that have meaning and relevance for each person and to work in partnership to devise individually tailored interventions. The occupational therapist works in collaboration with the client, families, caregivers, employers, teachers, coworkers, and colleagues to develop and deliver relevant and effective interventions. As far as possible, the client is engaged as an active partner throughout, and the intervention is carried out within his or her own living or working environments.

The core skills of the occupational therapist are collaboration with the client, assessment of function, problem solving, therapeutic use of activity, group work, and environmental adaptation. Activity is the main tool employed to achieve therapeutic goals: It can be used to develop and maintain skills, to improve occupational performance, to enhance self-esteem, and to increase social participation. Activities are chosen both for their potential to engage client participation and for their potential to meet treatment objectives. Throughout the intervention, activities are monitored and adapted to maintain client interest and therapeutic effectiveness.

Occupational therapists also use environmental adaptation to enhance functional and occupational performance. Changes to the client's environment have to be negotiated and agreed with the client and relevant others, taking into account the dynamics of the household or other setting. Environmental modifications may be made over time, as the client's circumstances

change. The therapist may also recommend the introduction of paid carers to improve the client's functional capacity and quality of life, or to relieve carers.

Occupational therapy has relevance for everyone who is experiencing occupational dysfunction and it is practiced in many countries throughout the world. In most countries, it is regulated as a health profession. The worldwide professional body for occupational therapy is the World Federation of Occupational Therapists: Member countries all have a national professional body and full member countries also have at least one approved program of occupational therapy education (White, 2007; Christiansen & Baum, 1997).

Occupational therapists are working in a wide range of public, private, and voluntary sector settings, although most occupational therapists work in the fields of health and social care. People of all ages, who have functional problems arising from physical, psychological, social, educational, economic, or other difficulties, participate in occupational therapy. Clients may be individuals, groups of people, communities, health and social care agencies, or other organizations. Individual clients may be hospital or community patients, schoolchildren, workers, compensation claimants, caregivers, homeless people, or anyone who is experiencing occupational dysfunction. Group clients can be families, coworkers, paid carers, or groups of patients. Some occupational therapists work in the areas of public health, occupational health, or health promotion. They may work in the field of law as expert witnesses or consultants, for example, carrying out assessments to ascertain the level of disability sustained following an accident (Radomski & Trombly, 2008). Occupational therapy contributes to the treatment of, and early recovery from, injury and disease, and is making an increasing contribution to health promotion and disease prevention. However, the profession's major role is in habilitation and rehabilitation: helping people to gain or regain the ability to perform their occupations and preventing secondary disability. Occupational therapists also work with people who have long-term or deteriorating health conditions, assisting them to remain active for as long as possible and slowing down the decline of functional ability (Christiansen & Baum, 1997).

Cross-References

▶ Occupational Therapy

References and Readings

Christiansen, C. H., & Baum, C. M. (1997). *Enabling function and well-being* (2nd ed.). Thorofare: Slack.

College of Occupational Therapists. (2009). *Curriculum guidance for pre-registration education*. London: Author.

Creek, J. (2003). *Occupational therapy defined as a complex intervention*. London: College of Occupational Therapists.

Fidler, G. S., & Fider, J. W. (1978). Doing and becoming: Purposeful action and self-actualization. *The American Journal of Occupational Therapy, 32*, 305–310.

Mattingly, C. (1994). Occupational therapy as a two-body practice: The body as machine. In C. Mattingly & M. H. Fleming (Eds.), *Clinical reasoning: Forms of inquiry in a therapeutic practice*. Philadelphia: FA Davis.

Radomski, M. V., & Trombly Latham, C. A. (Eds.). (2008). *Occupational therapy for physical dysfunction* (pp. 24–39). Philadelphia: Lippincott Williams & Wilkins.

Söderback, I. (Ed.). (2009). International handbook of occupational therapy interventions. New York: Springer.

White, E. (2007). When service users' views vary from those of their carers. In J. Creek & A. Lawson-Porter (Eds.), *Contemporary issues in occupational therapy: Reasoning and reflection* (pp. 115–126). Chichester: Wiley.

World Federation of Occupational Therapists. (2010). Statement on occupational therapy. Retrieved November 14, 2011, from www.wfot.org

Therapy, Physical

▶ Physical Therapy

Therapy, Speech

▶ Speech Therapy

Thoughts

► Cognitions

Thriving

► Perceived Benefits

Thrombosis

► Coagulation of Blood

Tinnitus

Linda C. Baumann and Alyssa Karel
School of Nursing, University of Wisconsin-Madison, Madison, WI, USA

Synonyms

Ringing in the ears

Definition

Tinnitus is a condition characterized by the perception of sound in the ears or head without the presence of an external source. Tinnitus itself is not a disease, but a symptom that can result from a number of different causes. Sounds heard can manifest in many different ways such as low to high pitched, heard in one or both ears, heard as a single noise or competing noises, or be heard intermittently or continuously. Sounds have been described as ringing, buzzing, blowing, humming, hissing, whooshing, hissing, and whistling among many others. Mild forms of tinnitus are very common and experienced by most people at some point in their lives. More severe forms, however, are less common and can lead to chronic sleep disturbance, anxiety, and depression.

Description

Tinnitus is generally categorized into two types: subjective tinnitus and objective tinnitus. Objective tinnitus is the less common of the two types and sound is not only heard by the patient, but also audible to other people, most often a clinician listening with a stethoscope or an ear tube. Pulsatile tinnitus is a common example of objective tinnitus. It is caused by muscle contractions or audible blood flow in arteries or veins (e.g., bruits) close to the inner ear that resonate as rhythmic pulsing in the ear. Subjective tinnitus is the most common form and is heard exclusively by the patient. This type of tinnitus has many causes and pathologies.

Although tinnitus is most often associated with abnormalities of the auditory or central nervous systems, it can also be caused by nonauditory etiologies. These include hypertension and cardiovascular disease, hypo- and hyperthyroidism, stress and fatigue, temporomandibular joint (TMJ) disorder, poor diet and physical inactivity, and wax buildup in the outer ear putting pressure on the tympanic membrane. Exposure to excessive noise is also a common cause of tinnitus, which can precede hearing loss and should therefore be an indicator of the need for protection from excessive noise exposure. Tinnitus associated with abnormalities of the auditory or central nervous systems including middle ear infections, damage to the inner ear, disorders that affect the central nervous system such as meningitis, encephalitis, and stroke; head and neck trauma; surgical injury; and tumors affecting the acoustic nerve (cranial nerve VIII); Meniere's disease (an inner ear disorder characterized by hearing loss vertigo, and tinnitus); and vestibular schwannoma (e.g., acoustic neuroma). Over 200 ototoxic medications are associated with inducing tinnitus. These medications include aspirin, some

antibiotics, diuretics, cancer chemotherapy drugs, and quinine.

With many causes of tinnitus, treatment of the underlying disease often alleviates symptoms. Drug therapies include benzodiazepines, anticonvulsants, antidepressants, vasodilators, tranquilizers, and antihistamines. Acamprosate, a drug used to treat alcohol dependence, has shown to have potential as a treatment, as well as zinc and gabapentin. Antiarrhythmic agents such as lidocaine have also shown to have tinnitus-suppressing qualities.

Hearing aides are another modality shown to benefit patients. Loss of hearing often increases awareness of tinnitus and a hearing aid, which amplifies external sound, often helps mask the perception of tinnitus. Wearable sound generators or tinnitus maskers are also used. These devices fit into the ear much like a hearing aid and deliver low-level sound directly into the ear.

Cochlear implant is a treatment used in patients whose tinnitus is accompanied by severe hearing loss. Electrical and magnetic stimulation treatments include transcranial magnetic stimulation and trans-electrical nerve stimulation. Cognitive behavioral therapy treatments include tinnitus retraining therapy (TRT), tinnitus activities treatment, sound therapies, auditory discrimination therapy, and neurofeedback. Alternative therapies for tinnitus have included acupuncture, hypnosis, craniosacral therapy, antioxidants, vitamin and herbal remedies.

References and Readings

Langguth, B., Hajak, G., Kleinjung, T., Cacace, A., & Moller, A. R. (Eds.). (2007). *Tinnitus: Pathophysiology and treatment*. Amsterdam: Elsevier.

Tinnitus. (2010a). *American academy of otolaryngology-head and neck surgery*. Retrieved February 18, 2011, from http://www.entnet.org/HealthInformation/tinnitus.cfm

Tinnitus. (2010b). *Medline plus medical encyclopedia*. Retrieved February 18, 2011, from http://www.nlm.nih.gov/medlineplus/ency/article/003043.htm

Tinnitus. (2010c). *National Institutes of Health: NIDCD*. Retrieved February 18, 2011, from http://www.nidcd.nih.gov/health/hearing/tinnitus

Tinnitus and Cognitive Behavior Therapy

Gerhard Andersson
Department of Behavioural Science and Learning, Linköping University, Linköping, Sweden

Synonyms

Hearing disturbances; Perception of internal noise (false)

Definition

Tinnitus is an auditory perceptual phenomenon that is defined as the conscious perception of internal noises without any outer auditory stimulation. The sounds may be very loud and bizarre, and the most common ones are heard like a high-pitched musical tone or a rushing sound like escaping steam or air. Other descriptions can be more complicated such as metallic sounds, multiple tones of varying frequencies, and mixtures between buzzing and ringing.

Tinnitus is in most cases a temporary sensation, which many people have experienced at least sometime in their life. However, it may develop into a chronic condition, and prevalence figures show that at least 10–15% of the general population have tinnitus. Fortunately, most persons do not have severe tinnitus. Only about 1–3% of the adult population has severe tinnitus, in the sense that it causes marked disruption of everyday activities, mood changes, reduced quality of life, and disrupted sleep patterns. Tinnitus has been reported in children, but in its severe form, it is more common in adults and in particular in the elderly.

Tinnitus is known to occur in association with almost all the dysfunctions that involve the human auditory system. This includes damage to the middle ear, the cochlea, the audiovestibular cranial nerve, and pathways in the brain from cochlear nucleus to primary auditory cortex.

A common distinction is often made between so-called objective (somatosounds, which can actually be heard from the outside) and subjective tinnitus (that are heard only by the afflicted person). Objective tinnitus represents a minority of cases. Subjective tinnitus has been linked to sensorineural hearing loss, caused by various deficits such as age-related hearing loss and noise exposure. Links to other conditions such as temporomandibular joint dysfunction have also been found. Tinnitus has been explained as the result of increased neural activity in the form of increased burst firing or as a result from pathological synchronization of neural activity. Other suggested mechanisms are hypersensitivity and cortical reorganization. With the advent of modern imaging techniques, it has been observed that tinnitus involves certain areas of the brain, particularly those that are related to hearing and processing of sounds. Some involvement of the brain's attentional and emotional systems has also been seen (Cacace, 2003).

Description

Distress and Tinnitus

What distinguishes mild from severe tinnitus is not easily established, apart from variations in subjective ratings of intrusiveness and loudness. In particular, in attempts to determine the handicap caused by tinnitus, it has not been possible to make the determination using the characteristics of the tinnitus itself (e.g., loudness, pitch, etc.). However, psychological factors are of major importance in determining the severity of tinnitus, and this has been observed in both clinical and epidemiological studies.

The problems experienced by tinnitus patients can be divided into four categories: hearing difficulties including noise sensitivity, emotional consequences, concentration problems, and insomnia. In addition, there might be interpersonal consequences and occupational difficulties (e.g., for a musician, admitting tinnitus can be regarded as a sign of weakness).

Hearing loss is the most common symptom that goes together with tinnitus and that can in itself be a great problem. Another common problem is noise sensitivity, which in its severe form can develop into hyperacusis, which is sensitivity to everyday sounds not regarded as loud by most people.

In its severe form, tinnitus is strongly associated with lowered mood and depression. Suicide caused by tinnitus is however rare. Most cases reported have had comorbid psychiatric disturbances. Anxiety, and in particular anxious preoccupation with somatic sensations, is an aggravating factor, and stress is often mentioned as a negative factor for tinnitus and in particular in association with major adverse life events, but the evidence for this notion is weak. Personality factors have been investigated, and associations have been reported between degree of optimism and tinnitus distress (Andersson, 1996) and between perfectionism and tinnitus distress (Andersson Airikka, Buhrman, & Kaldo, 2005).

Tinnitus patients often report difficulties with concentration, for example, with reading. Often this is perceived as auditory intrusions while trying to hold concentration on a task. Until recently, there have been few attempts to measure tinnitus patients' performance on tests of cognitive functioning, but recent research implies a role of the working memory system (Hallam, Shurlock, & McKenna, 2004). Another line of research has focused on the role of selective information processing. Finally, sleep problems represent a significant element in tinnitus patients' complaints and are often a driving reason for seeking help.

Theories

Among the most influential psychological theories on why tinnitus becomes annoying is Hallam et al.'s (1984) habituation model of tinnitus, which presents the notion that tinnitus annoyance is caused by lack of habituation, and the neurophysiological model by Jastreboff and coworkers (Jastreboff, 1990), which is a classical conditioning model where the tinnitus signal is conditioned to aversive reactions such as anxiety and fear. The latter model puts less emphasis on conscious mechanisms involved in tinnitus perception. Other researchers have endorsed

a cognitive-behavioral conceptualization of tinnitus, suggesting a major role for thoughts and beliefs regarding tinnitus (Andersson, 2002).

In clinical settings, management of tinnitus involves taking history of its characteristics such as onset, loudness, character, fluctuations, and severity. Audiological and neuro-otological measurements such as pure-tone audiometry, otoscopy, and brain stem audiometry are also included in routine assessment to exclude treatable conditions (Andersson, Baguley, McKenna, & McFerran, 2005).

Treatments

There is a long history of attempts to cure tinnitus, but surgical and pharmacological interventions have been largely without any success. When the aim is to reduce the suffering, treatment outcome is more promising, and psychologically informed treatments have been found to be helpful in randomized trials (Andersson & Lyttkens, 1999).

Among the psychosocial treatments, cognitive-behavioral therapy (CBT) is the most researched alternative. As for other medical conditions such as chronic pain, CBT for tinnitus distress is directed at identifying and modifying maladaptive behaviors and cognitions by means of behavior change and cognitive restructuring. The focus is on applying techniques such as applied relaxation in real-life settings. An overview of the techniques used in CBT for tinnitus is presented in the Table. There is now evidence from randomized trials that CBT can be effective for alleviating the distress caused by tinnitus in adults (e.g., Hesser, Weise, Zetterqvist Westin, & Andersson, 2011), including a trial on the use of CBT with older adults, and also that it works in a self-help format presented via the Internet (Kaldo et al., 2008). However, while the effects are promising, there is room for more improvement, and tinnitus is a typical example of an area where multidisciplinary input is necessary. Most recent development in CBT for tinnitus is to incorporate treatment procedures from acceptance and commitment therapy (ACT; Hayes, Strosahl, & Wilson, 1999).

Tinnitus and Cognitive Behavior Therapy, Table 1 Overview of cognitive-behavioral treatment for tinnitus

Case formulation
Structured clinical interview following audiological screening
Questionnaire assessment
Treatment rationale and information
Treatment presented in 6–10 sessions
Applied relaxation (1. progressive relaxation, 2. short progressive relaxation, 3. cue-controlled relaxation, and 4. rapid relaxation)
Positive imagery
Sound enrichment by means of external sounds
Hearing tactics and advice regarding noise sensitivity
Modification of negative thoughts and beliefs
Behavioral sleep management
Advice regarding concentration difficulties, exercises of concentration (mindfulness)
Exposure to tinnitus
Advice regarding physical activity
Relapse prevention
Follow-up
Interview and questionnaires

Conclusion

Tinnitus is a poorly understood phenomenon, and while the role of psychological factors is widely acknowledged, there is yet little research on basic mechanisms such as information processing bias, the role of psychopathology, and the influence of the tinnitus sound on working memory capacity. While there are few cases of tinnitus for which surgical and medical interventions might help, in most cases, there is no cure in the sense that the tinnitus sound will not disappear. However, longitudinal fluctuations of both loudness and severity of tinnitus have been observed, and health psychologists could benefit in the pursuit of an explanation why it is that tinnitus becomes bothersome only for a proportion of individuals. When it comes to methods to lessen the distress and to cope with the adverse consequences, such as lowered mood and sleep difficulties, CBT is a promising approach. However, the dissemination of CBT into audiological hospital settings has been slow, and there are very few clinical

psychologists working with tinnitus. Self-help methods are promising and could at least partly solve that problem, and there is also much to be done regarding preventive work as noise-induced hearing loss is the cause of tinnitus in one third of cases with recent onset of tinnitus (Table 1).

Cross-References

► Cognitive Behavioral Therapy (CBT)

References and Readings

Andersson, G. (1996). The role of optimism in patients with tinnitus and in patients with hearing impairment. *Psychology and Health, 11*, 697–707.

Andersson, G. (2002). Psychological aspects of tinnitus and the application of cognitive-behavioral therapy. *Clinical Psychology Review, 22*, 977–990.

Andersson, G., Airikka, M.-L., Buhrman, M., & Kaldo, V. (2005). Dimensions of perfectionism and tinnitus distress. *Psychology, Health & Medicine, 10*, 78–87.

Andersson, G., Baguley, D. M., McKenna, L., & McFerran, D. J. (2005). *Tinnitus: A multidisciplinary approach*. London: Whurr.

Andersson, G., & Lyttkens, L. (1999). A meta-analytic review of psychological treatments for tinnitus. *British Journal of Audiology, 33*, 201–210.

Cacace, A. T. (2003). Expanding the biological basis of tinnitus: Cross-modal origins and the role of neuroplasticity. *Hearing Research, 175*, 112–132.

Hallam, R. S., McKenna, L., & Shurlock, L. (2004). Tinnitus impairs cognitive efficiency. *International Journal of Audiology, 43*, 218–226.

Hallam, R. S., Rachman, S., & Hinchcliffe, R. (1984). Psychological aspects of tinnitus. In S. Rachman (Ed.), *Contributions to medical psychology* (Vol. 3, pp. 31–53). Oxford: Pergamon Press.

Hayes, S. C., Strosahl, K. D., & Wilson, K. G. (1999). *Acceptance and commitment therapy*. New York: Guilford Press.

Hesser, H., Weise, C., Zetterqvist Westin, V., & Andersson, G. (2011). A systematic review and meta-analysis of randomized controlled trials of cognitive-behavioral therapy for tinnitus distress. *Clinical Psychology Review, 31*(4), 545–553.

Jastreboff, P. J. (1990). Phantom auditory perception (tinnitus): Mechanisms of generation and perception. *Neuroscience Research, 8*, 221–254.

Kaldo, V., Levin, S., Widarsson, J., Buhrman, M., Larsen, H. C., & Andersson, G. (2008). Internet versus group cognitive-behavioral treatment of distress associated with tinnitus. A randomised controlled trial. *Behavior Therapy, 39*, 348–359.

Tiredness

► Fatigue

Tissue Repair

► Wound Healing

Tobacco

► Nicotine

Tobacco Advertising

Reiner Hanewinkel and Matthis Morgenstern
Institute for Therapy and Health Research, Kiel, Germany

Synonyms

Tobacco marketing; Tobacco promotion

Definition

Tobacco advertising is a form of communication by the tobacco industry with the aim of promoting tobacco products (typically cigarettes) and use. Different forms of advertising can be classified into "above the line" (ATL) and "below the line" (BTL) advertising. ATL advertising is traditional mass media advertising in print, television, radio, in cinemas, and on billboards. BTL advertising focuses more on specific target groups and uses less traditional advertising methods, such as sponsoring, promotion, event marketing, point-of-sale displays, product placements, direct marketing, ambient marketing, viral marketing, or brand stretching. Most research on the effects of tobacco advertising focuses on ATL advertising. However, the importance of BTL

tobacco advertising is growing in the light of bans or partial bans of traditional tobacco advertising in most countries.

Description

A fundamental premise for the tobacco industry to spend money into tobacco advertising is the assumption that it is effective. The term "effective" can refer to different levels or "outcomes." It can either mean that tobacco advertising increases the market share of a specific brand, given a fixed market size for tobacco products. It can also mean that tobacco advertising increases or stabilizes the market size, by recruiting new smokers and by stimulating current smokers not to quit or ex-smokers to relapse. Empirical research conducted by non-industry-funded researchers has mainly focused on the latter interpretation of effectiveness, which is the one with high impact from a public health perspective.

One type of study in this field has analyzed changes in the global or country-specific tobacco market size dependent on advertising spending or dependent on changes in tobacco policy (e.g. implementation of advertising bans). These studies are usually time series or interrupted time-series designs and use highly aggregated data. While many of the early econometric studies found no association between aggregate cigarette advertising spending and total market sales, there are also studies that found positive relations, especially if the aggregation of the data was reduced (Saffer & Chaloupka, 2000). Studies that compare countries with and without advertising bans or that conduct comparisons within a country before and after an advertising ban often find that bans reduce overall consumption of tobacco (Quentin, Neubauer, Leidl, & Konig, 2007). The effect is stronger in countries with comprehensive bans compared to partial bans.

A second type of studies uses individual-level data, looking at the effects of tobacco advertising on smoking behavior, mostly of young people. These studies are either experimental and quasi-experimental studies or cross-sectional and longitudinal observational studies. The effect of tobacco advertising is usually studied in terms of individual exposure to tobacco advertising. This is some form of induced exposure in the experimental studies or a measure of self-reported advertising exposure in the observational studies. Measures of exposure can be direct (e.g., advertising recall, brand recall, notice of advertising, liking of advertising, ownership of promotional items) or indirect (e.g., television screen times, reception and liking of specific television programs, movies, sports, or magazines). A 2006 systematic review of the empirical evidence based on individual-level studies found 29 studies from 5 continents with more than 300,000 participants (DiFranza et al., 2006). The authors concluded that there is strong evidence for a link between exposure to tobacco promotion and tobacco use of children and adolescents. Applying Hill's criteria for judging the likelihood of a causal relationship between exposure and behavior, the authors found that many of Hill's criteria of causation were fulfilled (Hill, 1965). They found that (1) children are exposed to tobacco promotion before the initiation of tobacco use (criterion temporality), (2) exposure increases the risk for initiation (criterion strength), (3) greater exposure results in higher risk (criterion dose–response), (4) the increased risk is robust (criterion consistency), (5) the risk is scientifically plausible (criterion plausibility), and (6) no other explanation can account for the evidence (criterion analogy). A recent study additionally confirmed the Hill criterion "specificity" (Hanewinkel, Isensee, Sargent, & Morgenstern, 2011).

It is less explicitly studied *how* this effect is mediated, i.e., how tobacco advertising leads to an increase in market size. However, tobacco advertising is not systematically different from other forms of advertising and can, therefore, be conceptualized within broader psychological and marketing theories. Most psychological theories of advertising can be classified as "hierarchy of effects" models (Vakratsas & Ambler, 1999). These models suggest that advertising is not directly influencing behavioral responses, but that the effects are always mediated by a mental process. In the broadest sense, this mental process is a change in object valence, the object being the brand, the product, the product group, or the advertising itself. The most common models used to

explain the effects of advertising are based on the information processing approach (McGuire, 1976). They assume that people are persuaded by the contents of the advertising and consciously follow a cognitive path which is mediated by preferences, attitudes, norms, and beliefs about the advertised object. Newer variants of these models are the so-called dual-process models which conceptualize two routes of information processing, a central route and a peripheral route (Eagly & Chaiken, 1993; Petty & Cacioppo, 1986). The central route is activated if advertising contents are thoughtfully elaborated and recipients have high involvement and attention. On the peripheral route, information is less thoughtfully processed (low involvement) which happens if advertisings are consumed rather casually. Recipients with low involvement are more influenced by peripheral or emotional characteristics of the advertising (e.g., attractiveness of the source, colors, music). Most recent psychological theories even go a step further and assume that conscious mental processes are not a necessary precondition for behavioral influences (Bargh, 2002; Harris, Bargh, & Brownell, 2009). From this perspective, advertising is a form of behavioral priming that automatically affects the perceiver. The term "automatic" implies that the consumer does not have to be aware of having seen the advertising and also does not have to be aware that she/he responded to it. Such conceptions of advertising effects have, of course, strong implications for prevention strategies as it may be very difficult to counteract unconscious advertising effects.

Cross-References

► Tobacco Control
► Tobacco Use

References and Readings

Bargh, J. A. (2002). Losing consciousness: Automatic influences on consumer judgment, behavior and motivation. *Journal of Consumer Research, 29*(2), 280–286.
DiFranza, J. R., Wellman, R. J., Sargent, J. D., Weitzman, M., Hipple, B. J., & Winickoff, J. P. (2006). Tobacco promotion and the initiation of tobacco use: Assessing the evidence for causality. *Pediatrics, 117*(6), e1237–e1248.
Eagly, A. H., & Chaiken, S. (1993). Process theories of attitude formation and change: The elaboration likelihood model and heuristic systematic models. In A. H. Eagly & S. Chaiken (Eds.), *The psychology of attitudes* (pp. 305–325). Fort Worth, TX: Harcourt Brace Jovanovich.
Hanewinkel, R., Isensee, B., Sargent, J. D., & Morgenstern, M. (2011). Cigarette advertising and teen smoking initiation. *Pediatrics, 127*(2), e271–e278.
Harris, J. L., Bargh, J. A., & Brownell, K. D. (2009). Priming effects of television food advertising on eating behavior. *Health Psychology, 28*(4), 404–413.
Hill, A. B. (1965). The environment and disease: Association or causation? *Proceedings of the Royal Society of Medicine, 58*, 295–300.
McGuire, W. J. (1976). Some internal psychological factors influencing consumer choice. *Journal of Consumer Research, 2*(4), 302–319.
Petty, R. E., & Cacioppo, J. T. (1986). *Communication and persuasion: Central and peripheral routes to attitude change*. New York: Springer.
Quentin, W., Neubauer, S., Leidl, R., & Konig, H. H. (2007). Advertising bans as a means of tobacco control policy: A systematic literature review of time-series analyses. *International Journal of Public Health, 52*(5), 295–307.
Saffer, H., & Chaloupka, F. (2000). The effect of tobacco advertising bans on tobacco consumption. *Journal of Health Economics, 19*(6), 1117–1137.
Vakratsas, D., & Ambler, T. (1999). How advertising works: What do we really know? *Journal of Marketing, 63*(1), 26–43.

Tobacco Cessation

► Smoking Cessation

Tobacco Control

Pekka Puska
National Institute for Health and Welfare (THL), Helsinki, Finland

Synonyms

Secondhand smoke; Smokeless tobacco; Smoking; Smoking cessation; Smoking prevention; Tobacco policy

Definition

Tobacco control includes all measures aiming at reduction of tobacco use and of its harmful consequences in the population.

Thus, tobacco control includes measures aiming at both prevention and cessation of use of all tobacco products – both smoking and use of smokeless tobacco.

Description

Introduction

The great health risks from smoking have been convincingly shown since the 1950s. It is now known that all forms of tobacco use, i.e., smoking and use of smokeless tobacco, are addictive and potentially lethal. Scientific evidence confirms that smokers have significantly elevated risks of death from many cancers, cardiovascular and respiratory diseases, and many other fatal conditions (US Department of Health and Education and Welfare, 1972). The harmful effects of secondhand smoke have also been convincingly established (Öberg, Jaakkola, Woodward, Peruga, & Prüss-Ustün, 2011).

Tobacco is a highly addictive substance that directly kills half of its users, as well as many nonsmokers exposed to secondhand smoke. There is no safe form of tobacco use or no safe level of exposure to secondhand smoke.

It is estimated that currently about 1 billion of men (nearly 50% of adult men) and about 250 million women (over 10% of women) in the world smoke. Smoking rates among men seem to have peaked, but among women, they are still increasing on global scale.

Tobacco kills currently annually some six million people (about 10% of world deaths), and with current trends some eight million people annually by 2030. Of the tobacco deaths some three fourth occur in low- and middle-income countries, and generally proportionally more among lower socioeconomic segments of the population. The economic costs of tobacco-related harms are enormous: both the direct costs to health services and the indirect societal costs (Shafey, Eriksen, Ross, & Mackay, 2009).

Health professionals started to warn about the harmful consequences of tobacco use already in the 1950s. Because of the disinformation and lobbying of the big tobacco industry, policy actions to reduce tobacco use started much later, generally only in the 1980s and the 1990s. A milestone was the adoption of the WHO Framework Convention on Tobacco Control (FCTC) in 2003 (World Health Organization [WHO], 2003). Currently, over 170 countries have ratified the convention that is a pioneering example of use of international law in the field of public health. FCTC covers all the main elements of tobacco control.

Elements of Tobacco Control

Reduction of Demand for Tobacco

Education and communication: Included are comprehensive educational and public awareness programs on the health risks and on the addictive nature of tobacco products and exposure to tobacco smoke. This includes also effective training programs on tobacco control to health workers, to other professional and community groups dealing potentially with tobacco control, as well as to decision makers.

Tobacco cessation: Stopping tobacco use is often difficult because of strong physical addiction to nicotine and to the psychosocial dependence to the habit. During the last few decades, pharmacological and nonpharmacological (psychological, educational) methods have been developed to effectively help smokers and other tobacco users to quit the habit. Tobacco control policies include measures to provide tobacco users access to cessation services.

Elimination of tobacco advertising and promotion: An important background for the global tobacco epidemic is the powerful push from the multinational tobacco industry in form of effective advertising, promotion, sponsorship, and lobbying of decision makers. Thus, important component of tobacco control is a comprehensive ban on advertising, promotion, and sponsorship. Here an international agreement is

especially important because of the cross-border spreading of advertising. It is also important to eliminate false or misleading messages about the tobacco products.

Price and tax measures: Price is an important aspect of use of any product. Accordingly, price and tax measures are effective and important means of reducing tobacco consumption, in particular among young persons.

Regulation on the contents of tobacco products: Although there is no safe tobacco products, authorities can introduce regulations on testing, measuring and levels of the contents and emissions of tobacco products. This can also include introduction of self-extinguishing cigarettes to reduce fires. National legislation can also regulate tobacco product disclosures.

Smoke-free environments: Exposure to tobacco smoke, especially indoors, is a health risk to everybody and especially to vulnerable population groups like children. At the same time, smoke-free environments discourage initiation and continuation of smoking. Thus, important elements of any tobacco control policies include prohibition of smoking in indoor workplaces, public transport, indoor public places, and also in other public places (e.g., stadia).

Packaging and labeling of tobacco products: Tobacco product packages and labels should not promote the product by any false, misleading, or deceptive messages. Such messages may include terms like "low tar," "light," "ultralight," or "mild." Tobacco products should carry large and clear health warnings in text or in form of pictures. Recently, also generic tobacco packages have been proposed.

Reduction of the Supply of Tobacco

Sale to minors: An important part of tobacco-related health work is prevention of tobacco use among children and youth, and moving the possible initiation to as late as possible. Thus, sale of tobacco to minors should be prohibited, and this legislation well enforced, e.g., by requiring the purchaser to provide appropriate evidence of age. Vending machines should be placed so that minors cannot use them. Regulations should also prohibit sale of tobacco products by minors, as well as sale of individual cigarettes or small cigarette packets.

Illicit trade: Surprisingly, large part of tobacco products used in the world is smuggled, manufactured illicitly, or counterfeited. Thus, elimination of illicit trade of tobacco products is important, and an issue in which international collaboration by authorities is especially needed.

Economic alternatives to tobacco business: Reduction of tobacco use calls also for reduction of tobacco growing. Thus, alternatives for tobacco growing should be encouraged, as well as also other viable alternatives for other tobacco-related occupations.

Other Aspects of Tobacco Control

Research, monitoring, and surveillance: Although the scientific base of tobacco control is very strong, further research is needed in several areas. It is also important that every country has own tobacco research. Monitoring of tobacco use trends in the population and its subgroups is crucial. It is also important to monitor many aspects of determinants and process of tobacco use as well as activities related to tobacco control.

Exchange of information: For the international collaboration and the reporting of the FCTC implementation exchange of tobacco control–related information is needed. This includes, e.g., information on legislative, administrative, and other tobacco control measures, as well information on tobacco use trends.

International collaboration in scientific, technical, and legal fields of tobacco control: Because of the global nature of the tobacco epidemic, also the tobacco control calls for strong international collaboration, much assisted by the international FCTC-related work.

Implementation of International Tobacco Control

The FCTC Convention Secretariat published in 2009 a summary report on the global progress in implementation of the FCTC (FCTC, 2009). The report concluded that the implementation levels vary substantially between different policy

measures. Overall, countries report high implementation rates for measures on packaging and labeling, sales to minors, and education, training, and public awareness. Rates remain low in areas like disclosure of marketing expenditures or programs for tobacco use cessation.

The implementation of tobacco control measures varies across different regions of the world. Also comparability of reports from different countries varies concerning both implementation measures and tobacco use data.

Overall, there seems to be notable progress in introduction and implementation of various tobacco control measures in most parts of the world.

Cross-References

- ▶ Health Promotion and Disease Prevention
- ▶ Public Health
- ▶ Risk Factors and Their Management
- ▶ Smoking Prevention Policies and Programs

References and Readings

FCTC. (2009). *Summary report on global progress in implementation of the WHO Framework Convention on Tobacco Control*. Geneva: Convention Secretariat. FCTC/2009.1.

Öberg, M., Jaakkola, M., Woodward, A., Peruga, A., & Prüss-Ustün, A. (2011). Worldwide burden of disease from exposure to second-hand smoke: A retrospective analysis of data from 192 countries. *The Lancet, 377*, 139–146.

Shafey, O., Eriksen, M., Ross, H., & Mackay, J. (2009). *The tobacco Atlas* (3rd ed.). Atlanta: American Cancer Society.

US Department of Health, Education and Welfare. (1972). *The health consequences of smoking. A report of the surgeon general:1972*. DHEW Publication No. (HSM) 72–7516. Washington DC: Author.

World Health Organization. (2003). *WHO framework convention on tobacco control*. Geneva: Author.

Tobacco Marketing

▶ Tobacco Advertising

Tobacco Policy

▶ Tobacco Control

Tobacco Promotion

▶ Tobacco Advertising

Tobacco Smoking and Health

▶ Smoking and Health

Tobacco Smoking Cessation

▶ Smoking Cessation

Tobacco Use

▶ Smoking Behavior

Tonic REM

▶ REM Sleep

Total Cholesterol

▶ Lipid

Total Cholesterol in the Blood

▶ Lipid, Plasma

Total Sleep Time

▶ Sleep Duration

Touch

▶ Massage Therapy

Traditional Chinese Medicine

▶ Acupuncture

Trail-Making Test

Romola S. Bucks
School of Psychology, The University of Western Australia (M304), Crawley, WA, Australia

Synonyms

Trails

Definition

This term refers to a widely used test assessing organized visual search, planning, attention, set shifting, cognitive flexibility, and divided attention (Rabin, Barr, & Butler, 2005), all capacities thought to be executive in nature. Originally developed by Partington (Brown & Partington, 1942), it was first published as part of the *Army Individual Test Battery* (1984). The test is currently available in public domain (see Lezak, Howieson, Loring, Hannay, & Fischer, 2004; Strauss, Sherman, & Spreen, 2006) and revised versions (e.g., Reynolds, 2002) and as part of a number of assessment batteries (e.g., Delis, Kaplan, & Kramer, 2001).

The standard trail-making test (TMT) contains two parts: Trails A and Trails B, which usually takes no more than 5–10 min to complete. In Trails A, the subject draws lines to connect consecutively numbered circles, drawn on a single A4 sheet (1-2-3. . .). In Trails B, the subject connects consecutively numbered and lettered circles, alternating between them (1-A-2-B-3. . .) on a second sheet. The subject is asked to connect the numbers, or numbers and letters, as fast as possible without lifting the pencil from the sheet. Revised versions (e.g., Delis et al., 2001; Reynolds, 2002) usually contain an equivalent to Trails B, plus up to four other subtests designed to help the assessor distinguish the cause(s) of difficulties in the switching task, such as number or letter sequencing, visual scanning, or motor deficits.

The main performance measure is time taken to complete the sequence, but errors are commonly recorded as they can also be clinically useful (Lezak et al., 2004). Because of the significant motor requirements of the task, normative data must be age-stratified (e.g., Mitrushina, Boone, & D'Elia, 1999; Tombaugh, 2004). Education-based norms are also recommended (Tombaugh, 2004). Generally, Trails B is thought to require more executive skills because it requires shifting between sequences (Kortte, Horner, & Windham, 2002). Evidence suggests that the difference between Trails A and B, or their ratio, may be "cleaner" indices of executive function by controlling for baseline motor, visual tracking, and sequencing abilities (Arbuthnott & Frank, 2000; see e.g., Hester, Kinsella, Ong, & McGregor, 2005, for difference and ratio norms). Indeed, fMRI evidence supports this view: Zakzanis, Mraz, & Graham (2005) found greater left frontal activation in the dorsolateral prefrontal cortex during Trails B than Trails A.

Both parts of the TMT are highly sensitive to dementia and brain injury, including Parkinson's (Goldman, Baty, Buckles, Sahrmann, & Morris, 1998) and Alzheimer's disease (Chen et al., 2000). Importantly, deficits in TMT performance predict everyday activities of daily living difficulties (Bell-McGinty, Podell, Franzen, Baird, & Williams, 2002) and mortality (Vazzana et al., 2010) and may indicate preclinical Alzheimer's dementia (Chen et al., 2000).

Cross-References

- ▶ Executive Function
- ▶ Neuropsychology

References and Readings

Arbuthnott, K., & Frank, J. (2000). Trail making test, part B as a measure of executive control: Validation using a set-switching paradigm. *Journal of Clinical and Experimental Neuropsychology, 22*, 518–528.

Bell-McGinty, S., Podell, K., Franzen, M., Baird, A. D., & Williams, M. J. (2002). Standard measures of executive function in predicting instrumental activities of daily living in older adults. *International Journal of Geriatric Psychiatry, 17*, 828–834.

Brown, R. R., & Partington, J. E. (1942). The intelligence of the narcotic drug addict. *Journal of General Psychology, 26*, 175–179.

Chen, P., Ratcliff, G., Belle, S. H., Cauley, J. A., DeKosky, S. T., & Ganguli, M. (2000). Cognitive tests that best discriminate between presymptomatic AD and those who remain nondemented. *Neurology, 55*, 1847–1853.

Delis, D., Kaplan, E., & Kramer, J. (2001). *Delis-Kaplan executive function scale*. San Antonio, TX: The Psychological Corporation.

Goldman, W. P., Baty, J. D., Buckles, V. D., Sahrmann, S., & Morris, J. C. (1998). Cognitive and motor functioning in Parkinson disease: Subjects with and without questionable dementia. *Archives of Neurology, 55*, 674–680.

Hester, R. L., Kinsella, G. J., Ong, B., & McGregor, J. (2005). Demographic influences on baseline and derived scores from the trail making test in healthy older Australian adults. *The Clinical Neuropsychologist, 19*, 45–54.

Kortte, K. B., Horner, M. D., & Windham, W. K. (2002). The trail making test, part B: Cognitive flexibility or ability to maintain set? *Applied Neuropsychology, 9*, 106–109.

Lezak, M. D., Howieson, D. B., Loring, D. W., Hannay, H. J., & Fischer, J. S. (2004). *Neuropsychological assessment* (4th ed.). New York: Oxford University Press.

Mitrushina, M. N., Boone, K. L., & D'Elia, L. (1999). *Handbook of normative data for neuropsychological assessment*. New York: Oxford University Press.

Rabin, L. A., Barr, W. B., & Butler, L. A. (2005). Assessment practices of clinical neuropsychologists in the United States and Canada: A survey of INS, NAN, APA Division 40 members. *Archives of Clinical Neuropsychology, 20*, 33–65.

Reynolds, C. R. (2002). *Comprehensive trail-making test*. Austin, TX: PRO-ED.

Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). *A compendium of neuropsychological tests: Administration, norms, and commentary*. New York: Oxford University Press.

Tombaugh, T. N. (2004). Trail making test A and B: Normative data stratified by age and education. *Archives of Clinical Neuropsychology, 19*, 203–214.

Vazzana, R., Bandinelli, S., Lauretani, F., Volpato, S., Lauretani, F., Di Iorio, A., et al. (2010). Trail making test predicts physical impairment and mortality in older persons. *Journal of American Geriatrics Society, 58*, 719–723.

Zakzanis, K. K., Mraz, R., & Graham, S. J. (2005). An fMRI study of the trail making test. *Neuropsychologia, 43*, 1878–1886.

Trails

- ▶ Trail-Making Test

Trait Anger

Judith Carroll
Cousins Center for Psychoneuroimmunology, University of California, Los Angeles, CA, USA

Synonyms

Hostile affect; Hostility

Definition

Trait anger is described as a dispositional characteristic where one experiences frequent anger, with varying intensity (e.g., mild irritability, intense rage), and is often accompanied by related negative emotions such as envy, resentment, hate, and disgust (Buss, 1961; Siegman & Smith, 1994). There is considerable construct overlap between hostile dispositions and trait anger, making it difficult to disentangle. Martin, Watson, and Wan (2000) have proposed a three-factor model of trait anger, which includes the anger-related affect, behavior (i.e., aggression), and cognitions (i.e., cynicism), similar to several of the subscales

of the Cook-Medley Hostility Scale (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989). A frequently used measure of trait anger is the Spielberger State-Trait Anger Expression Inventory (STAXI), which measures trait anger as having a proneness to experiencing anger either as a general tendency (*Anger temperament)*, or with provocation (*Anger Reactions*) (Spielberger, 1988, Spielberger & Sydeman, 1994). Furthermore, Speilberger describes three different styles of anger expression: (1) showing anger emotions (*Anger-Out*), (2) preventing anger from being expressed but still experiencing it internally (*Anger-In*), or (3) having the initial affective response but then regulating it well (*Anger-Control*) (Spielberger, 1988).

Behavioral medicine research has documented associations of trait anger, and the related constructs of hostility, with greater cardiovascular disease incidence and progression (al'Absi & Bongard, 2006; Chida & Steptoe, 2009; Miller, Smith, Turner, Guijarro, & Hallet, 1996; Siegman & Smith, 1994; Smith, Glaser, Ruiz, & Gallo, 2004). Although poor health behaviors are thought to partially explain these associations (Everson et al., 1997; Siegman & Smith, 1994), it is also likely that this trait disposition contributes to worse health through the repeated emotionally driven activation of the neuroendocrine stress response and its associated downstream biological effects, including increases in blood pressure, inflammation, and oxidative stress (Carroll et al., 2010, 2011; Greeson et al., 2009; Smith & Gallo, 1999; Suarez, Kuhn, Schanberg, Williams, & Zimmermann, 1998). Further work is needed to better define the mechanisms of this association.

Cross-References

- ▶ Anger Expression
- ▶ Anger, Measurement
- ▶ Anger-In
- ▶ Anger-Out
- ▶ Hostility, Cynical
- ▶ Hostility, Measurement of

References and Readings

al'Absi, M., & Bongard, S. (2006). Neuroendocrine and behavioral mechanisms mediating the relationship between anger expression and cardiovascular risk: Assessment consideration and improvements. *Journal of Behavioral Medicine, 29*, 573–591.

Barefoot, J. C., Dodge, K. A., Peterson, B. L., Dahlstrom, G., & Williams, R. B. (1989). The cook-medley hostility scale: Item content and ability to predict survival. *Psychosomatic Medicine, 51*, 46–57.

Buss, A. H. (1961). *The psychology of aggression*. New York: Wiley.

Carroll, J. E., Low, C. A., Prather, A. A., Cohen, S., Fury, J. M., Ross, D. C., et al. (2011). Negative affective responses to a speech task predict changes in interleukin (IL)-6. *Brain, Behavior, and Immunity, 25*, 232–238.

Carroll, J. E., Marsland, A. L., Jenkins, F., Baum, A., Muldoon, M. F., & Manuck, S. B. (2010). A urinary marker of oxidative stress covaries positively with hostility among midlife community volunteers. *Psychosomatic Medicine, 72*(3), 273–280.

Chida, Y., & Steptoe, A. (2009). The association of anger and hostility with future coronary heart disease: A meta-analytic review of prospective evidence. *Journal of the American College of Cardiology, 53*(11), 936–946.

Everson, S. A., Kauhanen, J., Kaplan, G. A., Goldberg, D. E., Julkunen, J., Tuomilehto, J., et al. (1997). Hostility and increased risk of mortality and acute myocardial infarction: The mediating role of behavioral risk factors. *American Journal of Epidemiology, 146*(2), 142–152.

Greeson, J. M., Lewis, J. G., Achanzar, K., Zimmerman, E., Young, K. H., & Suarez, E. C. (2009). Stress-induced changes in the expression of monocytic beta2-integrins: The impact of arousal of negative affect and adrenergic responses to the anger recall interview. *Brain, Behavior, and Immunity, 23*(2), 251–256.

Martinn R., Watson, D., & Wan, C. K. (2000). A three-factor model of trait anger: dimensions of affect, behavior, and cognition. *Journal of Personality*, *68*, 869–897.

Miller, T. Q., Smith, T. W., Turner, C. W., Guijarro, M. L., & Hallet, A. J. (1996). A meta-analytic review of research on hostility and physical health. *Psychological Bulletin, 119*, 322–348.

Siegman, A. W., & Smith, T. W. (Eds.). (1994). *Anger, hostility, and the heart*. Hillsdale, NJ: Erlbaum.

Smith, T. W. (1992). Hostility and health: Current status of a psychosomatic hypothesis. *Health Psychology, 11*(3), 139–150.

Smith, T. W., & Gallo, L. C. (1999). Hostility and cardiovascular reactivity during marital interaction. *Psychosomatic Medicine, 61*(4), 436–445.

Smith, T. W., Glazer, K., Ruiz, J. M., & Gallo, L. C. (2004). Hostility, anger, aggressiveness, and coronary heart disease: An interpersonal perspective on personality, emotion, and health. *Journal of Personality, 72*(6), 1217–1270.

Spielberger, C. D. (1988). *Manual for the state-trait anger expression inventory (STAXI)*. Odessa, FL: Psychological Assessment Resources.
Spielberger, C., & Sydeman, S. J. (1994). State-trait anxiety inventory and state-trait anger expression inventory. In M. E. Maruish (Ed.), *The use of psychological testing for treatment planning and outcome assessment* (pp. 292–321). Hillsdale, NJ: Erlbaum.
Suarez, E. C., Kuhn, C. M., Schanberg, S. M., Williams, R. B., Jr., & Zimmermann, E. A. (1998). Neuroendocrine, cardiovascular, and emotional responses of hostile men: The role of interpersonal challenge. *Psychosom Medicine, 60*(1), 78–88.

Trait Anxiety

Yori Gidron
Faculty of Medicine & Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

Trait anxiety refers to the stable tendency to attend to, experience, and report negative emotions such as fears, worries, and anxiety across many situations. This is part of the personality dimension of neuroticism versus emotional stability. Trait anxiety also manifests by repeated concerns about and reporting of body symptoms. Trait anxiety is characterized by a stable perception of environmental stimuli (events, others' statements) as threatening. Trait-anxious people often experience and express also state anxiety, in situations in which most people do not experience such responses. This bias is thought to reflect a cognitive-perceptual bias. At the perceptual level, there is an overattentional bias to threatening stimuli. At the cognitive level, there is a distorted negative interpretation of information congruent with and fostering anxious responses. Finally, at the level of memory, there is overrecall of threatening information. These three biases are common in people with a trait-anxious personality type and have important etiological roles in various types of affective disorders (Mathews & Macleod, 2005). Trait anxiety is commonly assessed with the state-trait anxiety inventory – trait version (Spielberger, Gorsuch, & Lushene, 1970), though other instruments exist as well. Trait anxiety is an important predictor and moderator in behavior medicine. For example, trait anxiety predicts functional recovery following spine surgery, risk of posttraumatic stress disorder, as well as adaptation to and risk of death following myocardial infarction (e.g., Szekely et al., 2007). These relationships could occur since trait anxiety is related to various coping strategies and to various neurophysiological responses. For example, high trait-anxious people demonstrate greater activity in the amygdala and reduced activity in the inhibitory dorsal anterior cingulate cortex, during extinction of fear responses (Sehlmeyer et al., 2011). This brain pattern can explain their increased vulnerability for psychological disorders and adaptation problems. As such, this psychological trait deserves attention in research and clinical applications of behavior medicine. The underlying causes, mechanisms for contributing to poor health outcomes, and ways for reducing the consequences of trait anxiety are important avenues of research for the benefit of clinical practice.

Cross-References

▶ Anxiety and Heart Disease
▶ Anxiety and Its Measurement
▶ Anxiety Disorder

References and Readings

Mathews, A., & MacLeod, C. (2005). Cognitive vulnerability to emotional disorders. *Annual Review of Clinical Psychology, 1*, 167–195.
Sehlmeyer, C., Dannlowski, U., Schöning, S., Kugel, H., Pyka, M., Pfleiderer, B., et al. (2011). Neural correlates of trait anxiety in fear extinction. *Psychological Medicine, 41*, 789–798.
Spielberger, C. D., Gorsuch, R. L., & Lushene, R. E. (1970). *Manual for the state-trait anxiety inventory*. Palo Alto, CA: Consulting Psychologists Press.
Székely, A., Balog, P., Benkö, E., Breuer, T., Székely, J., Kertai, M. D., et al. (2007). Anxiety predicts mortality and morbidity after coronary artery and valve surgery-a 4-year follow-up study. *Psychosomatic Medicine, 69*, 625–631.

Traits

▶ Personality

Trans Fats

▶ Fat, Dietary Intake
▶ Trans Fatty Acids

Trans Fatty Acids

Leah Rosenberg
Department of Medicine, School of Medicine, Duke University, Durham, NC, USA

Synonyms

Trans fats

Definition

Trans fatty acids are unsaturated fatty acids with one double bond in the *trans* structural configuration as opposed to the *cis* conformation. These differences in conformation likely have consequences in the development of atherosclerosis secondary to diets rich in trans fatty acids. Dyslipidemia and adverse health outcomes have been linked to frequent consumption of *trans* fatty acids. While *trans* fatty acids do appear in nature, the vast majority in industrialized-world diets are manufactured to promote shelf life stability and enhance flavor in prepared foods. Preventative approaches to cardiovascular disease prevention and management include elimination of *trans* fatty acid consumption (Curhan & Mitch, 2007). Certain municipalities, such as New York City, have recently enacted prohibitions against the use of *trans* fatty acids in restaurant food.

References and Readings

Curhan, G. C., & Mitch, W. E. (2007). Chapter 53-Diet and kidney disease. In *Section IX – Conservative and pharmacologic management of kidney disease* (Brenner and Rector's the kidney 8th ed.). Philadelphia: Saunders.

Transactional Model

▶ Cognitive Appraisal

Transcendental Meditation

Alan M. Delamater
Department of Pediatrics, University of Miami Miller School of Medicine, Miami, FL, USA

Synonyms

Attention training; Concentration; Contemplation; Meditation; Mental training

Definition

Transcendental meditation (TM) is a meditation technique that has its origins in the ancient Vedic tradition of India. In the 1960s, Maharishi Mahesh Yogi introduced this meditative technique to the western world in a simple, nonreligious fashion, and since then TM has been practiced by millions of people worldwide. A considerable amount of research has been conducted on the effects of TM on physiological and psychological outcomes. Overall, the results of this research indicate that the practice of TM has beneficial effects in individuals with chronic health conditions as well as healthy people.

TM is classified as a concentrative meditation technique. The method consists of twice-daily 20 min practice in which the individual focuses on their mantra which is individually prescribed by a certified instructor. The individual is

instructed to sit in a relaxed posture in a quiet environment and focus on the silent repetition of their mantra in their mind to the exclusion of other thoughts or feelings.

As concentration deepens, feelings of calm or tranquility are experienced. Research has shown that when individuals practice this type of meditation, they experience a restful hypometabolic state in which their respiration, heart-rate, blood pressure, muscle tension, and other indicators of sympathetic nervous system activation all decrease. This state of hypometabolic, restful alertness has been termed "the relaxation response." The relaxation response can be reliably elicited by the repetition of a mental stimulus (e.g., a mantra) while the individual adopts a relaxed mental attitude in a quiet environment.

Cross-References

- ▶ Meditation
- ▶ Mindfulness
- ▶ Relaxation

References and Readings

Anderson, J. W., et al. (2008). Blood pressure response to transcendental meditation: A meta-analysis. *American Journal of Hypertension, 21*(3), 310–316.

Benson, H. (1975). *The relaxation response*. New York: Morrow.

Chandler, H. M., et al. (2005). Transcendental meditation and postconventional self-development: A 10-year longitudinal study. *Journal of Social Behavior and Personality, 17*(1), 93–121.

Dillbeck, M. C., & Orme-Johnson, D. W. (1987). Physiological differences between transcendental meditation and rest. *American Psychologist, 42*, 879–881.

Goleman, D. (1988). *The varieties of the meditative experience*. New York: Tarcher.

Mahesh Yogi, M. (1963). *Transcendental meditation*. New York: New American Library.

Paul-Labrador, M., et al. (2006). Effects of randomized controlled trial of transcendental meditation on components of the metabolic syndrome in subjects with coronary heart disease. *Archives of Internal Medicine, 166*, 1218–1224.

So, K. T., & Orme-Johnson, D. W. (2001). Three randomized experiments on the holistic longitudinal effects of the transcendental meditation technique on cognition. *Intelligence, 29*(5), 419–440.

Walton, K. G., et al. (2004). Review of controlled clinical research on the transcendental meditation program and cardiovascular disease: Risk factors, morbidity, and mortality. *Cardiology in Review, 12*(5), 262–266.

Transducer

Yori Gidron
Faculty of Medicine and Pharmacy, Free University of Brussels (VUB), Jette, Belgium

Definition

In physics, a transducer is a device or system which converts one type of energy to another type. In biology, this term can refer to cells or intracellular elements which transform one form of input into another. Both are applicable for behavior medicine. Looking at devices, transducers are found in any machine which measures bodily parameters and depicts them electronically. A device measuring heart rate or pulse can detect changes in light in blood vessels, which reflect amount of blood as a function of one's heart rate. These changes in light are sensed, for example, by photoresistors, which are then translated to changes in electrical energy (current), which is then translated to digital numbers reflecting the rate of change in heart rate. Such devices are pivotal in medical diagnosis and in psychophysiological research. Another example of a device would be a galvanic skin conductance measure, which detects changes in electrical conductance of the skin, which reflects sympathetic activity and input into the skin. The conductance is translated into a digital representation, to reflect sympathetic activity. This too is used in psychophysiological research on stress responses.

Biologically, numerous transducers exist in the pathways of the sensory system and in cells. In the eyes, for example, the retina contains numerous photoreceptor cells that contain

molecules called opsins. These photoreceptor cells synapse onto neuronal pathways and, via signal transducers, convert light energy detected by the opsins to neuronal energy, for visual processing in the brain. In the auditory system, sound reaches the middle ear after being channeled by the ear's shape. The eardrum and bones carry vibrations to the inner ear, where physical movements are transformed to fluid movement in the cochlea. This fluid movement excites hair cells in the basilar membrane that generates, via transduction, neuronal signals to the auditory cortex for higher auditory processing. Another example is the neuroendocrine transducer, where a neuron, for example, in the pituitary gland, translates electrical stimulation in its input to secretion of hormones at its output. In recent years, the vagus nerve has been found to be a pivotal neuroimmune transducer since its paraganglia express receptors for interleukin-1. Upon signaling by that cytokine, neuronal information is carried to the brain via acetylcholine, thus translating immune to nerve information, which then triggers several negative feedback anti-inflammatory loops (Tracey, 2009). Transducers also play major roles in diseases. In cancer, for example, among multiple intracellular signaling pathways, the signal transducer and activator of transcription 3 (STAT3) is a transcription factor which is active upon extracellular activation by many signals including cytokines and growth factors. STAT3 plays a role in cell apoptosis and growth. In some cancers, constant activity of STAT3 is related to procarcinogenic activity and to poor prognosis (e.g., Alvarez, Greulich, Sellers, Meyerson, & Frank, 2006). Thus, transducers are omnipresent in the body (or in devices) and are crucial for communication between the body and the external world as well as between different types of signals inside the body, in relation to health and disease.

Cross-References

► Psychophysiological

References and Readings

Alvarez, J. V., Greulich, H., Sellers, W. R., Meyerson, M., & Frank, D. A. (2006). Signal transducer and activator of transcription 3 is required for the oncogenic effects of non-small-cell lung cancer-associated mutations of the epidermal growth factor receptor. *Cancer Research, 66*, 3162–3168.

Tracey, K. J. (2009). Reflex control of immunity. *Nature Reviews Immunology, 9*, 418–428.

Trans-fatty Acids

► Fat: Saturated, Unsaturated

Transfer RNA

► RNA

Transformational Coping

► Posttraumatic Growth

Translational Behavioral Medicine

Bonnie Spring, Michael James Coons, Jennifer Duncan, Alyson Sularz and Justina Deary
Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Synonyms

Implementation; Integrated behavioral medicine research, practice, policy; Research to practice translation

Definition

Translational behavioral medicine (TBM) is an approach that concerns the transfer of knowledge

from the psychosocial and biomedical sciences in order to develop behavioral interventions to improve health, evaluate the effectiveness of those interventions, and study and improve their implementation in practice and policy. The overarching objective of TBM is to advance, integrate, and actualize knowledge from the research, practice, and policy arenas to improve the health of individuals and communities. *Translational Behavioral Medicine: Practice, Policy, Research*, a scholarly professional journal devoted to the topic, was established in 2011 by the Society of Behavioral Medicine, with founding editor, Bonnie Spring.

Description

In 2001, the Institute of Medicine (IOM) published a report on the quality of health care in the United States. The IOM perceived a chasm between the health care Americans receive and the kind they could and should receive. They attributed this gap largely to inadequate translation of scientific discoveries into actual practices. An often-cited statistic is that it takes 15–20 years for a scientific discovery to influence clinical practice (Balas and Boren, 2000). Moreover, even when a research-supported treatment does become recognized as a best practice, practitioner adherence is highly variable.

"Translation" is the process of adapting theoretical principles and empirical findings from research so that these can be applied to the worlds of clinical and public health practice (Sung et al., 2003; Westfall, Mold, & Fagnan, 2007; Woolf, 2008). The translation process proceeds through a series of phases, as illustrated in the conceptual model by Westfall et al. (2007), shown in Fig. 1.

T1 Translation

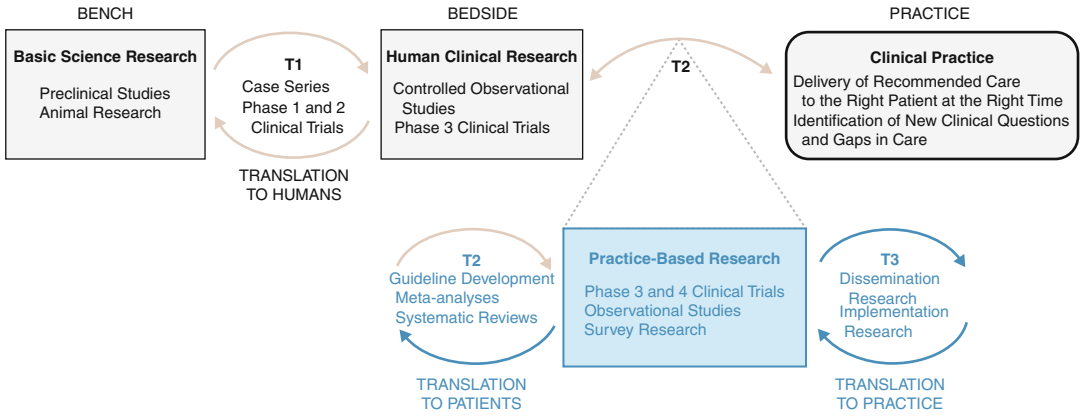
The first translational phase (T1) is focused on using knowledge obtained from the study of basic biological, psychosocial, and behavioral processes to inform the development and refinement of promising interventions for health conditions. T1 research is sometimes called "bench to bedside," as it provides the first link from basic science to human clinical studies. Basic scientists address fundamental questions about mechanisms that underlie human functioning. T1 researchers then apply these understandings of biopsychosocial mechanisms to develop efficacious behavioral interventions and assessments of how they work. T1 research can be performed using a number of different types of research designs.

Case Series

A case series (or clinical series) is a form of observational research design. In a T1 case series to develop a new treatment, a single individual or small group of individuals is observed (either prospectively or retrospectively using recorded information). The aim is to examine whether there is an association between exposure to the treatment elements and a clinical event (e.g., symptom improvement or remission). Usually applied in the earliest phases of treatment development, a series of cases may be gathered to establish "proof of concept" that a new treatment holds sufficient promise to warrant further study. An advantage of case series studies is that they capture clinical events in a naturalistic context. Disadvantages are that they examine small, highly selected samples of people who may be atypical, and can demonstrate only correlation, not causation.

Randomized Controlled Trials

Randomized controlled trials (RCTs) are the gold standard method of testing whether a treatment works. After participants are screened for inclusion and exclusion criteria, they are assigned randomly to two or more groups or conditions. In a two-group design, participants in one group receive the active intervention (e.g., smoking cessation treatment), and participants in the other group receive a control intervention (e.g., general health education) that is comparable in some elements (e.g., credibility, contact time) but inert in the active elements (e.g., specific skills training) thought responsible for the treatment's effect. The primary outcome might be change from baseline in the number of study participants who smoke. Because the group allocation is concealed until randomization has occurred,



Translational Behavioral Medicine, Fig. 1 Translational research phases from the NIH roadmap. Reprinted with permission from Westfall, Mold, & Fagnan (2007)

neither investigator nor patient can influence the treatment assignment, so that participants have an equal chance of being assigned to either the intervention or control group. This enables researchers to eliminate any bias that might otherwise occur in the group assignment.

Two different randomization procedures are employed in RCTs. They are: (1) fixed allocation randomization (which includes simple, blocked, or stratified randomization), and (2) adaptive randomization (which includes baseline adaptive- or response adaptive randomization). Simple randomization is analogous to repeated fair-coin tossing. However, this procedure is prone to creating imbalanced group sizes. Blocked randomization (also referred to as permuted block randomization) instead ensures that at no time during the randomization will the difference between group sizes be large, and at some points, groups will be equal. Stratified randomization helps to ensure the even distribution of certain factors (e.g., gender) between the groups or conditions. In adaptive randomization, the probability of being randomized to different groups changes as the study progresses. Altering the randomization procedure can help to overcome imbalances based on differences in participants' baseline characteristics (i.e., baseline adaptive randomization) or based on their responses at a later point in the study (i.e., response adaptive randomization).

In RCTs testing drug treatments, the use of identical appearing pills to contain active and inactive agents makes it possible to keep both participants and study personnel naïve to group assignment, a state of affairs referred to as double blind. When only participants are naive, the trial is described as being single blind. Blinding participants and personnel to study conditions helps to ensure that treatment effects are due to the intervention, rather than person-level factors (e.g., knowledge or expectancies about the treatment or outcome). However, double blinding is rarely feasible in trials of behavioral interventions: Both patients and interventionists usually know which treatment is being given. One important form of blinding that does remain feasible is blinding of outcome assessors. When blinded, the assessors who evaluate study outcomes are unaware of whether patients belong to the treatment or the control group.

RCTs are the gold standard for evaluating treatments because this design surpasses others in its internal validity. This means that differences between the study groups in their outcome can be attributed to the treatment, because the researchers held constant other extraneous variation between the groups. The presence of the control group enables the researcher to account for shared influences, such as being repeatedly assessed or receiving attention from professionals. Equally important is the need to establish treatment fidelity; that is, that the intended intervention was delivered as planned. Fidelity is induced by training and supervising therapists to

follow a treatment protocol (i.e., a treatment manual or algorithm) and is assessed by monitoring to ensure that critical intervention elements are delivered. The RCT's internal validity permits researchers to make causal inferences; that is, to attribute between group differences in patient outcomes to variation in the treatment. Its drawbacks are that RCTs are expensive and time consuming to implement, and that random assignment of study participants to conditions is not always feasible. Just as an RCT's validity is compromised by low-quality design or execution, its utility is undermined by incomplete reporting. To facilitate comprehensive, uniform reporting of RCTs, most scholarly journals in health have adopted the international CONSORT guidelines which guide the information to be reported when publishing a RCT (Moher et al., 2010).

Intervention Development via Optimization Design

Behavioral medicine interventions usually combine multiple treatment components brought together in a compilation intended to achieve maximal benefit. For example, an exercise intervention may combine individual coaching from an exercise physiologist with peer support groups, a free gym membership, and incentives for monitoring physical activity and reaching behavioral goals. Although the practice of testing a bundled treatment package in an RCT maximizes the likelihood of detecting a treatment effect, it can be inefficient for long-term policy. After a treatment package has been found effective, it remains unclear which treatment components have produced the positive effect, whether some treatment elements are inert and could be eliminated (potentially reducing costs), and whether the dose and timing of other components is optimal. Subsequent "dismantling" trials are needed to answer those questions.

Multiphase optimization strategy (MOST), adapted from engineering science, is a methodology designed to build new interventions from the ground up by first optimizing and evaluating the contribution of multiple intervention components. MOST follows a sequential, stepped approach to intervention development. The first step is to establish a conceptual, theoretical model of how the eventual intervention should produce benefit and apply the model to derive the intervention components to be examined. The second step involves experimentation to examine the impact of individual intervention components. That stage may be followed by further experimentation to refine and optimize the components (e.g., by modifying their dose, timing, format, or delivery channel). Once the individual treatment components have been optimized, the third step is to assemble the treatment package (the beta intervention) and confirm its efficacy via an RCT. If the trial proves successful, the fourth step is that the new intervention can be released and tested further for effectiveness.

Note that the MOST approach delays the RCT of a bundled treatment package until the third step in intervention development. A key feature of the MOST strategy is that each subsequent intervention will have been engineered, and empirically validated to be superior to the previous one on whatever optimization parameter the interventionist desires. For example, a treatment can be optimized to have no inactive components, to produce the maximum change attainable for a given level of financial resource or time, to maximize the number of people that can be exposed to an intervention, etc. By optimizing for a specific context, MOST emphasizes efficiency and careful management of resources to increase the implementation rate of science.

T2 Translation

The second translational phase (T2), sometimes called "bench to trench," is concerned with evaluating the effectiveness of interventions under conditions that become progressively less controlled and more representative of the general population and usual practice settings. For example, the studies conducted during T1 are usually experiments or phase I and phase II clinical trials. These trials are characterized by their strict control of extraneous variables. The intent of a phase I clinical trial is to pilot test the intensity, timing, duration, or format of an intervention, with participant safety being a primary outcome.

Consequently, phase I trials are often conducted with small samples of participants, who are relatively free of complex medical histories. Once patient safety is established, phase II trials are undertaken. The goal of a phase II clinical trial is to evaluate the efficacy of an intervention for the treatment of a specific, circumscribed problem. Efficacy testing is performed under optimal conditions; for example, in an academic medical setting, employing highly trained research staff as interventionists, and involving patients without co-occurring health conditions. The progression to T2 research introduces phase III trials, in which local staff in a community setting may deliver an intervention as part of their regular job duties. Such trials impose few exclusion criteria and enroll patients even if they have comorbidities. Phase III trials are often called studies of effectiveness (in contrast to efficacy) because they involve more "real world," less highly selected settings, interventionists, and patients.

Another component of T2 research involves the creation of systematic evidence reviews and practice guidelines. Unlike primary research, which involves the collection of new data, systematic evidence reviews are secondary research that culls and combines information from prior reports. The science of systematic reviewing is in itself a sophisticated and evolving field with many nuances that surround the unbiased acquisition of publications, extraction and analysis of data, and interpretation of results. Systematic reviews offer a means to evaluate whether the evidence about a treatment's effectiveness is strong and consistent enough to warrant widespread application in practice. If the data are plentiful and the studies sufficiently similar, a systematic review can provide evidence about whether a treatment's effects are broadly generalizable. In other words, the review can indicate whether there are boundary conditions or types of people for whom the treatment is less helpful or even contraindicated. The comprehensive, unbiased evidence base analyzed for a systematic review affords an excellent grounding from which experts can formulate practice guidelines. The dissemination of evidence-based guidelines concludes the T2 translational phase by conveying best research-tested practices to clinicians and policy makers.

T3 Translation

The third translational phase (T3) is also called Dissemination and Implementation (D & I) research. D & I studies examine how to facilitate the uptake of evidence-based (research-supported) interventions into routine, day-to-day provision of clinical care and public health services. In contrast to T1 and T2, which concern determining whether treatments work (and for whom), T3 research examines how to get effective treatments widely implemented in real-world settings. D & I research focuses on identifying and learning how to overcome barriers at the practitioner, institutional, and system levels that keep effective treatments from being used. Barriers may include limitations in clinician training or skills, lack of available resources for training, competing institutional priorities, or policy barriers (e.g., lack of insurance reimbursement).

Quasi-Experimental Designs

Quasi-experimental designs emerged from social science research because true experimentation (i.e., randomization of participants to groups or conditions) was sometimes challenging to implement. These designs gained popularity with the development of advanced statistical procedures to control for the effect of extraneous variables associated with group membership. In essence, these procedures help researchers to overcome some of the limitations of non-randomization. Quasi-experimental designs are often used when randomization of participants to groups or conditions is infeasible (or impossible). Suppose, for example, a researcher wished to study the effect of preventive care reimbursement policy on the frequency with which clinicians counsel patients for smoking and obesity. Ideally, one would randomize practices to different levels of preventive care coverage. However, that will not be feasible. Therefore, instead of being randomized, practices will be grouped into those where most patients have preventive care coverage versus those where most patients lack such coverage. Quasi-experimental designs are more susceptible than

RCTs to confounding variables that may affect the outcomes of interest. For example, universal preventive coverage may prevail in cooperative, single payer systems (e.g., Group Health, Kaiser Permanente) that are geographically and demographically distinct from practices where majority preventive care is rarer. It will not be possible to determine whether these extraneous differences or differences in insurance coverage account for observed variations in clinician counseling behavior.

Examples of quasi-experimental designs include: time-series designs (i.e., following a single group of participants longitudinally to obtain a large number of data points), single group pretest posttest design, and case–control design (i.e., observational design where participants selected for a certain condition, such as insomnia treated with cognitive behavioral therapy, are compared with a control group whose insomnia is untreated). The advantage of quasi-experimental designs is that they are broadly applicable and easy to implement. For policy interventions or other contexts that preclude randomization, quasi-experimental designs may be the only option available. Their disadvantage is that non-randomization prevents researchers from being able to make causal inferences about what generated the outcomes, particularly because confounding variables (both measured and unmeasured) offer alternative explanations.

Cross-References

- ▶ Evidence-Based Behavioral Medicine (EBBM)
- ▶ Research to Practice Translation

References and Readings

Balas, E. A., & Boren, S. A. (2000). Managing clinical knowledge for health care improvement. In: J. Bemmel, A.T. McCray (eds.). *Yearbook of Medical Informatics 2000: Patient-centered Systems*. (pp. 65–70). Stuttgart, Schattauer Verlagsgesellschaft mbH.

Dougherty, D., & Conway, P. H. (2008). The "3T's" road map to transform US health care: The "how" of high-quality care. *JAMA: The Journal of the American Medical Association, 299*, 2319–2321.

Hopewell, S., Dutton, S., Yu, L., Chan, A., & Altman, D. (2010). The quality of reports of randomized trials in 2000 and 2006: Comparative study of articles indexed in PubMed. *BMJ, 340*, c1432.

Moher, D., Hopewell, S., Schulz, K., Montori, V., & Gotzsche, P. (2010). Consort 2010 explanation and elaboration: Updated guidelines for reporting parallel group randomized trials. *BMJ, 340*, c869.

Spring, B. (2011). Translational behavioral medicine: A pathway to better health. *Translational Behavioral Medicine, 1*(1), 1–3.

Sung, N. S., Crowley, W. F. Jr, Genel, M., Salber, P., Sandy, L., Sherwood, L. M., Johnson, S. B., Catanese, V., Tilson, H., Getz, K., Larson, E. L., Scheinberg, D., Reece, E. A., Slavkin, H., Dobs, A., Grebb, J., Martinez, R. A., Korn, A., & Rimoin, D. (2003). Central challenges facing the national clinical research enterprise. *JAMA:The Journal of the American Medical Association*, *289*(10), 1278–1287.

Westfall, J. M., Mold, J., & Fagnan, L. (2007). Practice-based research-"Blue Highways" on the NIH roadmap. *JAMA: The Journal of the American Medical Association, 297*(4), 403–406.

Woolf, S. H. (2008). The meaning of translational research and why it matters. *JAMA: The Journal of the American Medical Association, 299*(2), 211–213.

Translational Research

▶ Research to Practice Translation

Transmethylation

▶ Methylation

Transtheoretical Model of Behavior Change

James O. Prochaska
Clinical and Health Psychology, University of Rhode Island, Kingston, RI, USA

Synonyms

Stages-of-change model

Definition

The Transtheoretical Model (TTM) construes behavior change as an intentional process that unfolds over time and involves progress through a series of six stages of change (Prochaska, DiClemente, & Norcross, 1992a). TTM integrates processes and principles of change from across leading theories, hence the name Transtheoretical.

Description

Precontemplation is the initial stage in which individuals are not intending to take action in the foreseeable future, usually assessed as the next 6 months.

People can be in this stage due to a lack of awareness of the health consequences of a behavior.

Or, they can be demoralized about their abilities to change, like millions of people who have tried to lose weight multiple times in multiple ways. This stage is often misunderstood to mean that these people do not want to change.

The history of demoralized individuals indicates that they want to change, but they have given up on their abilities to change.

Contemplation is the stage in which individuals are intending to change in the next 6 months, but not immediately in the next month. These individuals are more aware of the benefits or pros of changing, but can also be acutely aware of the cons, such as having to give up some of their favorite foods or having to risk failure. Decisional conflict between the pros and cons can lead to profound ambivalence reflected in the motto: "When in doubt, don't act." With smokers intending to quit for good in the next 6 months, without help, less than 50% will quit for 24 h in the next 12 months.

In the preparation stage, individuals are intending to take action in the next month. Their number one concern is, "If I act, will I fail?" The emphasis here is helping them to be well-prepared, because people know in growing up, the better prepared they are in academics or athletics, the more likely they are to reach their goal.

In the Action stage, change is typically overt and observable, with individuals having quit smoking, started exercising, or practicing stress management. This is the busiest stage, where people have to work the hardest to keep from regressing or returning to an earlier stage. Many people believe the worst risks for relapse will be over in a few days or few weeks. We find that people who progress through action work the hardest for about 6 months, which happens to represent the steepest part of relapse curves across addictions (Prochaska & DiClemente, 1983). So, Action is defined as 6 months being risk-free and we encourage individuals to think of this time as the behavior medicine equivalent of life-saving surgery. Following such surgery, would they give themselves 6 months to recover? Will they let others know they will not be at their best and will need more support? This is the type of priority needed to progress through this tough time.

Maintenance is the stage in which people are free from their problem for 6 months to 5 years. People are considered cured from cancer after 5 years without symptom remission. For many people, it may take 5 years to get free from behavioral causes of cancer. During this stage, individuals do not have to work as hard, but they do have to be prepared to cope with the most common causes of relapse. These causes are times of distress, when people are anxious, depressed, lonely, bored, or stressed. Average Americans cope with such distress by increasing unhealthy habits. We try to prepare people to cope with such temptations through healthy alternatives, like talking with a supportive person, walking, or relaxing.

Termination is the stage in which people are totally confident that they are never going back to their high-risk behavior and have no temptation to return. We have found that of alcoholics and smokers in their first 5 years of abstinence, about 20% have reached this criteria (Snow, Prochaska, & Ross, 1992). These people can put all of their change efforts into enhancing other aspects of

their lives. But, for many it may mean a lifetime of maintenance. The ideal goal is to have a new healthy behavior be automatic and under stimulus control, like taking their aspirin every day at the same time and place.

Applying TTM usually begins by assessing which stage the patient is in and then helping them set a realistic goal for now, like progressing to the next stage. Research shows that if we try to pressure patients to progress quickly from precontemplation to action, there can be unforeseen consequences of dropping out of treatment, stopping until treatment is over and then quickly relapsing or simply lying.

What are the principles for helping patients progress from precontemplation to contemplation? A meta-analysis of the pros and cons of changing for 48 health behaviors revealed some remarkable results from 140 studies from 10 countries in 9 languages (Hall & Rossi, 2008). The cons are clearly greater than the pros in precontemplation (PC) and the pros are clearly higher in contemplation (C). So, the first principle of progress is to raise the pros. With sedentary individuals, we might ask them if they like bargains and tell them that if so, physical activity (PA) is the bargain basement of behaviors. There is no other behavior from which they can get as many benefits as PA. We would ask them to list all the benefits that they believe they could get from regular PA, chart their list, and then challenge them to try to double the list. Most have five or six and we tell them there are over 60 scientific benefits and we only want them to find five more. If we see the list going up, it is like seeing cholesterol or blood pressure coming down. We know our behavior medicine is working.

In contemplation, the pros and cons are exactly tied, reflecting their profound ambivalence. From C to preparation (P), the cons come down, so the second principle is to help lower the cons. The number one con for PA is time, so some individuals lower this con by riding a lifecycle where they can multitask and read or review an article for work, read a book for pleasure, or catch up on the news. Others may volunteer to help coach their kid's soccer team and at the same time, get some PA, be with their child, do community service, meet more parents, and have fun. Fortunately, the cons have to decrease only half as much as the pros increase, so we put twice as much emphasis on raising the pros.

In the preparation stage, the pros clearly outweigh the cons, so individuals are not encouraged to take action until they have a favorable profile. Once in the action stage, they can use their growing list of pros to put other principles and processes of change into operation. When they write down on their "To-Do" list, "walking for my heart" they are making a daily commitment based on the process of self-liberation from Existential therapy. When they look at the list they are cued to action based on stimulus control from Behavior Therapy. When they scratch off their list, they are reinforcing themselves based on Skinnerian Theory. As they move from one pro to the next each week, like walking for my weight, my sleep, my self-esteem, my immune system, and my sex life, after a while they may be running. Over time, they are using PA to affirm so much of their body, selves, and others based on self-reevaluation from cognitive theory and self-psychology.

This description illustrates how different principles and processes are applied to produce progress at different stages of change. This integrative approach led to the development of computer-tailored interventions (CTIs) by which individuals are assessed on TTM variables related to their current stage. Their assessment is compared to a normative database and they can be given feedback on how they are applying principles and processes compared to peers who make the most progress. Over time, they can be given feedback compared to themselves, such as "Congratulations, you have progressed two stages, which means you have about tripled the chances you will be taking effective action in the next few months."

Such CTIs have been found in randomized population trials to be effective with a growing

range of problems, including smoking, exercise, diet, stress, depression, bullying, partner violence, and medication adherence. The percentage of those in the action or maintenance stage at long-term follow-up ranges from about 25% for smoking (Prochaska, Velicer, Fava, Rossi, & Tsoh, 2001), to about 45% for exercise and diet (Prochaska, Wright, & Velicer, 2008), to over 65% for stress (Evers et al., 2006) and medication adherence (Johnson et al., 2006). These results are with populations in which typically the majority, like 80%, would be labeled as unmotivated or not ready to change when we proactively reached out to them at home, school, work, or in clinics to offer them help matched to their personal needs. The results can be remarkably robust with very comparable outcomes with smoking, for example, with adolescents and older smokers, Hispanic and African-American smokers, and smokers with mental illness (Velicer, Redding, Sun, & Prochaska, 2007).

Similar interventions have been found to be just as effective when we treat populations for three or four behaviors at the same time (Prochaska, Velicer, Prochaska, Deluschi, & Hall, 2006). Individuals working on one behavior are just as effective as those working on two who are just as effective as those working on three. But, very few people are taking action on more than one behavior at a time because they are not ready. So, they can be progressing through early stages on two behaviors, for example, while they are working to maintain action on another single behavior. Over time, the outcome will have much greater impact on populations with multiple health risk behaviors who have the highest risks for morbidity, disability, mortality, lost productivity, and increased health care costs.

References and Readings

Evers, K. E., Prochaska, J. O., Johnson, J. L., Mauriello, L. M., Padula, J. A., & Prochaska, J. M. (2006). A randomized clinical trial of a population and transtheoretical model-based stress-management intervention. *Health Psychology, 25*, 521–529.

Hall, K. L., & Rossi, J. S. (2008). Meta-analytic examination of the strong and weak principles across 48 health behaviors. *Preventive Medicine, 46*, 266–274.

Johnson, S. S., Driskell, M. M., Johnson, J. L., Dyment, S. J., Prochaska, J. O., Prochaska, J. M., et al. (2006). Transtheoretical model intervention for adherence to lipid-lowering drugs. *Disease Management, 9*, 102–114.

Prochaska, J. O. (2008). Multiple health behavior research represents the future of preventive medicine. *Preventive Medicine, 46*, 281–285.

Prochaska, J. O., Butterworth, S., Redding, C., Burden, V., Perrin, N., & Leo, M. (2008). Initial efficacy of MI, TTM tailoring and HRI's with multiple behaviors for employee health promotion. *Preventive Medicine, 45*, 226–231.

Prochaska, J. O., & DiClemente, C. C. (1983). Stages and processes of self? change of smoking: Toward an integrative model of change. *Journal of Consulting and Clinical Psychology, 51*, 390–395.

Prochaska, J. O., DiClemente, C. C., & Norcross, J. C. (1992a). In search of how people change: Applications to the addictive behaviors. *American Psychologist, 47*, 1102–1114.

Prochaska, J. O., DiClemente, C. C., & Norcross, J. C. (1992b). In search of how people change: Applications to the addictive behaviors. *American Psychologist, 47*, 1102–1114.

Prochaska, J. O., Norcross, J. C., & DiClemente, C. C. (1994). *Changing for good*. New York: Morrow. Released in paperback by Avon, 1995.

Prochaska, J. O., Velicer, W. F., Fava, J. L., Rossi, J. S., & Tsoh, J. Y. (2001). Evaluating a population-based recruitment approach and a stage-based expert system intervention for smoking cessation. *Addictive Behaviors, 26*, 583–602.

Prochaska, J. J., Velicer, W. F., Prochaska, J. O., Deluschi, K. I., & Hall, S. M. (2006). Comparing intervention outcomes in smokers with single versus multiple behavior risks. *Health Psychology, 25*, 380–388.

Prochaska, J. O., Wright, J. A., & Velicer, W. F. (2008). Evaluating theories of health behavior change: A hierarchy of criteria applied to the transtheroetical model. *Applied Psychology: An International Review, 57*(4), 561–588.

Snow, M. G., Prochaska, J. O., & Rossi, J. S. (1992). Stages of change for smoking cessation among former problem drinkers: A cross-sectional analysis. *Journal of Substance Abuse, 4*, 107–116.

Velicer, W. F., Redding, C. A., Sun, X., & Prochaska, J. O. (2007). Demographic variables, smoking variables, and outcome across five studies. *Health Psychology, 26*(3), 278–287.

Trauma, Early Life

► Stress, Early Life

Traumatic Brain Injury

Mary Spiers and Emily W. Reid (Deceased)
Department of Psychology, Drexel University, Philadelphia, PA, USA

Synonyms

Brain damage; Brain injury; Brain trauma; Concussion; Head injury

Definition

Traumatic brain injury (TBI) is an acquired brain injury resulting in diffuse brain damage. Injury is caused by the direct impact of an external force or whiplash, which results in a rapid acceleration or deceleration of the brain against the skull. The rapid movement causes neurons to shear and tear, and the impact of the brain against the skull can result in bruising and bleeding. The trauma can cause secondary complications such as ischemia, increased blood pressure, or ruptured blood vessels. The impairments caused by TBI depend on its severity. Due to the diffuse nature of the injury, a variety of cognitive, emotional, and behavioral changes are often seen.

Description

Epidemiology of Traumatic Brain Injury

The CDC estimates that 1.5–2 million people suffer a traumatic brain injury (TBI) every year in the United States (Faul, Xu, Wald, & Coronado, 2010). Of those, an estimated 80% are mild in severity, 10% are moderate, and the remaining 10% have severe injuries (Kraus, McArthur, Silverman, & Jayaraman, 1996). A small proportion (estimated 275,000) are hospitalized, whereas 1.4 million are treated and released (Faul et al., 2010). This is likely to be an underestimate of incidence as many do not go to the emergency room, and it is estimated that 1.6–3.8 million sports-related TBIs occur each year, of which many are treated on the field and do not seek emergency room assistance (Brain Trauma Foundation, 2011). Approximately 2% of the US population currently live with disabilities from TBI and represent a significant public health challenge (Brain Trauma Foundation, 2011).

The majority of TBIs are caused by falls (35.2%), motor vehicle accidents (17.3%), striking or being struck by or against an object (16.5%), and assaults (10%) (Faul et al., 2010). Another common cause for military personnel is blast-related injury. Approximately 52,000 deaths each year are due to traumatic brain injury with motor vehicle accidents resulting in the largest number of fatalities (31.8%) (Faul et al., 2010).

TBI is the leading cause of death and disability for children and adults under the age of 44 (Brain Trauma Foundation, 2011). Those most vulnerable to TBI are children, ages 0–4; older adolescents, ages 15–19; and adults over 65 years old. However, the number of emergency room visits for ages 0–14 is twice as many as those for adults over 65 years of age (Faul et al., 2010). Across all age groups, men sustain at least twice as many head injuries as women (Faul et al., 2010), and males, ages 0–4, have the highest rates of TBI-related emergency visits, hospitalizations, and deaths.

TBI also has a strong economic impact. In a 1998 consensus report from NIH, an estimated $9–10 billion were spent on new TBI cases each year ("Consensus Conference," 1999). Financially, the lifelong care for a person with a TBI is estimated to be between $600,000 and $1.9 million (Elias & Saucier, 2006). The NIH acknowledges that these are likely underestimates of the actual cost, as these numbers do not reflect the lost earnings, costs to social services systems, and the value of time and forgone earnings of family members who care for persons with TBI (Elias & Saucier, 2006). Further complications to care include insurance coverage, access to care, ability to navigate the health system, and available family and community support ("Consensus Conference," 1999).

Mechanism of Injury

Traumatic brain injury results either from object penetration or from rapid acceleration or deceleration of the brain resulting in the classifications of open head and closed head injuries.

Open or Penetrating Head Injury

In an open or penetrating head injury, the skull and the covering of the brain, or meninges, are ruptured. These injuries occur when an object (e.g., bullet, knife, bone fragment) lodges or passes through the brain. Because the brain is exposed, infection is a concern. Also, because penetrating head injuries may create more focal damage than in a closed head injury, the pattern of behavioral deficits is dependent on location of injury. Aftereffects of the initial injury, including swelling and bleeding, may cause more global, though usually time-limited, effects due to intracranial pressure or inflammatory response.

Closed Head Injury

The majority of head injuries are closed head injuries. These injuries occur by either direct impact or whiplash and may be caused by, for example, motor vehicle accidents, sports-related concussions, falls, and war-related blast injuries. The brain undergoes a rapid acceleration or deceleration or both but without skull penetration.

Contusions and/or hematomas may be seen at the location of impact (i.e., coup) and the opposite side of the brain (i.e., contre-coup) owing to the acceleration and deceleration of the brain against the skull. Despite the location of impact, a pattern of diffuse injury is likely to occur, impacting the frontal lobes and temporal poles because of the jagged surface of the tentorial plates that hold those brain structures in place. The physical forces may shear, tear, and rupture neurons, blood vessels, and the meninges, or covering, of the brain.

Diffuse axonal injury (DAI) may occur with shearing or tearing of neurons, often as a result of head rotation or rapid deceleration. Consequently, the axon damage can result in fewer axonal connections and/or less efficient transmission from one axon to another (Lux, 2007). The neurons most vulnerable to this type of strain are those with long axons, usually white-matter tracts that connect distant brain regions. DAI produces two types of cell death, necrosis and apoptosis, which are the leading contributors to brain damage in closed head injuries. Both impede axonal transport, induce atypical metabolic changes, and cause the axon to swell. In DAI, the damage is widespread, regional, multifocal, and at times global, and the course of damage may change over time. The swelling leads to detachment downstream from other neurons. This pattern of deafferentation is considered to affect more neurons than those identified as originally damaged. Although the brain's plasticity does allow for new axonal growth in living neurons, spurious growth can cause additional complications by forming undesirable connections leading to behavioral disturbances.

Consequences of Traumatic Brain Injury

The neuropsychological and behavioral consequences of TBI are related to injury characteristics and severity. The injury characteristics of importance in the acute phase are length of loss of consciousness (LOC) and length of emergence into consciousness with accompanying degree of posttraumatic amnesia (PTA), characterized by confusion and disorientation. For example, LOC ranges from none to brief LOC in concussion and mild traumatic brain injury (MTBI) to weeks in severe head injury. Emergence into consciousness ranges from minutes to hours in MTBI to weeks to days in severe head injury. Corresponding recovery of neuropsychological functioning can range from days to years.

LOC and coma are directly associated with an injury to those areas of the brain, typically the lower brain stem and reticular activating system (RAS), that are involved in maintaining consciousness and arousal. Coma falls along a continuum related to depth of responsiveness and is typically assessed at regular intervals after an injury via the Glasgow Coma Scale (GCS) (Teasdale & Jennett, 1974). The GCS assesses best motor, verbal, and eye opening response at one point in time. Scores range from 15 (can obey motor commands, is oriented, and eyes are open)

to 3 (flaccid motor response, no verbal response, and no eye opening). Medically, coma is defined as a score of 3–8, which typically corresponds to a severe brain injury. Greater mortality is typically associated with scores below 7. Although initial severity of GCS is an important prognostic indicator for survival, other indicators, such as number of days to reach a GCS of 15, and length of PTA are added predictors for long-term neuropsychological outcome. Duration of PTA and education appear to be two of the best predictors of long-term functional outcome. On measures of global functioning and disability, those who had less education preinjury or a longer period of PTA had poorer outcomes 10 years after the injury (Ponsford, Draper, & Schonberger, 2008).

The recovery from coma is a process of "emergence" in which greater awareness of environmental stimuli occurs. Characteristic of traumatic brain injury is a loss of recall for the actual impact event. Since the injured person does not recall the event, he or she cannot usually give a reliable account of the length of LOC. This period of PTA is characterized by confusion and disorientation and typically includes both retrograde and anterograde amnesia. Retrograde amnesia is the impairment in the retrieval of information for events preceding the injury. Conversely, anterograde amnesia is impairment in encoding new memories after the injury.

The difference in diagnosis between mild, moderate and severe head injuries relates both to immediate injury severity as rated by the GCS and time related to resolution of symptoms. For example, MTBI is characterized by a GCS score of 13–15 and a fairly rapid resolution of LOC (less than 30 min) and PTA (less than 24 h). Moderate-level brain injuries are characterized by a GCS of 9–12, LOC between 30 min and 24 h, and PTA between 1 and 7 days. Severe head injuries are those with a GCS of 3–8, LOC longer than a day, and PTA longer than 7 days.

Neuropsychological Consequences of TBI

The neuropsychological consequences of TBI may include a number of deficits. However, those deficits that correspond to frontal and temporal injuries, as well as diffuse axonal injuries are most common. Specifically, these include difficulties in attention, memory, and language functions, executive dysfunction, and emotionality.

Regardless of injury severity, attentional deficits are common after TBI. Reports include a feeling of mental slowness, difficulty following conversations, losing a train of thought, and trouble with multitasking (Gronwall, 1987; Van Zomeren & Brouwer, 1994). The most universal consequence is a reduced ability to process information (McCullagh & Feinstein, 2005). Therefore, when tasks become more complex, reaction time becomes slower. As a result, a TBI sufferer may not appear cognitively impaired in simpler routine assessments, but the deficits may become more evident in the multicomponent tasks of daily life (Granacher, 2008; Lux, 2007). Deficits are seen across all types of attention processing, including selective, sustained, and divided attention with particular problems on tasks that require controlled rather than automatic processing (Park, Moscovitch, & Robertson, 1999).

In addition to attention and working memory difficulties, a deficit in episodic memory is a hallmark feature of TBI (Richardson, 2000). Memory impairment is one of the most frequent (Arcia & Gualtieri, 1993; King, Crawford, Wenden, Moss, & Wade, 1995) and long-lasting complaints with significant deficits found in many people 10 years postinjury (Zec et al., 2001) and poor employment prognosis 7 years postinjury (Brooks, McKinlay, Symington, Beattie, & Campsie, 1987). Furthermore, TBI patients tend to have impairments in prospective memory, or remembering to perform an intended action, which may lead to forgetting appointments, payment of bills, and medication taking (Kinsella et al., 1996). Adding further difficulty, patients tend to be less aware of their memory difficulties than those around them (McCullagh & Feinstein, 2005).

Those with TBI may also experience language and communication difficulties. Speech tends to be less productive and efficient, with less content in longer discourse, and with greater fragmentation (Hartley & Jensen, 1991). Additionally,

language difficulties may include trouble naming objects and, to a lesser extent, comprehension of complex commands (Levin, Grossman, & Kelly, 1976; Sarno, Buonaguro, & Levita, 1986).

Deficits in executive functioning influence functional, emotional, and social outcomes after TBI. The term "executive functioning" refers to higher-order capabilities that include goal setting, planning, initiating, sequencing, reasoning abilities, decision making, inhibiting responses, self-monitoring, and self-regulation (Stuss & Levine, 2002). Since these processes underlie many daily and social skills, such as impulse control, judgment, creativity, emotional regulation, and moral judgment, routine testing might not detect the degree of impairment evident in these areas (Zillmer, Spiers, & Culbertson, 2008). Verbal fluency tests, as a measure of executive dysfunction, however, consistently show impairment for TBI patients because they require organization of verbal retrieval and recall, self-monitoring aspects of cognition, and effortful self-initiation and inhibition of responses. (Henry & Crawford, 2004)

Emotional and behavioral complications are common after TBI and often lead to depressed and anxious mood, impulsivity, agitation, and amotivation (Vaishnavi, Rao, & Fann, 2009). Mood disturbances are considered the most common psychiatric complication of TBI and are often the most difficult adjustment for those who care for the TBI patient (Rosenthal, Christensen, & Ross, 1998). Approximately 10–60% of patients are depressed, reporting feelings of hopelessness, worthlessness, and anhedonia (Hurley & Taber, 2002). Also, they display somatic symptoms, such as sleep disturbances, reduced initiation, fatigue, and changes in appetite. TBI patients have reduced participation in leisure activities and have difficulty engaging in new hobbies (Rosenthal et al., 1998). Mood disturbances can cause significant emotional distress in patients with TBI, contributing to the disruption of social relationships.

In sum, while a pattern of neuropsychological deficits in TBI may show common features, the evaluation of any particular individual should include a consideration of the unique injury location and severity features in a context of previous levels of education, work, and social history.

Cross-References

- ▶ Brain Damage
- ▶ Brain, Injury
- ▶ Neuropsychology

References and Readings

Arcia, E., & Gualtieri, C. T. (1993). Association between patient report of symptoms after mild head injury and neurobehavioural performance. *Brain Injury, 7*, 481–489.

Brain Trauma Foundation. (2011). Facts about TBI in the USA. Retrieved March 3, 2011, from https://www.braintrauma.org/tbi-faqs/tbi-statistics/

Brooks, N., McKinlay, W., Symington, C., Beattie, A., & Campsie, L. (1987). Return to work within the first seven years of severe head injury. *Brain Injury, 1*, 5–19.

Consensus Conference Writing Group (1999). Consensus conference. Rehabilitation of persons with traumatic brain injury. NIH Consensus Development Panel on Rehabilitation of Persons With Traumatic Brain Injury. *Journal of the American Medical Association, 282*(10), 974–983. doi: jcf90001 (pii).

Elias, L. J., & Saucier, D. M. (2006). *Neuropsychology: Clinical and experimental foundations*. Boston: Pearson/Allyn & Bacon.

Faul, M., Xu, L., Wald, M. M., & Coronado, V. G. (2010). Traumatic brain injury in the United States: Emergency Department Visits, Hospitalizations and Deaths 2002–2006. Retrieved March 3, 2011, from www.cdc.gov/TraumaticBrainInjury

Granacher, R. P. (2008). *Traumatic brain injury: Methods for clinical and forensic neuropsychiatric assessment* (2nd ed.). Boca Raton: CRC Press/Taylor & Francis Group.

Gronwall, D. (1987). *Advances in the assessment of attention and information processing after head injury in neurobehavioral recovery from head injury*. New York: Oxford University Press.

Hartley, L. L., & Jensen, P. J. (1991). Narrative and procedural discourse after closed head injury. *Brain Injury, 5*(3), 267–285.

Henry, J. D., & Crawford, J. R. (2004). A meta-analytic review of verbal fluency performance in patients with traumatic brain injury. *Neuropsychology, 18*(4), 621–628. doi:2004-19607-003[pii]10.1037/0894-4105.18.4.621.

Hurley, R. A., & Taber, K. H. (2002). Emotional disturbances following traumatic brain injury. *Current Treatment Options in Neurology, 4*(1), 59–75.
King, N. S., Crawford, S., Wenden, F. J., Moss, N. E., & Wade, D. T. (1995). The rivermead post concussion symptoms questionnaire: A measure of symptoms commonly experienced after head injury and its reliability. *Journal of Neurology, 242*(9), 587–592.
Kinsella, G., Murtagh, D., Landry, A., Homfray, K., Hammond, M., & O'Beirne, L. (1996). Everyday memory following traumatic brain injury. *Brain Injury, 10*(7), 499–507.
Kraus, J. F., McArthur, D. L., Silverman, T. A., & Jayaraman, M. (1996). Epidemiology of brain injury. In R. K. Naravan, J. E. Wilberger, & J. T. Povlishock (Eds.), *Neurotrauma*. New York: McGraw-Hill.
Levin, H. S., Grossman, R. G., & Kelly, P. J. (1976). Aphasic disorder in patients with closed head injury. *Journal of Neurology, Neurosurgery & Psychiatry, 39*(11), 1062–1070.
Lux, W. E. (2007). A neuropsychiatric perspective on traumatic brain injury. *Journal of Rehabilitation Research and Development, 44*(7), 951–962.
McCullagh, S., & Feinstein, A. (2005). Cognitive changes. In J. M. Silver, T. W. McAllister, & S. C. Yudofsky (Eds.), *Textbook of traumatic brain injury* (1st ed., p. 321). Washington, DC: American Psychiatric.
Park, N. W., Moscovitch, M., & Robertson, I. H. (1999). Divided attention impairments after traumatic brain injury. *Neuropsychologia, 37*(10), 1119–1133. doi:S0028393299000342[pii].
Ponsford, J., Draper, K., & Schonberger, M. (2008). Functional outcome 10 years after traumatic brain injury: Its relationship with demographic, injury severity, and cognitive and emotional status. *Journal of International Neuropsychological Society, 14*(2), 233–242. doi:10.1017/S1355617708080272.
Richardson, J. (2000). *Clinical and neuropsychological aspects of closed head injury*. East Sussex, UK: Psychology Press.
Rosenthal, M., Christensen, B. K., & Ross, T. P. (1998). Depression following traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 79*(1), 90–103.
Sarno, M. T., Buonaguro, A., & Levita, E. (1986). Characteristics of verbal impairment in closed head injured patients. *Archives of Physical Medicine and Rehabilitation, 67*(6), 400–405.
Stuss, D. T., & Levine, B. (2002). Adult clinical neuropsychology: Lessons from studies of the frontal lobes. *Annual Review of Psychology, 53*, 401–433. doi:10.1146/annurev.psych.53.100901.13522053/1/401[pii].
Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness. A practical scale. *Lancet, 2*, 81–84.
Vaishnavi, S., Rao, V., & Fann, J. R. (2009). Neuropsychiatric problems after traumatic brain injury: Unraveling the silent epidemic. *Psychosomatics, 50*(3), 198–205. doi:50/3/198[pii]10.1176/appi.psy.50.3.198.
Van Zomeren, A., & Brouwer, W. (1994). *Clinical neuropsychology of attention*. New York: Oxford University Press.
Zec, R. F., Zellers, D., Belman, J., Miller, J., Matthews, J., & Femeau-Belman, D. (2001). Long-term consequences of severe closed head injury on episodic memory. *Journal of Clinical and Experimental Neuropsychology, 23*(5), 671–691.
Zillmer, E. A., Spiers, M. V., & Culbertson, W. C. (2008). *Principles of neuropsychology* (2nd ed.). Belmont, CA: Thomson/Wadsworth.

Treatment Group

▶ Experimental Group

Treatment of Fatigue

▶ Fatigue

Trier Social Stress Test

Robert Miller and Clemens Kirschbaum
Chair of Biopsychology, Technische Universität Dresden, Dresden, Saxony, Germany

Synonyms

Laboratory stress protocol; Psychosocial stress; TSST

Definition

The Trier Social Stress Test (TSST) is a procedure for induction of moderate psychosocial stress under laboratory conditions. It was introduced in 1993 by Kirschbaum, Pirke, and Hellhammer.

Description

Stress is one of the presumably most significant health problems of the twenty-first century (World Health Organization, 2001). Thus it has been of growing importance to gain insight into its underlying components using standardized methods, which reliably induce self-reported, behavioral, and biological stress responses in laboratory settings.

Two major biological systems significantly drive stress responses in mammals: the sympathetic-adrenal-medullary (SAM) axis and the hypothalamic-pituitary-adrenal (HPA) axis. Adequate methods and protocols are needed for reliable stimulation of biomarkers for these systems, to allow for investigations of psychosocial stress effects on the brain and peripheral tissues. While already an effort-driven response suffices for a sufficiently large SAM response, effective HPA psychological stressors, however, need to exert significant social-evaluative threat and uncontrollability upon the tested individual (Dickerson & Kemeny, 2004).

With the development of the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993), a laboratory protocol with social-evaluative threat, uncontrollability, and effort components became available for rapid and reliable activation of SAM, HPA axis, and other biological stress pathways. Over the past two decades, the TSST has been employed in many laboratories around the globe in stress research of healthy subjects and various clinical samples.

The TSST is a motivated performance task protocol being disguised as a job interview, which consists of a brief preparation period followed by two 5-min test periods during which a subject has to perform a free speech and solve an arithmetic problem, respectively. Upon arrival the subject is informed that he/she is supposed to take over the role of a job applicant who is invited for a personal interview with a selection committee. After ensuring that the subject fully understood the instructions, he/she is guided into a separate room where the selection committee is already seated at a desk upon entrance of the subject. The committee members (male and female confederates) wear white lab coats and are specially trained to withhold any positive or negative feedback during the whole procedure. Furthermore, the room is equipped with a separate desk and chair, a video camera, and a microphone being located approximately 2 m in front of the selection committee. After the initial 3-min preparation phase, during which the subject has the opportunity to structure the upcoming free speech on personal job-relevant traits, the committee asks the subject to step in front of the microphone and begin with the presentation. Most test subjects finish their talk after about 2–3 min of speech time and are encouraged to continue with their speech by one member of the committee. Upon the second speech interruption, the committee silently focuses their gaze on the subject for 20 s, before they begin to ask standard personal questions. After exactly 5 min the subject is asked to stop the speech and continue with the second task, which comprises continuous serial subtractions. The subject is told that upon each error, the committee would ask to start anew from the initial number. After another 5 min, the subject is asked to return to the experimenter who is already waiting outside the testing room to continue with study protocol and the assessment of relevant biomarkers.

With proper completion of the TSST about 70–85% (Kudielka, Hellhammer, & Kirschbaum, 2007) of all subjects reveal an increase in HPA activity as indicated by corticotropin-releasing hormone, adrenocorticotropic hormone (ACTH), serum, and salivary cortisol (Kirschbaum et al., 1993). While ACTH levels peak at or shortly after stress cessation, cortisol levels reach maximum values between 10 and 20 min thereafter. The TSST also activates the SAM, with significant responses in norepinephrine, epinephrine, salivary amylase, heart rate, blood pressure levels, and electrodermal activity. In addition, hemoconcentration, blood coagulation indicators, and transcription factor activation is seen after the TSST (Kudielka et al., 2007). Evaluation of

perceived stress level changes as measured with self-report scales also supports the validity of the protocol.

The methodological advantages of the TSST have led to its widespread use in psychoneuroendocrinological research and stimulated the development of adaptions for children (TSST-C; Buske-Kirschbaum et al., 1997), retirees (Kudielka et al., 1998), psychiatric patients (Brenner et al., 2009), and groups (TSST-G; von Dawans, Kirschbaum, & Heinrichs, 2011). Furthermore, a TSST-like placebo protocol has been developed, which is especially useful in studies with control group designs (Het, Rohleder, Schoofs, Kirschbaum, & Wolf, 2009). Apart from these, variations of the TSST have been employed with mixed results (i.e., insufficient/unreliable stress responses; Gold, Zakowski, Valdimarsdottir, & Bovbjerg, 2004; Gunnar, Frenn, Wewerka, & Van Ryzin, 2009; Kelly, Matheson, Martinez, Merali, & Anisman, 2007; Simoens et al., 2007).

Even with complete adherence to the standard TSST protocol, there is considerable intra- and interindividual variation in the stress response patterns. Certain demographic, biological, and psychological variables can change the magnitude and course of the biomarkers. Among other variables, chronic and acute nicotine or alcohol consumption, dietary status, pregnancy, lactation, physical exercise, or personality traits can lead to differences in HPA activation by the TSST. For a detailed review on potential confounds, Foley and Kirschbaum (2010) recently summarized the TSST literature with a special focus on genetic factors.

In within-subject experimental designs, the TSST may be used repeatedly. Although prior knowledge about the protocol, as well as repeated exposure with days, weeks, or months between sessions can lead to HPA response habituation (Kirschbaum et al., 1995), these effects can be bypassed by changing setting variables, i.e., the selection committee, and the test location with each session. For the assessment of SAM activity with repeated TSST exposure such adaptations are not necessary, since SAM biomarkers show comparable activation patterns even with five identical TSST repetitions. The same seems to apply to cytokines, blood coagulation indices, and parameters of hemoconcentration (Kudielka et al., 2007).

While the TSST has become a research tool frequently employed in many different areas of basic science and clinical research, a powerful stress protocol for use in imaging studies is still missing. Although an adapted version of a computer-based stress task, the "Montreal Imaging Stress Task" (Dedovic et al., 2005) was created for this purpose, numerous subjects show only small HPA responses or no significant cortisol rises at all with this protocol. A scanner-adapted version of the TSST may therefore prove useful to advance our understanding how stress affects the brain and peripheral tissues.

Cross-References

- ▶ Biomarkers
- ▶ Cortisol
- ▶ Imaging
- ▶ Stress
- ▶ Stress Responses
- ▶ Stressor

References and Readings

Brenner, K., Liu, A., Laplante, D. P., Lupien, S., Pruessner, J. C., Ciampi, A., et al. (2009). Cortisol response to a psychosocial stressor in schizophrenia: Blunted, delayed, or normal? *Psychoneuroendocrinology, 34*, 859–868.

Buske-Kirschbaum, A., Jobst, S., Wustmans, A., Kirschbaum, C., Rauh, W., & Hellhammer, D. H. (1997). Attenuated free cortisol response to psychosocial stress in children with atopic dermatitis. *Psychosomatic Medicine, 59*, 419–426.

Dedovic, K., Renwick, R., Mahani, N. K., Engert, V., Lupien, S. J., & Pruessner, J. C. (2005). The Montreal imaging stress task: Using functional imaging to investigate the effects of perceiving and processing psychosocial stress in the human brain. *Journal of Psychiatry & Neuroscience, 30*, 319–325.

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130*, 355–391.

Foley, P., & Kirschbaum, C. (2010). Human hypothalamus-pituitary-adrenal axis responses to acute psychosocial stress in laboratory settings. *Neuroscience and Biobehavioral Reviews, 35*, 91–96.

Gold, S. M., Zakowski, S. G., Valdimarsdottir, H. B., & Bovbjerg, D. H. (2004). Higher Beck depression scores predict delayed epinephrine recovery after acute psychological stress independent of baseline levels of stress and mood. *Biological Psychology, 67*, 261–273.

Gunnar, M. R., Frenn, K., Wewerka, S. S., & Van Ryzin, M. J. (2009). Moderate versus severe early life stress: Associations with stress reactivity and regulation in 10–12-year-old children. *Psychoneuroendocrinology, 34*, 62–75.

Het, S., Rohleder, N., Schoofs, D., Kirschbaum, C., & Wolf, O. T. (2009). Neuroendocrine and psychometric evaluation of a placebo version of the 'Trier Social Stress Test'. *Psychoneuroendocrinology, 34*, 1075–1086.

Kelly, O., Matheson, K., Martinez, A., Merali, Z., & Anisman, H. (2007). Psychosocial stress evoked by a virtual audience: Relation to neuroendocrine activity. *Cyberpsychology & Behavior, 10*, 655–662.

Kirschbaum, C., Pirke, K. M., & Hellhammer, D. H. (1993). The Trier Social Stress Test: A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology, 28*, 76–81.

Kirschbaum, C., Pruessner, J. C., Stone, A. A., Federenko, I., Gaab, J., Lintz, D., et al. (1995). Persistent high cortisol responses to repeated psychosocial stress in a subpopulation of healthy men. *Psychosomatic Medicine, 57*, 468–474.

Kudielka, B. M., Hellhammer, J., Hellhammer, D. H., Wolf, O. T., Pirke, K. M., Varadi, E., et al. (1998). Sex differences in endocrine and psychological responses to psychosocial stress in healthy elderly subjects and the impact of a 2-week dehydroepiandrosterone treatment. *The Journal of Clinical Endocrinology & Metabolism, 83*, 1756–1761.

Kudielka, B. M., Hellhammer, D. H., & Kirschbaum, C. (2007). Ten years of research with the Trier Social Stress Test-revisited. In E. Harmon-Jones & P. Winkielman (Eds.), *Social neuroscience: Integrating biological and psychological explanations of social behavior* (pp. 56–83). New York: The Guilford Press.

Simoens, V. L., Istók, E., Hyttinen, S., Hirvonen, A., Näätänen, R., & Tervaniemi, M. (2007). Psychosocial stress attenuates general sound processing and duration change detection. *Psychophysiology, 44*, 30–38.

von Dawans, B., Kirschbaum, C., & Heinrichs, M. (2011). The Trier Social Stress Test for groups (TSST-G): A new research tool for controlled simultaneous social stress exposure in a group format. *Psychoneuroendocrinology, 36*, 514–522.

World Health Organization. (2001). *The world health report 2001 – Mental health: New understanding, new hope*. Retrieved from http://www.who.int/entity/whr/2001/en/whr01_en.pdf. Accessed 27 March, 2012.

Triglyceride

Chad Barrett
Department of Psychology, University of Colorado Denver, Denver, CO, USA

Synonyms

Lipid

Definition

Triglycerides are a type of lipid (fat) which consist of glycerol and three molecules of fatty acid and are found in blood plasma and fat tissue. They are derived from fats and carbohydrates that are consumed. When calories are consumed, the body converts any calories not immediately used by tissues into triglycerides and transports them into fat cells for storage. Hormones regulate the release of triglycerides from fat tissues in order to provide energy for the body between meals. If the body uses fewer calories than are consumed in a day, then the surplus of calories can cause elevated levels of triglycerides (Welson, 2006).

According to the American Heart Association, the normal level of triglycerides is less than 150 mg/dL and the optimal level of triglycerides is 100 mg/dL or lower. Borderline high levels range from 150 to 199 mg/dL; high levels range from 200 to 499 mg/dL; very high levels range from 500 mg/dL and above. Elevated levels of triglycerides are often the result of being overweight, physically inactive, smoking, excessive consumption of alcohol, and diets high in fat and carbohydrates. High levels of triglycerides have been linked to atherosclerosis (hardening of the arteries) and increased risk of heart disease, stroke, metabolic syndrome (Triglycerides, 2010; What Your Cholesterol Levels Mean, 2011), and Alzheimer's disease (Altman & Rutledge, 2010). Interventions to lower triglycerides typically involve changes in lifestyle such as losing weight, adopting a more heart-healthy diet consisting of less fats and foods with added

sugars, engaging in regular exercise, quitting smoking, and reducing alcohol consumption (Haffner et al., 2005; Graves & Miller, 2003; Triglycerides, 2010). Omega-3 fatty acids can also help reduce triglyceride levels and reduce the risk of cardiovascular diseases and possibly even strokes (Kris-Etherton, Harris, & Appel, 2002).

Cross-References

- ▶ Cardiovascular Disease
- ▶ Cardiovascular Disease Prevention
- ▶ Cholesterol

References and Readings

Altman, R., & Rutledge, J. C. (2010). The vascular contribution to Alzheimer's disease. *Clinical Science, 119*, 07–421. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/20684749

Graves, K. D., & Miller, P. M. (2003). Behavioral medicine in the prevention and treatment of cardiovascular disease. *Behavior Modification, 27*, 3–25.

Haffner, S., Temprosa, M., Crandall, J., Fowler, S., Goldberg, R., Horton, E., et al. (2005). Intensive lifestyle. Intervention or metformin on inflammation and coagulation in participants with impaired glucose tolerance. *Diabetes, 54*, 1566–1572.

Kris-Etherton, P. M., Harris, W. S., & Appel, L. J. (2002). Fish consumption, fish oil, omega-3 fatty acids and cardiovascular disease. *Circulation*, *106*, 2747–2757. Retrieved from http://circ.ahajournals.org/content/106/21/2747.full

Triglycerides. (2010). Retrieved October 10, 2011, from http://www.heart.org/HEARTORG/GettingHealthy/NutritionCenter/Triglycerides_UCM_306029_Article.jsp

Welson, L. T. (2006). *Triglycerides and cholesterol research*. New York: Nova.

What your cholesterol levels mean. (2011). Retrieved October 10, 2011, from http://www.heart.org/HEARTORG/Conditions/Cholesterol/AboutCholesterol/What-Your-Cholesterol-Levels-Mean_UCM_305562_Article.jsp

tRNA

- ▶ RNA

TSST

- ▶ Trier Social Stress Test

Tumor Necrosis Factor-Alpha (TNF-Alpha)

Nicolas Rohleder
Department of Psychology, Brandeis University, Waltham, MA, USA

Synonyms

Cachectin

Definition

Tumor necrosis factor-alpha (TNF-alpha) belongs to the group of pro-inflammatory cytokines. Cytokines are chemical messenger molecules of the immune system, and the group of pro-inflammatory cytokines characterizes molecules that are secreted in response to inflammatory stimuli, and further promote inflammatory responses in target cells. The cytokine now known as tumor necrosis factor-alpha was first discovered as a molecule that appeared to be essential in the wasting syndrome associated with bacterial infection, and therefore initially referred to as cachectin. At the same time, another molecule was discovered that induced pronounced necrosis of certain tumors in organisms infected by gram-negative bacteria. This molecule turned out to be identical to cachectin, and both were then called tumor necrosis factor-alpha (Beutler & Cerami, 1989; Pennica et al., 1984).

TNF-alpha is one of the major products secreted by macrophages that are activated by inflammatory stimuli, and it acts through a family of different receptors that have some structural similarities and are present as

transmembrane proteins on a variety of target cells. There are two different categories of receptors, based on their effect on the target cell. The first category is characterized by intracellular signals preceding programmed cell death; these are most likely the receptors that mediate the tumor necrotic effects of TNF. The second category induces pro-inflammatory effects, for example, by stimulation of proliferation or transcription of further inflammatory mediators. On a systemic level, TNF-alpha plays a notable role in sepsis and in the induction of septic shock. Similar to the interleukins (IL)-6 and -18, TNF-alpha does also act on nonimmune tissues such as endothelial cells, adipocytes, muscle cells, the liver, and the gastrointestinal tract (Beutler & Bazzoni, 1998; Hehlgans & Pfeffer, 2005).

Of note, along with IL-1, TNF-alpha is an important mediator of CNS effects of peripheral inflammation. TNF-signaling into the CNS has been shown to activate the hypothalamus-pituitary-adrenal axis, to induce hyperalgesia, to reduce food intake, and to contribute to the well-described sickness behavior response (e.g., Besedovsky et al., 1991; Watkins, Goehler, Relton, Brewer, & Maier, 1995). This immune-to-CNS signaling function of TNF-alpha and other inflammatory cytokines plays an essential role in the control of peripheral inflammation during infectious and inflammatory diseases, and disruption of this loop leads to death in animal models of inflammatory diseases (Sternberg, 2006).

TNF-alpha and other inflammatory cytokines are further important as targets of CNS-to-immune signaling, and serve as a link between CNS states with disease-relevant pathophysiological factors. Inflammatory cytokine production is modulated by the sympathetic nervous system and the hypothalamus-pituitary-adrenal axis, and pro-inflammatory cytokines in blood are sensitive to acute and chronic stress, and found increased in depression and posttraumatic stress disorder (e.g., Rohleder, Marin, Ma, & Miller, 2009; Rohleder, Wolf, & Wolf, 2010; Steptoe, Hamer, & Chida, 2007). Inflammatory cytokines also increase with age and have been found to predict later life morbidity and mortality (e.g., Bruunsgaard et al., 2003).

Cross-References

- ▶ Depression
- ▶ Inflammation
- ▶ Posttraumatic Stress Disorder
- ▶ Psychoneuroimmunology
- ▶ Stress

References and Readings

Besedovsky, H. O., del Rey, A., Klusman, I., Furukawa, H., Monge Arditi, G., & Kabiersch, A. (1991). Cytokines as modulators of the hypothalamus-pituitary-adrenal axis. *The Journal of Steroid Biochemistry and Molecular Biology, 40*(4–6), 613–618.

Beutler, B., & Bazzoni, F. (1998). TNF, apoptosis and autoimmunity: A common thread? *Blood Cells, Molecules & Diseases, 24*(2), 216–230.

Beutler, B., & Cerami, A. (1989). The biology of cachectin/TNF – a primary mediator of the host response. *Annual Review of Immunology, 7*, 625–655.

Bruunsgaard, H., Ladelund, S., Pedersen, A. N., Schroll, M., Jorgensen, T., & Pedersen, B. K. (2003). Predicting death from tumour necrosis factor-alpha and interleukin-6 in 80-year-old people. *Clinical and Experimental Immunology, 132*(1), 24–31.

Hehlgans, T., & Pfeffer, K. (2005). The intriguing biology of the tumour necrosis factor/tumour necrosis factor receptor superfamily: Players, rules and the games. *Immunology, 115*(1), 1–20.

Pennica, D., Nedwin, G. E., Hayflick, J. S., Seeburg, P. H., Derynck, R., Palladino, M. A., et al. (1984). Human tumour necrosis factor: Precursor structure, expression and homology to lymphotoxin. *Nature, 312*(5996), 724–729.

Rohleder, N., Marin, T. J., Ma, R., & Miller, G. E. (2009). Biologic cost of caring for a cancer patient: Dysregulation of pro- and anti-inflammatory signaling pathways. *Journal of Clinical Oncology, 27*(18), 2909–2915.

Rohleder, N., Wolf, J. M., & Wolf, O. T. (2010). Glucocorticoid sensitivity of cognitive and inflammatory processes in depression and posttraumatic stress disorder. *Neuroscience and Biobehavioral Reviews, 35*(1), 104–114.

Steptoe, A., Hamer, M., & Chida, Y. (2007). The effects of acute psychological stress on circulating inflammatory factors in humans: A review and meta-analysis. *Brain, Behavior, and Immunity, 21*(7), 901–912.

Sternberg, E. M. (2006). Neural regulation of innate immunity: A coordinated nonspecific host response

to pathogens. *Nature Reviews: Immunology, 6*(4), 318–328.
Watkins, L. R., Goehler, L. E., Relton, J., Brewer, M. T., & Maier, S. F. (1995). Mechanisms of tumor necrosis factor-alpha (TNF-alpha) hyperalgesia. *Brain Research, 692*(1–2), 244–250.

Twin Studies

Jennifer Wessel
Public Health, School of Medicine, Indiana University, Indianapolis, IN, USA

Definition

The classic twin study builds on the fact that there are two kinds of twins that provide contrasting degrees of genetic relationship in siblings of the same age and family circumstances. Monozygotic (MZ) twins, or identical twins, have identical copies of all their genes. In contrast, dizygotic (DZ) twins, or fraternal twins, share, on average, only half of their genes by descent, as do ordinary siblings.

If there are genetic influences on the phenotype of interest, the MZ correlation will exceed that for the DZ twins. The greater the influence of the genes in determining individual differences in the phenotype, i.e., the greater the proportion of phenotypic variance attributable to genetic differences, the greater the difference between the MZ and DZ correlations will be. With some simplification of assumptions, twice the difference between the MZ and DZ correlations can be taken as an estimate of the heritability of the trait.

If genetic influences are the only cause of familial aggregation, if there is no nonadditive genetic variation, and if mating is random with respect to the characteristic under study, the correlation for DZ twins would be expected to be half that for MZ twins. However, if there are significant nonadditive genetic influences or there is significant competition between the twins or other contrast effects that accentuate the genetic differences of siblings, the DZ correlation may be less than half the MZ correlation. Conversely, assortative mating (like marrying like) or imitative or cooperative effects within the sibship, e.g., cooperative involvement in smoking or drinking behavior, may cause the DZ correlation to exceed half the MZ correlation.

Environmental influences on the phenotype of interest have two distinct characteristic consequences. First, if there are environmental influences that are shared by siblings growing up in the same home, e.g., socioeconomic status or parenting style, the MZ and DZ correlations will reflect this source of familial aggregation to the same extent. In the absence of genetic influences, these shared environmental influences would lead to equal MZ and DZ correlations. If there are genetic influences present, the shared environment will raise the DZ correlation relative to the MZ correlation. Another point to consider is individual environmental influences. If there are significant environmental influences that are unique to individuals, e.g., significant personal life events, familial aggregation will be attenuated and the MZ and DZ correlations will be reduced, although their relative magnitudes will continue to reflect the importance of genetic versus shared environmental causes of familial aggregation.

Historically, the twin study design has been a key tool for understanding behavioral genetics, but the use of twins has expanded to facilitate our understanding of the genetic contribution to a number of complex traits and diseases.

Cross-References

▶ Dizygotic Twins
▶ Monozygotic Twins

References and Readings

Nussbaum, R. L., Mc Innes, R. R., & Willard, H. F. (2001). *Genetics in medicine* (6th ed.). Philadelphia: W.B. Saunders Company.
Spector, T. D., Snieder, H., & MacGregor, A. J. (2000). *Advances in twin and sib-pair analysis* (1st ed.). London: Greenwich Medical Media.

Type 1 Diabetes

► Insulin-Dependent Diabetes Mellitus (IDDM)

Type 1 Diabetes Mellitus

Luigi Meneghini
Diabetes Research Institute, University of Miami, Miami, FL, USA

Synonyms

Autoimmune diabetes mellitus; Insulin-dependent diabetes mellitus (IDDM); Juvenile diabetes

Definition

Type 1 diabetes (T1DM) is an elevation in blood glucose due to insufficient production of insulin thought to result from cell-mediated autoimmune destruction of insulin-producing pancreatic beta cells. Autoimmunity is manifested by mononuclear cell invasion of islets (insulitis) and the production of islet-specific antibodies, such as GAD (Glutamic Acid Decarboxylase), IA-2 autoantibodies and ICA (Islet Cell Antibodies), detectable in ~85% of newly diagnosed patients. Genetic, environmental, and possibly other unknown factors contribute to disease susceptibility, which is most commonly manifested in the teenage years (hence the former designation of juvenile diabetes).

If not replaced, absolute insulin deficiency in T1DM results in severe hyperglycemia and diabetic ketoacidosis (DKA), which if left untreated can prove fatal (hence the designation insulin-dependent diabetes mellitus). Insulin replacement ideally takes the form of basal/bolus insulin therapy, delivered either via multiple daily insulin injections or an insulin pump infusion. Poorly controlled type 1 diabetes can lead to, among other complications, nephropathy (kidney damage manifested by excess protein in the urine) and end-stage kidney failure, retinopathy potentially leading to decreased visual acuity, and neuropathy and the risk of diabetic foot infections or ulcerations. Long-standing diabetes is also associated with increased risk for heart disease, stroke, and peripheral vascular disease. Maintaining good glycemic control, as reflected by an HbA1c of less than 7%, will prevent many of the chronic complications of the disease.

Cross-References

► Diabetes

References and Readings

Joslin, E. P., & Kahn, C. R. (2005). *Joslin's diabetes mellitus* (14th ed.). Philadelphia: Lippincott Williams & Willkins.

Type 2 Diabetes

► Type 2 Diabetes Mellitus

Type 2 Diabetes Mellitus

Elizabeth R. Pulgaron[1] and Alan M. Delamater[2]
[1]Department of Pediatrics, University of Miami, Miami, FL, USA
[2]Department of Pediatrics, University of Miami Miller School of Medicine, Miami, FL, USA

Synonyms

Non-insulin-dependent diabetes mellitus; Type 2 diabetes

Definition

In type 2 diabetes mellitus (T2D), high blood glucose (or hyperglycemia) is the result of the

pancreas producing insufficient quantities of insulin due to beta-cell dysfunction, as well as insulin resistance. Peripheral insulin resistance, in which cells resist the action of insulin at the receptor level, occurs early in the disease course. Initially this is compensated for by increased production of insulin, or hyperinsulinemia. Over time, however, insulin secretion declines, and hyperglycemia results. There is no single cause of T2D, although it is generally accepted to be the result of genetic, physiologic, and lifestyle factors, including obesity and physical inactivity.

Most cases of diabetes worldwide are due to T2D. Obesity and family history are well-known correlates of T2D, with over 85% of individuals being either overweight or obese at diagnosis, and most having a positive family history of T2D. The incidence of T2D is increasing dramatically, particularly among children and youth, most likely attributable to the increase in obesity among youth. This increasing incidence is expected to continue in the future unless significant prevention efforts are successfully implemented at the population level.

T2D is generally managed by prescription of oral medications such as Metformin to help reduce blood glucose, but insulin is also used in the treatment of T2D. Because of the association of obesity with insulin resistance, weight loss is another important goal of treatment, achieved through lifestyle modification of dietary habits and physical activity. The health complications associated with poorly controlled diabetes, whether from type 1 or T2D, include cardiovascular disease, renal disease, blindness, and limb amputations. Thus, management of T2D constitutes an important public health issue. In the past, T2D was referred to as non-insulin-dependent diabetes or adult onset diabetes. However, now that T2D is diagnosed at earlier ages in the life course and treatment often does utilize insulin, T2D is the accepted term.

Cross-References

► Insulin Resistance
► Type 1 Diabetes

References and Readings

Joslin, E. P., & Kahn, C. R. (2005). *Joslin's diabetes mellitus* (14th ed.). Philadelphia, PA: Lippincott Williams & Willkins.

Kaufman, F. R. (2002). Type 2 diabetes mellitus in children and youth: A new epidemic. *Journal of Pediatric Endocrinology and Metabolism, 15*, 737–744.

Type 2 Diabetes Prevention

► Diabetes Prevention Program

Type A Behavior

Shin-ichi Suzuki[1] and Yoshihiko Kunisato[2]
[1]Faculty of Human Sciences, Graduate School of Human Sciences, Waseda University, Tokorozawa-shi, Saitama, Japan
[2]Department of Psychiatry and Neurosciences, Hiroshima University, Minami-ku, Hiroshima, Japan

Definition

The type A behavior pattern is a personality type which is a risk factor for coronary artery disease. It was described by Friedman and Rosenman in 1959. The type A behavior pattern is defined as a complex set of action and emotion including floating hostility, sense of time urgency, impatience, intense achievement drive, and a desire for recognition and advancement. It has been reported that the type A behavior pattern associates with inadequacy and low self-esteem.

The association between type A behavior pattern and coronary artery disease has been examined for more than a few decades and strong epidemiological evidences in this association have appeared. However, successive studies have failed to find the association between type A behavior pattern and coronary artery disease. From these findings, hostility has been supposed to be the main psychosocial predictor of coronary artery disease instead of global type A behavior

pattern (Razzini et al., 2008; Trigo, Silva, & Rocha, 2005).

The type A behavior pattern is originally assessed by the structured interview. The interviewer must have training to be able to ask the question in an interview format before administering the structured interview. Also, some self-report questionnaires have been developed. For example, the Bortner Rating Scale and Framingham Type A Scale assess the type A behavior pattern. Although self-report questionnaires have been used widely, Friedman (1996) pointed out that inconsistencies of the association between type A behavior pattern and coronary artery disease were caused by the assessment method including the such as self-report questionnaires and the structured interview. Friedman proposed the type A videotaped clinical examination (VCE), which can detect the physical signs of type A behavior pattern and was valid for diagnosis of type A behavior pattern. The VCE method has predictive validity for myocardial infarctions.

The modification of type A behavior pattern has been proposed and summarized by Friedman (1996). The modification of type A behavior pattern consist of some components including the enhancement of self-esteem and the modification of floating hostility and sense of time urgency. Friedman et al. (1986) conducted a large randomized clinical trial to examine the effect of type A modification group therapy for myocardial infarction male patients. Their results showed that the coronary artery disease recurrence rate in treatment group was lower than that of control group.

Cross-References

- ▶ Behavioral Medicine
- ▶ Coronary Artery Disease
- ▶ Coronary Heart Disease
- ▶ Health Psychology
- ▶ Hostility, Cynical
- ▶ Personality
- ▶ Trait Anger

References and Readings

Friedman, M. (1996). *Type A behavior: Its diagnosis and treatment*. New York: Plenum Press.

Friedman, M., Thoresen, C. E., Gill, J. J., Ulmer, D., Powell, L. H., Price, V. A., et al. (1986). Alteration of type A behavior and its effect on cardiac recurrences in post myocardial infarction patients: Summary results of the recurrent coronary prevention project. *American Heart Journal, 112*, 653–65.

Razzini, C., Bianchi, F., Leo, R., Fortuna, E., Siracusano, A., & Romeo, F. (2008). Correlations between personality factors and coronary artery disease: From type A behaviour pattern to type D personality. *Journal of Cardiovascular Medicine (Hagerstown, Md.), 9*, 761–768.

Trigo, M., Silva, D., & Rocha, E. (2005). Psychosocial risk factors in coronary heart disease: Beyond type A behavior. *Revista Portuguesa de Cardiologia, 24*, 261–281.

Type A Behavior Pattern (TABP)

▶ Heart Disease and Type A Behavior

Type D Personality

Johan Denollet
CoRPS – Center of Research on Psychology in Somatic diseases, Tilburg University, Tilburg, The Netherlands

Synonyms

Distressed personality type

Definition

The Type D (distressed) personality refers to *a general propensity to psychological distress* (Denollet, Schiffer, & Spek, 2010) *that is defined by elevated scores on two broad personality traits*, *negative affectivity* (NA), and *social inhibition* (SI). NA refers to the tendency to experience negative emotions across time and

situations, and SI to the tendency to inhibit the expression of emotions and behaviors in social interaction (Denollet, 2005).

Description

Individuals with a Type D personality are more likely to report *feelings of dysphoria, tension, and worry* (Denollet, 2005). On an interpersonal level, Type D individuals tend to feel *insecure and inhibited* in the company of others, fearing rejection and disapproval (Denollet). Although these individuals may experience emotional difficulties, this may not be acknowledged by others given their inhibited behavior. Type D individuals are less likely to express their true thoughts and feelings and may keep other people at a distance.

A number of studies have reported that Type D was associated with an increased risk of *mortality and other adverse events in cardiac patients*, even after statistical adjustment for measures of depression or anxiety. In 1996, a paper published in the Lancet was one of the first reports on Type D personality as an independent predictor of mortality in patients with heart disease (Denollet et al., 1996). A meta-analytic review that summarized the findings from Type D studies that were published over a 15-year period (1995–2009) concluded that this personality profile may be related to adverse health outcomes in patients with a cardiovascular condition (Denollet et al., 2010). Another independent meta-analytic review confirmed that Type D was associated with adverse health outcomes among patients with cardiovascular disorder (O'Dell, Masters, Spielmans, & Maisto, 2011). There are also null studies that found no effect of Type D personality on mortality in patients with heart failure (Coyne et al., 2011; Pelle et al., 2010) or other cardiac conditions (Grande et al., 2011). However, depression or anxiety also failed to predict prognosis in these null studies.

The *prevalence* of Type D among patients with cardiovascular disease largely ranges between 25% and 35%. Type D personality and its two components, NA and SI, can be reliably assessed with the *DS14 self-report scale* (Denollet, 2005). The 14 items of the DS14 are rated on a five-point Likert scale, ranging from 0 (false) to 4 (true) and are divided into NA and SI subscales. The seven NA items cover the tendency to experience feelings of dysphoria, anxiety, and irritability. The seven SI items cover social discomfort, reticence, and lack of social poise. These personality measures have good internal consistency and are stable over time. Due to its brevity and the simplicity of the items, completing the DS14 takes only a few minutes and comprises little burden to patients. The DS14 has been validated in multiple languages, making it widely applicable. In the International HeartQoL study of 6,222 patients with ischemic heart disease, cross-cultural measurement equivalence was demonstrated for the Type D scale in 21 countries (Kupper et al., 2012).

Type D research is based on the notion that (a) research should examine *the way traits combine* in the determination of disease, and that (b) the *delineation of subtypes* may help to identify groups of patients who share a set of relevant characteristics in terms of clinical course. Only those individuals scoring positive on both NA and SI are classified as "Type D." Type D caseness is determined by a cutoff score $\geq$10 on both the NA and SI subscales. Some have argued that Type D personality is more accurately represented as a *dimensional* rather than as a *categorical* construct (Ferguson et al., 2009). The Type D construct does not infer a true taxon that is defined by discontinuity between groups on an underlying dimension; rather, individuals belong only probabilistically to Type D and non-Type D subgroups (Denollet et al., 2010). Therefore, dimensional and categorical approaches to Type D personality do not need to be mutually exclusive, but rather represent two different ways of capturing psychological tendencies of individuals (Chapman, Duberstein, & Lyness, 2007).

General distress, shared across anger, depression, and anxiety, partly accounts for the link between mind and heart (Denollet & Pedersen, 2009). The Type D personality profile identifies

individuals who are particularly vulnerable to this adverse effect of general distress. Hence, Type D personality is not a concurrent of standard psychological risk factors such depression, anxiety, or stress, but rather aims at the early identification of individuals who are inclined to experience these manifestations of distress over a longer period of time. At first glance, depression and Type D personality may appear quite similar, but there are some clear differences. While depression reflects psychopathology, Type D represents a normal personality construct. Accordingly, a narrative review of 29 studies showed that Type D personality and depression are distinct manifestations of psychological distress, with different and independent cardiovascular effects (Denollet et al., 2010). It is not surprising that there is some overlap between Type D and the neuroticism and extraversion traits of the Five Factor Model of personality. However, Type D still predicts health outcomes after controlling for these traits (Denollet et al.), and both the Five Factor and Type D models are related to health outcomes in primary care patients (Chapman et al., 2007).

Several biological and behavioral pathways may *explain the link* between Type D and health outcomes. Potential *biological pathways* associated with Type D personality include elevated levels of the stress hormone cortisol (Molloy et al., 2008), elevated biomarkers of inflammation (Conraads et al., 2006; Einvik et al., 2011), decreased capacity to repair vascular damage (Van Craenenbroeck et al., 2009), and reduced heart rate recovery after exercise (von Känel et al., 2009). Type D has also been related to cardiovascular effects during experimental stress, including higher cardiac output (Williams, O'Carroll, & O'Connor, 2009) and blood pressure (Habra, Linden, Anderson, & Weinberg, 2003), and lower heart rate variability (Martin et al., 2010).

Behavioral pathways may also mediate the relationship between Type D personality and adverse health outcomes (Williams et al., 2008). In the International HeartQoL study of cardiac patients from 21 different countries, Type D was associated with a higher prevalence of hypertension, smoking, and a sedentary lifestyle (Kupper et al., 2012). In the general population, Type D has also been linked to unhealthy behaviors such as smoking and physical inactivity (Einvik et al., 2011; Hausteiner et al., 2010). Type D individuals may show reluctance to consult clinical staff for cardiovascular symptoms (Schiffer, Denollet, Widdershoven, Hendriks, & Smith, 2007), and are not likely to seek care for their mental problems (Williams et al., 2008). In the medical care for patients with chronic condition, *adherence to treatment* may be of particular importance. Type D has been associated with poor adherence to treatment in patients with cardiac (Williams, O'Connor, Grubb, & O'Carroll, 2011) and sleep (Broström et al., 2007) disorders.

Type D personality has been mainly studied in cardiovascular patients, but there is evidence to suggest that Type D personality can also provide relevant information in *other populations* as well. Type D has been related to poor patient-reported health outcomes in patients with *other conditions*. In cancer survivors, for example, Type D personality has been associated with impaired quality of life and poor mental health (Mols, Thong, de Poll-Franse, Roukema, & Denollet, 2012). In the *general population*, Type D individuals have been shown to have an increased risk for clinically significant depression, panic disorder, and alcohol abuse (Michal, Wiltink, Grande, Beutel, & Brähler, 2011). In this study, Type D was also robustly associated with major stressors such as traumatic events and social isolation. These authors concluded that Type D as a frequent disposition is of high relevance for health care (Michal et al., 2011). In other studies of individuals without cardiovascular disease, Type D personality was related to unhealthy behaviors such as smoking and low physical activity (Einvik et al., 2011; Hausteiner et al., 2010).

The evidence so far seems to indicate that patients with a Type D personality profile are at increased risk of for a multitude of adverse health outcomes, particularly in the context of cardiac disease. However, there still are a number of unresolved issues. Evidence suggests that social inhibition modulates the adverse effect of

negative emotions on cardiac prognosis (Denollet et al., 2006) but more research is needed to test this model. Although to date the optimal treatment and the applicability of counseling options for Type D individuals are still unknown, screening may identify these high-risk patients. Overall, the findings of Type D research support the simultaneous use of specific and general measures of distress in cardiovascular research and practice. In this context, *screening for Type D personality with the DS14* (Denollet, 2005) may be useful to improve clinical research and practice in the context of cardiovascular disease and other chronic conditions.

Cross-References

- ▶ Negative Affectivity
- ▶ Social Inhibition

References and Readings

Broström, A., Strömberg, A., Mårtensson, J., Ulander, M., Harder, L., & Svanborg, E. (2007). Association of Type D personality to perceived side effects and adherence in CPAP-treated patients with OSAS. *Journal of Sleep Research, 16*, 439–447.

Chapman, B. P., Duberstein, P. R., & Lyness, J. M. (2007). The distressed personality type: Replicability and general health associations. *European Journal of Personality, 21*, 911–929.

Conraads, V. M., Denollet, J., De Clerck, L. S., Stevens, W. J., Bridts, C., & Vrints, C. J. (2006). Type D personality is associated with increased levels of tumor necrosis factor (TNF)-alpha and TNF-alpha receptors in chronic heart failure. *International Journal of Cardiology, 113*, 34–38.

Coyne, J. C., Jaarsma, T., Luttik, M. L., van Sonderen, E., van Veldhuisen, D. J., & Sanderman, R. (2011). Lack of prognostic value of Type D personality for mortality in a large sample of heart failure patients. *Psychosomatic Medicine, 73*, 557–562.

Denollet, J. (2005). DS14: Standard assessment of negative affectivity, social inhibition, and Type D personality. *Psychosomatic Medicine, 67*, 89–97.

Denollet, J., & Pedersen, S. S. (2009). Anger, depression and anxiety in cardiac patients: The complexity of individual differences in psychological risk. *Journal of the American College of Cardiology, 53*, 947–949.

Denollet, J., Pedersen, S. S., Ong, A. T., Erdman, R. A., Serruys, P. W., & van Domburg, R. T. (2006). Social inhibition modulates the effect of negative emotions on cardiac prognosis following percutaneous coronary intervention in the drug-eluting stent era. *European Heart Journal, 27*, 171–177.

Denollet, J., Schiffer, A. A., & Spek, V. (2010). A general propensity to psychological distress affects cardiovascular outcomes: Evidence from research on the Type D (distressed) personality profile. *Circulation: Cardiovascular Quality and Outcomes, 3*, 546–557.

Denollet, J., Sys, S. U., Stroobant, N., Rombouts, H., Gillebert, T. C., & Brutsaert, D. L. (1996). Personality as independent predictor of long-term mortality in patients with coronary heart disease. *The Lancet, 347*, 417–421.

Einvik, G., Dammen, T., Hrubos-Strøm, H., Namtvedt, S. K., Randby, A., Kristiansen, H. A., et al. (2011). Prevalence of cardiovascular risk factors and concentration of C-reactive protein in Type D personality persons without cardiovascular disease. *European Journal of Cardiovascular Prevention and Rehabilitation, 18*, 504–509.

Ferguson, E., Williams, L., O'Conner, C., Howard, S., Hughes, B. M., Johnston, D. W., et al. (2009). A taxometric analysis of Type D personality. *Psychosomatic Medicine, 71*, 981–986.

Grande, G., Romppel, M., Vesper, J. M., Schubmann, R., Glaesmer, H., & Herrmann-Lingen, C. (2011). Type D personality and all-cause mortality in cardiac patients – Data from a German cohort study. *Psychosomatic Medicine, 73*, 548–556.

Habra, M. E., Linden, W., Anderson, J. C., & Weinberg, J. (2003). Type D personality is related to cardiovascular and neuroendocrine reactivity to acute stress. *Journal of Psychosomatic Research, 55*, 235–245.

Hausteiner, C., Klupsch, D., Emeny, R., Baumert, J., Ladwig, K. H., & for the KORA Investigators. (2010). Clustering of negative affectivity and social inhibition in the community: Prevalence of Type D personality as a cardiovascular risk marker. *Psychosomatic Medicine, 72*, 163–171.

Kupper, N., Pedersen, S. S., Höfer, S., Saner, H., Oldridge, N., & Denollet, J. (2012). Cross-cultural analysis of Type D (distressed) personality in 6222 patients with ischemic heart disease: A study from the International HeartQoL Project. *International Journal of Cardiology*, in press.

Martin, L. A., Doster, J. A., Critelli, J. W., Lambert, P. L., Purdum, M., Powers, C., et al. (2010). Ethnicity and Type D personality as predictors of heart rate variability. *International Journal of Psychophysiology, 76*, 118–121.

Michal, M., Wiltink, J., Grande, G., Beutel, M. E., & Brähler, E. (2011). Type D personality is independently associated with major psychosocial stressors and increased health care utilization in the general population. *Journal of Affective Disorders, 134*, 396–403.

Molloy, G. J., Perkins-Porras, L., Strike, P. C., & Steptoe, A. (2008). Type D personality and cortisol in survivors of acute coronary syndrome. *Psychosomatic Medicine, 70*, 863–868.

Mols, F., Thong, M. S., de Poll-Franse, L. V., Roukema, J. A., & Denollet, J. (2012). Type D (distressed) personality is associated with poor quality of life and mental health among 3080 cancer survivors. *Journal of Affective Disorders, 136*, 26–34.

O'Dell, K. R., Masters, K. S., Spielmans, G. I., & Maisto, S. A. (2011). Does Type D personality predict outcomes among patients with cardiovascular disease? A meta-analytic review. *Journal of Psychosomatic Research, 71*, 199–206.

Pelle, A. J., Pedersen, S. S., Schiffer, A. A., Szabó, B. M., Widdershoven, J. W., & Denollet, J. (2010). Psychological distress and mortality in systolic heart failure. *Circulation: Heart Failure, 3*, 261–267.

Schiffer, A. A., Denollet, J., Widdershoven, J. W., Hendriks, E. H., & Smith, O. R. (2007). Failure to consult for symptoms of heart failure in patients with a Type D personality. *Heart, 93*, 814–818.

Van Craenenbroeck, E. M., Denollet, J., Paelinck, B. P., Beckers, P., Possemiers, N., Hoymans, V. Y., et al. (2009). Circulating CD34 + KDR + endothelial progenitor cells are reduced in chronic heart failure patients as a function of Type D personality. *Clinical Science, 117*, 165–172.

von Känel, R., Barth, J., Kohls, S., Saner, S., Znoj, H., Saner, G., et al. (2009). Heart rate recovery after exercise in chronic heart failure: Role of vital exhaustion and Type D personality. *Journal of Cardiology, 53*, 248–256.

Williams, L., O'Carroll, R. E., & O'Connor, R. C. (2009). Type D personality and cardiac output in response to stress. *Psychology and Health, 24*, 489–500.

Williams, L., O'Connor, R. C., Grubb, N., & O'Carroll, R. (2011). Type D personality predicts poor medication adherence in myocardial infarction patients. *Psychology and Health, 26*, 703–712.

Williams, L., O'Connor, R. C., Howard, S., Hughes, B. M., Johnston, D. W., Hay, J. L., et al. (2008). Type D personality mechanisms of effect: The role of health-related behavior and social support. *Journal of Psychosomatic Research, 64*, 63–69.

U

Unexplained Patient Complaints

► Medically Unexplained Symptoms

Unexplained Symptoms

► Medically Unexplained Symptoms

Unintentional Nonadherence

Tavis S. Campbell, Jillian A. Johnson and Kristin A. Zernicke
Department of Psychology, University of Calgary, Calgary, AB, Canada

Synonyms

Noncompliance

Definition

Unintentional nonadherence refers to a non-deliberate alteration in treatment (e.g., medications, exercise, diet). Unintentional nonadherence includes forgetting, poor manual dexterity, lack of understanding of requirements, losing medications, or not being able to afford treatment (DiMatteo, 2004). Unlike intentional nonadherence that is more strongly related to individuals' beliefs, unintentional nonadherence is more strongly related to demographics and clinical variables, such as age, socioeconomic factors, and stage of illness (DiMatteo, 2004).

Cross-References

► Nonadherence
► Noncompliance

References and Readings

DiMatteo, R. M. (2004). Variations in patients' adherence to medical recommendations: A quantitative review of 50 years of research. *Medical Care, 42*(3), 200–209.
Horne, R. (2007). Adherence to treatment. In S. Ayers, A. Baum, & C. McManus (Eds.), *Cambridge handbook of psychology, health and medicine* (pp. 417–423). Cambridge, MA: Cambridge University Press.
Wroe, A. I. (2002). Intentional and unintentional nonadherence: A study of decision making. *Journal of Behavioural Medicine, 25*(4), 355–372.

Unipolar Depression

Amy Wachholtz
Department of Psychiatry, University of Massachusetts Medical School, Worcester, MA, USA

Synonyms

Major depressive disorder

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

Definition

Unipolar depression is characterized by a combination of two types of symptoms: neurovegetative and emotional-cognitive. Neurovegetative symptoms are those symptoms that are directly related to the body (e.g., insomnia/hypersomnia, dysregulated eating, fatigue, and decreased energy). Emotional-cognitive symptoms involve those symptoms that are related to how a person processes information (e.g., suicidal ideation, decreased concentration, feeling worthless, anhedonia, and depressed mood). A combination of these depression symptoms must be unremitting for more than 2 weeks in order to be diagnosed with depression. Common treatments for depression include cognitive-behavioral psychotherapy, antidepressant medications, or a combination of these treatments. There are also a number of complementary treatments that are gaining empirical research support for use in combination with traditional approaches to enhance treatment outcomes. These include treatments such as exercise therapy, light therapy, and vitamin B and D supplements.

Unipolar depression may be seen in conjunction with a number of physical health issues (see ▶ Comorbidity) including chronic pain, cancer, physical trauma, cardiac issues (heart attack), chronic health concerns and terminal stage illnesses. Additionally, some medications used to treat medical disorders can mimic or trigger depressive symptoms such as beta-blockers and interferon treatments.

The term *unipolar depression* is used to differentiate between this disorder and *bipolar depression* which involves periods of mania in addition to depression. The term depression is also differentiated from an, which may include feeling depressive symptoms due to a specific life change (e.g., job loss, divorce, recent cancer diagnosis), or *bereavement* due to the loss of a loved one. However, both adjustment disorder and bereavement may progress to unipolar depression if symptoms are present for a prolonged period of time.

Cross-References

▶ Comorbidity

References and Readings

American Psychiatric Association. (2000). Major depressive disorders. In *Diagnostic and statistical manual of mental disorders DSM-IV-TR* (4th ed., pp. 369–375). Washington, DC: Author.

United States Department of Labor

▶ Job Classification

Units of Nature

▶ Ecosystems, Stable and Sustainable

Univariate Analysis

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

Univariate analyses analyze one outcome variable at a time.

Cross-References

▶ Multivariate Analysis

References and Readings

Campbell, M. J., Machin, D., & Walters, S. J. (2007). *Medical statistics: A textbook for the health sciences* (4th ed.). Chichester, UK: Wiley.

Unprotected Sex

► Sexual Risk Behavior

Upper Respiratory Infection (Mild)

► Common Cold

Upper Respiratory Infection (Mild): Cause

► Common Cold: Cause

Upper Respiratory Infection (Mild): the Stress Factor

► Common Cold: The Stress Factor

Urothelial Carcinoma of the Bladder

► Cancer, Bladder

Usual Care

Manjunath Harlapur[1] and Daichi Shimbo[2]
[1]Center of Behavioral Cardiovascular Health, Division of General Medicine, Columbia University, New York, NY, USA
[2]Center for Behavioral Cardiovascular Health, Columbia University, New York, NY, USA

Synonyms

Control group of a randomized trial; Usual care arm

Definition

Although the definition of usual care has not been standardized, it can include the routine care received by patients for prevention or treatment of diseases.

Description

In cardiology, the type of routine care can vary by disease type and severity, the practice in which the patient is seen, health care system, and individual physician. Major task forces such from the American College of Cardiology and American Heart Association have published guidelines on the diagnosis, prevention, and treatment of several cardiovascular diseases. These guidelines are based on expert opinion as well as on the strength of the evidence. The purpose of these guidelines is to ensure that these recommendations are disseminated to the practicing cardiology community, as the type and quality of diagnostic, preventive, and treatment strategies widely varies.

In randomized trials, there has been some debate about the advantages and disadvantages of including a usual care arm as a control group. The Declaration of Helsinki states that the "benefits, risks, burdens, and effectiveness of a new method should be tested against those of the best current prophylactic, diagnostic, and therapeutic methods." In theory, it makes sense that in a randomized controlled trial, the usual care arm should be defined by the "best current" method available. The main advantage, of course, is to test the intervention against what is currently available in evidence-based clinical practice. However, there has been disagreement on what should constitute "usual" care in a randomized controlled trial. First, in the community, usual care can sometimes include suboptimal or older practices. Thus, whether "usual care" should really be changed to "optimal care" or "evidence-based care" remains unclear. Second, the outcome in the usual care arm may be affected by the Hawthorne effect. It is difficult to blind the physicians and the patients to being in the usual care arm. The physicians or the patients may improve or modify their behavior after finding out that they are not in the active intervention arm. Third, because physician treatment patterns

vary, the components of the usual care arm and their effects on the outcome are difficult to quantify. Some investigators have advocated for proposing a standardized treatment plan for patients randomized to the usual care arm. Further, differences between the active intervention arm and the usual care arm could be minimized if the usual care arm contains the proposed intervention. Finally, the types and nature of treatment typically given by physicians in the usual care arm could change during the study period.

For example, in the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT-LLT), 10,355 persons, aged 55 years or older with hypertension and moderately elevated low-density lipoprotein cholesterol levels, were randomized in an unblinded fashion to pravastatin (a statin) or to usual care. During a mean follow-up of 5 years, there was no significant difference in all-cause mortality or coronary heart disease events. ALLHAT-LLT was unique as it was one of the few trials that did not show the beneficial effects of statin therapy on cardiovascular events in patients who were at risk for future events. The reasons for the lack of difference in outcomes between the two arms are unknown, but the inclusion of a usual care arm as a control group may have played a role. For example, ALLHAT-LLT was conducted during a period that several other randomized trials of statin therapy were published. Thus, over time, physicians could have treated patients in the control arm with statin therapy. In fact, there was a steady increase in the use of statins in the usual care arm: 8.2% at year 2, 17.1% at year 4, and 26.1% at year 6. In addition to statins, other treatments in the usual care arm could have played a role. For example, physicians could have disproportionately recommended non-pharmacologic strategies (i.e., exercise, diet, and weight reduction) to their patients in the usual care arm as well. Given that the types of and intensity of the treatments in the usual care arm were likely variable, ultimately, these reasons remain speculative. Overall, ALLHAT-LLT is one example of the potential limitations, described above that may be associated with a usual care arm.

Cross-References

- ▶ Randomized Clinical Trial
- ▶ Randomized Controlled Trial

References and Readings

ALLHAT Officers and Coordinators for the ALLHAT Collaborative Research Group. (2002). Major outcomes in moderately hypercholesterolemic, hypertensive patients randomized to pravastatin vs usual care: The antihypertensive and lipid-lowering treatment to prevent heart attack trial (ALLHAT-LLT). *Journal of the American Medical Association, 288*, 2998–3007.

Smelt, A. F., et al. (2010, July). How usual is usual care in pragmatic intervention studies in primary care? An overview of recent trials. *British Journal of General Practice, 60*(576), e305–e318.

Usual Care Arm

- ▶ Usual Care

Uutela, Antti

Antti Uutela
Department for Lifestyle and Health, National Institute for Health and Welfare (THL), Helsinki, Finland

Biographical Information

Antti Uutela was born in Tammela, Finland, on March 27, 1946. He is Doctor of Social Sciences

(social psychology) and works as research professor and department director at the National Public Health Institute THL Department of Lifestyle and Participation, and also as professor of public health at the University of Tampere, Finland. Before the current positions, he worked as researcher at the Academy of Finland, as lecturer and assistant professor at University of Helsinki, and chief of laboratory at the National Public Health Institute KTL, respectively.

Uutela was educated as a cognitive social psychologist with a special interest in the link of attitudes and behavior. The latter and interest in practical applications of social psychology led him to start working with health-related applications in 1975, first at the Department of Social Psychology, and from 1980 at the Department of Public Health of the Helsinki University. In 1986, Uutela established a connection to the National Institute of Public Health KTL and to the Finrisk study. This contact was to become a signpost for his later professional career, which also included project membership in the Health 2000 Study.

When accepting the position of chief of the new Health Behavior Research Unit at National Public Health Institute KTL in 1994, Uutela started to look for possibilities of establishing a national member society associated to the International Society of Behavioral Medicine. This emerged rapidly, and he became the founding President of the Finnish Section of Behavioral Medicine, a section of Finnish Society of Social Medicine in 1994, the year when the section was also accepted as a full member of ISBM.

Uutela has been a member of the Board of the Finnish section ever since; he is also a member of the Swedish Society of Behavioral Medicine, and the Society of Behavioral Medicine. He has been active in the International Society of Behavioral Medicine in officer positions and organization of international meetings since 1996. He was the President of ISBM from 2004 to 2006, President-elect from 2002 to 2004, and immediate Past President and chair of the Awards committee from 2006 to 2008. He started his officer career in ISBM by becoming the treasurer of society for the period 1996–2002. He is the current Chair of the Finance Committee of ISBM (2008–2012). On several occasions, he has represented the Finnish section in the Governing Council. Dr. Uutela has belonged to the Editorial Board of IJBM from 1996 onward, been a program committee member for ISBM international congresses in 1996–2006, chair of the local organizing committee (ICBM, Helsinki 2002), and program track chair (1996–2000). He was the Poster session chair for the 2008 and 2010 meetings.

In addition to his work in behavioral medicine, Uutela has been involved in academic research and education, in several national and international committees and expert duties in health promotion, including program chair of the Nordic Public Health Conference in 2011. He is currently President of the Finnish Society of Sport Sciences LTS and a Board member of the Foundation for Sport and Health Sciences LIKES.

Major Accomplishments

Upon becoming the Unit chief at KTL, Uutela assumed leadership of the working age and senior citizens' lifestyle monitoring research, which produced information for public health planning and evaluation in the general population, its subgroups and regionally. This monitoring system is one of the oldest in the world, an excellent basis for evaluation and planning of national health policy, and national and international lifestyle statistics. The system has given rise to similar systems, e.g., in the Baltic countries of Europe. Uutela led study several projects funded by the Academy of Finland related to socioeconomic health inequalities and health promotion. From 2001 onward, he has participated and led intervention studies in the Good Old Age in Lahti region (GOAL) community intervention framework aimed at improving functional capacity of the aging population and preventing type 2 diabetes. He has been in charge of the nutrition intervention study focusing on Finnish male conscripts and also contributed to the upper primary school nutrition intervention at the National Public Health Institute. He has more than 300 publications.

During his ISBM presidency, Uutela worked actively to broaden the global basis of the Society: Asia, Central and Eastern Europe, and Middle and Southern America were especially targeted. In June 2005, the Governing Council of ISBM gathered in Mexico City in association with the Latin-American Regional Meeting in Behavioral Medicine. The Central European Society of Behavioral Medicine to started form with a network meeting in Targu-Mures, Romania, in November 2005. Contacts between ISBM and South America were strengthened during the President's visit to Caracas, Venezuela, in association to the Third Venezuelan Congress of Behavioral Medicine in October. A Thai Forum of Behavioral Medicine was held in Bangkok in December 2005 as a gateway to the 2006 ICBM there. Uutela with his fellow executives worked toward improvement of the administration and budgeting of ISBM. As an important item, in 2006, special funds in ISBM budgeting were devoted to developmental funds of ISBM to increase chances for global development and participation of behavioral medicine. The highly successful Bangkok ICBM, with Brian Oldenburg and Marc Gellman as program chairs, and Naiphinich Kotchabhakdi as chair of local organization, led to acceptance of the Central-Eastern Society of Behavioral Medicine as an ISBM member, and in a change of by-laws regarding individual membership.

V

Validity

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Definition

The primary objective of an experimental or nonexperimental research study is to obtain a valid estimate of the treatment effect of interest. Validity can be divided into considerations of internal validity and external validity (Rothman, Greenland, & Lash, 2008).

Both experimental and nonexperimental studies require consideration to be paid to study design, data acquisition, data management, and analysis. If all of these are of optimum quality and there are no imperfections in the study, the study is deemed valid and the correct result is provided. Any imperfections lead to bias of various types.

Internal validity addresses the validity of inferences concerning the source population, and external validity addresses the validity of inferences to the general population, an issue also known as generalizability.

Cross-References

- ▶ Bias
- ▶ Confounding Influence
- ▶ Experimental Designs
- ▶ Generalizability
- ▶ Nonexperimental Designs
- ▶ Statistical Inference

References and Readings

Kleinbaum, D. G., Sullivan, K. M., & Barker, N. D. (2007). *A pocket guide to epidemiology*. New York: Springer.

Rothman, K. J., Greenland, S., & Lash, T. L. (2008). Validity in epidemiologic studies. In K. J. Rothman, S. Greenland, & T. L. Lash (Eds.), *Modern epidemiology* (3rd ed., pp. 128–147). Philadelphia: Lippincott Williams & Wilkins.

Variability

- ▶ Variance

Variance

J. Rick Turner
Cardiovascular Safety, Quintiles, Durham, NC, USA

Synonyms

Variability

Definition

Variance is a sophisticated measure of dispersion that takes into account the position of every data

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

point about a central value, typically the mean. It is therefore a measure of the variability within a data set.

Imagine the following data set: 6, 8, 10, 12, and 14. The mean is the sum of all the values divided by the total number of values, i.e., $50/5 = 10$. How can we find a measure that will capture the totality of the dispersion of the numbers around the mean? An initial thought is to calculate the arithmetic distance each number, or score, lies from the mean, and sum these values. This leads to the following: -4, -2, 0, 2, and 4, the sum of which is 0. That is, the total deviation of the scores from the mean is 0.

This is actually true for any such calculation for any data set. The mathematics of calculating the mean ensures that the sum of the deviations of any set of scores from its mean is always zero. So, this strategy does not help convey the degree of dispersion around a central value.

However, one extra step takes us to a useful strategy: This involves squaring all of the deviations. Given that a negative number multiplied by another negative number produces a positive value, we now get the following for our original data set: 16, 4, 0, 4, and 16, which sum to 40. This value is known as the sum of squares. If most of the scores in a data set tend to be close to the mean, the sum of squares (the variance) will be relatively low. The converse is true when most of the scores tend to be relatively further away from the mean. It is therefore possible to have two data sets with identical means and yet very different sums of squares.

One further step is required to calculate the variance: The sum of squares is divided by the value that is one less than the number of scores in the data set. This value is called the degrees of freedom, and is discussed in the entry titled "▶ Degrees of Freedom." In this case, the sum of squares, 40, is divided by $(5 - 1)$, i.e., 4. The variance is therefore 10, which is coincidentally the same as the mean in this case.

In most cases, one further step will be taken: the standard deviation will be calculated as the square root of the variance, as discussed in the entry titled "▶ Standard deviation."

Cross-References

▶ Degrees of Freedom
▶ Standard Deviation

Vascular Abnormalities, Function

Jonathan Newman
Columbia University, New York, NY, USA

Description

Vascular function can be described as the function of the vascular endothelium, the monolayer of cells lining the intimal surface of the entire circulatory system. The surface of the vascular endothelium in one individual is enormous and has been estimated to contain roughly 3×10^{13} cells, covering the surface of more than six tennis courts. This section will review the cardinal domains of vascular function in cardiovascular disease; will describe putative pathogenic abnormalities in vascular function, and will briefly review measures to assess vascular function.

The regulation of vascular tone and hemostasis/coagulation are two primary domains of vascular function important in the pathogenesis of cardiovascular disease (CVD). There are other physiologically important vascular functions, including complex metabolic properties and the regulation of vascular permeability, but these domains are outside of this review. The regulation of vascular tone is a delicate balance of opposing vasoconstrictive and vasodilatory functions. The endothelial cells lining the vasculature have vasodilatory and vasoconstrictive properties and synthesize some local active molecules that have both local and systemic effects on vascular tone. The vasoconstrictive and vasodilatory properties of the endothelium that help maintain normal hemodynamics are a crucial portion of the response to local (vascular trauma, compromise) or systemic (congestive heart failure, hypertension) abnormalities. Vasodilation and

vasodilatory function is reviewed elsewhere (see ▶ Vasodilation, Vasodilatory Functions). Vasoconstriction is mediated in part by the endothelium-derived molecules endothelin-1, thromboxane-A_2, and platelet activating factor. Importantly, the vasodilatory and vasoconstrictive properties of the endothelium also have anti- and prothrombotic activities, respectively. This leads to delicate balance of vascular function in which the vasodilatory properties also promote blood fluidity through the inhibition of coagulation, whereas the vasoconstrictive properties of the endothelium promote platelet activation and hemostasis. Thus, the regulation of vascular tone interacts crucially with the other primary domain of vascular function, coagulation, and hemostasis, and in most normal circumstances, the endothelium constitutively expresses an antithrombotic surface to the blood that inhibits platelet aggregation and clot formation. When the endothelium is activated or compromised, however, it is capable of quickly becoming prothrombotic, activating platelets and promoting coagulation.

Given its importance, a number of different modalities have been developed and investigated to assess vascular function.

There are, however, two main categories of assessment.

The first and "gold standard" methodology is coronary angiography (catheterization) to measure coronary circulation with infusion of vasoactive medications, such as adenosine, to measure coronary blood flow and resistance.

However, this technique is invasive, expensive, and not without risk.

Therefore, relying on the principle that endothelial dysfunction is a systemic disorder, vascular function can be measured in other arterial beds in a less invasive manner.

The most widely studied method is flow-mediated dilation (FMD) of the brachial artery using high-resolution ultrasound.

During the measurement of FMD, brachial artery diameter is measured before and after an increase in wall stress induced by reactive hyperemia (increased blood flow) seen following inflation of a sphygmomanometer cuff proximal to the brachial artery for 5 min at high pressures, up to 200 mmHg.

The amount of dilation seen largely reflects endothelial function and the availability of important vasodilatory molecules, such as nitric oxide (NO).

FMD is predictive of the extent and severity of coronary atherosclerosis and is an independent predictor of prognosis.

Brachial artery FMD is also moderately correlated with coronary artery FMD.

However, FMD is operator dependent and requires significant training.

It is also influenced by catecholamines, levels of hormones such as estrogen and progesterone, stress level, and sleep deprivation.

Further, it remains unclear whether the assessment of FMD provides information that is additional to traditional risk factors.

A recent technology that utilizes similar methods of assessing changes following reactive hyperemia is finger-pulse plethysmography.

This technology relies on changes in pulse wave amplitude following reactive hyperemia in the finger.

These changes in amplitude are filtered, amplified, displayed, and stored, and a threshold has been identified that may have utility in identifying endothelial dysfunction within the coronary circulation.

Measurement of both brachial FMD and finger-pulse plethysmography has largely been restricted to research use, but investigation into the potential clinical applications of these modalities is ongoing.

References and Readings

Gori, T., Parker, J. D., & Manzel, T. (2010). Flow-mediated constriction: Further insight into a new measure of vascular function. *European Heart Journal, 32*, 784–787.

Lerman, A., & Zeiher, A. M. (2005). Endothelial function: Cardiac events. *Circulation, 111*, 363–368.

Moncada, S., & Higgs, E. A. (Eds.). (2006). *The vascular endothelium*. New York: Springer.

Munzel, T., Sinning, C., Post, F., Warnholtz, A., & Schulz, E. (2008). Pathophysiology, diagnosis and prognostic implications of endothelial dysfunction. *Annals of Medicine, 40*, 180–196.

Vascular Endothelial Growth Factor (VEGF)

Alexandra Erdmann and Erin Costanzo
Department of Psychiatry, Carbone Cancer Center, University of Wisconsin-Madison, Madison, WI, USA

Definition

Vascular endothelial growth factor (VEGF) is a key regulatory molecule that promotes growth of new blood vessels. VEGF plays an important role in normal physiological processes that require increased vascularization to bring oxygen and nutrients to tissues, including embryonic and postnatal development and wound healing (Ferrara, Gerber, & LeCouter, 2003). VEGF can also contribute to disease processes. For example, upregulation of VEGF has been linked to development of intraocular neovascular syndromes, inflammatory disorders, and brain edema, among others (Ferrara et al., 2003). VEGF also plays a critical role in tumor growth and development. Specifically, VEGF recruits endothelial cells to the tumor site, allowing for growth of capillary networks that enable tumor development by increasing the supply of oxygen and nutrients essential for cell division and growth (Antoni et al., 2006; Ferrara et al., 2003). The vascularization of a tumor in its early stages allows the mass to grow beyond a critical point and to eventually metastasize, or spread through the circulatory system to other tissues and organs (Kerbel, 2000). Consequently, anti-VEGF agents have been developed for cancer treatment, with promising results from recent clinical trials (e.g., Escudier et al., 2007).

Of relevance to behavioral medicine, there is evidence that stress can affect VEGF production. Specifically, stimulation of in vitro ovarian, melanoma, and nasopharyngeal tumor cell lines with stress hormones such as norepinephrine, epinephrine, and cortisol leads to increased production of VEGF. Effects appear to occur via β-adrenergic signaling pathways and can be moderated by β-blockers, such as propranolol (Lutgendorf et al., 2003; Yang et al., 2006, 2009). These results suggest a potential pathway by which stress-related psychosocial factors may affect tumor development and progression. New investigations are determining the extent to which β-blockers may be promising therapeutic options (Costanzo, Sood, & Lutgendorf, 2011). These studies pinpoint a physiological pathway by which behavioral interventions targeting stress may have the potential to improve cancer outcomes.

Cross-References

- ▶ Cancer, Ovarian
- ▶ Cancer, types of
- ▶ Cortisol
- ▶ Cytokines
- ▶ Epinephrine
- ▶ Stress

References and Readings

Antoni, M. H., Lutgendorf, S. K., Cole, S. W., Dhabhar, F. S., Sephton, S. E., McDonald, P. G., et al. (2006). The influence of bio-behavioural factors on tumour biology: Pathways and mechanisms. *Nature Reviews Cancer, 6*, 240–248.

Costanzo, E. S., Sood, A. K., & Lutgendorf, S. K. (2011). Biobehavioral influences on cancer progression. *Immunology and Allergy Clinics of North America, 31*, 109–132.

Escudier, B., Pluzanska, A., Koralewski, P., Ravaud, A., Bracarda, S., Szczylik, C., et al. (2007). Bevacizumab plus interferon alfa-2a for treatment of metastatic renal cell carcinoma: A randomized, doubleblind phase III trial. *Lancet, 370*, 2103–2111.

Ferrara, N., Gerber, H., & LeCouter, J. (2003). The biology of VEGF and its receptors. *Nature Medicine, 9*, 669–676.

Kerbel, R. (2000). Tumor angiogenesis: Past, present, and the near future. *Carcinogenesis (London), 21*, 505–515.

Lutgendorf, S. K., Cole, S., Costanzo, E., Bradley, S., Coffin, J., Jabbari, S., et al. (2003). Stress-related mediators stimulate vascular endothelial growth factor secretion by two ovarian cancer cell lines. *Clinical Cancer Research, 9*, 4514–4521.

Yang, E. V., Kim, S. J., Donovan, E. L., Chen, M., Gross, A. C., Webster Marketon, J. I., et al. (2009). Norepinephrine upregulates VEGF, IL-8, and IL-6

expression in human melanoma tumor cell lines: Implications for stress-related enhancement of tumor progression. *Brain, Behavior, and Immunity, 23*, 267–275.
Yang, E. V., Sood, A. K., Chen, M., Li, Y., Eubank, T. D., Marsh, C. B., et al. (2006). Norepinephrine upregulates the expression of vascular endothelial growth factor, matrix metalloproteinase (MMP)-2, and MMP-9 in nasopharyngeal carcinoma tumor cells. *Cancer Research, 66*, 10357–10364.

Vascular Headache

► Migraine Headache

Vasoconstriction

Leah Rosenberg
Department of Medicine, School of Medicine, Duke University, Durham, NC, USA

Definition

Vasoconstriction is the process by which smooth muscle causes contraction and narrowing along the vessel length. The mechanism of vasoconstriction is mediated by intracellular calcium levels and a variety of calcium-binding proteins, particularly calmodulin (Barrett, Barman, Boitano, & Brooks, 2010).

Vasoconstriction is a physiologic cardiovascular regulatory mechanism that has essential functions throughout the body. Vasoconstriction may be triggered locally in the vasculature or from afar by upstream mediators in response to a variety of physical and emotional stimuli. It is also an example of autoregulation, when organism homeostasis is maintained through balance of vasoconstriction and vasodilatory mechanisms. While most changes are in response to blood flow of the vascular bed or an intrinsic reaction to stretch, there are many other stimuli that induce acute and chronic changes. In the event of vascular injury or compression, there is local response thought to be mediated by serotonin released by platelets that have accumulated proximate to the injured tissue. In addition to serotonin, there are a variety of other vasoconstrictor proteins that have systemic effects.

Several mechanisms have been developed to measure vasoconstriction in the central as well as peripheral vasculature. These are mostly utilized in research settings and have been put into mainstream clinical practice. However, the noninvasive measurement of vascular tone may soon prove to have indications for identifying patients at increased risk for complications like heart attack and stroke.

Cross-References

► Coronary Vasoconstriction

References and Readings

Barrett, K. E., Barman, S. M., Boitano, S., & Brooks, H. (2010). Chapter 33. Cardiovascular regulatory mechanisms. In K. E. Barrett, S. M. Barman, S. Boitano, & H. Brooks (Eds.), *Ganong's review of medical physiology* (23rd ed.). New York: McGraw-Hill.

Vasodilation, Vasodilatory Functions

Jonathan Newman
Columbia University, New York, NY, USA

Definition

Vasodilation refers to the opening or enlargement of blood vessels as a result of relaxation in the smooth muscle cells lining the arteries and to a lesser extent, the veins of the human body. Vasodilation is largely the reverse of the vasoconstriction, which refers to the narrowing of the same blood vessels throughout the human body.

Description

In general, vasodilation leads to a decrease in resistance in that vascular structure and a subsequent

increase in blood flow. This relationship can be illustrated by examining the hemodynamic relationships of total peripheral resistance (TPR). Total peripheral resistance is equal to the difference between mean arterial pressure (MAP) and mean venous pressure (MVP), divided by the cardiac output (CO), represented by the equation: TPR = (MAP − MVP)/CO. In general, resistance in tubular structures, like blood vessels, is inversely proportional to the radius of that tube (blood vessel) raised to the 4th power (Resistance = $1/\text{radius}^4$). Therefore, any increase in the radius of a blood vessel produces a significant decrease in the resistance of the vessel in question. In general, vasodilation works to decrease TPR through the actions of local and systemic factors that relax the smooth muscle cells in and around arteries and arterioles, increasing the size of blood vessels through a change in their radius.

Vasodilation plays an important role in the maintenance of body temperature through the vasodilation of superficial blood vessels and the release of heat into the cooler air surface surrounding the human body. Vasodilation is mediated by local or paracrine factors secreted by the endothelial cells themselves such as nitric oxide, bradykinin, potassium, and adenosine diphosphate (a breakdown product of working muscles). Vasodilatory function has an important role in the pathophysiology and treatment of cardiovascular disease and impaired vasodilation, and vasodilatory function has been demonstrated to be an important component of atherosclerotic coronary artery disease (see ▶ Vascular Abnormalities, Function). Impaired vasodilation is thought to reflect reduced nitric oxide bioavailability. Further, the promotion or stimulation of vasodilation with different cardioactive medications has proven therapeutic benefit in conditions such as mitral regurgitation, aortic regurgitation, hypertension, coronary artery disease, and congestive heart failure.

In general, the stimulation of vasodilation may help to reduce the amount of regurgitant blood flow seen in aortic and mitral regurgitation. Acutely, intravenous vasodilation in these conditions may reduce ventricular pressures and increase forward flow, potentially improving ventricular function. It is not clear if the hemodynamic improvement seen with vasodilator use in regurgitant valvular lesions is due to the reductions in blood pressure alone or through a combination of factors. In heart failure, the use of vasodilators encompasses both arterial and venous vasodilation, typically with agents such as hydralazine (arteriolar dilation) and nitrates (venodilation). The next effect of the use of these two vasodilators is to reduce the amount of a forward (preload) and "backward" (afterload) pressure on the heart, thereby reducing myocardial work and improving function. The antihypertensive effect of vasodilators is due mostly to the reduction arterial tone and therefore blood pressure. In coronary disease, nitrates can have profound anti-ischemic effects through their actions as both arterio- and venodilators, though the main effects of vasodilation in patients with coronary disease may be through decreasing ventricular filling (preload) which decreases wall tension, cardiac work, and hence myocardial oxygen demand, and may in turn lessen anginal symptoms.

In conclusion, vasodilation and vasodilatory function is an important component in both normal cardiovascular function and in the pathogenesis of disease. As described, the promotion of vasodilation is a useful therapeutic target for the treatment of a wide range of cardiovascular diseases.

Vasopressin

George J. Trachte
Academic Health Center, School of Medicine-Duluth Campus, University of Minnesota, Duluth, MN, USA

Synonyms

Antidiuretic hormone

Definition

Background: Vasopressin (antidiuretic hormone) is essential for regulating the osmolarity of blood

and also influences blood pressure. It has numerous behavioral influences such as reinforcing bonding between mating pairs, enhancing memory, and promoting aggressive behavior.

Physiological Relevance: Vasopressin is required for concentration of urine. It is present in all terrestrial animals, presumably to allow water retention. The absence of vasopressin results in a condition called diabetes insipidus, characterized by excretion of large volumes of dilute urine and the need to consume large quantities of water to survive. Vasopressin typically influences cells in the collecting duct of the kidney to promote water retention and concentrate the urine. Urine concentration cannot occur naturally in its absence.

Vasopressin also is essential for bond formation in a number of species. Mating bonds occurring in normally monogamous rodents are prevented by blockade of vasopressin V1a receptors in the brain. It is suspected to have a similar role in humans. Memory also is enhanced by vasopressin in a number of species but the physiological relevance of this potential action remains to be determined in humans. Finally, vasopressin constricts blood vessels to elevate blood pressure and this action might be important in hypovolemic states such as hemorrhagic shock.

Vasopressin actions are mediated by at least three separate receptors termed V1a, V1b, and V2. The V1a receptor mediates vasoconstrictor effects of vasopressin whereas V2 receptors mediate the renal effects to concentrate urine. The exact role of V1b receptors has not been determined at this point.

Control of Release/Secretion: Vasopressin is synthesized in the supraoptic and paraventricular nuclei of the hypothalamus and is transported to the posterior pituitary gland within magnocellular neurons. Vasopressin is synthesized as a 168 amino acid preprohormone that is converted to a 145 amino acid prohormone with the nine amino acids of the amino terminal representing the final vasopressin molecule. Vasopressin is stored in the posterior pituitary and released in response to plasma osmolarity exceeding 280 mM, sexual activity, or hypotension.

Localization/Molecular Biology: The preprovasopressin gene is located on chromosome 20 in humans. It is expressed in the supraoptic and paraventricular nuclei of the hypothalamus. Another site of synthesis is the heart, although the hypothalamic nuclei are believed to be the primary sites of synthesis.

Behavioral Actions: Vasopressin has at least three prominent behavioral actions, including: promotion of pair bonding; improvement of memory; and increased aggression toward sexual rivals. The pair bonding data are derived primarily from monogamous rodents who fail to develop pair bonds when vasopressin V1a receptors are blocked. Similar actions are suspected in humans and vasopressin is referred to as a "bonding" hormone. Memory also is improved in humans and other species by infusions of vasopressin and V1a receptor antagonists can impair memory in some species. Finally, vasopressin increases aggression in males toward other males. It is viewed as a monogamy hormone, enhancing bonding with a mate and children while reinforcing protective behaviors.

Cross-References

▶ Blood Pressure
▶ Hypothalamus

Vegetative Nervous System

▶ Autonomic Nervous System (ANS)

Venereal Diseases

▶ Sexually Transmitted Diseases (STDs)

Ventromedial Nucleus

▶ Hypothalamus

Video Applications

▶ Williams LifeSkills Program

Vigilance

▶ Coffee Drinking, Effects of Caffeine

Visceral Adiposity

▶ Central Adiposity

Visceral Nervous System

▶ Autonomic Nervous System (ANS)

Visualization

▶ Guided Imagery

Vital Exhaustion

Douglas Carroll
School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, UK

Definition

Vital exhaustion is a prodromal constellation of symptoms including physical exhaustion and feelings of hopelessness preceding major coronary heart disease events such as myocardial infarction.

Description

Vital exhaustion was a concept first proposed by Appels some 25 years ago. Appels argued that exhaustion was not simply premonitory to cardiac events, reflecting established pathology and representing early warnings, the historic clinical view. Rather, he contended that the syndrome of vital exhaustion was casually related to subsequent events. The causal pathway was hypothesized to be the neuroendocrine mechanisms typically invoked as the link between psychosocial exposures and heart disease. Support for a causal role for vital exhaustion was gleaned from a number of subsequent observational epidemiological studies demonstrating an association between measure of exhaustion and subsequent all-cause mortality and cardiac disease mortality and morbidity. Unfortunately, such evidence is necessarily only indicative and provides insufficient surety of the direction of causation. Reverse causation, where heart disease that has not yet been formally diagnosed leads to symptoms of exhaustion, must remain a possibility. A growing literature illustrates how the inflammatory processes implicated in atherosclerosis may contribute to feeling of fatigue and depression. In addition, confounding by some unmeasured or poorly measured variable in these observational studies cannot be wholly dismissed.

The proper test of causation is intervention: a randomized control trial, whether patients are randomly allocated to a exhaustion treatment invention or to a condition in which no such treatment is available. Some 5 years ago, Appels and his colleagues reported the outcome of such an intervention. Participants were over 700 cardiac disease patients who had undergone successful angioplasty, a procedure for the surgical repair of block coronary arteries. Patients were randomized to a 6-month exhaustion intervention comprising among other things relaxation training and stress management, or to usual care. At the 18-month, but not the 6-month, follow-up, fewer intervention than usual care patients reported feeling exhausted and fewer were depressed. However, the intervention did not reduce the likelihood of patients having a recurrent cardiac disease event.

Cross-References

▶ Depression
▶ Heart Disease

References and Readings

Appels, A., Bar, F., van der Pol, G., Erdman, R., Assman, M., Trijsburg, W., et al. (2005). Effects of treating exhaustion in angioplasty patients on new coronary events. Results of the randomised Exhaustion Intervention Trial (EXIT). *Psychosomatic Medicine, 67*, 217–223.

Appels, A., & Mulder, P. (1989). Fatigue and heart disease. The association between 'vital exhaustion' and past, present, and future heart disease. *Journal of Psychosomatic Research, 33*, 727–738.

Carroll, D., Phillips, A. C., & Macleod, J. (2006). Intervening for exhaustion. *Journal of Psychosomatic Research, 61*, 9–10.

Vital Status

► Mortality

Vitality

Christiane A. Hoppmann[1] and Denis Gerstorf[2]
[1]Department of Psychology, University of British Columbia, Vancouver, BC, Canada
[2]Institute of Psychology, Humboldt University, Berlin, Germany

Definition

The concept of active life expectancy revolves around the fundamental question of how many of the years added to life through advances in average life expectancy are spent in reasonably good health. A comprehensive and multifaceted view of health considers a variety of different physical, mental, and psychological health aspects.

Description

Overview

Human life expectancies have almost doubled over the last century. As a result, an unprecedented number of individuals can expect to reach old age. This historically new situation challenges us to better understand the factors that may contribute to vitality in old age. This entry aims to shed light on two important questions: First, it introduces the concept of active life expectancy to address the fundamental question of how many of those years added to life are spent in reasonably good health. Second, it argues for a comprehensive multifaceted view on vitality in old age that takes into account important psychological variables, including well-being, social engagement, and cognitive functioning. It concludes by alluding to key challenges that this line of work has to confront in the future.

Active Life Expectancy

Recent increases in human life expectancies and longevity are a great achievement, due to many different factors including improved public health and medical advances in Western countries (► Longevity). Those demographic trends also pose important societal challenges such as the question whether the gains in quantity of life are accompanied by gains in (or at least maintenance of) quality of life. A central construct in this regard is active (or healthy) life expectancy, which is frequently defined as the number of years individuals can expect to live without chronic disability (Crimmins, Hayward, & Saito, 1996). Research using this concept has advanced our knowledge regarding segments of the population who benefit or who are excluded from such trends. This line of inquiry has also helped put some of the earlier enthusiasm about increasing longevity into perspective. For example, although women can typically expect to live longer than men, they often spend a greater portion of their lives in disabling conditions. Likewise, individuals with higher socioeconomic status do not only live longer, but they also enjoy old age in better health than individuals with lower socioeconomic status. Moreover, there are notable racial differences in active life expectancies with African Americans typically spending more years with disabilities than non-Blacks in the United States (Crimmins et al., 1996). More generally, research on active life expectancy has revealed important insights into which demographic strata can be expected to spend the years added to life in reasonably good health and which strata do not.

Apart from identifying key population-level predictors of vitality, this line of research has also fueled important discussions about big-picture trends. Different propositions have been put forward regarding future changes in morbidity-mortality dynamics. The probably most prominent example is the compression of morbidity hypothesis (Fries, 1980), according to which increases in active life expectancy occur at a faster rate than increases in life expectancy. As a consequence, the years people spend in ill health are compressed into an increasingly shorter period at the very end of life. Conversely, other scholars have proposed that further increases in longevity would result in increased disease prevalence, ultimately leading to an expansion of morbidity (Gruenberg, 1977). Thirty years later, the verdict is still out. It appears as if individual perceptions of the years spent in good health are indeed increasing at the population level. In contrast, trends for the years spent in disability differ by severity. Years spent with severe disabilities are declining, whereas years spent with less severe forms are on the rise (Christensen, Doblhammer, Rau, & Vaupel, 2009). More generally, some researchers are skeptical about the possibility to successfully compress morbidity (e.g., Crimmins et al., 1996) whereas others report promising findings regarding delayed disabilities among more recently born cohorts of older adults (e.g., Manton, Gu, & Lowrimore, 2008). Taken together, the epidemiological literature has posed big-picture questions regarding how increasing longevity impacts disease prevalence and has raised awareness to tremendous heterogeneity in how well older adults from different backgrounds can expect to live their last years of life.

Psychological Indicators of Vitality

A comprehensive understanding of vitality in old age fundamentally depends on how health is defined. Specifically, it has been proposed that the time is due to move away from disease-centered definitions of health (Ryff & Singer, 2000). For example, the WHO defines health as "a state of complete physical, mental and social well-being" (Official Records of the World Health Organization, 1948). This definition dovetails with notions of successful aging that emphasize the importance of older adults maintaining their cognitive and physical functioning as well as actively engaging with life (Rowe & Kahn, 1997). To provide a more holistic account of health with aging, social scientists have thus embarked on a search for psychological factors that contribute to a more comprehensive understanding of vitality in old age that goes beyond the mere absence of disease. For example, it has been shown that emotional well-being is closely linked with physical health (Pressman & Cohen, 2005). In a similar vein, social relationships seem to be associated with older adults' physical and mental health (Hoppmann & Gerstorf, 2009), and associations between cognitive functioning and physical health in old age are a well-established research finding (Schaie, 2005). Hence, psychological research promises to broaden the scope of what constitutes vitality in old age by pointing to the important role of such diverse factors as emotional well-being, motivational processes, and cognition.

In addition to such normative accounts of vitality, there is also a rich literature on interindividual differences in self-regulatory processes. Importantly, this line of research takes into account that older adults may be able to maintain their well-being when confronted with losses in functional health. For example, it has been shown that strategies of selective optimization with compensation, self-efficacy, and control processes are positively associated with health in old age (e.g., Baltes & Smith, 2004; Seeman, Unger, McAvay, & Mendes de Leon, 1999; Wrosch, Schulz, & Heckhausen, 2004).

Identifying key psychological factors that distinguish older adults who spend their final years in reasonably good health from those who suffer from disease is important because it allows us to move beyond a recognition of well-established risk factors of late-life disability (e.g., being male, low socioeconomic status, member of a minority) that are in fact unalterable. Hence, recognizing that psychological constructs are key ingredients of or central contributors to vitality in old age may offer important insight into factors

that may be amenable to intervention and where growth might be possible until old age (Ryff & Singer, 2000).

Future Directions

In a final step, we would like to highlight two key societal challenges that are intrinsically linked with the concept of vitality. First, it is going to become increasingly important to recognize the practical implications of (changes in) vitality. For example, many developed countries currently face a heated debate about whether or not to increase mandatory retirement age. Importantly, it seems as if more flexible and individualized retirement decisions are necessary so as to accommodate the specific needs of the aging population. Some older adults may simply not be well enough to continue working beyond age 65, whereas other 65+ year olds may benefit from staying in the work force for longer and would profit from work-related cognitive stimulation and social participation (Pinquart & Schindler, 2007).

Second, it is well possible that positive secular trends that have repeatedly been reported for earlier points in life such as retirement age may be offset or even reversed in more advanced ages or at the end of life. In fact, there is empirical evidence that later-born cohorts may experience steeper end-of-life declines in cognitive abilities than earlier-born cohorts (Gerstorf, Ram, Hoppmann, Willis, & Schaie, 2011). One way to interpret such results is that members of later-born cohorts may have survived diseases that would have resulted in death among members of earlier-born cohorts. However, the previously higher levels of (cognitive) functioning were not maintained for this manufactured survival time (Olshansky, Hayflick, & Carnes, 2002). More generally, it remains unclear if it will ultimately be possible to successfully compress morbidity into increasingly shorter time periods at the end of life. We have to keep in mind that humans were not designed to enjoy such long post-reproductive lives as it is often the case today and that chance plays a much greater role in determining health during the post-reproductive as compared to the reproductive years of life (Finch & Kirkwood, 2000).

Cross-References

▶ Longevity

References and Readings

Baltes, P. B., & Smith, J. (2004). Lifespan psychology: From developmental contextualism to developmental biocultural co-constructivism. *Research in Human Development, 1*, 123–144.

Christensen, K., Doblhammer, G., Rau, R., & Vaupel, J. W. (2009). Ageing populations: The challenges ahead. *Lancet, 374*, 1196–1208.

Crimmins, E. M., Hayward, M. D., & Saito, Y. (1996). Differentials in active life expectancy in the older population of the United States. *Journals of Gerontology: Social Sciences, 51*, 111–120.

Finch, C. E., & Kirkwood, T. B. L. (2000). *Chance, development, and aging*. New York, NY: Oxford University Press.

Fries, J. F. (1980). Aging, natural death, and the compression of morbidity. *New England Journal of Medicine, 303*, 1369–1370.

Gerstorf, D., Ram, N., Hoppmann, C. A., Willis, S. L., & Schaie, K. W. (2011). Cohort differences in cognitive aging and terminal decline in the Seattle Longitudinal Study. *Developmental Psychology, 47*(4), 1026–1041.

Gruenberg, E. F. (1977). The failures of success. *Milbank Memorial Fund Quarterly/Health and Society, 55*, 3–24.

Hoppmann, C., & Gerstorf, D. (2009). Spousal interrelations in old age – A mini review. *Gerontology, 55*(449), 459.

Manton, K. G., Gu, X., & Lowrimore, G. R. (2008). Cohort changes in active life expectancy in the U.S. elderly population: Experience from the 1982–2004 national long-term care study. *Journals of Gerontology: Social Sciences, 63*, 269–281.

Olshansky, S. J., Hayflick, L., & Carnes, B. A. (2002). Position statement on human aging. *Journals of Gerontology Series A: Medical and Biological Sciences, 57A*, B292–B297.

Pinquart, M., & Schindler, I. (2007). Changes of life satisfaction in the transition to retirement: A latent-class approach. *Psychology & Aging, 22*, 442–455.

Pressman, S. D., & Cohen, S. (2005). Does positive affect influence health? *Psychological Bulletin, 131*, 925–971.

Rowe, J. W., & Kahn, R. L. (1997). Successful aging. *The Gerontologist*, *37*, 433–440.

Ryff, C. D., & Singer, B. (2000). Interpersonal flourishing: A positive health agenda for the new

millennium. *Personality and Social Psychology Review, 4*, 30–44.
Schaie, K. W. (2005). *Developmental influences on intelligence: The Seattle Longitudinal Study*. New York, NY: Oxford University Press.
Seeman, T. E., Unger, J. B., McAvay, G., & Mendes de Leon, C. F. (1999). Self-efficacy beliefs and perceived declines in functional ability: MacArthur Studies of Successful Aging. *Journals of Gerontology: Psychological Sciences, 54B*, P214–P222.
World Health Organization. (1948). *Official records*, No. 2. New York.
Wrosch, C., Schulz, R., & Heckhausen, J. (2004). Health stresses and depressive symptomatology in the elderly: A control-process approach. *American Psychological Society, 13*, 17–20.

VO_2max Test

► Maximal Exercise Stress Test

Vocational Assessment

► Readiness for Return-to-Work (RRTW)

Vocational Evaluation

► Functional Versus Vocational Assessment

Vocational Testing

► Functional Versus Vocational Assessment

W

Waist Circumference

► Waist Girth
► Waist Size

Waist Circumference (WC)

Tavis S. Campbell, Jillian A. Johnson and Kristin A. Zernicke
Department of Psychology, University of Calgary, Calgary, AB, Canada

Synonyms

Girth

Definition

Waist circumference (WC) is considered a measure of the relative health risk associated with excess abdominal fat. Abdominal fat contains higher amounts of visceral fat, a fat that is produced by the liver, turned into ► cholesterol, and released into the bloodstream where it can form plaque on artery walls. Excess abdominal fat is associated with high ► cholesterol, ► high blood pressure, and ► cardiovascular disease.

Waist circumference may be a better indicator of obesity-related diseases than ► body mass index (BMI) (Cawley, 2006), especially among certain populations (e.g., elderly persons). Certain ethnic groups are genetically predisposed to store more fat in the abdomen, even at healthy weights (e.g., non-Hispanic blacks, people of Asian descent).

It is measured by first locating the upper hipbone and lowest rib, followed by placing the end of a measuring tape between these two points and wrapping horizontally around the abdomen.

Men with a waist circumference greater than 102 cm (40 in.) and women with a waist circumference exceeding 88 cm (35 in.) are considered to be at increased risk for developing obesity-related health problems, including type II ► diabetes, ► hypertension, and ► cardiovascular disease.

Cross-References

► Body Mass Index

References and Readings

Cawley, J. H. (2006). *Beyond BMI: The value of more accurate measures of fatness and obesity in social science research*. Cambridge, MA: National Bureau of Economic Research.

Heart and Stroke Foundation. (2010, January). *Healthy waists*. Retrieved April 8, 2011 from http://www.heartandstroke.com/site/c.ikIQLcMWJtE/b.3876195/

National Heart Lung and Blood Institute. (2011, April). *Assessing your weight and health risk*. Retrieved April 8, 2011 from http://www.nhlbi.nih.gov/health/public/heart/obesity/lose_wt/risk.htm

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

Waist Girth

Christopher Shaw
Institute of Sport, Exercise and Active Living, Victoria University, Melbourne, Australia

Synonyms

Waist circumference

Definition

Waist girth is related to abdominal visceral fat and predicts a number of disease risk factors associated with obesity.

Description

Obesity which is explained by an increase in body fat is associated with a clustering of risk factors known as the metabolic syndrome and originates from an imbalance between energy intake and energy expenditure. However, there is substantial evidence that body fat distribution rather than total fat mass per se plays a more important role in the development of such risk factors. Gynoid obesity (commonly referred to as "pear shaped") reflects adipose tissue accumulation around the hips and buttocks, whereas android obesity (more commonly referred to as "apple shaped") reflects increased abdominal fat deposition. Android obesity increases with age, is more prominent in males, and poses a significantly greater risk for the development of hypertension, dyslipidemia, insulin resistance, atherosclerosis, type 2 diabetes, coronary artery disease, and certain types of cancer and higher rates of mortality than gynoid obesity. Girth measurements can be used to assess such differences in body fat distribution, and measurement of waist girth specifically represents abdominal obesity. Therefore, it may be a more relevant measure for anyone looking to use simple anthropometric measurements to predict the risk of obesity-related diseases across populations or in response to interventions over time. A variety of gender and age-specific equations are also available to predict body fat percentage from waist girth measurements.

The measurement of waist girth can be used in conjunction with hip circumference to measure waist-hip ratio which is a common method used to predict many health hazards associated with obesity. However, waist girth is more predictive of obesity-related risk factors than waist-to-hip ratio as it is a more accurate reflection of deep visceral adipose tissue (Roche, Heymsfield, & Lohman, 1996). Furthermore, waist girth measurements also correlate well with the gold standard CT or MRI techniques for the measurement of visceral abdominal fat. The reason that this adipose tissue site is such a risk factor is complex but is likely related to it being more active to lipolytic stimuli such as catecholamines, expression of adrenoreceptors and receptors for insulin, glucocorticoids, and testosterone. Abdominal fat also expresses and releases a multitude of peptides and inflammatory cytokines which are also likely to be involved.

The standardized measurement of waist girth is described by the American College of Sports Medicine (*ACSM's Guidelines for Exercise Testing and Prescription*, 2006). Measurements should be made with a flexible, inelastic tape without compression of the subcutaneous adipose tissue. Measurements should be performed with the participant standing with their feet together and arms by their side. A horizontal measure is then taken at the narrowest part of the torso, typically between the bottom of the ribs and the iliac crest. Duplicate measures should be taken and repeated if the variation exceeds 5 mm.

Waist girth alone, or in combination with other anthropometric measures, has been used to classify and evaluate disease risk. However, different cut off points have been suggested depending on the specific population studied. For example, a waist circumference over 99 cm for males and over 89 cm for females is associated with a high risk for the development of obesity-related disease (American College of Sports Medicine, 2006). Further, a disease risk classification is also available taking into account both BMI and waist

circumference (see McArdle, Katch, & Katch, 2001). This describes a higher risk of disease when a BMI over 25 kg/m^2 is combined with a waist circumference greater than 102 or 88 cm for males and females, respectively.

Cross-References

- ▶ Waist Circumference
- ▶ Waist Size

References and Readings

American College of Sports Medicine. (2006). *ACSM's guidelines for exercise testing and prescription* (7th ed.). Philadelphia: Lippincott Williams & Wilkins.

McArdle, W. D., Katch, F. I., & Katch, V. L. (2001). *Exercise physiology: Energy, nutrition and human performance* (5th ed.). Philadelphia: Lippincott Williams & Wilkins.

Roche, A. F., Heymsfield, S. B., & Lohman, T. G. (1996). *Human body composition*. Champaign, IL: Human Kinetics.

Waist Size

Kazuo Hara
Department of Metabolic Diseases, Graduate School of Medicine, The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Synonyms

Waist circumference

Definition

There are several lines of evidence that suggest subjects with a large waist size are at increased risk of developing type 2 diabetes and cardiovascular diseases, even after adjustment for body mass index (BMI), an indicator of total adiposity (Hu et al., 2007; Winter et al., 2008; Yusuf et al., 2005). The large waist size reflects accumulation of excess abdominal fat, also known as central obesity, and is reported to be a primary risk factor for glucose intolerance, dyslipidemia, and high blood pressure, which together is now defined as metabolic syndrome (Kadowaki et al., 2006). Therefore, the measurement of waist size is essential for the screening of metabolic syndrome. Two international definitions for metabolic syndrome are currently used in daily practice, one provided by the International Diabetes Federation (IDF) (IDF Worldwide Definition of the Metabolic Syndrome) and the other by the National Cholesterol Education Program (NCEP) (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, 2001). The former criterion requires waist size above a certain cut point for a diagnosis of metabolic syndrome, whereas the latter one does not require a large waist size if the subject has three or more of the following: raised fasting plasma glucose concentrations, elevated blood pressure, elevated levels of triglycerides, and/or reduced levels of high-density lipoproteins.

The IDF recommends that the cut point for waist size used to define central obesity should vary according to ethnic group, and they proposed the following European-specific cut points for waist size: 94 and 80 cm for men and women, respectively (Alberti, Zimmet, Shaw, & IDF Epidemiology Task Force Consensus Group, 2005). Nonetheless, further extensive investigations must be performed before more suitable cut points can be established for use in clinical practice to accurately predict cardiovascular diseases.

The method of measurement must also be standardized. Waist size is usually measured at a level midway between the lowest rib and the iliac crest (Han, van Leer, Seidell, & Lean, 1995). However, the waist size is measured at the umbilical level in some countries, such as Japan, for screening metabolic syndrome (Matsuzawa, 2005). It is essential that the subjects being measured are not holding their breath and that the tape measure is not at an angle around their waist.

Cross-References

- ▶ Obesity
- ▶ Waist to Hip Ratio

References and Readings

Alberti, K. G., Zimmet, P., Shaw, J., & IDF Epidemiology Task Force Consensus Group. (2005). The metabolic syndrome – A new worldwide definition. *Lancet, 366*(9491), 1059–1062.

Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. (2001). Executive summary of the third report of the national cholesterol education program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (adult treatment panel III). *Journal of American Medical Association, 285*(19), 2486–2497.

Han, T. S., van Leer, E. M., Seidell, J. C., & Lean, M. E. (1995). Waist circumference action levels in the identification of cardiovascular risk factors: Prevalence study in a random sample. *British Medical Journal, 311*(7017), 1401–1405.

Hu, G., Tuomilehto, J., Silventoinen, K., Sarti, C., Männistö, S., & Jousilahti, P. (2007). Body mass index, waist circumference, and waist-hip ratio on the risk of total and type-specific stroke. *Archives of Internal Medicine, 167*(13), 1420–1427.

IDF Worldwide Definition of the Metabolic Syndrome. Retrieved form http://www.idf.org/idf-worldwide-definition-metabolic-syndrome

Kadowaki, T., Yamauchi, T., Kubota, N., Hara, K., Ueki, K., & Tobe, K. (2006). Adiponectin and adiponectin receptors in insulin resistance, diabetes, and the metabolic syndrome. *Journal of Clinical Investigation, 116*(7), 1784–1792.

Matsuzawa, Y. (2005). Metabolic syndrome – Definition and diagnostic criteria in Japan. *Journal of Atherosclerosis and Thrombosis, 12*(6), 301.

Winter, Y., Rohrmann, S., Linseisen, J., Lanczik, O., Ringleb, P. A., Hebebrand, J., et al. (2008). Contribution of obesity and abdominal fat mass to risk of stroke and transient ischemic attacks. *Stroke, 39*(12), 3145–3151.

Yusuf, S., Hawken, S., Ounpuu, S., Bautista, L., Franzosi, M. G., Commerford, P., et al. (2005). Obesity and the risk of myocardial infarction in 27,000 participants from 52 countries: A case-control study. *Lancet, 366*(9497), 1640–1649.

Waist to Hip Ratio

Kazuo Hara
Department of Metabolic Diseases, Graduate School of Medicine, The University of Tokyo, Bunkyo-ku, Tokyo, Japan

Synonyms

WHR

Definition

The waist hip ratio (WHR) is the ratio of waist circumference to hip circumference. It was reported that the mortality and the risk of coronary artery disease are positively correlated with WHR both in men and women (Lapidus et al., 1984; Larsson et al., 1984). It is well documented that subjects with high WHR and upper body obesity present insulin resistance (Peiris, Mueller, Smith, Struve, & Kissebah, 1986). According to the standard defined by World Health Organization (WHO) in 1999 about metabolic syndrome, in which type 2 diabetes mellitus, glucose tolerance, and insulin resistance are required items, obesity is defined as WHR >0.9 in men and WHR >0.85 in women (World Health Organization, 1999).

Cross-References

▶ Waist Size
▶ Obesity

References and Readings

Lapidus, L., Bengtsson, C., Larsson, B., Pennert, K., Rybo, E., & Sjöström, L. (1984). Distribution of adipose tissue and risk of cardiovascular disease and death: A 12 year follow up of participants in the population study of women in Gothenburg, Sweden. *British Medical Journal, 289*, 1257–1261.

Larsson, B., Svardsudd, K., Welin, L., Wilhelmsen, L., Björntorp, P., & Tibblin, G. (1984). Abdominal adipose tissue distribution, obesity, and risk of cardiovascular disease and death: 13 year follow up of participants in the study of men born in 1913. *British Medical Journal, 288*, 1401–1404.

Peiris, A. N., Mueller, R. A., Smith, G. A., Struve, M. F., & Kissebah, A. H. (1986). Splanchnic insulin metabolism in obesity. Influence of body fat distribution. *Journal of Clinical Investigation, 78*(6), 1648–1657.

World Health Organization. (1999). *Definition, diagnosis and classification of diabetes mellitus and its complication. Part 1: Diagnosis and classification of diabetes mellitus*. Geneva, Switzerland: Department of Noncommunicable Disease Surveillance.

Warmth

▶ Interpersonal Circumplex

Water Pill

► Diuretic

Ways of Coping Checklist (WCCL)

Susan Folkman
Department of Medicine, School of Medicine, University of California San Francisco, San Mateo, CA, USA

Definition

The Ways of Coping Checklist (WCCL) is a measure of coping based on Lazarus and Folkman's (1984) stress and coping theory. The WCCL contains 66 items that describe thoughts and acts that people use to deal with the internal and/or external demands of specific stressful encounters. Usually the encounter is described by the subject in an interview or in a brief written description saying who was involved, where it took place, and what happened. Sometimes a particular encounter, such as a medical treatment or an academic examination, is selected by the investigator as the focus of the questionnaire. Subjects respond on a 4-point Likert scale (0 = does not apply and/or not used; 3 = used a great deal), the extent to which the item was used in the specific stressful encounter.

Subscales

Factor analysis of data from a study of stressful encounters reported by a community sample of middle-aged married couples indicated eight scales: planful problem-solving, positive reappraisal, seeking social support, distancing, self-controlling, escape-avoidance, accepting responsibility, and confrontive coping (Folkman, Lazarus, Gruen, & DeLongis, 1986). The eight scales use 50 of the 66 items. The additional items are retained on the WCCL because some investigators find them useful. Subsequent factor analyses indicated variability in factor structure. Some factors such as planful problem-solving, positive reappraisal, escape-avoidance, and distancing are relatively stable. Other factors such as self-controlling, accepting responsibility, seeking social support, and confrontive coping are less stable. For a critical review of the WCCL, see Schwarzer & Schwarzer (1996).

Scoring

There are two systems for scoring the WCCL. The "Raw Score" method is to sum the ratings for each scale. This method provides a score for amount of each type of coping used in the specified event.

The "Relative Score" method, introduced by Vitaliano, Maiuro, Russo, and Becker (1987), controls for the variability in scale length. Relative scores are computed by first obtaining the mean item score for each scale. Once the mean effort (ME) is obtained for each scale, the relative effort is calculated by dividing the ME for the particular scale by the sum of the MEs for each of the scales.

Interpretation

The WCCL is descriptive, not diagnostic. Typically, scores are correlated with outcomes of interest, such as depressive symptoms, distress, positive mood, or a relevant behavior.

Investigators ask if there are population norms for comparison purposes. Stress and coping theory (Lazarus & Folkman, 1984) views coping as contextual, which means that there is no "standard" amount of coping that can be used as a norm. For example, certain types of coping (e.g., planful problem-solving) are used more in situations that are controllable while other types (e.g., distancing) are used more in situations that have to be accepted.

The Ways of Coping Checklist can be obtained through Mind Garden Publishers (www.mindgarden.com).

References and Readings

Folkman, S., Lazarus, R. S., Gruen, R. J., & DeLongis, A. (1986). Appraisal, coping, health status, and psychological symptoms. *Journal of Personality and Social Psychology, 50*, 571–579.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.
Schwarzer, R., & Schwarzer, C. (1996). A critical survey of coping instruments. In M. Zeidner & N. S. Endler (Eds.), *Handbook of coping: Theory, research, applications* (pp. 107–132). Oxford, UK: Wiley.
Vitaliano, P. P., Maiuro, R. D., Russo, J., & Becker, J. (1987). Raw versus relative scores in the assessment of coping strategies. *Journal of Behavioral Medicine, 10*, 1–18.

Web-Based Studies

▶ Internet-Based Studies

Weight

▶ Body Mass Index
▶ Lifestyle Changes

Weight Loss

▶ Lifestyle Changes

Weight Loss Surgery

▶ Bariatric Surgery

Weight: Control, Gain/Loss/ Reduction, Maintenance, Monitoring

Shuji Inada
Department of Stress Science and Psychosomatic Medicine, Graduate School of Medicine, The University of Tokyo, Tokyo, Japan

Definition

Weight is the mass of one's body. Weight is usually measured by a scale in kilograms, which are base units of mass in the international systems of unit. Weight is used as barometer of adiposity or condition of nutrition or index of growth in children. Weight is useful for intrapersonal comparisons, while body mass index (in adults) or Rohrer's index (in children), which is adjusted for body height, is used for interpersonal comparisons. Weight is also applied for index of water balance in patients who have water imbalance such as congestive heart failure or renal failure.

Since weight measured by a scale is affected by meal, urine, and stool, it is better to measure on the same time after bladder emptying and defecation.

Description

Weight Control

Weight control is to keep, increase, or decrease one's weight within a certain range. Weight category is classified with body mass index as follows (also see ▶ Body Mass Index) (Table 1).

Since overweight and obese population is increasing, "weight control" often means "weight loss." Weight control is essential to prevention and treatment of many diseases, especially of metabolic disease (Must et al., 1999; Strazzullo et al., 2010).

Weight Gain/Loss/Reduction/Maintenance

Weight gain/loss depends on an energy balance under a normal water balance. Energy intake is usually derived from food intake. Energy consumption includes basal metabolism, consumption for physical activity, and consumption for thermogenesis. Energy intake exceeding energy consumption results weight gain, and energy consumption exceeding energy intake results weight loss.

Diet is the most important for weight reduction, and exercise is often combined with diet. Exercise alone has minimal effect for body weight (Franz et al., 2007). Sibtramine and Orlistat are approved by FDA for use in obesity patients (see ▶ Obesity: Prevention and Treatment). Bariatric surgery is performed to severe obese patients (see ▶ Bariatric Surgery).

Weight: Control, Gain/Loss/Reduction, Maintenance, Monitoring, Table 1 The international classification of adult underweight, overweight, and obesity according to BMI (WHO, 2006)

Classification	Principal cutoff point
Underweight	<18.5
Normal	18.5–24.99
Overweight	25–29.99
Obese class I	30–34.99
Obese class II	35–39.99
Obese class III	>40

Behavioral therapy is applied to weight management and achieves preferable short-term outcome. Behavioral management of obesity includes self-monitoring of diet, physical activity, and/or body weight; stimulus control; cognitive restructuring; goal setting; and problem solving (Johnston, Tyler, & Foreyt, 2007).

Maintenance of reduced weight is more difficult than weight reduction. Some regain of weight is inevitable for nonsurgical weight-loss therapy (Franz et al., 2007).

Monitoring

Self-monitoring is an important element of behavioral weight-loss program. Self-monitoring of dietary intake, physical activity, and/or body weight achieves self-evaluation of the weight-loss behavior, one's awareness of weight and behavior, and self-reinforcement of preferable behavior (Burke, Wang, & Sevick, 2011). While paper-pencil diaries are conventionally used for self-monitoring, internet-based diaries or PDA-based diaries are recently developed for easier recording and less-biased data recording (Burke et al., 2011; Fukuo et al., 2009).

Cross-References

- ▶ Bariatric Surgery
- ▶ Basal Metabolic Rate
- ▶ Body Mass Index
- ▶ Obesity: Prevention and Treatment
- ▶ Self-monitoring

References and Readings

Burke, L. E., Wang, J., & Sevick, M. A. (2011). Self-monitoring in weight loss: A systemic review of the literature. *Journal of American Dietetic Association, 111*, 92–102.

Franz, M. J., vanWormer, J. J., Crain, A. L., Boucher, J. L., Histon, T., Caplan, W., et al. (2007). Weight-loss outcomes: A systematic review and meta-analysis of weight-loss clinical trials with a minimum 1-year follow-up. *Journal of American Dietetic Association, 107*, 1755–1767.

Fukuo, W., Yoshiuchi, K., Ohashi, K., Togashi, H., Sekine, R., Kikuchi, H., et al. (2009). Development of a hand-held personal digital assistant-based food diary with food photographs for Japanese subjects. *Journal of American Dietetic Association, 109*, 1232–1236.

Johnston, C. A., Tyler, C., & Foreyt, J. P. (2007). Behavioral management of obesity. *Current Atherosclerosis Reports, 9*, 448–453.

Must, A., Spadano, J., Coakley, E. H., Field, A. E., Colditz, G., & Dietz, W. H. (1999). The disease burden associated with overweight and obesity. *Journal of American Medical Association, 282*, 1523–1529.

Strazzullo, P., D'Elia, L., Cairella, G., Garbagnati, F., Cappuccio, F. P., & Scalfi, L. (2010). Excess body weight and incidence of stroke meta-analysis of prospective studies with 2 million participants. *Stroke, 41*, e418–e426.

WHO. (2006). *Global database on body mass index*. Retrieved 31 January, 2011 from http://apps.who.int/bmi/index.jsp

Weighted Sample

Jane Monaco
Department of Biostatistics, The University of North Carolina at Chapel Hill, Chapel Hill, NC, USA

Definition

In a weighted sample, not all sample observations contribute equally to the estimate of a population parameter.

Investigators are often interested in estimating quantities (such as means, counts, or proportions) in a population by using a representative sample selected from that population. Probability samples, defined as samples in which each sampling unit has

a known, nonzero probability of selection based on the sampling design, allow investigators to compute estimates of population parameters. The most straightforward type of probability sampling design, a simple random sample (SRS), is a selection method in which each sample has the same probability of being selected. In an SRS, the probability of selection of each member in the population is the same.

The estimation of the population mean is straightforward for the SRS design. Let n = sample size, N = population size. Also, let $\{Y_1, \ldots, Y_N\}$ be the population values and $\{y_1, \ldots, y_n\}$ be the sample values. We define the overall sampling fraction as $f = \frac{n}{N}$. Then $\bar{Y} = \frac{1}{N}\sum_{i=1}^{N} Y_i$ the population mean, can be estimated by the statistic,

$$\bar{y} = \frac{1}{n}\sum_{i=1}^{n} y_i$$

Under this SRS design, each sample observation, y_i, contributes equally to the estimate, $\bar{y}$, of the population mean.

More complicated sampling designs, such as stratified sampling, may be chosen by investigators for various reasons including potential efficiency and the ability to use different sampling methods for different strata. In stratified sampling, the population is grouped by some characteristic (such as gender, geographic location, or age category), and a sample is selected within each subgroup separately. In this stratified design, the probability of selecting an individual is likely not the same for all individuals, but rather depends on the individual's subgroup (stratum). For example, in a study of illicit drug use among adolescents in a particular city, the population could be stratified into two age groups, middle school and high school. The probability of selection of a particular student will depend on the sample size and population size within that student's age group. Therefore, the sample statistics using a stratified design must be weighted to account for the unequal selection probability of observations.

To compute a weighted sample mean for a stratified sample, first consider the partition of the population into H mutually exclusive strata. Let N_h = the population size in the h^{th} stratum, n_h = the sample size in the h^{th} stratum so that $N = \sum_{h=1}^{H} N_h$ and $n = \sum_{h=1}^{H} n_h$. The stratum specific sampling fraction is $f_h = \frac{n_h}{N_h}$. We can compute each stratum-specific mean, $\bar{y}_h$, as the average of the n_h units in the h^{th} stratum. The weighted sample mean is computed as weighted sum of the stratum specific means: $\bar{y}_w = \frac{1}{N}\sum_{h=1}^{H} N_h \bar{y}_h$. This weighted sample mean can be shown to provide an unbiased estimate of the population mean, $\bar{Y}$.

Cross-References

▶ Probability

References and Readings

Foreman, E. K. (1991). *Survey Sampling Principles*. New York: M. Dekker.

Kish, L. (1965). *Survey Sampling*. New York: Wiley.

Korn, E. L., & Graubard, B. I. (1995). Examples of differing weighted and unweighted estimates from a sample survey. *The American Statistician, 49*(3), 291–295.

Weiss, Stephen M.

Stephen M. Weiss
Department of Psychiatry and Behavioral Sciences, Miller School of Medicine, University of Miami, Miami, FL, USA

Biographical Information

Stephen Weiss was born in northern New Jersey on June 17, 1937. He received his B.A. in Psychology from the University of Maryland (1959) and his Master's (AM) in Psychology

from Temple University (1961). He completed his clinical psychology internship and residency in the Department of Medical Psychology at the University of Oregon Medical School (1962–1963) and received his PhD in Psychology (doctoral minor: Cultural Anthropology) from the University of Arizona (1965). Returning to school after nearly 30 years, he completed his MPH in International Health at Johns Hopkins University School of Hygiene and Public Health in 1993.

Weiss has had several "careers" over the past 45+ years. He began his academic career with appointments including the following: assistant professor, University of Arizona (1964–1967); assistant professor, Johns Hopkins University School of Medicine (1967–1970); associate professor and professor (adjunct), Uniformed Services University of the Health Sciences (1978–1993); and adjunct appointments at the Johns Hopkins University School of Hygiene and Public Health (1976–1998) and the NIH Graduate School (Foundation for Advanced Education in the Sciences) (1979–1994). During "intermissions" in his academic career, Weiss spent 5 years (1969–1974) with the US peace corps (including 2 years in the Ivory Coast in West Africa as director of Training for Africa) as deputy director to the Family Health International AIDS Control and Prevention Project (1991–1993) and nearly 20 years at NIH, directing the behavioral medicine program at the National Heart, Lung and Blood Institute (NHLBI: 1974–1991) and as senior advisor at the National Institute of Mental Health (1993). He joined the faculty of the University of Miami School of Medicine in late 1993 as professor in the Department of Psychiatry and Behavioral Sciences, serving as vice chair for research from 2002 to 2009.

Major Accomplishments

Throughout his career(s), Weiss has focused his professional and scientific energies on one principal issue: to better understand how biological, behavioral, and psychosocial factors *interact* in the etiology, treatment, and prevention of chronic illness.

The first 30 years were focused on cardiovascular health and disease. Beginning with his doctoral dissertation, "Psychological Adjustment Following Open Heart Surgery," he continued this work at Johns Hopkins University School of Medicine and subsequently at NHLBI. His program development mandate at NIH enabled him to advance the "biobehavioral" perspective in behavioral and biomedical science venues. For example, when he arrived at NIH (1974), there was *very* little biobehavioral research related to the prevention and control of chronic disease. Upon becoming acquainted with the NIH system of "peer review," it became obvious that one of the principal reasons for this concerned the *lack* of "peer review" for grant applications attempting to combine biological and behavioral perspectives. He demonstrated the need for a "Behavioral Medicine" study section to the senior officials responsible for grant review by screening over 7,000 grant applications from a single review cycle and identifying over 50 applications requiring both behavioral and biomedical review expertise. Established the following year (Joseph Matarazzo was the first Chair), the Behavioral Medicine study section soon proved its relevance to the NIH mission and was permanently chartered after a 3-year trial period.

During this same period, Weiss, with the active participation of a small group of similarly inclined "biobehaviorists" (e.g., Neal Miller, Joseph Matarazzo, Redford Williams, Neil Schneiderman, Gary Schwartz, Herbert Benson, Margaret Chesney, Thomas Coates, and Judith Rodin to name but a few), convened several conferences and workshops which stimulated the establishment of the fields of behavioral medicine (e.g., the Yale Conference on Behavioral Medicine; the Institute of Medicine/National Academy of Sciences Working Group on Behavioral Medicine) and health psychology (e.g., the Arden House Conference on Education and Training in Health Psychology).

These activities led to the formation and growth of several professional organizations (including the Society of Behavioral Medicine;

the Academy of Behavioral Medicine Research; the Division of Health Psychology of the American Psychological Association; the International Society of Behavioral Medicine). He served as President of each of these organizations and was instrumental in establishing new scientific journals representing these areas during his stewardship of these organizations (e.g., *Journal of Behavioral Medicine; Annals of Behavioral Medicine; Health Psychology; International Journal of Behavioral Medicine*).

Discussions between Weiss and David Hamburg, president of the Institute of Medicine of the National Academy of Sciences, in the late 1970s concerning the importance of the biobehavioral perspective in understanding the multifaceted, multilayered nature of chronic illness stimulated two major "health and behavior" efforts by the Institute of Medicine (IOM) to highlight the need for expanding NIH research efforts in this area. Two seminal volumes were published by the IOM Committee on Health and Behavior over a 20-year period synthesizing important scientific findings and providing direction for future research. Weiss served as consultant to this Committee, as well as being invited to provide testimony to several related IOM committees in the ensuing years.

During the 1980s, Weiss became active in international health scientific exchanges, participating as scientific liaison to the US-USSR Scientific Exchange in Cardiovascular Behavioral Medicine for over 10 years, and in a similar capacity with the US-Israel Scientific Exchanges on Hypertension and Coronary Heart Disease. He returned to the Johns Hopkins University School of Hygiene and Public Health to complete the MPH in International Health, a course of study that had important implications for his professional career direction. During this program, he became acutely aware of the growing catastrophe of the HIV/AIDS pandemic, particularly in the developing world. Based on this experience (and his newly minted MPH degree), he set out to explore what role he might play in challenging this public health disaster in Africa, where he had spent 2 years in the early 1970s working with the US peace corps.

Retiring from the NIH in 1991, Dr. Weiss began to search for a meaningful professional direction in HIV/AIDS. He spent 2 years with Family Health International and NIMH in HIV/AIDS administrative and program advisory capacities. Recognizing the major differences in the epidemics in the developed vs. developing worlds, he realized that new and unique approaches would be required to establish effective prevention strategies for HIV transmission. It took several years, however, before sufficient momentum was achieved in scientific circles to stimulate the NIH to establish an aggressive extramural research program in the search for new barriers to sexual transmission of the virus.

The advent of the female condom and growing interest in chemical barriers (microbicides) stimulated the need for a "critical mass" of research to answer important questions of safety, efficacy, acceptability, and effectiveness of these new sexual barrier products. Designation of HIV/AIDS as a "threat to our national security" by former President Clinton produced a "sea change" in the federal government's willingness to invest in international studies and demonstration projects to contain the epidemic in the developing world.

The critical need to develop *acceptable* barrier products for the varied populations at high risk of HIV infection suggested that biological efficacy must be coupled with product acceptability studies to ultimately demonstrate the effectiveness and impact of such strategies in containing the epidemic. He received NIH funding to address many of these issues in the USA, Africa, and India. In addition to being P.I. on two large-scale US trials of behavioral interventions with HIV+ women, over the last 16 years, he also has been funded to conduct several studies in Zambia with HIV+ and HIV− men, women, and couples and also to serve as PI, Co-PI, Protocol Co-Chair, and site investigator for an additional five studies on sexual risk reduction and medication adherence in Zambia, South Africa, and India.

Since settling in Miami nearly 20 years ago, Weiss has continued to lead an active professional life. Nonetheless, he still has made time for his passion for flying (commercial license, instrument, multiengine, with over 5,600 flight hours), as well as the occasional fishing trip on

his small boat. He has four grown children and nine growing grandchildren, whom he and his wife, Deborah, visit regularly, thanks to his aging, but trusty, Beech Bonanza. He has served as mentor to many junior faculty, residents, and fellows within the medical school through his "Grantsmanship 101" seminar and open invitations to join his research program. He has authored/coauthored over 120 papers, monographs, and scientific reviews, in addition to 10 edited volumes on health and behavior. He currently serves on the editorial boards of several scientific journals and health publications and regularly participates as a reviewer on NIH study sections.

When asked what the single most influential factor in his professional preparation was, Weiss answered with one word: *mentorship*. He attributes the guidance he received from strong mentors like Joe Matarazzo and Neal Miller, his psychologist role models during graduate training, as instrumental in preparing him for the many challenges he has faced in his varied, multifaceted professional journey.

Cross-References

- ▶ Behavioral Medicine
- ▶ HIV Infection

References and Readings

Jones, D. L., Weiss, S. M., Chitalu, N., et al. (2008). Acceptability and use of sexual barrier products and lubricants among HIV-seropositive Zambian men. *AIDS Patient Care and STDs, 22*, 1015–1020.

Kapiga, S., Kelly, C., Weiss, S., Daley, T., Peterson, L., Leburg, C., et al. (2009). Risk factors for incidence of sexually transmitted infections among women in South Africa, Tanzania and Zambia: Results from HPTN 055 study. *Sexually Transmitted Diseases, 36*(4), 199–206.

Lopez, E. J., Jones, D. L., Villar-Loubet, O. M., Arheart, K. L., & Weiss, S. M. (2010). Violence, coping, and consistent medication adherence in HIV-positive couples. *AIDS Education and Prevention, 22*, 61–68.

Schneiderman, N., Weiss, S. M., & Kaufmann, P. G. (Eds.). (1989). *Handbook of research methods in cardiovascular behavioral medicine*. New York: Springer.

Schwartz, G., & Weiss, S. (1977). What is behavioral medicine. *Psychosomatic Medicine, 39*(6), 377–381.

Weiss, S. M. (1966). Psychological adjustment following open heart surgery. *The Journal of Nervous and Mental Disease, 143*(4), 363–368.

Weiss, S. M., Fielding, J., & Baum, A. (Eds.). (1991). *Perspectives in behavioral medicine: Health at work* (p. 219). Hillsdale, NJ: Erlbaum.

Weiss, S. M., Jones, D. L., Lopez, M., Villar-Loubet, O., & Chitalu, N. (2011). The many faces of translational research: A tale of two studies. *Translational Behavioral Medicine, 1*, 327–330.

Weiss, S. M., Tobin, J. N., Antoni, M., Ironson, G., Ishii, M., Vaughn, A., et al. (2011). Enhancing the health of women living with HIV: The SMART/EST women's project. *International Journal of Women's Health, 3*, 63–77.

Welfare

▶ Well-Being: Physical, Psychological, Social

Well-Being

▶ Williams LifeSkills Program

Well-Being: Physical, Psychological, Social

Sarah D. Pressman, Tara Kraft and Stephanie Bowlin
Department of Psychology, University of Kansas, Lawrence, KS, USA

Synonyms

Happiness; Health; Positive emotions; Welfare; Wellness

Definition

Well-being is a multi-faceted construct best described as a state of physical, psychological, and social health.

Description

Well-being is a broad term that encompasses what it means to be functioning as a healthy person across multiple domains. Although words like "well-being (WB)," "health," and "happiness" permeate scientific literature, they are frequently variably defined from one study to the next, with many researchers relying on a single construct, such as "happiness" or "high quality of life" to provide an adequate definition. Rather than constricting WB to one equivocal definition, a review of the literature suggests that it is most comprehensively defined and productively discussed as a combination of the following three components: psychological well-being (PsWB), social well-being (SWB), and physical well-being (PWB).

Psychological Well-Being (PsWB)

Psychological well-being (PsWB) and "happiness" have historically been synonymous and equally indefinite in literature across academic disciplines. Contemporary scientific definitions of PsWB, however, encompass three substantial, coherent domains: emotional experience, cognitive evaluation of life satisfaction, and human flourishing (e.g., Diener, Lucas, & Oishi, 2002; Keyes, Shmotkin, & Ryff, 2002). Although PsWB is frequently referred to as synonymous with Subjective WB (a cognitive evaluation of satisfaction with life), some models of PsWB consider cognitive evaluation a related yet distinct subcomponent of a larger construct of PsWB. Both models will be further discussed below.

Early in the study of emotion, Bradburn (1969) clarified that the absence of negative emotion does not indicate the presence of positive emotion; thus, experiencing both frequent positive emotion and infrequent negative emotion are essential to *feeling* "happy." Instruments to measure positive and negative emotion at both the short-term "state" and long-term "trait" level ask individuals to rate the degree to which they experience a range of emotions, varying in valence (e.g., happy vs. sad) and arousal (e.g., excitement vs. calm) and have led to valuable research on emotion. For example, Fredrickson's (2001) notable "Broaden and Build" theory states that positive emotions increase WB by broadening our cognitive range and building lasting resources that can be utilized in times of need. For example, when experiencing positive emotion, individuals are able to identify a wider range of activities they would like to engage in and are more likely to set and reach goals. Furthermore, the manipulation and natural expression of positive emotion in experimental settings has been shown to improve post-stress cardiovascular recovery, possibly resulting in protective effects against the negative physiological consequences of stress.

As outlined above, PsWB frequently involves and is debatably defined as a cognitive evaluation of satisfaction with life (for a review, see Diener, Suh, Lucas, & Smith, 1999) partnered with positive and negative emotion ratings. This model of PsWB is based on ratings of WB from the respondent's own perspective. For example, although an outside person may judge circumstances to be unfortunate, if a respondent reports high life satisfaction, he or she has high Subjective WB. Furthermore, this model assumes that this report has some amount of stability over time; however, reports can vary with momentary experience and events. Measures connecting these two evaluative components are associated with many positive outcomes, such as hope, optimism, and greater income. Interestingly, twin studies have revealed correlations between genes and PsWB, indicating a possible genetic influence on the "set point" for happiness. Since a variety of factors (e.g., genetics, culture, age, gender, emotional experience) can influence life satisfaction ratings, Diener and colleagues have suggested that "happiness" is best understood through a discussion of the factors that influence Subjective WB.

Ryff (1989) has differentially argued that PsWB includes happiness ("hedonic" WB, or the experience of pleasure) and "eudaimonic" WB. Eudaimonic WB is concerned with human flourishing and encompasses constructs related to engagement with and evaluation of challenging life events, including self-acceptance, purpose and meaning in life, sense of mastery over one's

environment, positive interpersonal relationships, personal growth, and autonomy. Proponents of this approach argue that this combination of factors provides a more complete picture of happiness, or "optimal" WB than emotional or cognitive/evaluative WB components alone and have found this optimal balance to increase with age, education, and the presence of certain personality characteristics (i.e., high extraversion and conscientiousness). While Ryff's work suggests that a comprehensive discussion of PsWB must consider the combination of factors that influence both Subjective WB and human flourishing more broadly, it is somewhat problematic when contemplating research questions attempting to determine the critical components of WB. For example, interpersonal relationships (a component of eudaimonic WB) may have independent pathways (e.g., neuroanatomy, hormonal) to PWB as compared to more emotional constructs within eudaimonia (e.g., autonomy, acceptance) whose paths may have more in common with hedonic measures. It seems important then to consider these subcomponents and their differential mechanisms separately in order to understand how PsWB "gets under the skin" to alter physical health.

Social Well-Being (SWB)

Humans are inherently social creatures who evolved in groups that helped ensure the survival of the species. As a result, there is a deeply engrained need to belong and feel supported. When these social needs are met, individuals experience SWB. There is no firmly agreed upon definition of SWB, but generally it can be considered to be a *multifactorial* construct that includes different components of the social environment that when evaluated together result in an overall positive assessment of one's social life. No single measure completely captures SWB, and as a result, many different types of social characteristics must be considered.

The most basic measures of the social environment are collected via integration measures that assess the extent to which people feel like they belong to their communities. This is typically measured by measures that enumerate important social roles (e.g., spouse, close friend). This work has a rich history dating back to the industrial revolution when Durkheim noted that individuals were more likely to commit suicide upon leaving their families for industrial positions in other cities. Since then, the finding that isolated individuals are at risk for detrimental health outcomes is one of the most pervasive and robust in health psychology. For example, an early study from Alameda County, CA (Berkman & Syme, 1979) revealed that those who lacked ties to others were up to three times more likely to die over a 9-year follow-up. Broader social networks (e.g., online networks of friends) are generally less strongly tied to WB since the presence of distant social contacts has less of an immediate and regular impact on one's life. What seems to be critical is the support and resources that your network provides. Related then is the concept of social support, or the perception that emotional and objective resources (e.g., information, tangible aid) are available if and when negative events occur. Research has indicated that those who report having support in times of need have lower physiological stress responses, better immune function, and overall better health and resilience to health problems, resulting in broad WB benefits.

Other work focuses more closely on the perception and quality of social ties. For example, loneliness is an indicator of low SWB but can be relatively independent from objective network size. The hallmark of loneliness is not being alone but the accompanying distress related to the *perception* of isolation. The relationships approach would state that SWB is present when an individual can have satisfactory close relationships and also shows evidence of secure attachment (i.e., a strong emotional bond between two people). If a romantic partner is missing or an individual is insecurely attached, then high SWB would not be expected. There are a number of other related constructs, such as feelings of love and intimacy, social acceptance, and measures of social conflict and hostility, that are also tied to positive and negative appraisals of WB. When considering these feelings and perceptions or relationships, it is critical to also consider

differences in social needs. For example, individuals high in extraversion will have greater needs for regular social contact than someone low on this personality trait. By definition, extraverts feel "better" when in the presence of social others and may therefore require greater interaction to achieve SWB as compared to their more introverted counterparts.

Finally, the social milieu contributes to SWB. Groups (e.g., clubs, volunteer groups, religious organizations) provide important feelings of belongingness, support, and contribution, which promote SWB. Those individuals who endorse group membership roles as well as those who report engaging in various social activities generally have higher WB. Beyond close groups, the broader environment also impacts SWB. Social capital describes the resources available to individuals through their membership in their community (e.g., civic associations, civic engagement, perceptions of trust and sharing in the community). Higher social capital leads to belonging, increased social participation, and has been tied to crime prevention and better societal health. This type of social engagement is critical for SWB as it likely has a trickle-down effect onto many of the previously discussed measures.

From the above examples, it is clear that SWB arises from a number of places. It is unlikely that any one characteristic uniquely predicts SWB. It is also important to consider the interaction between internal factors (e.g., attachment style, personality) and external characteristics of the social environment. Two individuals may live in the same social environment yet experience their social world in different manners, illustrating the importance of individual perception. Future research should consider which components are most critical to SWB and study more closely the interactions between different measurement types in terms of how they predict WB outcomes.

Physical Well-Being

Physical health researchers generally adhere to a medical model that conceptualizes health as the absence of disease, despite the definition in 1948 by the World Health Organization for health as "a state of complete physical, mental and social WB and *not* merely the absence of disease or infirmity." Physical well-being (PWB) then, is something more than "not sick" and may even reflect individuals realizing their fullest wellness potential, as echoed by the model of positive health by Seligman (2008). Compatible with the PWB model, the wellness continuum model (Sheridan & Radmacher, 1992) anchors one end of their model by disease, disability, and death and the other end by optimal human functioning where a person is maximally healthy, has resources for resisting disease, and is capable of experiencing joy (Sheridan & Radmacher, 2003). If the medical model idea of health were captured in the continuum model, it would end at the neutral/zero point with no room or definition of WB beyond "disease-absent." Despite multiple models suggesting that there is something beyond the absence of illness, health is still primarily measured by the presence or absence of symptoms, illness and disease, physiological markers, and reports of physical health typically topping off at "I feel very healthy." Reports of positive physical functioning beyond this "zero" mark are not typically gathered by health practitioners or researchers.

To determine *objective* wellness, researchers and practitioners frequently rely on assessments of biomarkers to determine disease status and risk. These might include factors like immune function, endocrine hormones, or cardiovascular function, which are important indicators of disease status. While there are general guidelines about what levels indicate "healthy" functioning, these are not frequently tied to current WB. For example, someone may be hypertensive as assessed by blood pressure readings, be a genetic carrier of a serious disease, or even have a blocked artery, but still report feeling healthy and asymptomatic. Furthermore, while biological markers are *possible* correlates of physical health, conclusive statements often cannot be drawn based on physiological measures alone (e.g., lowered immune cell counts). This raises the importance of *subjective* assessments of health when considering PWB.

Self-rated health (SRH), a formalized measure of subjective health, has been used in research

studies and suggests that individuals have insight into their health beyond the capabilities of biological measurement tools. Specifically, the commonly used Short-Form Health Survey (SF-36; Ware & Sherbourne, 1992) asks questions such as, "In general, would you say your health is excellent, very good, fair, or poor?" Several of these questions take into account individual perspectives concerning what "health" means to the individual, and even ask for reports comparative to the past. Additionally, questions such as "Have you felt calm/peaceful/full of pep/happy?" are tapping physical health via constructs that influence reports of PWB but are not direct physical measures. Interestingly, poor SRH is independently associated with increased mortality in diverse socioeconomic groups, age groups, in men and women, over time, even after accounting for objective health status at baseline (e.g., Lekander, Elofsson, Neve, Hansson, & Unden, 2004). Researchers are now investigating whether these nonspecific "sickness" symptoms (weakness, listlessness, decreased motivation) are mediated by stress-induced dysfunctional immune patterns (e.g., high inflammation). Given that positive traits and events are tied to healthy immune function (for further reading, see Pressman & Cohen, 2005), it is plausible that positive reports of SRH are occurring via generalized positive feelings that alter the perception of the body and that these positive feelings are representative of an objectively healthy physiology. At this point, the full mapping of these connections with SRH, biomarkers, emotion, and objective longitudinal health outcomes has not been done.

Problematic in definitions based on biomarkers and disease indicators is determining whether individuals suffering from chronic illness can have high PWB. Based on biological assessment, if an individual is diagnosed with cancer, they have low PWB. However, cancer patients frequently survive their cancer, find benefit in the experience of their illness, build relationships with others, and become optimistic about their future. During treatment, they will clearly experience poor PWB (e.g., side effects of radiation or chemotherapy); however, at some point, they will begin to recover. Self-report measures of PWB may be able to track this based on changes in perceptions of symptoms and health; however, biological measures may be more deficient at determining at what point the person's PWB is on the positive end of the spectrum (i.e., it is unlikely that PWB can be measured by a simple assessment of circulating cancer cells). To truly understand WB in this population and many others, it is clear that there is a need for PWB tools that assess both "harder" biological outcomes partnered with "softer" self-report measures.

Conclusions and Future Directions

Although themes of happiness, physical health, and social involvement have long been studied in philosophy, social psychology theory, and cultural analysis, it has only been in the last 10 years that WB has seen an upswing in interdisciplinary scientific study. Because of this, the scientific community is still working on definitions and operationalizations of critical constructs in the field. Currently, significant overlap exists between WB measures making it difficult to separate and understand these constructs. For example, individuals with serious illness by definition have poor PWB, but it is also likely that PsWB and SWB additionally suffer since illness will block engagement in activities that promote these WB types. Similarly, current measures of depression and loneliness frequently include questions tapping *both* PsWB and SWB revealing the extent to which these constructs overlap. Measures need to be improved to reflect the specific underlying components of WB in order to better understand the unique ways that these different WBs impact one another as well as outside outcomes. Finally, it is also critical to consider that in many cases, there is benefit to the overlap of these constructs. When considering interventions to improve PWB, it is frequently via SWB (e.g., support) and PsWB (e.g., relaxation) that impressive improvements are seen, ranging from physiological alterations to increased life span. There is clearly therapeutic value to considering

ALL types of WB and the interrelations that they have with one another. A thorough understanding of the underlying constructs of WB will enable scientists, health professionals, and individuals of all walks of life to better promote and increase WB at multiple levels, with implications for both personal health and public policy decisions.

Cross-References

- ▶ Emotions: Positive and Negative
- ▶ Happiness and Health
- ▶ SF-36

References and Readings

Berkman, L. F., & Syme, L. (1979). Social networks, host resistance, and mortality: A nine-year follow-up study of Alameda County residents. *American Journal of Epidemiology, 109*, 186–204.

Bradburn, N. M. (1969). *The structure of psychological well-being*. Chicago: Aldine.

Cacioppo, J. T., & Patrick, B. (2008). *Loneliness: Human nature and the need for social connection*. New York: W.W. Norton.

Cohen, S., Underwood, L. G., & Gottlieb, B. H. (2000). *Social support measurement and intervention: A guide for health and social scientists*. New York: Oxford University Press.

Diener, E., Lucas, R. E., & Oishi, S. (2002). Subjective well-being: The science of happiness and life satisfaction. In C. R. Snyder & S. J. Lopez (Eds.), *Handbook of positive psychology* (pp. 63–73). New York: Oxford University Press.

Diener, E., Suh, E. M., Lucas, R. E., & Smith, H. L. (1999). Subjective well-being: Three decades of progress. *Psychological Bulletin, 125*, 276–302.

Fredrickson, B. L. (2001). The role of positive emotions in positive psychology: The broaden-and-build theory of positive emotions. *American Psychologist, 56*, 218–226.

Fredrickson, B. (2002). Positive emotions. In C. R. Snyder & S. J. Lopez (Eds.), *Handbook of positive psychology* (pp. 120–134). New York: Oxford University Press.

Keyes, C. L. M., Shmotkin, D., & Ryff, C. D. (2002). Optimizing well-being: The empirical encounter of two traditions. *Journal of Personality and Social Psychology, 82*, 1007–1022.

Lekander, M., Elofsson, S., Neve, I. M., Hansson, L. O., & Unden, A. L. (2004). Self-rated health is related to levels of circulating cytokines. *Psychosomatic Medicine, 66*, 559–563.

Lyubomirsky, S., King, L., & Diener, E. (2005). The benefits of frequent positive affect: Does happiness lead to success? *Psychological Bulletin, 131*, 803–855.

Pressman, S. D., & Cohen, S. (2005). Does positive affect influence health? *Psychological Bulletin, 131*, 925–971.

Putnam, R. D. (1985). *Bowling alone: The collapse and revival of American community*. New York: Simon & Schuster.

Ryff, C. (1989). Happiness is everything, or is it? Explorations on the meaning of psychological well-being. *Journal of Personality and Social Psychology, 57*, 1069–1081.

Seligman, M. E. P. (2008). Positive health. *Applied Psychology, 57*, 3–18.

Sheridan, C. L., & Radmacher, S. A. (1992). *Health psychology: Challenging the biomedical model*. New York: Wiley.

Sheridan, C. L., & Radmacher, S. A. (2003). Significance of psychosocial factors to health and disease. In L. A. Schein, H. S. Bernard, H. I. Spitz, & P. R. Muskin (Eds.), *Psychosocial treatment for medical conditions: Principles and techniques*. New York: Brunner-Routledge.

Ware, J. E., & Sherbourne, C. D. (1992). The MOS 36-item short-form health survey (SF-36): I. Conceptual framework and item selection. *Medical Care, 30*, 473–483.

Wellbutrin®

- ▶ Bupropion (Wellbutrin, Zyban)

Wellness

- ▶ Well-Being: Physical, Psychological, Social

Whitehall Study

Mark Hamer
Epidemiology and Public Health, Division of Population Health, University College London, London, UK

Definition

The original Whitehall study of British civil servants began in 1967 and showed a steep inverse

association between social class, as assessed by grade of employment, and mortality from a wide range of diseases. The Whitehall II study was later established in 1985 to identify causal pathways linking socioeconomic position to pathophysiological changes and clinical disease. The Whitehall II is an ongoing prospective cohort study of 10,308 British white-collar workers employed in the civil service (Marmot et al., 1991). The study is primarily interested in the pathways explaining social inequalities via characteristics of the work environment, such as job strain, and health-related behaviors including physical activity, smoking, and diet. Data are collected at regular intervals through a combination of self-administered questionnaires and clinical examination. In the 20 years separating the two studies, there has been no diminution in social class difference in morbidity: there remains an inverse association between employment grade and prevalence of common diseases such as heart disease, cancer, and respiratory diseases. Self-perceived health status and symptoms are worse in participants from lower-status jobs. There are also clear employment-grade differences in health-risk behaviors including smoking, diet, and exercise, in economic circumstances, in possible effects of early-life environment as reflected by height, in social circumstances at work (e.g., monotonous work characterized by low control and low satisfaction), and in social supports. These key findings appear to partly explain the social disparities in health (Stringhini et al., 2010). The Whitehall II study currently has available data for nearly 30 years of follow-up, and many of the participants are now in their seventh and eighth decades of life. Thus, the focus of the study is gradually shifting toward aging since many of the participants are now retired from full-time employment.

Social position can be viewed as an indicator of chronic lifetime stress exposure since people of lower social status tend to experience adverse psychosocial factors including greater financial strain, lower control at work, chronic neighborhood and domestic stresses, and more limited social networks. The Whitehall psychobiology studies have been conducted in healthy subsamples ($N = 250–500$) of the main study cohort, in 2001 and 2006, and were specifically undertaken to examine the biological processes that might explain the association between psychosocial stress and coronary heart disease (Hamer, O'Donnell, Lahiri, & Steptoe, 2010; Steptoe et al., 2002). In these studies, healthy men and women from the Whitehall II cohort (stratified by grade of employment as a marker of social status) underwent psychophysiological stress testing involving measurement of cardiovascular and biological responses to laboratory-induced mental stressors. Results suggested that lower social status individuals were characterized not so much by heightened cardiovascular responsiveness, but by impaired poststress recovery, or sustained activation after termination of stressors. Results from the most recent study have suggested that stress responsivity is also associated with subclinical coronary atherosclerosis. Population-based psychophysiological studies with detailed biological measures have contributed to a better understanding about stress and disease pathways.

Cross-References

- ▶ Heart Disease and Cardiovascular Reactivity
- ▶ Psychophysiologic Reactivity
- ▶ Psychophysiologic Recovery

References and Readings

Hamer, M., O'Donnell, K., Lahiri, A., & Steptoe, A. (2010). Salivary cortisol responses to mental stress are associated with coronary artery calcification in healthy men and women. *European Heart Journal, 31*, 424–429.

Marmot, M. G., Davey Smith, G., Stansfeld, S., Patel, C., North, F., Head, J., et al. (1991). Health inequalities among British civil servants: The Whitehall II study. *Lancet, 337*, 1387–1393.

Steptoe, A., Feldman, P. J., Kunz, S., Owen, N., Willemsen, G., & Marmot, M. (2002). Stress responsivity and socioeconomic status: A mechanism for increased cardiovascular disease risk? *European Heart Journal, 23*, 1757–1763.

Stringhini, S., Sabia, S., Shipley, M., Brunner, E., Nabi, H., Kivimaki, M., et al. (2010). Association of socioeconomic position with health behaviors and mortality. *Journal of the American Medical Association, 303*, 1159–1166.

Whole-Genome Association Study (WGAS)

▶ Genome-Wide Association Study (GWAS)

WHR

▶ Waist to Hip Ratio

Williams LifeSkills Program

Virginia P. Williams[1] and Redford B. Williams[2]
[1]Williams LifeSkills, Inc., Durham, NC, USA
[2]Department of Psychiatry and Behavioral Sciences, Division of Behavioral Medicine, Duke University, Durham, NC, USA

Synonyms

Cognitive-behavioral stress management training; Coping skills training; Hardiness; Heart patients; Incarcerated youths; Online training; Pastors; Resilience; Resilience training; Teens; Telephone coaching; Video applications; Well-being

Definition

Williams LifeSkills is a protocol-driven manualized training program that aims to improve stress coping and interpersonal relationship skills. It focuses on acquiring ten skills in two categories: (1) six skills for handling problematic situations: logkeeping, think before you act to decide between action and deflection, deflection, problem solving, assertion, and saying no; and (2) four skills for improving relationships with others and oneself: speaking, listening, empathy, and increasing positives over negatives. The program has been evaluated in several clinical trials, with resulting evidence of its efficacy on psychosocial and biological markers. There are several delivery options. The initial standard was face-to-face workshops of 5–12 participants, with a trained facilitator. The video version consists of a 70-minute video covering the ten skills, with an accompanying workbook; this video/workshop can be incorporated into a workshop or used as a stand-alone intervention, with or without telephone coaching by a trained facilitator. There is also an online version, either freestanding or with submission of exercises. One-on-one coaching is another option. The initial program has been expanded to include a number of specialized versions that maintain focus on the ten skills: for caregivers with a relative with Alzheimer's, teens delivered by high school teachers, a specialized teen version for incarcerated youths, pastors who otherwise are usually supporting others, and heart patients.

Description

The Williams LifeSkills copyrighted program evolved over the last two decades as a means of translating research by Redford Williams and others (Williams, 2008) that has documented the health-damaging effects of psychosocial factors like hostility/anger, depression, social isolation, job stress, and stressful life situations into a behavioral intervention that can reduce levels, and hopefully the health-damaging effects, of these psychosocial risk factors. Williams LifeSkills, Inc., was founded as a commercial entity in 1996 by Redford and Virginia Williams with the goals of developing and refining this behavioral intervention, doing research to test its efficacy and effectiveness in both controlled and observational clinical trials, and marketing behavioral intervention products shown to work in this research. The founders of Williams LifeSkills have written three books for a lay audience that also serve to describe the program as it evolved (Williams & Williams, 1993, 1997, 2006). While the programs are presented as wellness training for increasing participants' effectiveness in the increasingly stress-filled modern world, they also provide a first-level intervention for individuals with mild to moderate anxiety,

depression, and hostility. The intervention has also been used with outpatients with a wide variety of diagnoses, both to help them cope with their illness and to reduce distress over other issues in their lives.

The face-to-face workshop has been used extensively in the United States, Brazil, Singapore, Hungary, and China. Intervention groups are quite varied in these varied settings: some examples are general stress management, patients with oncological problems, medical students, college students; heart patients, corporations, pastors, and health professionals. It also has been incorporated into a general intervention for heart patients in Germany. Facilitators are initially trained for at least five days, spending the first two days as participants and the last three focusing on practicing supervision of the interactive sections of the program. There is an accompanying participant text (about 40 pages long) and a workbook (about 15 pages long), so the text can either be given to a participant or used again. Participants begin by learning to write up log entries, emphasizing the objective facts, their thoughts and feelings, behaviors, and consequences. They then apply their own examples to practice the other skills. The program thus involves iteration without repetition, since new layers of training are being added. Facilitators are trained to encourage participation, within a set of ground rules, and to emphasize improvement through practice, rather than aiming for perfection. The entire program is designed to fit together, with algorithms to address which skills to apply. As a result, the program can be used in almost any situation a participant might face.

The generic LifeSkills program has also been adapted for specific groups. The LifeSkills course for caregivers focuses on a special video created to address problems particular to caring for someone with a disabling memory disorder. A 121-page workbook accompanies the video. Also part of the program is a manual for five sessions of telephone coaching by a LifeSkills facilitator. All of the materials are oriented to the ten LifeSkills. There is an adaptation for teens in which the emphasis is on issues faced by teens. There is an extensive Facilitator's Manual for high school teachers, which is incorporated into the week-long training that prepares them to deliver the training to students in the high school setting. Teens can either report a personal log entry or draw a role-playing card. Separate packets of cards accompany each skill. Teens have a special text and workbook. The teen program has been modified for incarcerated teens. The same general format is used as in the school program, but the role-playing cards are different. Some of the illustrations and workbook examples also are modified. In the United States, prison educators are trained to be the facilitators. In China, the program is being tested, using psychiatrists and psychologists as facilitators. The program for medical personnel has assumed different forms in the United States, Hungary, and China. The program for heart patients also has assumed different forms in the United States and Singapore (also adapted a number of clinical trials have documented the efficacy of the LifeSkills program in its various forms in the United States and Canada). The first study to document benefits of an early version – based on *Anger Kills* (Williams & Williams, 1993) – of the LifeSkills workshop was a small randomized controlled trial (RCT) in males following a heart attack. Results showed that hostility after the end of training as assessed by both structured interview and questionnaire as well as resting diastolic blood pressure were reduced significantly more in patients in the active arm than those randomized to usual care (Gidron, Davidson, & Bata, 1999). Both these improvements were maintained or enhanced at follow-up two months later. Because this study was conducted in Canada, it was possible to do a follow-up assessment of the clinical course over the six months post-training. Compared to usual care, men in the active arm showed significant reductions in both hospital stay and medical care costs over the six months follow-up (Davidson, Gidron, Mostofsky, & Trudeau, 2007). Another RCT evaluating benefits of the LifeSkills video found significantly larger reductions in both trait anxiety and perceived stress among the active arm participants compared to those randomized to wait list control, both two weeks after the end of training and

after six months follow-up (Kirby, Williams, Hocking, Lane, & Williams, 2006). In an RCT evaluating the LifeSkills workshop augmented by the LifeSkills video, hypertensive employees of a large urban medical center who were randomized to LifeSkills training showed significantly larger, clinically significant decreases in systolic blood pressure compared to those receiving usual care according to JNC-7 guidelines (Clemow, Pickering, Davidson, & Liriano, 2009). A controlled (alternating assignment to active vs. wait list arms), clinical trial of the LifeSkills video adapted for use by caregivers of a relative with Alzheimer's disease showed that those in the LifeSkills for Caregivers video arm, who also received weekly telephone coaching calls, showed significantly larger decreases in depressive symptoms, trait anxiety, perceived stress, and systolic and diastolic blood pressure that were maintained or enhanced at 6 months follow-up (Williams et al., 2010). In addition to replicating findings of psychosocial improvement following LifeSkills training, this study extends the demonstration of benefits to include a potential biological mechanism whereby stress damages health – higher blood pressure.

The LifeSkills program has also been adapted for use in Singapore and Hungary. In Singapore, Bishop, Kaur, and Tan (2005) conducted a RCT of the LifeSkills workshop in a sample of 59 male Singaporean (60% Chinese) patients following coronary bypass surgery. In addition to robust improvements with regard to both negative and positive psychosocial risk factors, blood pressure and heart rate at both rest and in response to anger recall were significantly decreased in the men randomized to LifeSkills workshop training, with all improvements maintained or enhanced at three months follow-up. Effect sizes for these psychosocial and cardiovascular improvements in the LifeSkills workshop arm were moderate to large. In Hungary, an observational trial of the LifeSkills workshop by Stauder et al. (2010) found that distressed persons in the Budapest metro area who participated in the workshop showed a pattern of psychosocial improvements that is remarkably similar to those observed by Bishop et al. (2005) on the other side of the world. Effect sizes in Hungary were similarly moderate to large. Dr. Stauder and colleagues at Semmelweis University in Budapest are now conducting RCTs of the LifeSkills workshop in workplace settings in Hungary.

Evidence for the effectiveness of Williams LifeSkills products in real-world settings comes from two observational trials. In one, conducted by Campo et al. (2008), second-year medical students who participated in the WLSR workshop over a two-day period showed significant reductions in hostility and in the use of placating and avoidant responses in difficult patient scenarios following the training. In contrast, a comparison group of second-year students the following year who did not receive WLSR training showed slight increases in both hostility and placating and avoidant responses. In an observational trial of the LifeSkills workshop among employees of one of our corporate clients, at the end of training, there were significant decreases in depressive symptoms and state and trait anxiety and increased social support; except for social support, all these improvements were maintained or enhanced at follow-up six months later (Williams, Brenner, Helms, & Williams, 2009).

The LifeSkills reach continues to expand, since the skills acquired contribute to resiliency and hence can be applied in situations where potential high stress needs to be inoculated against.

Cross-References

- ▶ Alzheimer's Disease
- ▶ Cognitive Behavioral Therapy (CBT)
- ▶ Coping
- ▶ Hardiness and Health
- ▶ Internet-based Interventions
- ▶ Resilience

References and Readings

Bishop, G. D., Kaur, D., & Tan, V. L. M. (2005). Effects of a psychosocial skills training workshop on psychophysiological and psychosocial risk in patients undergoing coronary artery bypass grafting. *American Heart Journal, 150*, 602–609.

Campo, A. E., Williams, V., Williams, R. B., Segundo, M. A., Lydston, D., & Weiss, S. M. (2008). Effects of LifeSkills training on medical students' performance in dealing with complex clinical cases. *Academic Psychiatry, 32*(3), 188–193.

Clemow, L. P., Pickering, T. G., Davidson, K. W., & Liriano, C. (2009). Multi-component stress management for hypertensives: For whom does it work best? *Psychosomatic Medicine, 71*, A–127.

Davidson, K. W., Gidron, Y., Mostofsky, E., & Trudeau, K. J. (2007). Hospitalization cost offset of a hostility intervention for coronary heart disease patients. *Journal of Consulting and Clinical Psychology, 75*, 657–662.

Gidron, Y., Davidson, K. W., & Bata, I. (1999). The short-term effects of a hostility-reduction intervention on male coronary heart disease patients. *Health Psychology, 18*, 416–420.

Kirby, E. D., Williams, V. P., Hocking, M. C., Lane, J. D., & Williams, R. B. (2006). Psychosocial benefits of three formats of a standardized behavioral stress management program. *Psychosomatic Medicine, 68*, 816–823.

Stauder, A., Konkolÿ Thege, B., Kovács, M. E., Balog, P., Williams, V. P., & Williams, R. B. (2010). Worldwide stress: Different problems, similar solutions? Cultural adaptation and evaluation of a standardized stress management program in Hungary. *International Journal of Behavioral Medicine, 17*, 25–32.

Williams, R. B. (2008). Psychosocial and biobehavioral factors and their interplay in coronary heart disease. *Annual Review of Clinical Psychology, 4*, 349–365.

Williams, V. P., Bishop-Fitzpatrick, L., Lane, J. D., Gwyther, L. P., Ballard, E. L., Vendittelli, A. P., et al. (2010). Video-based coping skills to reduce health risk and improve psychological and physical well-being in Alzheimer's Disease family caregivers. *Psychosomatic Medicine, 72*(9), 897–904.

Williams, V. P., Brenner, S. L., Helms, M. J., & Williams, R. B. (2009). Coping skills training to reduce psychosocial risk factors for medical disorders: A field trial evaluating effectiveness in multiple worksites. *Journal of Occupational Health, 51*, 437–442.

Williams, R. B., & William, V. P. (1993). *Anger kills: Seventeen strategies for controlling the hostility that can harm your health*. New York: Times Books. Trade paperback edition published by Harper-Collins, Spring, 1994.

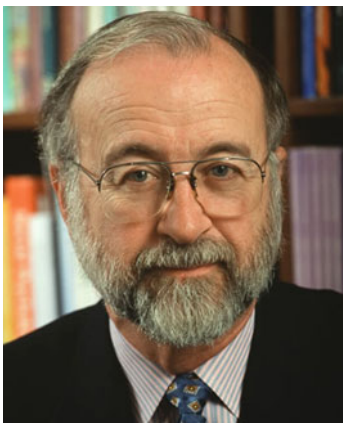
Williams, V. P., & Williams, R. B. (1997). *LifeSkills: 8 Simple ways to build stronger relationships, communicate more clearly, improve your health, and even the health of those around you*. New York: Times Books/Random House. Paperback, April, 1999.

Williams, R. B., & Williams, V. P. (2006). *In control*. New York: Rodale Books.

Williams Redford B. Jr.

Redford B. Williams
Department of Psychiatry and Behavioral Sciences, Division of Behavioral Medicine, Duke University, Durham, NC, USA

Biographical Information



Redford B. Williams, Jr., was born in Raleigh, NC, on December 14, 1940. Following graduation from Northampton High School in Eastville, Virginia, in June 1959, he entered Harvard College, receiving his A.B. degree in 1963. He married Virginia Carter Parrott in 1963, and they have two children and three grandchildren. Williams received his M.D. degree from Yale in 1967, where he completed his internship and residency in internal medicine in 1970. Following 2 years as a clinical associate at NIMH in Bethesda, MD, he was appointed assistant professor in the Departments of Psychiatry and Medicine at Duke University Medical Center, Durham, NC, in 1972, advancing to the rank of professor in 1978. Since 1985, he has been director of the Behavioral Medicine Research Center at Duke. He was named Head, Division of Behavioral Medicine in the Department of Psychiatry and Behavioral Sciences at Duke in 1990. In 1991, he was appointed professor of Psychology at Duke and adjunct professor of Epidemiology in

the School of Public Health at the University of North Carolina, Chapel Hill. He has served on several Medical Center committees over the years, including the Awards Committee (Chair), the Conflict of Interest Committee, and the Department of Psychiatry and Behavioral Sciences Appointments, Promotion, and Tenure Committee. Williams has been continuously funded by the NIH since receiving a Research Scientist Development Award from NIMH in 1974; he has been principal investigator on a program project grant from the NHLBI that began in 1986, continuing till April, 2015. In 1996, he collaborated with Virginia Parrott Williams to found Williams LifeSkills, Inc., a company with the mission of developing, testing, and marketing behavioral intervention products aimed at reducing the health-damaging effects of psychosocial risk factors such as hostility, depression, and social isolation. Williams has served as the president of the leading scientific societies in the field of psychosomatic/behavioral medicine, including the Society of Behavioral Medicine (1983–1984), the American Psychosomatic Society (1992–1993), the Academy of Behavioral Medicine Research (1995–1996), and the International Society of Behavioral Medicine (2006–2008). He was the inaugural recipient of the Society of Behavioral Medicine's Distinguished Scientist Award (1992). Williams is grateful for the mentorship he has received from George Goethals, Stan King, and John Spiegel at Harvard; Pat McKegney and Mickey Willard at Yale; Irv Kopin and Lyman Wynne at NIMH; Bud Busse and Saul Schanberg at Duke; and numerous colleagues and students at each step along the way over the years.

Major Accomplishments

Beginning with publication of two papers in 1965–1967 reporting effects of interview variables on blood pressure reactivity that were found in his research as a medical student at Yale, Williams has been continuously engaged in research aimed at identifying psychosocial factors that increase risk of cardiovascular disease and the biobehavioral mechanisms that mediate that risk. In the mid-1970s, following training in the assessment of type A behavior by Ray Rosenman and Meyer Friedman, Williams began a still ongoing project that initially pinpointed hostility as the toxic core of the type A behavior pattern. Working over the ensuing years with his Duke psychiatry and cardiology colleagues, Williams has made important contributions to our understanding of the role played by psychosocial factors such as hostility, depression, social isolation, and low socioeconomic status in the pathogenesis and course of cardiovascular disease, as well as the role of mediators such as increased sympathetic and HPA axis function, decreased parasympathetic function, and unhealthy lifestyle behaviors.

In the mid-1980s, Williams began the process of translating research findings on the health-damaging effects of psychosocial risk factors into behavioral interventions to prevent and/or ameliorate those effects. He has collaborated with his spouse of 48 years, Virginia Parrott Williams, to develop the Williams LifeSkills coping skills training program and evaluate its effects on psychosocial risk factors and biobehavioral mechanisms. Published clinical trials have documented a broad range of benefits of this program: decreased levels of hostility, anger, depression, anxiety, and perceived stress; decreased levels of blood pressure and heart rate at rest and during lab challenge; and increased levels of satisfaction with social support and satisfaction with life. These benefits persist over 6 months after the end of training and have been observed in distressed groups like patients following coronary artery bypass surgery and caregivers of a relative with Alzheimer's disease and in diverse cultural settings ranging from the USA to Singapore, to China, and to Hungary.

Beginning in the late 1990s, Williams has extended the focus of his research to include a search for genetic variants that increase one's vulnerability to the effects of environmental stressors on psychosocial risk factors and biobehavioral mechanisms. Based on his hypothesis that dysregulated central nervous system, serotonin plays an important role in the clustering of

psychosocial and biobehavioral risk factors in certain individuals; this work focused initially on genes that regulate serotonin functions. A typical finding emerging in this ongoing work is an association between the more functional long allele of the serotonin promoter 5HTTLPR polymorphism and increased cardiovascular reactivity to acute mental stress, suggesting that the long allele will be associated with increased risk of coronary heart disease – a prediction that has been confirmed in three independent case-control studies in Asia and Europe. Williams and his colleagues at Duke and elsewhere are currently working to test the hypothesis that genetic variants found associated – whether directly or via gene x environment interactions – with predisease endophenotypes such as increased neuroendocrine, sympathetic nervous system, and cardiovascular reactivity to stress, and increased expression of the metabolic syndrome are also associated with incidence of cardiovascular disease and/or type 2 diabetes in large cohorts of both healthy persons and persons with coronary heart disease.

Another important contribution over the years has been Williams' mentoring of students and postdoctoral fellows who have gone on to make their own important contributions to the field of behavioral medicine: Norman Anderson, Gary Bennett, James Blumenthal, Beverly Brummett, Shin Fukudo, Jim Lane, Linda Luecken, Marcellus Merritt, Len Poon, and Ed Suarez.

Cross-References

► Williams LifeSkills Program

References and Readings

Anderson, N. B., Muranaka, M., Williams, R. B., Lane, J. D., & Houseworth, S. J. (1988). Racial differences in blood pressure and forearm vascular responses to the cold face stimulus. *Psychosomatic Medicine, 50*, 57–63.

Barefoot, J. C., Dahlstrom, W. G., & Williams, R. B. (1983). Hostility, CHD incidence and total mortality: A 25-year follow-up study of 255 physicians. *Psychosomatic Medicine, 45*, 59–63.

Brummett, B. H., Babyak, M. A., Siegler, I. C., Shanahan, M., Harris, K. M., Elder, G. H., et al. (2011). Systolic blood pressure, socioeconomic status, and biobehavioral risk factors in a nationally representative US young adult sample. *Hypertension, 58*, 161–166.

Brummett, B. H., Boyle, S. H., Siegler, I. C., Kuhn, C., Ashley-Koch, A., Jonassaint, C. R., et al. (2008). Effects of environmental stress and gender on associations among symptoms of depression and the serotonin transporter gene linked polymorphic region (5-HTTLPR). *Behavior Genetics, 38*, 34–43.

Brummett, B. H., Muller, C. L., Collins, A. L., Boyle, S. H., Kuhn, C. M., Siegler, I. C., et al. (2008). 5-HTTLPR and gender moderate changes in negative affect responses to tryptophan infusion. *Behavior Genetics, 38*, 476–483.

Fukudo, S., Lane, J. D., Anderson, N. B., Kuhn, C. M., Schanberg, S. M., McCown, N., et al. (1992). Accentuated vagal antagonism of beta-adrenergic effects on ventricular repolarization: Differential responses between Type A and Type B men. *Circulation, 85*, 2045–2053.

Kirby, E. D., Williams, V. P., Hocking, M. C., Lane, J. D., & Williams, R. B. (2006). Psychosocial benefits of three formats of a standardized behavioral stress management program. *Psychosomatic Medicine, 68*, 816–823.

Kring, S. I., Brummett, B. H., Barefoot, J., Garrett, M. E., Ashley-Koch, A. E., Boyle, S. H., et al. (2010). Impact of psychological stress on the associations between apolipoprotein E variants and metabolic traits: Findings in an American sample of caregivers and controls. *Psychosomatic Medicine, 72*(5), 427–433.

McKegney, F. P., & Williams, R. B. (1967). Psychological aspects of hypertension: II. The differential influence of interview variables on blood pressure. *The American Journal of Psychiatry, 123*, 1539–1543.

Siegler, I. C., Peterson, B. L., Barefoot, J. C., & Williams, R. B., Jr. (1992). Hostility during late adolescence predicts coronary risk factors at mid-life. *American Journal of Epidemiology, 136*, 146–154.

Stauder, A., Konkolÿ Thege, B., Kovács, M. E., Balog, P., Williams, V. P., & Williams, R. B. (2010). Worldwide stress: Different problems, similar solutions? Cultural adaptation and evaluation of a standardized stress management program in Hungary. *International Journal of Behavioral Medicine, 17*, 25–32.

Williams, R. B. (1994). Neurobiology, cellular and molecular biology, and psychosomatic medicine. *Psychosomatic Medicine, 56*, 308–315.

Williams, R. B., Barefoot, J. C., Blumenthal, J. A., Helms, M. J., Luecken, L., Pieper, C. F., et al. (1997). Psychosocial correlates of job strain in a sample of working women. *Archives of General Psychiatry, 54*, 543–548.

Williams, R. B., Barefoot, J. C., Califf, R. M., Haney, T. L., Saunders, W. B., Pryor, D. B., et al. (1992).

Prognostic importance of social and economic resources among medically treated patients with angiographically documented coronary artery disease. *Journal of the American Medical Association, 267*, 520–524.

Williams, V. P., Bishop-Fitzpatrick, L., Lane, J. D., Gwyther, L. P., Ballard, E. L., Vendittelli, A. P., et al. (2010). Video-based coping skills to reduce health risk and improve psychological and physical well-being in Alzheimer's disease family caregivers. *Psychosomatic Medicine, 72*(9), 897–904.

Williams, R. B., Bittker, T. E., Buchsbaum, M. S., & Wynne, L. C. (1975). Cardiovascular and neurophysiologic correlates of sensory intake and rejection: I. Effect of cognitive tasks. *Psychophysiology, 12*, 427–433.

Williams, R. B., & Chesney, M. A. (1993). Psychosocial factors and prognosis in established coronary artery disease. The need for research on interventions. *Journal of the American Medical Association, 270*, 1860–1861.

Williams, R. B., & Eichelman, R. (1971). Social setting: Influence upon physiological responses to electric shock in the rat. *Science, 174*, 613–614.

Williams, R. B., Haney, T. L., Blumenthal, J. A., & Kong, Y. (1980). Type A behavior, hostility, and coronary atherosclerosis. *Psychosomatic Medicine, 42*, 539–549.

Williams, R. B., Kimball, C. P., & Willard, H. N. (1973). The influence of interpersonal interaction upon diastolic blood pressure. *Psychosomatic Medicine, 34*, 194–198.

Williams, R. B., Lane, J. D., Kuhn, C. M., Melosh, W., White, A. D., & Schanberg, S. M. (1982). Type A behavior and elevated physiological and neuroendocrine responses to cognitive tasks. *Science, 218*, 483–485.

Williams, R. B., Marchuk, D. A., Gadde, K. M., Barefoot, J. C., Grichnik, K., Helms, M. J., et al. (2003). Serotonin-related gene polymorphisms and central nervous system serotonin function. *Neuropsychopharmacology, 28*, 533–541.

Williams, R. B., Marchuk, D. A., Siegler, I. C., Barefoot, J. C., Helms, M. J., Brummett, B. H., et al. (2008). Childhood socioeconomic status and serotonin transporter gene polymorphism enhance cardiovascular reactivity to mental stress. *Psychosomatic Medicine, 70*, 32–39.

Williams, R. B., & McKegney, F. P. (1965). Psychological aspects of hypertension: I. The influence of experimental interview variables on blood pressure. *The Yale Journal of Biology and Medicine, 38*, 265–273.

Williams, R. B., Surwit, R. S., Siegler, I. C., Ashley-Koch, A. E., Collins, A. L., Helms, M. J., et al. (2010). Central nervous system serotonin and clustering of hostility, psychosocial, metabolic and cardiovascular endophenotypes in men. *Psychosomatic Medicine, 72*, 601–607.

Williams, R.B. & William, V.P. (1993). *Anger kills: Seventeen strategies for controlling the hostility that can harm your health*. New York: Times Books. Trade paperback edition published by Harper-Collins, Spring, 1994

Williams, V. P., & Williams, R. B. (1998). *LIFESKILLS: 8 simple ways to build stronger relationships, communicate more clearly, improve your health, and even the health of those around you*. New York: Times Books/Random House, Spring. Paperback, April, 1999.

Willingness-to-Pay (WTP)

▶ Benefit Evaluation in Health Economic Studies

Women's Cardiovascular Health

▶ Women's Health

Women's Health

Michael O'Hara
Department of Psychology, University of Iowa, Iowa City, IA, USA

Synonyms

Women's cardiovascular health; Women's mental health; Women's reproductive health; Women's well-being

Definition

A recent Institute of Medicine committee approached women's health as a concept that has expanded beyond the reproductive system and includes "... health conditions that are specific to women, are more common or more serious in women, have distinct causes or manifestations in women, have different outcomes or treatments in women, or have high morbidity or mortality in women (pp. 2–3)"

(Institute of Medicine [IOM], 2010). Examples of these conditions include breast cancer, cardiovascular disease, cervical cancer, depression, HIV/AIDS, and osteoporosis, among others.

Description

History

Historically, women's health referred to women's reproductive health – the menstrual cycle, ▶ pregnancy, labor, delivery, and ▶ menopause. It is only in the past three decades that women's health has been understood to include the full complement of women's health concerns. For example, it was common for women of reproductive potential to be excluded from clinical trials because of the possibility of unknown teratogenic effects of the medication under study. In fact, in 1977, the FDA restricted women of childbearing potential from being in clinical trials. This policy was only reversed in 1993. Moreover, it was assumed that results of clinical trials with men could be extrapolated to women. This was an untested assumption that is now known to be untrue. Additionally, investigators were concerned that the hormonal fluctuations associated with the menstrual cycle would influence study findings in unknown ways. For all of these reasons, the great bulk of health research until recently has either excluded women or included them in small numbers or has not analyzed the data for sex differences. These deficiencies are slowly being remedied.

There have been a number of important milestones in women's health. For example, the publication of *Our Bodies, Ourselves* in 1970 by the Boston Women's Health Book Collective was a landmark event in women advocating for women's health. This organization and its work continue today. It argues that it has brought several key ideas into the public discourse on women's health (Our Bodies Ourselves [OBOS], 2011).

- "That women, as informed health consumers, are catalysts for social change
- That women can become their own health experts, particularly through discussing issues of health and sexuality with each other
- That health consumers have a right to know about controversies surrounding medical practices and about where consensus among medical experts may be forming
- That women comprise the largest segment of health workers, health consumers, and health decision-makers for their families and communities, but are underrepresented in positions of influence and policy making
- That a pathology/disease approach to normal life events (birthing, menopause, aging, death) is not an effective way in which to consider health or structure a health system"

Change in traditional concepts of women's health did not occur quickly, and it was not until 1980s that clear recognition of the importance of a women's health perspective came into focus. In the forward to a seminal 1985 Public Health Service Task Force report on women's health, Edward Brandt, assistant secretary for health said "This report does not focus strictly on the diseases and problems unique to women in the traditional sense, that is, reproductive problems, but rather is devoted to assessing the problems of women's health, in the context of the lives women in America lead today (p. 74)." Going on the report states: "Good health requires a safe and healthful physical and social environment, an adequate income, safe housing, good nutrition, access to preventive and treatment services appropriate to the persons to be served, and a population that is educated and motivated to maintain healthful behaviors" (p. 77).

In 1990, the Congressional Caucus for Women's Issues (CCWI) pushed through legislation entitled the Women's Health Equity Act. This act created the Office for Women's Health Research at the NIH. It also created a gynecology research program and a Center for Women's Health Research at the NIH. Finally, it required the NIH director to report on progress on women's health and research and that a database be developed of research on women's health and to require the inclusion of women and minorities in NIH trials. The authors of the bill pointed to the then recent Harvard study of 22,000 men (and no women), which demonstrated that daily aspirin could prevent heart attacks as a case of blatant

discrimination against women. The CCWI continued to be very active in subsequent congressional sessions in pushing through many additional laws aimed at improving women's health and including women in biomedical research.

Subsequent to the to the Women's Health Equity Act, there has been a great deal of legislation that bears on improving women's health through improved services and research. Most recently, the Patient Protection and Affordable Care Act (Public Law 111–148) formally codified the Office of Women's Health Research at the NIH, and it formally established an Office of Women's Health in the director's office of the Agency for Health Care Research and Quality (AHRQ), Centers for Disease Control and Prevention (CDC), Food and Drug Administration (FDA), Health Resources and Services Administration (HRSA), and Substance Abuse and Mental Health Services Administration (SAMSA). Additionally, it also established a Department of Health and Human Services Coordinating Committee on Women's Health and a National Women's Health Information Center.

Advocacy Matters: The Example of Breast Cancer

Until the 1970s, there was very little public discussion of breast cancer. Often, it was a cause of shame in families. There were several important landmarks in the 1970s that set the stage for the current strong advocacy around breast cancer prevention and treatment. Already mentioned, *Our Body, Ourselves*, helped set the stage for women taking control of their health. In 1974, the first lady, Betty Ford, was diagnosed with breast cancer. She spoke about her experiences in public, and this encouraged others to do the same. At about the time, Rose Kushner, a journalist, was diagnosed with breast cancer. She objected to the then common practice in which a tumor biopsy and radical mastectomy were performed in a single surgical operation while the patient was under anesthesia. Instead she insisted on a two-step procedure in which biopsies and surgery were undertaken as separate procedures, allowing a woman time to consider her options. This was very unpopular with the medical establishment. However, through persistent advocacy, her efforts resulted in a 1977 NIH panel (of which she was a member), concluding that the two-step procedure that she recommended should be adopted, and that in primary breast cancer, the standard treatment should be a total simple mastectomy rather than the more common radical mastectomy.

The first major advocacy organization was the Susan G. Komen Breast Cancer Foundation in 1982. The work of the Foundation has been described as priming the market (step 1), which means drawing attention to breast cancer through personal stories and by communicating the impact of breast cancer in stark and concrete terms (Braun, 2003). Important in this effort was portraying that there was hope in the form of prevention and effective treatment. In a related manner, efforts were made to engage consumers (step 2) by sponsoring activities, such as the "Race for the Cure" established in 1983. These activities brought positive media attention and raised money for further advocacy. Political action (step 3) in 1980s and 1990s led to a number of legislative, regulatory, and funding accomplishments, for example, the army took on the task of breast cancer research; the Breast and Cervical Cancer Early Detection Program was established; and federal funding for breast cancer research increased fivefold in the 1990s. Currently, over $700,000,000 dollars in federal funds is allocated to breast cancer research each year. The final step in breast cancer advocacy is that the movement has gone mainstream (step 4). There is little question that breast cancer is firmly entrenched in the American consciousness. The ubiquitous pink ribbons and strong support for breast cancer research in the congress and the strong corporate support and the continuing proliferation of strong national and local advocacy groups speak to the effectiveness of breast cancer advocacy by women over the past 30 years.

Progress in Women's Health Research

In 2010, the Institute of Medicine released a report entitled *Women's Health Research: Progress,*

Pitfalls, and Priorities. The committee considered some select diseases of importance to women and characterized the nature of progress that has been made in prevention and treatment. Breast cancer, cervical cancer, and cardiovascular disease are conditions on which research has contributed to major progress. For example, the age-adjusted mortality from invasive breast cancer dropped from 33.1 per 100,000 women in 1990 to 22.8 per 100,000 in 2007 due to increased screening and improved adjuvant therapies. Despite these gains, the incidence of breast cancer is higher today than in 1975. However, there has been a significant decrease in estrogen-positive breast cancers since 2002, probably because of the large decline in hormone replacement therapy for menopause.

HIV/AIDS is an example of a condition in which scientific research has led to some progress in women's health despite the early heavy research emphasis on men. The proportion of AIDS cases represented by women has been increasing steadily since 1985, and women now represent about 27% of cases. Like the case for men, prevention entails decreasing high-risk sexual behavior. However, women suffer from their own high-risk sexual behavior as well as that of their partner, which makes prevention more challenging. Antiretroviral therapy has made a tremendous impact for women (as well as men). However, women seem to be less tolerant of the side effects of these therapies, and treatment toxicity remains a significant problem for women suffering from HIV/AIDs. Treatment and side effects for pregnant women and the offspring can also be problematic.

Autoimmune disorders as a group are common (third only to ▶ cardiovascular disease and cancer). Examples of autoimmune disorders include ▶ diabetes, thyroid disease, Graves' disease, lupus, rheumatoid arthritis, and multiple sclerosis. They affect 5–8% of population, but over 78% of those affected are women. Advances in knowledge have occurred over the past 20 years, but these advances have not led to treatment advances beyond the treatment of symptoms. These disorders do not have the mortality associated with cardiovascular disease and cancer, but they are associated with significant morbidity and reduced quality of life.

Women's Mental Health

Mental health problems represent a significant burden for women. For example, globally, among women aged 15–44 years, unipolar depressive disorders are the second leading cause of disability (measured in disability-adjusted life years) after HIV/AIDS. Moreover, schizophrenia, bipolar affective disorder, suicide attempts, and ▶ panic disorder are in the top 15 conditions causing disability in young women. Depressive and anxiety disorders are much more common in women than men – for depression, the ratio is approximately 2 to 1 around the world. These disorders are found in all societies, not just the societies in the developed nations. In addition to the suffering experienced by women due to mental health problems, there is a large and increasing literature linking maternal mental health problems to behavioral and mental health problems in the offspring. Effective pharmacological and psychotherapeutic interventions exist for depressive and anxiety disorders. Nevertheless, women often go untreated in part because of lack of access but also because the women's depressive and anxiety disorders are often undetected in primary care settings such as prenatal and postnatal health care and routine gynecological care. Major initiatives are underway to develop more effective methods of detecting ▶ depression and anxiety among women and primary care and to provide more efficacious and accessible treatment for these women.

Cross-References

- ▶ Breast Cancer
- ▶ Cardiovascular Disease
- ▶ Clinical Trial
- ▶ Depression
- ▶ Diabetes
- ▶ Menopause
- ▶ Panic Disorder
- ▶ Pregnancy

References and Readings

Boston Women's Health Book Collective. (1973). *Our bodies, ourselves: A book by and for women*. New York: Simon and Schuster.

Braun, S. (2003). The history of breast cancer advocacy. *The Breast Journal, 9*, S101–S103.

Institute of Medicine (IOM). (2010). *Women's health research: Progress, pitfalls, and promise*. Washington, DC: The National Academies Press.

National Cancer Institute. *Cancer research funding*. Accessed April 23, 2011, from http://www.cancer.gov/cancertopics/factsheet/NCI/research-funding

Our Bodies Ourselves. Accessed April 17, 2011, from http://www.ourbodiesourselves.org/about/default.asp

US Department of Health and Human Services (HHS). (1985). Women's health. Report of the public health service task force on women's health issues. *Public Health Reports, 100*, 73–106.

World Health Organization (WHO). (1946). *Preamble to the Constitution of the World Health Organization as Adopted by the International Health Conference*, New York, June, 19–22, 1946; Signed on 22 July 1946 by the Representatives of 61 States (Official Records of the World Health Organization, No. 2, p. 100) and Entered into Force on April 7, 1948. Accessed April 17, 2011, from http://www.who.int/about/definition/en/print.html

World Health Organization (WHO). (2001). *The world health report: 2001: Mental health: New understanding, new hope*. Geneva: Author.

Women's Health Initiative (WHI)

Jonathan Newman
Columbia University, New York, NY, USA

Definition

The Women's Health Initiative (WHI) is a long-term, domestic health study that has focused on preventing heart disease, colorectal cancer, and osteoporotic fractures in post-menopausal women, all of which are major causes of death and disability in older women.

Description

The WHI is a multimillion-dollar study sponsored by the National Institutes of Health (NIH) and the National Heart, Lung, and Blood Institute, involving 161,308 women aged 50–79. The study has two major parts: a set of randomized clinical trials and an observational component. The WHI clinical trials (CT) include three overlapping components: the hormone therapy (HT) trials, dietary modification (DM) trials, and the calcium and vitamin D (CaD) trial. Each trial is a randomized comparison among post-menopausal women, age 50–79 at enrollment, who could be randomized into one, two, or three of the CT trials. Women who declined or were unable to participate were invited to participate in the observational study (OS), including 93,976 participants.

The HT trial is one of the most noteworthy within the WHI, and it consists of two separate trials, one for women who were post-hysterectomy at the time randomization (estrogen-alone trial) and one for women with a uterus (estrogen plus progestin trial). Both the hormone therapy trials and the CaD trial had a 1:1 double-blinded, placebo-controlled randomization, while the DM trial randomized 40% of women to a sustained low-fat diet and 60% to self-selected dietary behavior.

After 5.6 years of follow-up, the estrogen plus progestin (E+P) trial was stopped in 2002 because of increased risks of cardiovascular disease and breast cancer among women randomized to active treatment. While at a lower risk for colon cancer and fracture, women on estrogen and progestin had a higher risk of heart disease, stroke, blood clots, and certain types of breast cancer. After stopping the E+P trial, the WHI continued to follow up the women involved. Three years after stopping hormone therapy with estrogen and progestin, the women no longer had an increased risk of cardiovascular disease nor a lower risk of colon cancer compared to women who were randomized to placebo. Further follow-up revealed a nearly twofold increase in the incidence of breast cancer among women who had taken this hormone combination for 5 or more years. However, once estrogen and progestin hormone therapy was stopped, the risk of breast cancer decreased significantly,

independent of rates of mammography in the population as a whole during this time.

The estrogen-alone trial was stopped in 2004 after 6.8 years of follow-up. It was found that estrogen alone did not affect the risk of heart disease; however, the risk of stroke was increased by 12 cases per 10,000 women taking estrogen alone and tended to increase the incidence of deep vein thrombosis as well. Similar to the results of the E+P HT trial, estrogen alone decreased the occurrence of both hip and total bone fractures. Colorectal cancer rates were unaffected, and there was a small suggestion of a reduction in breast cancer incidence. In a follow-up study, there was an indication that estrogen alone might decrease the amount of coronary artery calcium, a useful subclinical marker of atherosclerotic risk. Taking into account all the diseases studied during the estrogen-alone trial, the WHI investigators concluded that estrogen should not be used to prevent heart disease specifically, nor chronic diseases overall in postmenopausal women without a uterus.

The DM trials, while largely negative in terms of the effects of fat and caloric restriction on either breast or colorectal cancer in postmenopausal women, did suggest that reductions in saturated and trans fat, along with higher intakes of fruit and vegetables, may have a protective effect on the incidence of cardiovascular disease in these women. The separate CaD trials, while failing to show a protective effect of calcium and vitamin D on the occurrence of hip fracture in all postmenopausal women, did find a protective effect on the occurrence of hip fractures in women over the age of 60. The WHI trialists have recognized that the dose of calcium and vitamin D may have been insufficient to prevent hip fractures and may have contributed to the largely negative findings of this trial. Further, in the CaD trial, there were no beneficial effects of calcium and vitamin D on the occurrence of colon cancer. However, there were small (1%) but significant protective effects on maintenance of bone mineral density seen in women taking calcium plus vitamin D supplements.

References and Readings

Beresford, S., Johnson, K., Ritenbaugh, C., Lasser, N., Snetselaar, L., Black, H., Anderson, G., Assaf, A., Bassford, T., Bowen, D., Brunner, R., Brzyski, R., Caan, B., Chlebowski, R., et al. (2006). Low-fat dietary pattern and risk of colorectal cancer: The women's health initiative randomized controlled dietary modification trial. *Journal of the American Medical Association, 295*, 643–654.

Jackson, R., LaCroix, A., Gass, M., Wallace, R., Robbins, J., Lewis, C., Bassford, T., Beresford, S., Black, H., Blanchette, P., Bonds, D., Brunner, R., Bryzski, R., Caan, B., et al. (2006). Calcium plus vitamin D supplementation and the risk of fractures. *NEJM, 354*(7), 669–683.

The Women's Health Initiative Steering Committee. (2004). Effects of conjugated equine estrogen in postmenopausal women with hysterectomy. The women's health initiative randomized controlled trial. *Journal of the American Medical Association, 291*, 1701–1712.

The Writing Group for the WHI Investigators. (2002). Risks and benefits of estrogen plus progestin in healthy post-menopausal women: Principal results of the Women's health initiative randomized controlled trial. *Journal of the American Medical Association, 288*(3), 321–333.

Women's Mental Health

▶ Women's Health

Women's Reproductive Health

▶ Women's Health

Women's Well-Being

▶ Women's Health

Work

▶ Job Diagnostic Survey

Work Autonomy

► Job Satisfaction/Dissatisfaction

Work Engagement

► Job Demands

Work Fulfillment/Non-fulfillment

► Job Satisfaction/Dissatisfaction

Work Performance Feedback

► Job Satisfaction/Dissatisfaction

Work Satisfaction/Dissatisfaction

► Job Satisfaction/Dissatisfaction

Work Tasks

► Workload

Work, Lipids, and Fibrinogen (WOLF) Study

William Whang
Division of Cardiology, Columbia University Medical Center, New York, NY, USA

Definition

The WOLF study is a prospective cohort study that was started to analyze the role of adverse occupational conditions in cardiovascular risk and disease development in employed Swedish men and women. Occupational health units carried out baseline screening of employees from approximately 60 companies from 1992 to 1998, including a clinical exam and blood samples. The initial study of 10,382 subjects from WOLF found no associations between job strain and serum total cholesterol and plasma fibrinogen (Alfredsson et al., 2002). Additional studies have examined relationships between cardiac risk and leisure time (Fransson, Alfredsson, de Faire, Knutsson, & Westerholm, 2003) and managerial leadership behaviors (Nyberg et al., 2009).

References and Readings

Alfredsson, L., Hammar, N., Fransson, E., et al. (2002). Job strain and major risk factors for coronary heart disease among employed males and females in a Swedish study on work, lipids and fibrinogen. *Scandinavian Journal of Work, Environment & Health, 28*, 238–248.

Fransson, E. I., Alfredsson, L. S., de Faire, U. H., Knutsson, A., & Westerholm, P. J. (2003). Leisure time, occupational and household physical activity, and risk factors for cardiovascular disease in working men and women: The WOLF study. *Scandinavian Journal of Public Health, 31*, 324–333.

Nyberg, A., Alfredsson, L., Theorell, T., Westerlund, H., Vahtera, J., & Kivimaki, M. (2009). Managerial leadership and ischaemic heart disease among employees: The Swedish WOLF study. *Occupational and Environmental Medicine, 66*, 51–55.

Working Memory

David Pearson
School of Psychology, University of Aberdeen, Aberdeen, UK

Definition

The term “working memory” describes temporary memory systems involved in tasks such as reasoning, learning, and understanding. Examples of everyday tasks that rely on working memory include performing mental arithmetic or

remembering a shopping list. While linked to the concept of short-term memory (STM), theories of working memory place greater emphasis on simultaneous storage and processing of information. Memory systems responsible for STM can be regarded as forming part of an overall working memory system. Working memory not only temporarily stores information but also manipulates it during complex cognitive activities. Theories of working memory are highly influential in the fields of cognitive psychology, neuroscience, and behavioral medicine.

Description

An influential model of working memory is the multicomponent approach first proposed by Baddeley and Hitch in 1974. The original model was tripartite in nature and comprised three separate, limited-capacity components: the phonological loop, the visuospatial sketchpad, and the central executive. The phonological loop and sketchpad are modality-specific "slave systems" that enable individuals to retain verbal speech-based material and visuospatial material, respectively. The phonological loop consists of a passive phonological store containing information that is rehearsed by an active articulatory mechanism closely linked to the production of speech (Baddeley, 2007). A similar distinction has been made within the visuospatial component between a passive visual store and an active spatial mechanism involved during the planning and execution of movement (Logie, 2003; Pearson, 2007). Both the phonological loop and sketchpad are controlled by the central executive, a modality-free system responsible for strategic cognitive control, the coordination of tasks carried out in parallel, and scheduling and planning during multitasking (Law, Logie, & Pearson, 2006; Logie, Cocchini, Della Sala, & Baddeley, 2004). The original tripartite model was subsequently modified with the addition of a fourth component, the episodic buffer, which functions to bind together information from working memory and long-term memory into unitary multimodal representations (Baddeley, 2000).

An alternative to the multicomponent approach regards working memory as an activated portion of long-term memory rather than as a separate system. According to Cowan (1999), working memory comprises a set of cognitive processes that maintain information in a highly accessible state. Working memory representations are embedded within long-term memory, with the activation of representations controlled by attentional processes. The focus of attention is capacity limited and able to hold up to four activated representations (Cowan, 2005). The temporary activation of representations in working memory can be maintained by either continued attention or rehearsal in a verbal subsystem (Cowan, 1999).

The interaction between storage and processing in working memory is further explored by the time-based resource sharing model proposed by Barrouillet, Bernadin, & Camos (2004). In this model, rehearsal in working memory is carried out by an attentional mechanism also implicated during concurrent processing. Rehearsal must therefore take place during small time intervals in which task processing does not place demands upon attention. Forgetting in working memory is predicted by the model to increase with overall cognitive load, in which both the rate of individual processing steps during a task and their duration have a high density.

Theories of working memory have widespread application in behavioral medicine. Evidence suggests that schizophrenia is linked to a reduced ability to maintain information in working memory (Park & Holzman, 1992), with deficits also being found in patients' biological relatives (Myles-Worsley & Park, 2002). Working memory deficits have also been documented in patients suffering from bipolar affective disorder (Hammar & Ardal, 2009), Asperger's syndrome (Cui, Gao, Chen, Zou, & Wang, 2010), multiple sclerosis (McCarthy, Beaumont, Thompson, & Peacock, 2005), Alzheimer's disease (MacPherson, Della Sala, Logie, & Wilcock, 2007), and attention deficit hyperactivity disorder (Holmes, Gathercole, Place, Alloway, Elliott, & Hilton, 2010). Working memory has also been linked with theories of craving and addiction

(Kavanagh, Andrade, & May, 2005) and the occurrence of intrusive memories associated with post-traumatic stress disorder (Holmes, Brewin, & Hennessey, 2004; Pearson, Ross, & Webster, 2012; Pearson & Sawyer, 2011).

Cross-References

- ▶ Executive Function
- ▶ Posttraumatic Stress Disorder

References and Readings

Baddeley, A. D. (2000). The episodic buffer: A new component of working memory? *Trends in Cognitive Sciences, 4*(11), 417–423.

Baddeley, A. D. (2007). *Working memory, thought, and action*. Oxford: Oxford University Press.

Baddeley, A. D., & Hitch, G. J. (1974). Working memory. In G. Bower (Ed.), *The psychology of learning and motivation* (Vol. VIII, pp. 47–90). New York: Academic Press.

Barrouillet, P., Bernardin, S., & Camos, V. (2004). Time constraints and resource sharing in adults' working memory spans. *Journal of Experimental Psychology: General, 133*, 83–100.

Cowan, N. (1999). An embedded-processes model of working memory. In A. M. P. Shah (Ed.), *Models of working memory* (pp. 62–101). Cambridge: Cambridge University Press.

Cowan, N. (2005). *Working memory capacity*. Hove: Psychology Press.

Cui, J. F., Gao, D. G., Chen, Y. H., Zou, X. B., & Wang, Y. (2010). Working memory in early-school-age children with Asperger's Syndrome. *Journal of Autism and Developmental Disorders, 40*(8), 958–967.

Hammar, A., & Ardal, G. (2009). Cognitive functioning in major depression – a summary. *Frontiers in Human Neuroscience, 3,* Article No. 26.

Holmes, E. A., Brewin, C. R., & Hennessy, R. G. (2004). Trauma films, information processing, and intrusive memory development. *Journal of Experimental Psychology: General, 133*(1), 3–22.

Holmes, J., Gathercole, S. E., Place, M., Alloway, T. P., Elliott, J. G., & Hilton, K. A. (2010). The diagnostic utility of executive function assessments in the identification of ADHD in children. *Child and Adolescent Mental Health, 15*(1), 37–43.

Kavangh, D. J., Andrade, J., & May, J. (2005). Imaginary relish and exquisite torture: The elaborated intrusion theory of desire. *Psychological Review, 112*(2), 446–467.

Law, A. S., Logie, R. H., & Pearson, D. G. (2006). The impact of secondary tasks on multitasking in a virtual environment. *Acta Psychologica, 122*(1), 27–44.

Logie, R. H. (2003). Spatial and visual working memory: A mental workspace. In D. Irwin & B. H. Ross (Eds.), *The psychology of learning and motivation* (Vol. 42, pp. 37–38). New York: Academic Press.

Logie, R. H., Cocchini, G., Della Sala, S., & Baddeley, A. D. (2004). Is there a specific executive capacity for dual task coordination? Evidence from Alzheimer's disease. *Neuropsychology, 18*(3), 504–513.

MacPherson, S. E., Della Sala, S., Logie, R. H., & Wilcock, G. K. (2007). Specific AD impairment in concurrent performance of two memory tasks. *Cortex, 43*(7), 858–865.

McCarthy, M., Beaumont, J. G., Thompson, R., & Peacock, S. (2005). Modality-specific aspects of sustained and divided attentional performance in multiple sclerosis. *Archives of Clinical Neuropsychology, 20*(6), 705–718.

Myles-Worsley, M., & Park, S. (2002). Spatial working memory deficits in schizophrenia patients and their first degree relatives from Palau, Micronesia. *American Journal of Medical Genetics, 114*(6), 609–615.

Park, S., & Holzman, P. S. (1992). Schizophrenics show spatial working memory deficits. *Archives of General Psychiatry, 49*(12), 231–246.

Pearson, D. G. (2007). Visuospatial rehearsal processes in working memory. In N. Osaka, R. H. Logie, & M. D'Esposito (Eds.), *The cognitive neuroscience of working memory* (pp. 231–246). Oxford: Oxford University Press.

Pearson, D. G., Ross, F. D. C., & Webster, V. L. (2012). The importance of context: Evidence that contextual representations increase intrusive memories. *Journal of Behavior Therapy and Experimental Psychiatry, 43*, 573–580.

Pearson, D. G., & Sawyer, T. (2011). Effects of dual task interference on memory intrusions for affective images. *International Journal of Cognitive Therapy, 4*(2), 122–133.

Workload

Karen Jacobs[1], Miranda Hellman[2], Jacqueline Markowitz[2] and Ellen Wuest[2]

[1]Occupational Therapy, College of Health and Rehabilitation Science, Sargent Collage, Boston University, Boston, MA, USA

[2]Boston University, Boston, MA, USA

Synonyms

Work tasks

Definition

There is no one widely accepted definition of workload. Hart and Staveland (1988) describe workload as "the perceived relationship between the amount of mental processing capability or resources and the amount required by the task." Another definition is that it represents the relationship between a group or individual human operator and task demands. In simpler terms, it is the volume of work expected of a person. According to Wickens (1984), "the main objective of assessing and predicting workload is to achieve evenly distributed, manageable workload and to avoid overload or underload."

It can be measured in terms of many factors such as the amount of work accomplished over a period of time (number of hours worked or the number of assignments in a course), level of production, or the physical or cognitive demands of the work being performed (working with a person who speaks a different language than your own). There can be constraints to reaching one's workload such as lack of resources, motivation, and sufficient amount of time.

Cross-References

- ▶ Physical Functioning
- ▶ Social Support

References and Readings

Hart, S. G., & Staveland, L. E. (1988). Development of NASA-TLX (Task Load Index): Results of empirical and theoretical research. In P. A. Hancock & N. Meshkati (Eds.), *Human mental workload* (pp. 77–106). New York: Elsevier Science Publishers B.V (North Holland).

Spector, P., & Jex, S. (1998). Development of four self-report measures of job stressors and strain: Interpersonal conflict at work scale, organizational constraints scale, quantitative workload inventory, and physical symptoms inventory. *Journal of Occupational Health Psychology, 3*(4), 4356–4367.

Wickens, C. D. (1984). Processing resources in attention. In R. Parasuraman & D. R. Davies (Eds.), *Varieties of attention* (pp. 63–102). New York: Academic Press.

Work-Related Health

▶ Occupational Health

Work-Related Stress

▶ Job Related to Health

Worksite Health Promotion

Ellinor K. Olander
Applied Research Centre in Health and Lifestyle Interventions, Coventry University, Coventry, West Midlands, UK

Synonyms

Organizational health promotion

Definition

Worksite health promotion refers to interventions/programs implemented in the workplace that aim to promote health and improve employee well-being.

There are numerous benefits associated with implementing interventions/programs in the worksite setting including a great potential to reach a large number of individuals, provision of existing social support for behavior change or existing infrastructure making it easy to contact individuals or assess their behavior/health. Examples of worksite health promotion programs include those that target smoking, physical activity, stress management, weight reduction, and nutrition. Most worksite health promotion programs target individual behavior (such as encouraging individuals to stop smoking); others may target the physical environment (e.g., encouraging stair instead of elevator use) or organizational change/policy (such as not selling unhealthy food in the worksite restaurant).

Cross-References

- ▶ Nutrition
- ▶ Physical Activity
- ▶ Smoking Cessation

References and Readings

Chenoweth, D. (2006). *Worksite health promotion* (2nd ed.). Champaign, IL: Human Kinetics.

O'Donnell, M. P. (2001). *Health promotion in the workplace* (3rd ed.). Albany, NY: Delmar Cengage Learning.

Pronk, N. P. (Ed.). (2009). *ACSM's worksite health handbook: A guide to building healthy and productive companies (American College of Sports Med)* (2nd ed.). Champaign, IL: Human Kinetics.

World Health Organization (WHO)

Shekhar Saxena and M. Taghi Yasamy
Department of Mental Health and
Substance Abuse, World Health Organization,
Geneva 27, Switzerland

Basic Information

The World Health Organization (WHO) was established in 1948 and is the directing and coordinating authority on international health within the United Nation's system (World Health Organization, 2006). It is responsible for providing leadership on global health matters, shaping the health research agenda, setting norms and standards, articulating evidence-based policy options, providing technical support to countries, and monitoring and assessing health trends (About WHO).

WHO's constitution echoes a clear understanding about psychosocial aspects of health. It states that "Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity" (World Health Organization, 2007). It also lays emphasis from the beginning on the "happiness, harmonious relations and security to all people…" (World Health Organization, 2007).

Major Impact on the Field

WHO has a strong relationship with the Ministries of Health of the member states and this provides an opportunity for advocating for mental health and integration of mental health into general health care systems of countries.

For decades WHO has been generating tools and data on epidemiology of mental disorders, classification of mental disorders, and mapping available resources and services. WHO's ATLAS Mental Health and the WHO Assessment Instrument for Mental Health systems (WHO_AIMS) are well-known resources which are being widely used by mental health planners and policy makers across the globe (http://www.who.int/mental_health/evidence/atlas/en/; Saxena, Sharan, & Garrido, Saraceno, 2006; WHO_AIMS).

In 2001, when Dr. Gro Harlem Brutland was the Director General, WHO's theme was chosen "mental health, stop exclusion, dare to care." The World Health Report title was selected as "Mental Health: New Understanding, New Hope." The report focused on the fact that mental health is crucial to the overall well-being of individuals, societies, and countries. The report advocated the policies that were required to ensure that stigma and discrimination are broken down and that effective intervention and treatment are put in place (WHO, 2001).

WHO promotes the rights of people with mental illness and supports participation of service users at all stages of planning, implementation, and evaluation of mental health interventions (Funk, 2005). WHO has also produced intervention guidelines for a wide range of mental, neurological, and substance abuse disorders. In 2008, WHO's Director General launched the Mental Health Gap Action Programme (mhGAP) (World Health Organization, 2008), and in 2010 the mhGAP Intervention Guide (World Health Organization, 2010). The program and the related guidelines and tools will help poor resource countries in providing basic evidence-based mental health care through non-specialized health providers.

WHO is also currently developing the International Classification of Diseases (ICD-11) and is

collaborating with a wide group of professionals including psychologists to develop the mental and behavioral disorders chapter in ICD-11.

Cross-References

- ▶ Community-Based Health Programs
- ▶ Health Care
- ▶ Health Care Access
- ▶ Health Care System
- ▶ Health Care Utilization
- ▶ Health Economics
- ▶ Health Policy/Health-Care Policy
- ▶ Health Promotion and Disease Prevention
- ▶ Health Systems
- ▶ Lifestyle, Healthy
- ▶ Prevention: Primary, Secondary, Tertiary
- ▶ Primary Care
- ▶ Primary Care Providers
- ▶ Public Health
- ▶ Quality of Life
- ▶ Quality-Adjusted Life Years (QALYs)
- ▶ Reproductive Health
- ▶ Risk Factors and Their Management
- ▶ Tobacco Control

References and Readings

About WHO, Retrieved March 01, 2011, from http://www.who.int/about/en/

Funk, M. (2005). Advocacy for mental health: Roles for consumer and family organizations and governments. *Health Promotion International, 21*(1), 70–75.

Saxena, S., Sharan, P., Garrido, M., & Saraceno, B. (2006). World Health Organization's Mental Health Atlas 2005: Implications for policy development. *World Psychiatry, 5*(3), 179–184.

WHO (2001). *The world health report 2001 – mental health: New understanding, new hope*. Geneva: Author.

WHO_AIMS, *General information*. Retrieved May 25, 2011, from http://www.who.int/mental_health/evidence/WHO-AIMS/en/

World Health Organization. *Project atlas: Resources for mental health*. Retrieved May 25, 2011, from http://www.who.int/mental_health/evidence/atlas/en/

World Health Organization. (2006). *Working for health: An introduction to World Health Organization*. Geneva: Author.

World Health Organization. (2007). *Basic documents* (46th ed.). Geneva: Author.

World Health Organization. (2008). *mhGAP: Mental Health GAP Action Programme: Scaling up care for mental, neurological and substance use disorders*. Geneva: Author.

World Health Organization. (2010). *mhGAP intervention guide for mental, neurological and substance use disorders in non-specialized health settings. version 1.0*. Geneva: Author.

Worldview

- ▶ Meaning (Purpose)

Worry

J. F. Brosschot and Bart Verkuil
Clinical, Health and Neuro Psychology, Leiden University, Leiden, Netherlands

Synonyms

Intrusive thoughts; Perseverative cognition; Repetitive thinking; Rumination

Definition

Worry is defined as "*a chain of thoughts and images, negatively affect-laden and relatively uncontrollable. The worry process represents an attempt to engage in mental problem-solving on an issue whose outcome is uncertain but contains the possibility of one or more negative outcomes. Consequently, worry relates closely to fear process*" (Borkovec, Robinson, Pruzinsky, & DePree, 1983). Worry can impact health in several ways. First, it is a form of the so-called perseverative cognition (see term), which is defined as "the ongoing cognitive representation of psychological stressors (threat)" (Brosschot, Gerin, & Thayer, 2006). Perseverative cognition is believed to be responsible for a large part of the health impact of psychological stressors, because it prolongs the physiological responses to these

stressors. Second, disease-specific worry can lead to maladaptive outcomes in the long or short term, for example, in the aftermath of surgery or other intrusive medical treatments (Verkuil, Brosschot, Gebhardt, & Thayer, 2010). Third, severe worry about illness can take the form of the anxiety disorder hypochondriasis, or disorders such as somatization or somatoform disorders. In a milder form of illness, worry can increase the number and severity of medically unexplained symptoms, presumably via the excessive activation of illness-related memory networks, that bias perception and behavior in the direction of experiencing and reporting symptoms (Brosschot, 2002; Brown, 2004). Fourth, a symptom-specific, catastrophizing form, for example "pain catastrophizing," appears to play a role in the maintenance and exacerbation of symptoms. Catastrophizing thoughts about a symptom such as pain may drive the vicious circle that leads from pain to catastrophizing to immobility to more pain, etc. (Vlaeyen & Linton, 2000). Last but not least worry and related perseverative thinking styles, such as rumination, play a causal or maintaining role in several forms of psychopathology (Watkins, 2008).

Cross-References

- ▶ Anxiety and Its Measurement
- ▶ Intrusive Thoughts
- ▶ Negative Thoughts
- ▶ Perseverative Cognition

References and Readings

Borkovec, T. D., Robinson, E., Pruzinsky, T., & DePree, J. A. (1983). Preliminary exploration of worry: Some characteristics and processes. *Behavioral Research Therapy, 21*, 9–16.

Brosschot, J. F. (2002). Cognitive-emotional sensitization and somatic health complaints. *Scandinavian Journal of Psychology, 43*, 113–121.

Brosschot, J. F., Gerin, W., & Thayer, J. F. (2006). The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *Journal of Psychosomatic Research, 60*, 113–124.

Brown, R. J. (2004). Psychological mechanisms of medically unexplained symptoms: An integrative conceptual model. *Psychological Bulletin, 130*, 793–812.

Verkuil, B., Brosschot, J. F., Gebhardt, W., & Thayer, J. F. (2010). When worries make you sick: A review of perseverative cognition, the default stress response and somatic health. *Journal of Experimental Psychopathology, 1*(1), 87–118.

Vlaeyen, J. W., & Linton, S. J. (2000). Fear-avoidance and its consequences in chronic musculoskeletal pain: A state of the art. *Pain, 85*(3), 317–332.

Watkins, E. R. (2008). Constructive and unconstructive repetitive thought. *Psychological Bulletin, 134*(2), 163–206.

Worship

▶ Prayer

Wound Healing

Christopher G. Engeland
Center for Wound Healing and Tissue Regeneration, University of Illinois, Chicago, IL, USA

Synonyms

Tissue repair

Definition

Wound healing pertains to the repair and regeneration of damaged tissue.

Stages of Wound Healing

Tissue repair involves three interdependent and overlapping phases: (1) the inflammatory phase (hours to days) in which blood flow to the injured area is decreased, a blood clot forms, inflammatory cells (e.g., neutrophils, monocytes) are recruited to the site of injury, and bacterial clearance occurs; (2) the proliferative phase

(days to weeks) in which fibroblasts, epithelial cells, and endothelial cells are recruited and proliferate for the rebuilding process which involves wound contraction, reepithelialization, and angiogenesis; and (3) the remodeling phase (weeks to months) in which the connective tissue matrix begun in the previous phase is fully formed and restructured (for review, see Engeland & Marucha, 2009). Psychological stress can impair healing through its effects on each of these phases.

Description

Overview

It is generally accepted that a patient's state of mind can modulate immune responses (for reviews, see Christian et al., 2009; Hawkley, Bosch, Engeland, Cacioppo, & Marucha, 2007), affecting factors such as healing rates and surgical outcomes. Numerous psychological factors such as stress, anxiety, depression, hostility, and anger can relate to poor healing, such as slowed wound closure which increases the risk of infection and other complications. Conversely, factors such as positive mood, optimism, and positive social interactions can improve healing outcomes.

Stress Pathways

The primary effects of stress on wound healing occur through central activation of the hypothalamic pituitary adrenal (HPA) axis and the sympathetic nervous system (SNS). HPA activation promotes the systemic release of glucocorticoids (GCs) (e.g., cortisol in humans) from the adrenal glands. GCs are potently anti-inflammatory and immunosuppressant and act as the main brake on inflammation. By dysregulating the release and function of GCs, chronic stress can result in higher inflammatory reactions following immune activation (e.g., response to injury). Activation of the SNS by stress results in vasoconstriction (caused by norepinephrine), which limits blood flow to injury sites resulting in hypoxia and lowering available nutrient supplies to tissues. Vasoconstriction may also delay the arrival of immune cells, such as neutrophils and monocytes, to the site of injury, further impeding the healing process. Through these two pathways, stress dysregulates immune functions and alters early wound repair. The end result is impaired bacterial clearance, prolonged inflammation, higher risk of infection, and greater postsurgical (or post-injury) complications (for reviews see Marucha & Engeland, 2007; Engeland & Marucha, 2009).

Stress and Anxiety

Since 1995, numerous studies have demonstrated that stress negatively affects healing and this notion is now widely accepted. Stressors such as caregiving for an Alzheimer's patient and university examinations, along with preoperative and perceived stress, have each been shown to slow dermal healing (for reviews, see Devries et al., 2007; Engeland & Graham, 2011). For instance, stress associated with caregiving slowed the healing of skin wounds by 24%. Such delays are typically accompanied by elevated cortisol levels and reduced inflammation, and similar stress-induced changes are mirrored in animal research (Engeland & Marucha, 2009). Aside from skin, stress affects mucosal inflammation and is a known risk factor for periodontal disease. Stress from university examinations in American dental students slowed the healing of experimental oral mucosal wounds by 40%. It is of note that a stressor as predictable and transient as examinations should delay healing to such a degree in professional students. Interestingly, stress appears to delay healing in mucosal tissues by increasing early inflammation, rather than by inhibiting inflammation as has been shown in skin wounds (Engeland & Graham, 2011). Clinically, efforts should be made to reduce both pre- and postsurgical stress and to avoid scheduling elective surgery during or shortly after a period of stress (e.g., university examinations).

Depression and Other Behavioral Constructs

Psychosocial factors other than stress and anxiety also affect immunity. Higher depressive symptoms, marital disagreements, lower levels of anger control, and higher levels of hostility have

all been related to slowed healing in skin. Similarly, subclinical depression (dysphoria) relates to slower healing of mucosal wounds. Conversely, positive mood, optimism, and positive social communications relate to faster healing times in skin (for review, see Engeland & Graham, 2011), suggesting a positive role for behavioral interventions on wound healing (see below).

Pain

Pain usually accompanies healing and can be viewed as a unique stressor with both physiological and psychological components. Pain can alter immunity directly by promoting cytokine release and indirectly through activation of stress pathways and changes in behavior. Through these different mechanisms, pain exacerbates the negative effects of stress on healing. Greater pain from elective gastric bypass surgery has been related to slower healing of an experimental skin wound placed on the day of surgery. Importantly, pain and stress are not additive but synergistic in their effects. Not only does each negatively affect healing, but each promotes the other through physiological and immune responses and through changes in mood and behavior. For instance, pain can lead to maladaptive health behaviors such as loss of sleep, inactivity, and alcohol use, each of which has been linked with poorer healing. Not only pain, but also its anticipation, can be a tangible source of anxiety for surgical patients, as well as concerns about the healing process. Efforts to minimize not only pain, but also these sources of anxiety, are encouraged (for review, see Engeland & Graham, 2011).

Ageing

It is well accepted that ageing alters skin morphology. Aged skin undergoes reductions in vascularization, granulation tissue, collagen, elastin, mast cells, fibroblasts, and epidermal turnover (reduced ~50% as one ages from 20 to 70 years). In general, the elderly have slower healing times, increased rates of infection and wound dehiscence, and reduced wound strength. This is primarily caused by reductions in reepithelialization, angiogenesis, macrophage infiltration, and collagen deposition. In women, menopause exacerbates these effects. It is important to note that wound healing is slowed but not impaired in the elderly and the eventual healing outcome is similar to that of young adults. This appears to hold true for mucosal healing as well. Also, for reasons unknown, wounds in the aged heal better aesthetically with less scarring. However, risk factors for impaired healing occur more commonly in the elderly, such as comorbidity, malnutrition, immobility, and obesity. Stress is also more common in the aged and negatively impacts tissue healing to a greater degree than in young adults. Prior to surgery, each of these risk factors for impaired healing should be tested for, monitored and treated vigorously to ensure maximal healing outcomes. In the elderly, the presence of any of these risk factors should serve as a red flag to the clinician (for review, see Engeland & Gajendrareddy, 2011).

Chronic Wounds

Most wound healing studies have been conducted in a controlled laboratory setting on acute wounds which heal relatively quickly (for a review of wound healing models, see Bosch, Engeland, & Burns, 2011). Clinical studies, which involve more naturally occurring wounds (e.g., diabetic foot ulcers, surgical wounds, accidents), provide valuable information about how such factors affect larger and more chronic wounds where poor outcomes may have more serious implications. Higher levels of depression and anxiety have been related to slower healing of chronic leg ulcers, with patients who scored in the top 50% of these measures being four times more likely to be categorized as slow healers. Thus, psychological stress has been shown to exert negative effects on the healing of both acute and chronic wounds.

Skin Barrier Recovery

The skin is an essential barrier for providing pathogen resistance and limiting water loss. This barrier plays an important factor in numerous skin diseases (e.g., psoriasis, atopic

dermatitis) and in dermal healing. Repair to this barrier can be evaluated through tape stripping, in which cellophane tape is repeatedly applied to and removed from a dermal area (e.g., forearm). An evaporimeter is then used to assess transepidermal water loss (TEWL) over time. Stress from public speaking (i.e., Trier Social Stress Test) slows barrier recovery times and is associated with increased levels of cortisol and inflammation. Similar delays in barrier recovery times have been demonstrated with stress stemming from marital dissolution, university examinations, and sleep deprivation. Higher positive mood relates to faster recovery times, indicating the negative effect of stress on skin barrier repair is buffered by positive emotion. Skin barrier function, which is an important factor in numerous skin diseases (e.g., psoriasis, atopic dermatitis) and in dermal healing, undergoes slower recovery/repair during stress (for review, see Engeland & Graham, 2011).

Health Behaviors

Stress and pain can affect wound healing indirectly through behavioral alterations such as poor sleep and nutrition, reduced exercise, increased consumption of alcohol or nicotine, and self-neglect. For example, sleep deprivation can disrupt macrophage/lymphocyte functions and alter inflammation, thereby hindering immunity. Such changes can themselves promote stress, often in the form of anxiety or depression, which further impact on health creating a downward spiral effect. Smoking, which often increases during times of stress, reduces vitamin C and oxygen levels in blood, impairs collagen deposition and macrophage function, and alters turnover of the extracellular matrix during healing. It has been shown that smoking intervention 6–8 weeks before surgery reduces postoperative morbidity including wound-related complications. In addition, interventions aimed at lowering stress prior to surgeries such as patient education, massage therapy, and relaxation with guided imagery can have positive effects on postsurgical outcomes. Similarly, a 4-week exercise regimen was shown to speed healing rates by 25% in experimental skin wounds, independent of perceived stress. Emotional disclosure interventions, which involve participants writing about traumatic personal events, have been shown to positively affect immunity and speed up the healing of experimental wounds in skin. Wounds were 11% smaller compared to control subjects that wrote about time management (for reviews, see Engeland & Marucha, 2009; Engeland & Graham, 2011). Given the impact that such behaviors and interventions have on health, reducing negative behaviors and facilitating positive behaviors will help to optimize immune responses, wound healing, and postsurgical outcomes.

Cross-References

- ▶ Aging
- ▶ Anxiety
- ▶ Corticosteroids
- ▶ Depression
- ▶ Pain
- ▶ Stress

References and Readings

Bosch, J. A., Engeland, C. G., & Burns, V. E. (2011). Psychoneuroimmunology in vivo: Methods and principles. In J. Decety & J. T. Cacioppo (Eds.), *The Oxford handbook of social neuroscience* (pp. 134–148). New York: Oxford University Press.

Christian, L. M., Deichert, N. T., Gouin, J. P., Graham, J. E., & Kiecolt-Glaser, J. K. (2009). Psychological influences on endocrine and immune function. In G. G. Berntson & J. T. Cacioppo (Eds.), *Handbook of neuroscience for the behavioral sciences* (pp. 1260–1279). Hoboken, NJ: Wiley.

Devries, A. C., Craft, T. K., Glasper, E. R., Neigh, G. N., & Alexander, J. K. (2007). 2006 Curt P. Richter award winner social influences on stress responses and health. *Psychoneuroendocrinology, 32*, 587–603.

Engeland, C. G., & Gajendrareddy, P. K. (2011). Wound healing in the elderly. In M. Katlic (Ed.), *Cardiothoracic surgery in the elderly: Evidence based practice* (pp. 259–270). Berlin: Springer.

Engeland, C. G., & Graham, J. E. (2011). Psychoneuroimmunological aspects of wound healing and the role of pain. In D. Upton (Ed.), *Psychological impact of pain in patients with wounds* (pp. 87–114). London: Wounds UK Limited, A Schofield Healthcare Media Company.

Engeland, C. G., & Marucha, P. T. (2009). Wound healing and stress. In R. D. Granstein & T. A. Luger (Eds.), *Neuroimmunology of the skin: Basic science to clinical relevance* (pp. 233–247). Berlin: Springer.

Hawkley, L. C., Bosch, J. A., Engeland, C. G., Cacioppo, J. T., & Marucha, P. T. (2007). Loneliness, dysphoria, stress, and immunity: A role for cytokines. In N. P. Plotnikoff, R. E. Faith, & A. J. Murgo (Eds.), *Cytokines: Stress and immunity*. Boca Raton: CRC Press.

Marucha, P. T., & Engeland, C. G. (2007). Stress, neuroendocrine hormones, and wound healing: Human models. In R. Ader, D. Felten, & N. Cohen (Eds.), *Psychoneuroimmunology* (pp. 825–835). San Diego: Academic Press.

Written Disclosure

▶ Expressive Writing and Health

X

X-Ray Computed Tomography

► CAT Scan
► Computerized Axial Tomography (CAT) Scan

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

Y

Years of Potential Life Lost (YPLL)

► Life Years Lost

Yoga

Melissa M. A. Buttner
Department of Psychology, University of Iowa, Iowa City, IA, USA

Definition

Yoga is an ancient tradition originating in India and is derived from a Sanskrit word meaning "to yoke" or "to unite" (Desikachar, 1999). In Western culture, yoga is considered a form of physical activity and, more recently, a mind-body intervention designed to support overall health and well-being. In its full expression, yoga integrates three basic components: breath (*pranayama*), physical poses (*asanas*), and meditation (*dhyana*).

Description

Origins

Yogic philosophy traces back to 3000 BCE, with roots spanning four periods known as the Vedic (2000–600 BC), Preclassical (600–200 BC), Classical (200 BC), and Postclassical periods (Feuerstein, 2001). The Preclassical period is associated with the emergence of the *Upanishads* (200 texts influencing Hindu philosophy) and the creation of the *Bhagavad Gita*, considered to be among yoga's oldest written texts. It was during this time that the foundations of meditation and the concept of *samadhi* (a path to enlightenment) surfaced (Feuerstein, 2001). Patanjali's *Yoga Sutras* was compiled during the Classical period (Iyengar, 1993) and contains 196 sutras describing raja yoga and its underlying eight-limbed path, or *ashtanga* yoga: *yama* (ethical guidelines), *niyama* (spiritual observances), *asana* (physical poses), *pranayama* (breathing exercises), *pratyahara* (control of the senses), *dharana* (concentration), *dhyana* (meditation), and *samadhi* (state of bliss) (Iyengar, 2001). During the Postclassical period, *Hatha* yoga emerged, the most common form of yoga practiced in Western culture today (Feuerstein, 2001).

Complementary and Alternative Medicine (CAM)

Complementary and alternative medicine (CAM) is a form of medicine consisting of systems, practices, and consumer products that are outside the realm of traditional or conventional forms of medicine (Barnes, Bloom, & Nahin, 2008). Acceptance of this nontraditional form of medicine is growing. Findings from a 2008 survey on Americans' use of CAM published by the National Center for Complementary and Alternative Medicine (NCCAM) showed that *yoga* was one of the top ten most commonly used CAM therapies among adults, with 6.1% indicating use

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

Yoga, Table 1 Application of yoga as a therapeutic intervention

Psychological	Physical	Physiological
Anxiety	Arthritis	High blood pressure
ADHD	Cancer	High cortisol levels
Depression	Carpal tunnel syndrome	Increased heart rate
Eating disorders	Diabetes	PNS/SNS activity
OCD	Epilepsy	Poor circulation
Perimenopausal symptoms	Infertility	
Premenstrual syndrome	Insomnia	
PTSD	Irritable bowel syndrome	
Stress	Multiple sclerosis	
	Obesity	
	Pain (chronic)	
	Pregnancy (complications)	
	Sciatica	
	Scoliosis	
	Sinusitis	

ADHD attention deficit hyperactivity disorder, *OCD* obsessive compulsive disorder, *PTSD* post-traumatic stress disorder, *PNS* parasympathetic nervous system, *SNS* sympathetic nervous system

of yoga in the past year (Barnes et al., 2008). Preference for yoga as a CAM option over that of conventional forms of medicine may be associated with characteristics including decreased stigma, a more gentle form of treatment with minimal side effects, and flexibility in personalizing treatment (Uebelacker et al., 2010).

Yoga's Role in Behavioral Medicine

The past few decades have witnessed a confluence of Western medical science and psychological theories with ideas from Eastern practices such as yoga, which has led to the recent emergence of yoga as a mind-body intervention. In contrast with traditional forms of medicine, yoga offers a holistic approach to treating physical and mental health issues (Hayes & Chase, 2010) and is currently being used to address various health conditions (see Table 1 for a detailed list). Recent reviews of the yoga literature suggest that yoga has beneficial effects on pain syndromes, cardiovascular, autoimmune and immune conditions, and on pregnancy (Field, 2011). Further, there is evidence to suggest that yoga yields physiological benefits as evidenced in studies showing links between yoga and decreased heart rate and blood pressure, as well as physical effects, such as weight loss and increased muscle tone (Field, 2011). More well-established physiological effects of yoga include stress reduction (Ross & Thomas, 2010), with psychological effects evidenced by increased mindfulness and reduced symptoms of depression and anxiety (Field, 2011; Uebelacker et al., 2010) (see Figs. 1 and 2 for illustrations of yoga poses).

Stress

Stress has adverse effects as evidenced in many health conditions. Empirical evidence for yoga in combating these adverse effects suggests that controlled breathing helps to focus and relax the mind through activation of the autonomic nervous system (ANS), which may work to counteract stress (Harinath et al., 2004). To understand the role of stress in disease and the effects of relaxation associated with a yoga practice in prevention and recovery, it is important to understand the function of the ANS. The ANS is responsible for regulating the heart, intestines, and other internal organs through a synergistic relationship between the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS) (McCall, 2007). In general, when activity is high in the SNS, it will be low in the PNS, and vice versa. The SNS, along with the

Yoga, Fig. 1 *Warrior II Pose* (*Virabhadrasana II*) requires weight bearing and helps to strengthen and stretch the legs and ankles, stimulates abdominal organs, and increases stamina. This pose is recommended for carpal tunnel syndrome, flat feet, osteoporosis, and sciatica (McCall, 2007)

Yoga, Fig. 2 *Cobra Pose* (*Bhujangasana*) helps to strengthen the spine, stretch the chest and lungs, shoulders, and abdomen. It is recommended for stress, fatigue, and sciatica (McCall, 2007)

stress hormones cortisol and andrenaline, is responsible for generating a response to stressful situations, leading to physiological changes such as increased heart rate and blood pressure (McCall, 2007). Increased mobilization of energy and additional blood and oxygen flowing to the body will enable a person to respond to the stressor, otherwise known as the classic "fight or flight" response (McCall, 2007). Conversely, the PNS slows the heart rate and lowers blood pressure to support recovery from a stressful situation, thereby functioning as a restorative mechanism that is often referred to as the "relaxation response" (Benson, 2000).

The three basic components of yoga – physical postures, breathing, and meditation – interact

with the PNS and SNS as potential mechanisms by which yoga has a calming effect on the nervous system, potentially leading to decreased stress reactivity (Ross & Thomas, 2010). For example, ujjayi breathing (a form of slow yoga breathing characterized by an oceanic sound) is hypothesized to increase activation of the PNS, leading to a calm but alert state while simultaneously having a restorative effect on the body (McCall, 2007). Practicing more rigorous forms of breathing (e.g., *kapalabhati* breathing) and poses (e.g., sun salutations) triggers a sympathetic response similar to that found in traditional forms of aerobic activity. In contrast to a typical aerobic workout, however, a more invigorating yoga practice followed by a sequence of relaxing poses will stimulate the PNS and restore energy (McCall, 2007).

Mindfulness

The most commonly used definition of *mindfulness* is that of Jon Kabat-Zinn's, who defined it as "paying attention in a particular way: on purpose, in the present moment and nonjudgmentally" (Kabat-Zinn, 1994, p. 4). Support for mindfulness as an agent of change comes by way of its role as an active component of evidence-based psychotherapies (e.g., acceptance and commitment therapy and mindfulness-based cognitive therapy). The practice of mindfulness allows one to experience disengaging from evaluative thinking in the presence of negative stimuli through cultivation of an attitude of curiosity and attention to ongoing reactions to emotions, thoughts, and feelings (Shapiro et al., 2008). In depressed patients, for example, a repetitive focus on negative feelings (rumination) is a typical way of responding and is implicated in the onset and maintenance of depression. *Mindfulness* cultivated in a yoga practice may counteract ruminative thinking by offering participants an alternative focus, such as the breath and physical sensations experienced in the body (Uebelacker et al., 2010). Through a regular yoga practice, yoga practitioners naturally begin to adopt a lifestyle guided by yogic philosophies such as self-acceptance, compassion, and spirituality. In general, the more one commits to the practice, the greater the benefits tend to be, and healthier habits begin to form (McCall, 2007).

Yoga Styles

There is a preponderance of *Hatha* yoga styles practiced in the West today, with popular forms including *Anusara, Ashtanga, Bikram, Iyengar, Power, Vinyasa flow*, and *Viniyoga*. Most styles generally incorporate mindfulness with physical activity. Yoga, however, is distinct from traditional forms of physical activity with its focus on breath, and the intentional linking of breath with movement of the body through a sequence of postures (McCall, 2007). In addition, there are some forms of *Hatha* yoga that focus on the therapeutic application of poses for various medical conditions, including *Anusara*, *Iyengar*, and *Viniyoga*.

Anusara yoga was developed by John Friend who integrated his Iyengar background with a "heart-centered" approach to teaching (Friend, 2006). The focus in this style of yoga is on the physical poses and precision of alignment; however, chanting, pranayama, and meditation are also woven into many classes (Friend, 2006). Although the application of Anusara yoga is not well established in the empirical literature, characteristics such as a focus on alignment may support its use as a therapeutic tool (McCall, 2007).

Iyengar yoga, developed by B.K.S. Iyengar, is rooted in Patanjali's *Yoga Sutras* and is known for its therapeutic application based on a large body of literature supporting the benefits of Iyengar yoga and its emphasis on precision and alignment (Iyengar, 1995). Iyengar yoga is often employed in research trials examining the effectiveness of yoga for treating mood disorders such as depression and anxiety. This particular style of yoga incorporates the use of props (bolsters, blankets, blocks) to make yoga accessible to beginners, despite limited experience and flexibility, and to help assist in guiding the student into the posture (Iyengar, 2001). In this style of yoga, the poses are typically held for a longer duration of time relative to other forms of yoga such as Vinyasa flow. It is thought that holding the poses allows for proper alignment that is the hallmark

of Iyengar yoga (Iyengar, 2001). Yoga therapy performed according to this lineage of yoga can only be performed by instructors certified at a Junior Intermediate II level or above (see IYNAUS website for instructors and their level of certification). Iyengar yoga is known to have the highest standards in terms of teacher training and certification. Benefits of Iyengar yoga can be seen with many health conditions, ranging from depression to stress-related illnesses (Shapiro et al., 2007).

Viniyoga is a highly therapeutic and gentle style of yoga founded by T.K.V. Desikachar, in which poses and sequences are modified according to the needs of the student (Desikachar, 1999). Students practicing this style of yoga will flow from one pose to the next, holding the poses for a brief period of time, minimizing the risk of injury and making this style of yoga suitable for students with chronic diseases (McCall, 2007). In addition, pranayama breathing and chanting are integrated into a typical class.

Application of Yoga

Currently, there is no reliable system for identifying the most effective style of yoga for any one individual. Despite the number of studies examining the beneficial effects of yoga, most studies do not compare different styles of yoga or the mechanisms of action by which one particular style of yoga is effective (Uebelacker et al., 2010). In general, it is recommended that the elements of a yoga class (e.g., relaxation, rigorous flow with challenging poses), in addition to the quality of instruction, be considered when starting a yoga practice (McCall, 2007). The length of a yoga class may range from 60 to 90 minute, with each posture being held for a few seconds to 1 minute, depending on the style of yoga.

For physically fit individuals with no serious medical conditions contraindicated with yoga, most yoga classes are appropriate. For beginning students, learning yoga in a group environment or through private instruction with a certified instructor is preferable; however, individuals may also learn using audiotapes or DVDs. Individuals presenting with serious medical conditions seeking yoga as a form of therapy should consult with a certified yoga instructor specializing in the medical issue being treated (Shapiro et al., 2008). An increasing number of classes are now being offered that are tailored to the needs of specific populations, such as pregnant and postpartum women. Women practicing yoga during and after pregnancy are advised to attend classes with an instructor knowledgeable in providing modifications specific to the perinatal population. If a woman established a rigorous yoga practice prior to conceiving, it is recommended that she transition to a gentler style of yoga until after childbirth (McCall, 2007).

Cross-References

- ▶ Complementary and Alternative Medicine
- ▶ Exercise
- ▶ Meditation
- ▶ Mindfulness
- ▶ Physical Activity and Health
- ▶ Physical Activity Interventions

References and Readings

Barnes, P. M., Bloom, B., & Nahin, R. (2008). *Complementary and alternative medicine use among adults and children: United States, 2007* (CDC National Health Statistics Report No. 12). Accessed November 27, 2010.

Benson, H. (2000). *The relaxation response*. New York: Harper Paperbacks.

Cope, S. (2006). *The wisdom of yoga: A seeker's guide to extraordinary living*. New York: Bantam Dell.

Desikachar, T. K. V. (1999). *The heart of yoga*: *Developing a personal practice* (revised ed.). Rochester, VT: Inner Traditions International.

Feuerstein, G. (2001). *The yoga tradition: Its history, literature, philosophy and practice*. Prescott, AZ: Hohm Press.

Field, T. (2011). Yoga clinical research review. *Complementary Therapies in Clinical Practice, 17*, 1–8.

Friend, J. (2006). *Anusara yoga teacher training manual* (9th ed.). The Woodlands, TX: Anusara.

Harinath, K., Malhotra, A. S., Pal, K., Prasad, R., Kumar, R., Kain, T. C., Rai, L., & Sawhney, R. C. (2004). Effects of Hatha yoga and Omkar meditation on cardiorespiratory performance, psychologic profile, and melatonin secretion. *Journal of Alternative and Complementary Medicine*, *10*, 261–268.

Hayes, S. C. (1999). *Acceptance and commitment therapy: An experiential approach to behavior change*. New York: Guilford Press.
Hayes, M., & Chase, S. (2010). Prescribing yoga. *Primary Care, 37*, 31–47.
International Association of Yoga Therapists. Accessed November 29, 2010.
Iyengar, B. K. S. (1993). *Light on the yoga sutras of Patanjali*. London: Aquarian Press.
Iyengar, B. K. S. (1995). *Light on yoga*. New York: Schocken Books.
Iyengar, B. K. S. (2001). *Yoga: The path to holistic health*. London: Dorling Kindersley.
Iyengar Yoga National Association of the United States (IYNAUS). Retrieved from www.iynaus.org. Accessed November 29, 2010
Kabat-Zinn, J. (1994). *Wherever you go, there you are: Mindfulness meditation in everyday life*. New York: Hyperion.
Kripalu Center for Yoga and Health. Retrieved from www.kripalu.org. Accessed November 29, 2010
McCall, T. (2007). *Yoga as medicine: The yogic prescription for health and healing*. New York: Bantam Dell.
National Center for Complementary and Alternative Medicine (NCCAM). Retrieved from http://nccam.nih.gov/. Accessed November 29, 2010
Ross, A., & Thomas, S. (2010). The health benefits of yoga and exercise: A review of comparison studies. *The Journal of Alternative and Complementary Medicine, 16*, 3–12.
Shapiro, D., Cook, I. A., Davydov, D. M., Ottaviani, C., Leuchter, A. F., & Abrams, M. (2007). Yoga as a complementary treatment of depression: Effects of traits and moods on treatment outcome. *Evidence-based Complementary and Alternative Medicine: eCAM, 4*, 493–502. doi:10.1093/ecam/nel114.
Shapiro, S. L., Oman, D., Thoresen, C. E., Plante, T. G., & Flinders, T. (2008). Cultivating mindfulness: Effects on well-being. *Journal of Clinical Psychology*, *64*, 840–862.
Uebelacker, L. A., Epstein-Lubow, G., Gaudiano, B. A., Tremont, G., Battle, C. L., & Miller, I. W. (2010). Hatha yoga for depression: Critical review of the evidence for efficacy, plausible mechanisms of action, and directions for future research. *Journal of Psychiatric Practice, 16*, 22–33.
Yoga Alliance. Retrieved from www.yogaalliance.org. Accessed November 29, 2010
Yoga Journal. Retrieved from www.yogajournal.com. Accessed November 29, 2010
Yoga Research and Education Center (YREC). Retrieved from http://www.yrec.org/. Accessed November 29, 2010

Youth Life Orientation Test (Y-LOT)

► Optimism and Pessimism: Measurement

Z

Z Distribution

► Standard Normal (Z) Distribution

Zoloft®

► Selective Serotonin Reuptake Inhibitors (SSRIs)

Z-Score

► Standard Normal (Z) Distribution

Zung Depression Inventory

Maria Kleinstäuber
Department of Clinical Psychology and Psychotherapy, Johannes Gutenberg-University of Mainz, Mainz, Germany

Synonyms

Zung depression rating scale (ZDRS); Zung depression scale; Zung self-assessment depression scale; Zung self-rating depression scale (SDS)

Definition

The Zung Depression Inventory (SDS) is a 20-item self-rating scale concerning affective, cognitive, behavioral, and somatic symptoms of depression (Zung, 1965). Half of the items are worded positively, half are phrased negatively. The scale was developed on the basis of the most commonly found diagnostic criteria of depression and patient interviews. Subjects rate each item according to how they felt during the preceding week. The response categories range from (1) "none or a little of the time" to (4) "most or all of the time." The scale takes on average 5 min to complete. An index for the SDS is derived by summing the item scores. This summary score is then divided by a maximum possible score of 80. The SDS index ranges from 0.25 to 1. The following cutoffs are recommended by the author of the scale: Individuals with an index below 0.62 are considered normal, with an index of 0.62–0.74 are considered to suffer from a mild depression, and with an index of 0.75–0.86 are considered to suffer from a moderate to marked depression. Indices of 0.87 and above indicate severe depression. The SDS is available in about 30 languages.

The little available data examining the reliability of the SDS reveals satisfactory *split-half reliability* ($r = 0.73$) and *internal consistency* (Cronbach's alpha = 0.79). *Concurrent validity* of the scale could be demonstrated by correlating the SDS with other standardized self-rating scales of depression like the Beck Depression Inventory

M.D. Gellman & J.R. Turner (eds.), *Encyclopedia of Behavioral Medicine*,
DOI 10.1007/978-1-4419-1005-9,

and the Depression Scale of the Minnesota Multiphasic Personality Inventory. Results of correlations between the SDS and clinician-rated instruments like the Hamilton Rating Scale for Depression are less consistent. *Factor analyses* of the SDS revealed three main factors: positive symptoms/well-being, negative effect, and somatic symptoms. Sensitivity of the SDS in differentiating between depressed and nondepressed subjects was found to be adequate. The influence of different demographical variables on the SDS index was also examined. There seem to be higher SDS scores for individuals being older than 64 or younger than 20 years in non-patient groups. Associations between SDS score and age in psychiatric populations could not be demonstrated clearly. Women in patient as well as non-patient groups seem to have slightly higher scores than males. Furthermore, SDS scores are slightly negatively correlated with education (range: $r = -.08$ to $-.28$).

The SDS has not only been used in psychiatric settings and in clinical research to monitor for treatment effectiveness but also in behavioral medical settings. Specific difficulties have to be considered when the SDS is used for screening depressive symptoms in medically ill patients. For example, somatic symptoms of depression can be confounded with symptoms of the organic illness or side effects of medication. This can lead to an overestimation of depression (e.g., fatigue or insomnia can also be a symptom of cancer or can be a side effect of pain medication). Especially in diseases influencing cognitive functions, the use of reverse coding introduces complexity. In summary, evidence of validity supports the use of the SDS as screening tool but not as a diagnostic measure of depressive disorder.

Cross-References

- ▶ Beck Depression Inventory (BDI)
- ▶ Reliability and Validity

References and Readings

Hedlund, J. L., & Vieweg, B. W. (1979). The Zung self-rating depression scale: A comprehensive review. *Journal of Operational Psychiatry, 10*(1), 51–64.

Zung, W. W. (1965). A self-rating depression scale. *Archives of General Psychiatry, 12*(1), 63–70.

Zung, W. W. (1967). Factors influencing the self-rating depression scale. *Archives of General Psychiatry, 16*(5), 543–547.

Zung, W. W. (1986). Zung self-rating depression scale and depression status inventory. In N. Sartorius & T. A. Ban (Eds.), *Assessment of depression* (pp. 221–231). New York: Springer.

Zung Depression Rating Scale (ZDRS)

▶ Zung Depression Inventory

Zung Depression Scale

▶ Zung Depression Inventory

Zung Self-Assessment Depression Scale

▶ Zung Depression Inventory

Zung Self-Rating Depression Scale (SDS)

▶ Zung Depression Inventory

Zyban®

▶ Bupropion (Wellbutrin, Zyban)

List of Entries